

THIRD EDITION

Chiropractic Technique

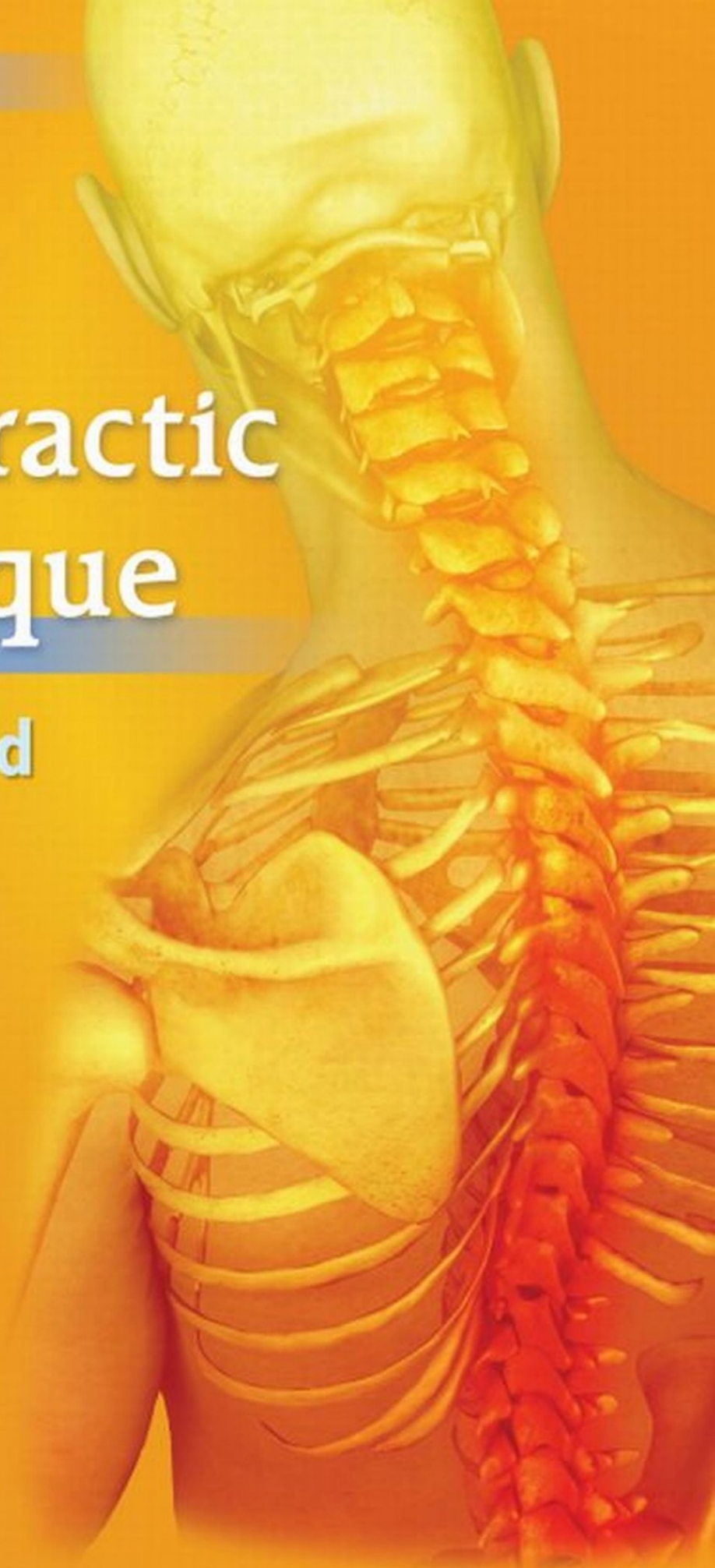
Principles and
Procedures

THOMAS F.
BERGMANN

DAVID H.
PETERSON



MOSBY
ELSEVIER



THIRD EDITION

Chiropractic Technique

Principles and Procedures

THOMAS F.

Bergmann, DC, FICC

Professor, Chiropractic Methods Department
Clinic Faculty, Campus Clinic
Northwestern Health Sciences University
Bloomington, Minnesota

DAVID H.

Peterson, DC

Professor,
Division of Chiropractic Sciences
Western States Chiropractic College
Portland, Oregon

with 1340 illustrations

ELSEVIER
MOSBY

ELSEVIER
MOSBY

3251 Riverport Lane
St. Louis, Missouri 63043

Chiropractic Technique Principles and Procedures
Copyright © 2011, 2002, 1993 by Mosby, Inc., an affiliate of Elsevier Inc.

978-0-323-04969-6

All rights reserved. No part of this publication may be reproduced or transmitted in any form or by any means, electronic or mechanical, including photocopying, recording, or any information storage and retrieval system, without permission in writing from the publisher. Permissions may be sought directly from Elsevier's Rights Department: phone: (+1) 215 239 3804 (US) or (+44) 1865 843830 (UK); fax: (+44) 1865 853333; e-mail: healthpermissions@elsevier.com. You may also complete your request on-line via the Elsevier website at <http://www.elsevier.com/permissions>.

Notice

Knowledge and best practice in this field are constantly changing. As new research and experience broaden our knowledge, changes in practice, treatment and drug therapy may become necessary or appropriate. Readers are advised to check the most current information provided (i) on procedures featured or (ii) by the manufacturer of each product to be administered, to verify the recommended dose or formula, the method and duration of administration, and contraindications. It is the responsibility of the practitioner, relying on their own experience and knowledge of the patient, to make diagnoses, to determine dosages and the best treatment for each individual patient, and to take all appropriate safety precautions. To the fullest extent of the law, neither the Publisher nor the [Editors/Authors] [delete as appropriate] assumes any liability for any injury and/or damage to persons or property arising out of or related to any use of the material contained in this book.

The Publisher

Library of Congress Cataloging-in-Publication Data

Bergmann, Thomas F.

Chiropractic technique: principles and procedures / Thomas F.

Bergmann, David H. Peterson. – 3rd ed.

p. ; cm.

Peterson's name appears first on the earlier edition.

Includes bibliographical references and index.

ISBN 978-0-323-04969-6 (hardcover : alk. paper)

1. Chiropractic. 2. Manipulation (Therapeutics) I. Peterson, David H., 1952- II. Title.

[DNLM: 1. Manipulation, Chiropractic—methods. 2. Chiropractic—methods. WB 905.9 B499c 2011]

RZ255.B47 2011

615.5'34—dc22

Proudly sourced and uploaded by [StormRG]
Kickass Torrents | TPB | ExtraTorrent | h33t

Vice President and Publisher: Linda Duncan

Senior Editor: Kellie White

Associate Developmental Editor: Kelly Milford

Publishing Services Manager: Catherine Jackson

Project Manager: Sara Alsup

Designer: Charlie Seibel

Printed in the United States of America

Last digit is the print number: 9 8 7 6 5 4 3 2 1

Working together to grow
libraries in developing countries

www.elsevier.com | www.bookaid.org | www.sabre.org

ELSEVIER

BOOK AID
International

Sabre Foundation

The authors wish to dedicate the third edition of this text to the students who have taught them much and provided the motivation for completing this text, and the rewards associated with their more than 30 years in chiropractic education.

This page intentionally left blank

FOREWORD

Ah! Where to begin? I've written books, chapters, papers, and editorials, but I think that writing a foreword can be the most challenging task. On the one hand so great is my admiration of this Third Edition of Dr. Tom Bergmann's and Dr. Dave Peterson's *Chiropractic Technique* that I have to restrain myself from writing what may substitute for an introductory chapter. On the other hand I've been given a rare opportunity to introduce readers to an extraordinary textbook, and I want to use my space wisely.

First, the traditional and well-deserved laudatory comments: this third edition of a now legendary chiropractic textbook offers old and new readers an encyclopedic treatise of chiropractic manual methods (*Principles and Procedures* as described in the subtitle) referenced with the most up-to-date evidence, lavishly illustrated, occasionally controversial but always rational, and true-to-form for these experienced authors, eminently readable. The most valuable addition is the availability of the text in electronic format (e-book), and the access to the Evolve website with video demonstrations of all assessment procedures and adjustive techniques. I can only imagine how valuable such an aid might have been during my own chiropractic education in the early 1970s. At that time we mostly learned from inconsistent personal instruction and crude drawings of static positioning. However, by using this wonderful reference work the next generation of chiropractors promises a whole new standard of consistency of care, not to mention the opportunity for instructors to design authentic and consistent assessment of their learners. Each of the chiropractic manual and manipulative procedures has been named to concur with common practice and especially the nomenclature used by the National Board of Chiropractic Examiners, which adds to the utility of this text for all students.

Chiropractic history is a special interest of mine, and I can admit to the fact that many common manipulative procedures are founded on a long tradition of empirical evidence, some dating back centuries. This said, our current understanding (and hence,

our refinement) of these procedures is based on modern sciences such as biomechanics and kinesiology, engineering, and diagnostic imaging. Elements of manipulative technique such as the idea of "pre-stressing" an articulation have acquired a new importance and allow for the first significant refinements of some manipulative techniques in many years. Staying abreast of such developments is the professional responsibility of every practicing chiropractor. This textbook provides a comprehensive reference for maintaining currency in the art and science of our field.

In adding my final comment about this new textbook, perhaps I will be a little controversial myself. In Chapter 3, the authors state that the concept of subluxation serves as a defining principle as well as the source of contentious debate and disagreement within the profession. I agree with this statement and I think that many modern and scientifically based chiropractors recognize the significance of this statement and the nature of the double-edged sword of this phenomenon we have historically known as the subluxation. Another double-edged sword is the great variety of chiropractic professional approaches and practices that are observed around the world today. Many have added to our diversity and sparked debate and the development of better, more effective care. In my view, however, this has also contributed to an often dogmatically based divisiveness, a lack of clear consensus on scope of practice and professional standards. It is my sincere hope and expectation that this textbook will contribute to a more visible consistency of approach to care in future generations of chiropractors, not forgetting those currently in practice with many years of practice remaining. Such a consistency of our professional approach to patient care is absolutely essential if we are to assume a rightful role in our nation's health care system. Were it in my power, I would insist that every single chiropractor and chiropractic student own and study this book and put into practice what Bergmann and Peterson have so masterfully described.

Michael R. Wiles, DC, Med

This page intentionally left blank

PREFACE

The third edition of *Chiropractic Technique* thoroughly discusses the use of the manual therapies with emphasis on thrust techniques in an unbiased and rational manner, based on and supported by current evidence, and continues its focus on teaching students of chiropractic and manual therapy. It is a practical and comprehensive presentation of the fundamental and advanced skills necessary to evaluate joint function and to deliver thrust and nonthrust techniques. As such it should help standardize the teaching and application of these procedures.

The third edition is also available as an electronic text. This feature allows for the addition of video demonstrations for the evaluative and adjustive procedures. From the inception of this text the authors have realized the enormous teaching value of having video clips to accompany the evaluation and treatment procedures. This feature provides the visual content that is so important for the development of manual skills and self-directed learning.

Also new to the third edition is the naming of each of the adjustive procedures. It is necessary to assign a clear and specific name to each technique procedure for teaching and testing purposes. The adjustive techniques have been given names that are based on the involved joint/region, patient position, contact used by the clinician, body part contacted, and any necessary additional information (i.e., push, pull, with distraction, etc.), as well as the induced joint movement. These names follow the patterns used by the U.S. National Board of Chiropractic Examiners and are designed to be helpful in the teaching and testing for competence.

The organization of the third edition remains the same with each chapter able to stand on its own. It is not necessary to read the information in one chapter to understand the material in another.

Chapter 1 provides an updated look at the past, present, and future aspects of the profession of chiropractic. It also draws attention to other professions that incorporate manipulative therapy and includes expanded information on the philosophical roots of the chiropractic profession.

Chapter 2 presents the musculoskeletal anatomy and basic biomechanical principles necessary to understand and apply chiropractic adjustive procedures. It has been updated with additional information on the effects of loads on all forms of connective tissue as well as the relationship between forces applied to the body and the consequences of those forces on human motion. Chapters 3 and 4 have been revised and supported with current references.

Chapter 3 is a comprehensive discussion of the basis for evaluation of joint dysfunction identifying important, relevant, and defensible concepts for the role that the musculoskeletal system plays in health and disease. It takes a critical look at the chiro-

practic manipulable lesion historically labeled as subluxation and also commonly referred to as joint dysfunction. Clearly this topic is one of passion and contention. We have attempted to discuss issues including definitions and theoretical models that have supportive evidence. Moreover, we discuss and describe the various evaluative procedures used to identify the presence of joint dysfunction with a corresponding Appendix demonstrating the known reliability and validity of the procedures.

Chapter 4 reviews the current understanding of manipulative mechanics, providing insight into current research and theoretical models of effects, or what happens when various forms of manual therapy are applied. We believe this chapter is very important to anyone seeking to become a user of thrust manipulation as it presents information relating to adjustive vectors, forces, and which joints and tissue may receive the majority of applied force. It suggests that what we say we have been doing and what we really are doing may be two entirely different things.

Chapters 5 and 6 have been updated with some new procedures, and other procedures have been modified. However, the significant change is in the layout of these two chapters. They are designed as a practical manual with technique descriptions that are closely associated with the illustrations and grouped by patient position. The names are changed to reflect the U.S. National Board of Chiropractic Examiners format.

Chapter 7 presents information on the application of mobilization, traction, and soft tissue procedures. Clearly, the High Velocity, Low Amplitude (HVLA) form of manual procedure is not indicated or tolerated by all patients and other forms of manual therapy should be applied. This chapter provides the rationale and description for many of nonthrust techniques.

We continue to believe that the text's distinguishing strong point is its comprehensive and extensively researched rational approach to the application of chiropractic adjustive techniques. The breadth of the topics covered makes it ideally suited as both a core teaching text for chiropractic students and a reference text for anyone using manual and manipulative therapy.

We are very pleased with the adoption of this text at a number of national and international chiropractic institutions and that the U.S. National Board of Chiropractic Examiners lists it as a reference for tests on chiropractic practice. Our goal for this text is to have it be a comprehensive source to assist in the standardization of teaching chiropractic diagnostic and adjustive methods.

Thomas F. Bergmann, DC., FICC
David H. Peterson, DC

This page intentionally left blank

ACKNOWLEDGMENTS

A third edition of a textbook can only occur through continued acceptance and use. Thanks must therefore go to all of the students, faculty, and practitioners who have found *Chiropractic Technique* a valuable educational resource.

We wish to acknowledge the roles of many individuals in the production of this edition: the photographic talents of Glen Gumaer on the First Edition, Arne Krogsven on the Second Edition, and Greg Steinke on the Third Edition; Nick Lang for the graphic artwork in the First Edition and Jeanne Roberts in the Second Edition; Dr. Janice Justice, Dr. Fred Rhead, Dr. Janet Preckel, Dr. Lolin Fletcher, and Dr. Andrew Baca for serving as models in the First Edition; Dr. Stacy Thornhill, Dr. Sarah Macchi, Dr. Torbin Jensen, Dr. Arin Grinde, and Brian Hansen for serving as models in the Second Edition; Andrea Albertson, Lindsey Baillie, Matt Christenson, Ayman Hassen, David Landry, Christine Rankin,

Kristen Roney, Haj Soufi, Kory Wahl, and Pler Yang for serving as models in the Third Edition.

Appreciation and gratitude goes to Dr. Stacy Thornhill and Dr. Joe Cimino for their expertise in differentiating and defining the various soft tissue techniques, to Dr. Tom Davis for the concepts of distractive and motion-assisted procedures, and to Dr. Bill Defoyd for his insight and suggestions concerning McKenzie methods.

Finally, we would like to express sincere gratitude to all of the individuals at Elsevier, Inc., who have maintained faith in us and this book to see it through to a third edition. Specifically, we thank Kellie White, Kelly Milford, and Sara Alsup.

D.P. and T. B.

This page intentionally left blank

CONTENTS

CHAPTER 1: GENERAL OVERVIEW OF THE CHIROPRACTIC PROFESSION	1	CHAPTER 5: THE SPINE: ANATOMY, BIOMECHANICS, ASSESSMENT, AND ADJUSTIVE TECHNIQUES	145
The Past	1	Structure and Function of the Spine	145
The Present	3	Evaluation of Spinal Joint Function	146
The Future	9	Identification of Joint Subluxation/ Dysfunction Syndrome	151
Conclusions	10	Cervical Spine	152
		Thoracic Spine	188
CHAPTER 2: JOINT ANATOMY AND BASIC BIOMECHANICS	11	Thoracic Adjustments	211
Fundamental Concepts, Principles, and Terms	11	Lumbar Spine	233
Joint Function	20	Pelvic Joints	262
Mechanical Forces Acting on Connective Tissue	23	CHAPTER 6: EXTRASPINAL TECHNIQUES	283
Properties of Connective Tissue	26	Role of the Peripheral Joints	283
Models of Spine Function	33	Temporomandibular Joint	283
		Shoulder	294
CHAPTER 3: JOINT ASSESSMENT PRINCIPLES AND PROCEDURES	35	Elbow	315
The Manipulable Lesion	36	Wrist and Hand	326
Subluxation	36	Hip	337
Vertebral Subluxation Complex	37	Knee	349
Joint Subluxation/Dysfunction Syndrome	47	Ankle and Foot	364
Spinal Listings	47	CHAPTER 7: NONTHRUST PROCEDURES: MOBILIZATION, TRACTION, AND SOFT TISSUE TECHNIQUES	381
Clinical Evaluation of Joint Subluxation/ Dysfunction Syndrome	47	Joint Mobilization	381
Clinical Documentation	82	Manual Traction-Distrraction	384
		McKenzie Method	387
CHAPTER 4: PRINCIPLES OF ADJUSTIVE TECHNIQUE	84	Cranial Manipulation	391
Classification and Definition of Manual Therapies	84	Soft Tissue Manipulation	393
Joint Manipulative Procedures	84	Conclusions	418
Soft Tissue Manipulative Procedures	88	GLOSSARY	419
Indications for Adjustive Therapy	89	APPENDIX 1: NAMED CHIROPRACTIC TECHNIQUES	426
Mechanical Spine Pain	89	APPENDIX 2: COMPILATION OF RELIABILITY STUDIES ON JOINT EVALUATION PROCEDURES	429
Joint Subluxation/Dysfunction Syndromes	90	APPENDIX 3: COMPILATION OF VALIDITY STUDIES ON MOTION PALPATION	440
Contraindications to and Complications of Adjustive Therapy	92	REFERENCES	441
Effects of Adjustive Therapy	105	INDEX	469
Application of Adjustive Therapy	120		

This page intentionally left blank

GENERAL OVERVIEW OF THE CHIROPRACTIC PROFESSION

OUTLINE

THE PAST

Philosophic Roots

THE PRESENT

Basic Principles

1
3
3
3

Chiropractic Education

Licensure

Scope of Practice

Patient Access and Chiropractic

Utilization

5
6
6
6
6

Research

Standard of Care and Guidelines

THE FUTURE

CONCLUSIONS

7
8
9
10

The chiropractic profession is only a little more than a century old, but manipulation in its various forms has been used to treat human ailments since antiquity. Although no single origin is noted, manual procedures are evident in Thai artwork dating back 4000 years. Ancient Egyptian, Chinese, Japanese, and Tibetan records describe the use of manual procedures to treat disease. Drawings demonstrate the application of this treatment form from the time of the ancient Greeks through the middle ages in various parts of the eastern and western world. Manipulation was also a part of the North and South American Indian cultures. Certainly, Hippocrates (460–355 BC) was known to use manual procedures in treating spinal deformity, and the noted physicians Galen (131–202 AD), Celsus, and Orbasius alluded to manipulation in their writings. The nineteenth century witnessed a rise in popularity of American and English “bonesetters,” the most well known being Mr. Hutton, who influenced the thoughts and writing of Sir James Paget and Wharton Hood. Bonesetters were often called upon to provide treatment for many types of maladies. Bonesetting was often practiced by families. It evolved from a lay practice developed from the peasant revival of manipulation after it went underground during the seventeenth century.

It was not until the days of Daniel David Palmer and Andrew Taylor Still, the founders of chiropractic and osteopathy, that these procedures were codified into a system. Palmer and Still both became acquainted with bonesetters and bonesetting techniques. In addition, the two men practiced magnetic healing, a reflex therapy that on occasion used powerful paraspinal massage.¹ Bonesetting and magnetic healing were instrumental in the founding of chiropractic and osteopathy. The early days of chiropractic and osteopathy represented major attempts to place manual procedures on firmer ground, and although the major developments in manual manipulative procedures in the late nineteenth century were largely American, developments were also occurring in other locations around the globe. At the same time, bonesetters were working in the United States and England and continued to do so into the early twentieth century. Bonesetters continue today to have an effect on health care delivery in Japan. While chiropractic was developing in the United States under the leadership of D.D. Palmer and his son, B.J. Palmer, medical manipulators from around the world were also making significant advances, as were early osteopathic researchers. The works of Mennell, Cyriax, Paget, and others are important in this regard.

Both chiropractic and osteopathy chose to focus on the musculoskeletal system, although in philosophically divergent ways. Andrew Still placed great emphasis on the somatic component of disease, largely involving the musculoskeletal system, and on the relationship of structure to function. Palmer postulated that subluxation, or improper juxtaposition of a vertebra, could interfere with the workings of the human nervous system and with innate intelligence, that power within the body to heal itself. Both emphasized the role the musculoskeletal system played in health and disease.

Coulter has described the historical concepts of chiropractic that initially defined the young and growing profession, and the emergence of a developing philosophy of care.² He suggested that chiropractic distinguished itself as a primary contact healing art by advocating for an alternative type of care, and advancing the specific philosophic tenets of critical rationalism, holism, humanism, naturalism, therapeutic conservatism, and vitalism in the care of patients. Many of these tenets have been well established and significantly advanced by the profession.³

However, to succeed in an environment as dynamic and volatile as health care, it is critical to distinguish between those aspects of a given profession that are vital to it and those aspects that are inessential and often create costly distractions.⁴ To begin to understand the aspects of the chiropractic profession that are either vital or inessential to the profession's identity, a look at the past, the present, and the future is necessary.

THE PAST

Daniel David Palmer (1845–1913; also known as D.D. Palmer) is considered the “father” of chiropractic. He came to the United States from Port Perry, Ontario, Canada, in 1865. He spent the next 20 years in such various occupations as farming, beekeeping, and store sales. In 1885, he opened a practice as a magnetic healer in the city of Davenport, Iowa, although he had no formal training in any healing art.

During the nineteenth century, various forms of spiritualistic and metaphysical speculation existed, all of which piqued Palmer's curiosity. He studied and was influenced by Mesmer's concept of animal magnetism and Mary Baker Eddy's spiritual concepts used in her Christian Science healing. During this same time, Thoreau and Emerson's transcendentalist philosophy, which emphasized a

love of nature and independence of thought, provided a supportive environment for the pioneers of new healing methods, including D.D. Palmer.⁵ Palmer was able to blend recognized spiritual and metaphysical concepts together with then-current scientific principles to create a unique ethos for the chiropractic healing art.⁶

His formulation of chiropractic practice and theory purportedly developed from his application of a manual thrust, which he called an *adjustment*, to Harvey Lillard in September 1895 (coincidentally and significantly, the same year that Roentgen discovered the x-ray). This event has moved beyond that of a simple tale to an apocrypha. As the story goes, this manual adjustment was directed to the fourth thoracic vertebra and resulted in the restoration of Mr. Lillard's lost hearing. From the reasoning used to devise this treatment, Palmer then applied similar lines of thought to other individuals with a variety of problems, each time using the spinous process of a vertebra as a lever to produce the adjustment. Palmer was the first to claim use of the spinous and transverse processes of the vertebrae as levers for manual adjustment of the spine—in effect, short lever contacts. This constituted the initiation of chiropractic as an art, a science, and a profession. Palmer wrote:

I am the originator, the Fountain Head of the essential principle that disease is the result of too much or not enough functioning. I created the art of adjusting vertebrae using the spinous and transverse processes as levers, and named the mental act of accumulated knowledge, function, corresponding to the physical vegetative function—growth of intellectual and physical—together, with the science, art and philosophy of Chiropractic.⁷

From this nearly chance opportunity came the outlines of the profession. Palmer developed the concept of a “subluxation” as a causal factor in disease, through the pressure such “displacements” would cause to nerve roots. Within 2 years of the initial discovery, Palmer had started the Chiropractic School and Cure and soon had his first student. By the year 1902, Palmer's son, Bartlett Joshua (usually referred to as B.J.), had enrolled in his father's school and 2 years later had gained operational control of the institution, becoming president in 1907. He maintained this post until his death in 1961.

Animosity between father and son developed. Palmer clearly stated that the only principle added by B.J. Palmer was that of greed and graft; he aspired to be the discoverer, developer, and fountainhead of a science brought forth by his father while he was a lad in his teens.⁷ The elder Palmer left the school of his name and traveled around the country, forming at least four other chiropractic schools in California, Oregon, and Oklahoma. He was also placed in jail for a short time for practicing medicine without a license. Although he might have been able to avoid jail by paying a small fine, he believed he had a more important principle to uphold. Palmer was not the last to be jailed for this crime; the process of jailing chiropractors for practicing medicine without a license continued into the next two decades.⁸ Preoccupation with the legal right to practice chiropractic no doubt led the profession to focus resources on political, ideologic, and economic concerns, rather than on research that might have influenced medical scientists.⁹

D.D. Palmer died in 1913 after enjoying only a short reconciliation with his son, B.J., who had by that time led the original Palmer School for nearly 7 years. In 1906, D.D. Palmer had already forsaken education at the original Palmer School. That year was also significant because it marked the first philosophic differences within the fledgling chiropractic profession. John Howard, one of the first graduates of the Palmer School, was unable to accept many of the philosophic beliefs relative to health care that B.J. Palmer was now openly espousing. B.J. had by then begun to preach that subluxation was the cause of all disease. Howard therefore left the Palmer School and founded the National School of Chiropractic not far from Palmer's school in Davenport. As Beideman had noted,¹⁰ Howard wanted to teach chiropractic “as it should be taught” and therefore moved the school to Chicago, believing that chiropractic education required coursework in the basic and clinical sciences, including access to laboratory, dissection, and clinics. These two schools (now colleges) still exist today.

Willard Carver, a longtime friend of D.D. Palmer and the attorney who defended him when he was arrested for practicing medicine without a license, decided to take up the profession of chiropractic as well. After D.D. sold the chiropractic school to B.J., Carver began to distance himself from the Palmers. He never had a strong relationship with B.J., and because of disagreements on the nature of subluxation and scope of practice, he began his own school in Oklahoma.

Carver viewed chiropractic practice in a manner opposed to that of the Palmers. Carver followed what he called a *structural approach*, which was essentially a systems approach to subluxation. In his view, disrelations in spinal joints were the result of compensatory patterns and adaptations arising from other subluxations. He was also an advocate for other therapeutic procedures beyond adjustment that were at times outside the common scope of chiropractic practice, such as physiotherapy. This put him very much at odds with the Palmerite approach to chiropractic.

Carver is equally well known for his legal and legislative efforts on behalf of the profession. Not only did he establish schools of chiropractic in several cities, he also wrote eight influential early chiropractic texts, published a college journal (*Science Head News*) that provided perspectives different from the prevailing Palmer view, and helped establish licensing laws for the chiropractic profession where none existed before.

Other chiropractic institutions were also being founded all over the country, and there was more and more internecine warfare among practitioners. B.J. Palmer had set himself up as the protector of a fundamental form of chiropractic (today referred to as *straight chiropractic*).

From 1910 to 1926, Palmer lost many important administrators, most of whom left to form their own institutions. Furthermore, from 1924 until his death in 1961, he was a titular leader only, keeping the flame for a fundamentalist minority and battling with most of the profession, which he saw as inevitably following the osteopathic moth into the seductive medical flame.¹¹

Regardless of the philosophic issues that were debated then, and that still divide the profession today, it is possible that without B.J. Palmer's missionary zeal and entrepreneurial brilliance, the chiropractic profession would not exist as it is today. B.J.'s title as the “developer” of chiropractic was honestly earned.

PHILOSOPHIC ROOTS

Spiritualism, which developed in the United States in the 1840s and is based on the simple premise that humans are dual beings consisting of a physical and a spiritual component, spawned a large array of interrelated religious, healing, and paranormal investigative groups.¹² Spiritualists believed that the physical element (the body) disintegrates at death, but the spiritual element (the soul, spirit, personality, consciousness, etc.) continues exactly as it was, but in another plane of existence: the “spirit world” or heaven.

American transcendental notions also evolved during this period and helped formulate the influential philosophy of Ralph Waldo Emerson. Emerson’s concept of a dual mind, which incorporates both innate and educated elements, were very similar to D.D. Palmer’s postulates and probably had a significant influence on Palmer’s early health care philosophy and theories.¹³ D.D. Palmer stressed the concept and importance of the innate mind and its role in self-regulation and restoration of health. He stated that “spirit and body” compose a dualistic system with “innate and educated mentalities,” which look after the body physically within its surrounding environments.¹³ This idea of innate intelligence forms a critical part of a 1956 work by B.J. Palmer in which he states, “Innate is the ONE eternal, internal, stable, permanent factor that is a fixed and reliable entity, does not fluctuate up and down scales to meet idiosyncrasies.”¹⁴

D.D. Palmer and his early followers emphasized health and the absence of disease over the management of disease. Early chiropractic theory emphasized the important role of the neuromusculoskeletal (NMS) system, specifically the spine, in treating and preventing disease. The concept was that a structural problem within the spine contributes to altered musculoskeletal and neurologic function and diminishes the ability of the body to heal itself.¹⁵ Palmer asserted that either too much or too little nerve energy is “dis-ease.” Moreover, he believed that disease was the result of internal imbalances involving hyperfunction or hypofunction of organs and systems rather than the result of something external that invades the body.

Osteopathy was also emerging at the same time and within the same philosophic environment. Andrew Taylor Still, the father of osteopathy, was a strong believer in Spiritualism. He stated, “We say *disease* when we should say *effect*; for disease is the effect of a change in the parts of the physical body. *Vis medicatrix naturae* to the *osteopath* declared that disease in an abnormal body was just as natural as is health when all parts are in place.”¹⁶

In addition to his interest in Spiritualism, D.D. Palmer also dabbled in other occult philosophies of his day. He first began his practice as a magnetic healer in Burlington, Iowa, and would in years to come write extensively on his thoughts about intrinsic “inner forces.” He went on to label the inner forces and their self-regulating effects as *innate intelligence*. He reasoned that health could be maintained if the body’s innate intelligence was functioning properly. Diseases were viewed as conditions resulting from either an excess or deficiency of this function.¹⁷ In contemporary health care the body’s ability to self-regulate and maintain internal equilibrium is referred to as *homeostasis*.

The early chiropractic focus on the philosophy of chiropractic and its distinct model of health care did not eliminate internal

debate concerning the need for scientific training and investigation. From the early days of chiropractic’s founding, there were diverging views and debates between those stressing *vitalism* (the belief that the principles that govern life are different from the principles of inanimate matter) and those stressing a scientific approach to practice. D.D. Palmer believed, as noted by Waagen and Strang, that both approaches (the vitalists and the scientists) were important and that the concept of innate intelligence, which formed the early cornerstone of the philosophy of chiropractic, could be incorporated with a scientific approach to chiropractic.¹⁵

Many of the early chiropractic debates and divergent positions concerning its philosophy and health care model persist. As a result, the philosophy of chiropractic suffers from a lack of clarity and understanding of its boundaries.¹⁸⁻²³ There is no demonstrable evidence that a body of content has been agreed upon. It is imperative that the chiropractic profession clearly delineate what exactly is meant by the *philosophy of chiropractic* and codify its content.²⁴

THE PRESENT

BASIC PRINCIPLES

The broad chiropractic model of health care is one of holism. In this model, health is viewed as a complex process in which all parts and systems of the body strive to maintain homeostatic balance against a dynamic environment of internal and external change. The human body is perceived as being imbued at birth with an innate ability (innate intelligence) to respond to changes in its internal and external environment. Earlier health care pioneers saw this as proof of the healing power of nature, *vis medicatrix naturae*. This concept emphasizes the inherent recuperative powers of the body in the restoration and maintenance of health and the importance of active patient participation in treating and preventing disease. The presence of an inherent ability within the organism to influence health and disease has been described by many different health care disciplines and is listed in Table 1-1.

TABLE 1-1 Names Given to the “Subtle” Energy of the Body Believed to Influence the Body in Health and Disease

Energy Name	Originator
Prana	Hindu
Chi	Chinese
Xi	Japanese
Libido	Freud
Orgone energy	Reich
Elan vitale	Bergson
Innate intelligence	Chiropractic
<i>Vis medicatrix naturae</i>	Medicine
Biochemicals of emotion	Pert

Broad-scope chiropractic care is committed to holistic health care and working with patients to optimize their health. Although the chiropractic profession's major contribution to overall health is through the evaluation and treatment of NMS disorders, it is common for chiropractic physicians to also counsel patients on other lifestyle issues such as diet, nutrition, exercise, and stress management.

The contemporary practice of chiropractic maintains its focus on the evaluation and conservative treatment of NMS disorders and the important relationship between the functioning of the NMS system and overall well-being and health. Dysfunction or disease of the musculoskeletal system in any form is viewed as having the potential to create disorders of the locomotor system that may lead to impaired functioning of the individual. This model is supported by the underlying principle that stresses the important interrelationship that exists between structure and function of the human body.

In addition to specializing in the adjustive (manipulative) treatment of disorders of the spinal and extremity joints, it is common for chiropractors to include other treatment procedures in patient management and health promotion. Common therapies applied include dietary modification, nutritional supplementation, physical therapies, and exercise.

The chiropractic profession considers the musculoskeletal system to be a clinically neglected component of the body, although musculoskeletal disorders are common and account for significant amounts of lost time at work and recreation. The musculoskeletal system therefore deserves full consideration and evaluation whenever patients are seen, regardless of the complaint causing them to seek care.

The musculoskeletal system should be viewed as part of the whole body and subject to the same intensive diagnostic evaluation as any other system in the body. The musculoskeletal system is involved in so many alterations of function that it demands such attention and should not be removed from consideration in diagnosis, even when the initial problem appears removed from the musculoskeletal system.

Moreover, the human musculoskeletal system accounts for more than half of the body's mass and is its greatest energy user. The large amounts of energy required by the musculoskeletal system must be supplied through the other systems in the body. If the musculoskeletal system increases its activity, an increased demand is placed on all the other body systems to meet the new, higher energy demands. Chiropractic notes that the presence of disease or dysfunction within the musculoskeletal system may interfere with the ability of the musculoskeletal system to act efficiently, which in turn requires greater work from the other systems within the body.

An important principle of chiropractic is that because the nervous system is highly developed in the human being and influences all other systems in the body, it therefore plays a significant role in health and disease. Although the exact nature of the relationship between dysfunction of the musculoskeletal system and changes in neurologic input to other body systems is not known, an enduring basic principle of chiropractic is that aberrations in structure or function can have an effect on health and the body's sense of well-being. The nervous system's effects on the body's

ability to fight disease through the immune response demonstrate this concept.²⁵

The nervous system also communicates with the endocrine system to maintain a state of *homeostasis*, defined simply as physiologic stability. This tendency of the body to maintain a steady state or to seek equilibrium despite external changes, referred to as *ponos* by Hippocrates, is the underlying theme in Palmer's original concept of innate intelligence influencing health.

Manual procedures and, specifically, the adjustment are applied to address local NMS disorders and to improve NMS function. A consequence of improved NMS function may be improvement in the body's ability to self-regulate, thereby allowing the body to seek homeostasis and improved health. In Haldeman's outline of this process, manipulative therapy improves the function of the musculoskeletal system, which then causes a change in the input from the nervous system, which in turn may have a positive effect on other NMS tissue, organ dysfunction, tissue pathologic condition, or symptom complex.²⁶ Reflex mechanisms that support these ideas have indeed been documented, although the effects of manipulation on these reflexes have yet to be adequately assessed and demonstrated.²⁷⁻³⁰

Palmer developed his model of the effects on the nervous system through the belief that subluxation affects the tone of the body. In this model, *tone* refers to the efficiency of the nervous system and to the ability of the body to self-regulate its processes properly. This view was in opposition to the medical thought of the day, which focused on the germ theory and its relationship to disease.

Although many of the early forebears in chiropractic postulated subluxations as the root cause of all health care disorders and a "one cause, one cure" approach to health, the monocausal theory of disease has now been rejected by the overwhelming majority of practicing chiropractors. Chiropractors today certainly accept the existence and reality of germs and the role they play in creating disease. Both the chiropractic and medical paradigms recognize the health of the individual and his or her resistance to infection as critical factors. Furthermore, the chiropractic profession views the host's susceptibility as depending on a multitude of factors. The chiropractic model postulates that the presence of joint dysfunction or subluxation may be one such factor serving as a noxious irritant to lower the body's ability to resist disease. Within this paradigm, removal of joint dysfunction or subluxation becomes an important consideration for optimal health.

The value and importance of adhering to early chiropractic philosophic models of health and disease are debated. Some argue for strict adherence to an early fundamental paradigm because of fear that divergence from fundamental core values will lead to the dilution of the profession's unique health care approach. Others argue that unwavering adherence to a particular belief system creates a climate of anti-intellectual dogma that retards the profession from investigating and differentiating effective from ineffective diagnostic and treatment procedures. Many of the historic philosophic chiropractic tenants are considered to fall within the realm of a belief system that can neither be refuted nor confirmed through research. Certainly the profession's early adherence to its core principles helped established the profession as a unique and valuable branch of the healing arts. These core values continue to

support the profession's conservative approach to health care and its emphasis on the body's inherent recuperative powers. However, it is probable that unwavering adherence to core values does create a climate that inhibits professional self-appraisal and clinical research. Questions concerning clinical effectiveness and whether "chiropractic works" are not answerable with philosophic debate.

Technically, philosophy asks questions about the nature of truth (epistemology), reality (metaphysics), the good (ethics), and the beautiful (aesthetics).³¹ None of these is susceptible to empirical scientific inquiry. Proving that "chiropractic works" has been a loudly expressed goal of the profession that offends scientific sensibilities. Concepts based on faith or intuition must not be confused with scientific theory validated by empirical data or facts.

A profession, with all its procedures and practices, cannot be demonstrated to "work." It has not been said that research proves that medicine or dentistry works; rather, specific studies are cited identifying that a specific procedure is effective for a specific condition. Research done to "prove" something works will be looked on suspiciously because there is a clear demonstration of bias. Furthermore, chiropractic must be viewed as a profession, not a procedure. It is important to be aware of the philosophic assumptions underlying conceptions of reality and truth but not confuse them with the search for scientific truths, which are never absolute but remain forever tentative and approximate.³¹ The traditional language of the philosophy of chiropractic might be revised to more closely coincide with the current language in the biologic and life sciences without loss of appropriate philosophic meaning.³²

CHIROPRACTIC EDUCATION

Although organized medicine rejected chiropractic from its outset, occurrences within medicine had a major effect on the development of the chiropractic profession. The Flexner Report, released in 1910, had a profound effect on chiropractic education.³³ This report was highly critical of the status of medical education in the United States. It recommended that medical colleges affiliate with universities to gain educational support. As Beideman has noted, it took the chiropractic profession nearly 15 years from the time of that report to begin the same types of changes that medicine underwent to improve its education.³⁴

The changes were not long in coming, however, once their need was recognized. These improvements ultimately led to the creation of the Council on Chiropractic Education (CCE), which later was recognized by the U.S. Department of Education (then the Department of Health, Education and Welfare) as the accrediting agency for the chiropractic profession.

By the late 1960s, the CCE had required its accredited institutions to use a 2-year preprofessional educational experience as a requirement for matriculation. In 1968, the doctor of chiropractic (DC) degree became a recognized professional degree, and in 1971 the CCE became an autonomous body. In addition to national accreditation by CCE and the U.S. Department of Education, regional accrediting bodies have reviewed chiropractic college programs, and all but two of the programs within the United States have achieved accreditation. The self-evaluation and accreditation process allowed chiropractic institutions to upgrade their professional

standards to an unprecedented degree. The requirements of the CCE govern the entire educational spectrum of chiropractic education, mandating that certain information must be imparted to the student body and providing a way to monitor compliance and to provide guidance to an individual college. The effect has been salubrious. Today, all CCE-accredited institutions require a minimum of 3 years of college credits (90 semester hours and 134 quarter hours) for matriculation. Prerequisite coursework includes 24 semester hours in basic sciences, including biology, chemistry, and physics, and 24 semester hours in humanities and social science. Included in the entrance requirements are 1 year of biology, general chemistry, organic chemistry, and physics.

All CCE-accredited institutions teach a comprehensive program incorporating elements of basic science (e.g., physiology, anatomy, and biochemistry), clinical science (e.g., laboratory diagnosis, radiographic diagnosis, orthopedics, neurology, and nutrition), and clinical intern experience. The chiropractic educational program is a minimum of 4 years, totaling an average of 4800 classroom hours. The first and second years are devoted primarily to basic sciences, chiropractic principles, and technique skill development. The third year emphasizes clinical and chiropractic sciences and prepares students for the transition into their fourth year and practical clinical experience treating the public in the college clinics. Government inquiries and comparative evaluations have determined that the coursework and hours of instruction in the basic sciences are very similar between chiropractic and medical schools. Chiropractic students on average spend more hours in anatomy and physiology and fewer hours in public health. In the clinical arena chiropractic students have very limited training in pharmacology and critical care, but have significantly more training in clinical biomechanics, NMS diagnosis, manual therapy, and exercise rehabilitation.

For the process of accreditation, the CCE established specific standards with which a chiropractic educational institution must comply to achieve and maintain accreditation.³⁵ Care has been taken to ensure that accreditation requirements are consistent with the realities of sound planning practices in the DC program. The word *requirements* signifies a set of conditions that must be met for CCE accreditation to be awarded. In recognition of their potential uniqueness, each program may be given some latitude in the means by which they meet some requirements. However, compliance with all requirements must be fulfilled by each accredited entity.

Although standardization of curriculum created an environment that ensures the public that most graduates of CCE institutions have been provided a competent education, each college does not necessarily teach its students the same scope of chiropractic manipulative techniques. Educational and philosophic differences between schools can dramatically affect the curriculum and the range of diagnostic and therapeutic procedures taught at each college. The result is different products and practice approaches among graduates of different schools. The major distinction between college programs rests with those that ascribe to evidence-based education and those that rely on joint "subluxation-based" or "philosophy-based" education.

Each institution must teach its students to adjust, but the procedures and intent taught at one college may differ from those

taught at other institutions. Although all these forms of chiropractic adjustive techniques have many elements in common, their approaches may differ substantially. A graduate of one college may find it difficult to share information with the graduate of a different college that teaches some alternate form of an adjustive procedure. Furthermore, a plethora of techniques is available in the form of postgraduate seminars, many of which are not governed by a regulatory body or accrediting procedure that would ensure an adequate scholastic level or competence.

Interested and probing chiropractors who noticed regularity in their results and began to ask why those results occurred founded the majority of chiropractic technique systems. This was largely a “bootstrapping” effort; the impetus to gain new knowledge and then disseminate it was largely self-driven. These approaches typically developed into systems of diagnosis and treatment (“system techniques”). These early commendable efforts are limited by the fact that they are often based on a biologically questionable or singular and simplistic rationale with little or absent systematic clinical research investigation. The human body is a very complex and integrated organism, and to rely on a single evaluative or treatment procedure without substantiated clinical justification is not considered sound clinical practice. This text hopes to improve the educational environment by providing a foundation of fundamental standards and psychomotor skills that are common to all adjustive thrust techniques. A list of most of the named chiropractic techniques is provided in Appendix 1, and many forms of chiropractic technique systems are described in the book, *Technique Systems in Chiropractic* by Robert Cooperstein and Brian Gleberzon (Elsevier 2004).

Chiropractic education continues to be innovative and to advance, as demonstrated by the growing adoption of evidence-based practice (EBP) content into chiropractic education. In 1999 the National Center for Complementary and Alternative Medicine (NCCAM) established an R25 Education Project Grant Program to encourage expanded knowledge of complementary and alternative medicine (CAM) in medical education. The initial round of funding was focused on medical schools and required them to pair with CAM professions in the development of medical curricula that would increase CAM literacy in medical school graduates and residents.

Beginning in 2005 a new round of NCCAM R25 educational grants was announced. This round of funding, the CAM Practitioner Research Education Project Grant Partnership, was focused on CAM health care institutions and on increasing the quality and quantity of evidence-based clinical research content in their curricula. The grant required that CAM institutions pair with a research-intensive institution with the goal of improving CAM students’ EBP skills. In the first round of funding, five institutions were awarded partnership grants. Two of the originally funded institutions were chiropractic colleges (National University of Health Sciences and Western States Chiropractic College) and subsequent rounds of funding have awarded to grants to two additional chiropractic institutions (Northwestern Health Sciences University and Palmer Chiropractic College).

LICENSURE

To become licensed, practitioners must pass four national board examinations. Part I tests basic science knowledge, part II evaluates

clinical science subjects, part III is a written clinical competency examination, and part IV is a practical objective structured competency examination, which tests candidates on x-ray interpretation and diagnosis, chiropractic technique, and case management. In addition to the national boards, most states require candidates to take a jurisprudence examination covering that state’s practice act and administrative rules.

Today, chiropractic is approved under federal law in all 50 states, in the Canadian provinces, and in a majority of foreign countries. Chiropractic practice in the United States is regulated by state statute and by each state’s board of chiropractic examiners. Chiropractic practice acts define the practice of chiropractic locally and establish regulations for licensure, discipline, and scope of practice for all 60 jurisdictions in North America.

There is significant variation and diversity of definitions in state practice acts and the interpretation of what constitutes each state’s practice act and scope of practice are profound and bewildering.³⁶ This diversity and variability undermine the desire of many chiropractors to be regarded as a unified profession with clearly established standards of practice and treatment.³⁷ A survey of practice acts revealed a broad scope of chiropractic practices, but also demonstrated a lack of consensus within the profession, which causes confusion for the profession itself, for those seeking services from the profession, and for those who conduct business with members of the profession.³⁸

SCOPE OF PRACTICE

Chiropractors are licensed as primary contact portal of entry providers in all 50 states. They are trained to triage, differentially diagnose, and refer nonchiropractic cases. Chiropractors use standard physical examination procedures with an emphasis on orthopedic, neurologic, and manual examination procedures. Chiropractors are licensed to take x-rays in all 50 states and, when indicated, can order special tests if permitted by state law (e.g., blood work, imaging).

Although there is wide variation in therapeutic scope of practice from state to state, nearly all chiropractors use a variety of manual therapies with an emphasis on specific adjustive techniques. Therapeutic alternatives range from manual therapy, physical therapy, and spinal adjustments to exercise and nutritional and dietary counseling.

Chiropractors view themselves as specialists in NMS care but also as complementary and alternative caregivers for a number of other chronic conditions. In these situations chiropractors typically incorporate other therapeutic intervention such as counseling on diet, nutrition, and lifestyle modification. Management or comanagement of patients with hypertension, diabetes, or dyslipidemia are a few examples.

PATIENT ACCESS AND CHIROPRACTIC UTILIZATION

Chiropractic is the largest CAM profession with approximately 60,000 practitioners, and the most widely used CAM profession (30% of annual CAM visits). Approximately 11% of the population uses chiropractic services each year, and it is estimated that

one third of the population has seen a chiropractor at some point in their lifetimes. Nearly all chiropractors surveyed (98%) state that they refer patients to medical doctors, and a North Carolina study indicates 65% of medical doctors have referred to a chiropractor at some point in their career. A majority of chiropractors (77%) state they have had a referral from a medical doctor. Insurance coverage for chiropractic is quite extensive. Chiropractic is included under Medicare and Medicaid laws with worker's compensation coverage in all 50 states. Approximately 50% of health maintenance and 75% of private health insurance plans cover chiropractic.

Recent legislation has greatly expanded chiropractic services in the Department of Defense and Veterans Administration (VA) health care programs. This legislation was prompted by an independent demonstration project funded by the U.S. Department of Defense on Chiropractic Health Care. This project produced data confirming the cost-effectiveness of chiropractic services, with patients reporting chiropractic care to be as good as or better than medical care for selected musculoskeletal conditions. In late 2001, the U.S. Congress enacted a bill to provide chiropractic services for the military on a comprehensive and permanent basis. Chiropractic services are in the process of being established in all communities in the United States and worldwide where there are active U.S. military personnel.

A report from the Veterans Health Administration Office of Public Health and Occupational Hazards cites musculoskeletal injuries as the number-one complaint (41.7%) among U.S. veterans of Iraq and Afghanistan.³⁹ By working in concert with medical doctors and other health care providers at VA facilities, chiropractors could have an influence on the upsurge of joint and back pain among U.S. veterans.

Chiropractic access to hospital services has expanded during the last several decades. This expansion was initiated by the successful outcome of a long antitrust case that the profession waged against organized medicine. The outcome of the Wilk trial on February 7, 1990, in the Seventh Circuit U.S. Court of Appeals found the American Medical Association (AMA) guilty of an illegal conspiracy to destroy the competitive profession of chiropractic. This decision arose from a suit brought by five chiropractors alleging that the AMA, along with several other organizations involved in health care, conspired to restrain the practice of chiropractic through a sustained and unlawful boycott of the chiropractic profession. This was despite the fact that chiropractic care had been found to be, in some cases, as effective or more effective in treating certain NMS-related health problems.

Although opposition to inclusion of chiropractic was initially profound, it has been gradually waning. Staff privileges are being sought and gained by more and more chiropractors.

Use of CAM services has increased dramatically during the last several decades.^{40,41} "Recent estimates based on the 2002 National Health Interview Survey reveal that 62.1% of U.S. adults used CAM therapies during the previous year."⁴² Within the CAM community, chiropractic accounts for the largest provider group and the greatest number of patient visits.^{40,41} The growing evidence base and expanding demand for CAM services has stimulated the medical community to recognize that CAM literacy should be an essential part of medical education. Surveys have

indicated that an overwhelming percentage of medical college faculty and students want information about CAM and integrative therapy in their school's curriculum.⁴² More recent surveys indicated that the amount of time devoted to CAM education has increased and that medical students are more confident in their understanding and ability to counsel patients about CAM therapies.⁴² The number of prestigious medical universities interested in integrative and CAM therapies has increased dramatically during the preceding 5 years, with membership in the Consortium of Academic Health Centers for Integrative Medicine increasing from 11 to 39 schools.⁴²

RESEARCH

Federal recognition and funding increased dramatically during the 1990s, with a number of institutions receiving federally funded grants and monies allocated for the development of a research center and annually funded research workshops. The National Workshop for Developing the Chiropractic Research Agenda (or Research Agenda Conference) occurred in the summer of 1996. Five specific areas of chiropractic research were examined: clinical research, basic research, educational research, outcomes research, and health services research. For each topic area, a group of specialists met to develop specific recommendations. Barriers to research and opportunities for research were discussed at length; obviously, one desire of the attendees was to find ways to overcome those identified barriers. The proceedings have been published. A continuation grant from the Health Resource Service Administration was approved for the program coordinators, ensuring that this work would move forward into the future.

Opportunities for funding chiropractic research expanded in 1998 when congress established the NCCAM at the National Institutes of Health (NIH). The centers were designed to stimulate, develop, and support research on CAM for the benefit of the public. Complementary and alternative health care and medical practices are those health care and medical practices that are not currently an integral part of conventional medicine. The list of procedures that are considered CAM changes continually as CAM practices and therapies that are proven safe and effective become accepted as "mainstream" health care practices. NCCAM has the roles of exploring CAM healing practices in the context of rigorous science, training CAM researchers, and disseminating authoritative information. Funding is made available through the NIH, and grants have been awarded to chiropractic institutions.

In 2006 a group of the profession's leading researchers undertook a comprehensive decade review of the research accomplishments and status of chiropractic research. They concluded, "During the past decade, the work of chiropractic researchers has contributed substantially to the amount and quality of the evidence for or against spinal manipulation in the management of low back pain, neck pain, headache, and other conditions."⁴³

They recommended that the profession and its education institutions should strengthen its efforts to promote chiropractic research, with a focus on translating research findings into practice and a focus on evidence-based health care and best practices and their dissemination.

STANDARD OF CARE AND GUIDELINES

In early 1990, the profession held its first Consensus Conference on the validation of chiropractic methods and standard of care.⁴⁴ The conference brought together researchers, academicians, technique developers, politicians, and others from all walks of chiropractic life to develop systems to assess the validity of chiropractic procedures. The program addressed a variety of topics related to technique validation, followed by several roundtable and panel discussions related to the way such validation might occur.

The first major chiropractic-sponsored critical assessment of chiropractic methods was the professionally commissioned 1992 RAND report.⁴⁵ This project was designed to look at the clinical criteria for the use of spinal manipulation for low back pain as delivered by both chiropractors and medical doctors. The project involved four stages of study: one to review the literature concerning manipulation and low back pain, a second to convene a panel of back pain experts from a variety of disciplines to rate the appropriateness of a number of indications for the use of manipulation in treating low back pain, a third to convene a second panel solely composed of chiropractors to rate those same indications, and a fourth to analyze the services of practicing chiropractors.⁴⁶

The expert panels found that there was clear support for the use of spinal manipulation in treating acute low back pain of mechanical origin with no signs of nerve root involvement. Conclusions of the fourth stage were that the proportion of chiropractic spinal manipulation was judged congruent with appropriateness criteria similar to proportions previously described for medical procedures.⁴⁶

A similar project with parallel results examined the appropriateness of manipulation of the cervical spine.^{47,48} The effect of the studies rests with the importance of a multidisciplinary panel of experts being able to determine that spinal manipulation is appropriate for specific clinical problems of the lumbar and cervical spine.

Another consensus process, the Mercy Conference,⁴⁹ so-called because it occurred at the Mercy Center in California, was a consensus conference that brought together chiropractic clinical experts to look at the issue of standards of practice. This conference began the arduous task of looking at the full range of chiropractic procedures, diagnostic as well as clinical. The two questions that needed to be asked were, Are there any scientific data to support a conclusion about the use of a test or a procedure, and In the absence of such data, was there a consensus of opinion on the use of that test or procedure?

A list of the chapters in the published proceedings gives an idea of the scope of coverage of this conference and the guidelines it produced:

- History and physical examination
- Diagnostic imaging
- Instrumentation
- Clinical laboratory
- Record keeping and patient consents
- Clinical impressions
- Modes of care
- Frequency and duration of care
- Reassessment

- Outcome assessment
- Collaborative care
- Contraindications and complications
- Preventive and maintenance care and public health
- Professional development

Although not without great controversy, this conference had a significant effect on professional practice patterns. In an effort to maintain the momentum generated by the Mercy Conference and generate current and equitable evidence-based guidelines, the Council on Chiropractic Guidelines and Practice Parameters (CCGPP) was formed in 1995. CCGPP was delegated to examine all existing guidelines, parameters, protocols, and best practices in the United States and other nations in the construction of this document.

CCGPP researches and rates evidence that is compiled in a summary document for the chiropractic profession and other related stakeholders. The information contained in the eight clinical chapters covered in this project is being assembled by CCGPP as a literature synthesis. Appropriate therapeutic approaches will consider the literature synthesis as well as clinical experience, coupled with patient preferences in determining the most appropriate course of care for a specific patient. After several years of work the CCGPP research teams have completed a number of chapters and have posted them on the Internet for comment.

The 1990s also produced two additional and significant independent analyses concerning the management of back pain—the Manga report and the Agency for Health Care Policy and Research (now the Agency for Healthcare Research and Quality – AHRQ) *Guidelines for Acute Low Back Problems in Adults*.⁵⁰ Both had very positive implications for chiropractic care.

The Manga report⁵¹ examined the effectiveness and cost-effectiveness of chiropractic management for low back pain in the province of Ontario. Perhaps of greatest interest to the profession was the first executive finding: “On the evidence, particularly the most scientifically valid clinical studies, spinal manipulation applied by chiropractors is shown to be more effective than alternative treatments for LBP [low back pain].” They further concluded that chiropractic manipulation was safe and “far safer than medical management of LBP.” Chiropractic care was determined to be more cost-effective than medical care.

The authors concluded that increased use of chiropractic services would lead to a significant reduction in costs, fewer hospitalizations, and reduced chronic disability. Ultimately, recommendations were made to fully insure chiropractic services under the Ontario Health Insurance Plan, to extend hospital privileges, and to increase funding for chiropractic research and education.

In a follow-up study, Manga and Angus concluded that “there is an overwhelming body of evidence indicating that chiropractic management of low back pain is more cost-effective than medical management” and that “there would be highly significant cost savings if more management of low back pain was transferred from physicians to chiropractors.”⁵²

AHRQ published its guideline number 14, which discusses the management of low back pain.⁵⁰ This document represents a synthesis of the best evidence regarding the assessment and management of acute low back pain in the adult population of the United States. It employed a panel of experts drawn from the professions involved in treating low back pain, and this certainly

included chiropractic involvement. There were a number of principal conclusions:

- The initial assessment of patients with acute low back problems focuses on the detection of “red flags.”
- In the absence of red flags, imaging studies and other testing of the patient are usually not helpful during the first 4 weeks of low back pain.
- Most notably for the chiropractic profession, relief of discomfort can be accomplished most safely with nonprescription medication or spinal manipulation.
- Bed rest in excess of 4 days is not helpful and may be harmful to the patient.
- Patients need to be encouraged to return to work as soon as possible.
- Patients suffering from sciatica recover more slowly, but further evaluation can be delayed; furthermore, 80% of patients with sciatica recover without the need for surgery.

A 4-year study of comprehensive data from 1.7 million members of a managed care network in California identified that access to managed chiropractic care may reduce overall health care expenditures through several effects, including “(1) positive risk selection; (2) substitution of chiropractic care for traditional medical care, particularly for spine conditions; (3) more conservative, less invasive treatment profiles; and (4) lower health service costs associated with managed chiropractic care. Systematic access to managed chiropractic care not only may prove to be clinically beneficial but also may reduce overall health care costs.”⁵³

THE FUTURE

The chiropractic profession has labored long and hard to get to where it is, and the future holds exciting opportunities and challenges. First among its challenges is reaching consensus concerning its scope of practice and professional identity. Practitioners need to determine if they wish to continue to be viewed primarily as back pain specialists or expand the perception of chiropractic patient management skills to include such arenas as extremity disorders, sports medicine, functional medicine, and diet and nutritional counseling.

It is clear to the authors that the profession has the foundations, capacity, and expertise to expand the public’s perception and awareness of its more extensive skill set, especially in the arena of extremity dysfunction and disorders. An expanded professional image can only be accomplished through professional consensus. For this to occur, the profession must move beyond petty philosophic differences and work toward clinically demonstrating that its graduates and practitioners can safely and effectively treat a wide variety of health care disorders.

Chiropractors must provide a consistent brand and quality of care wherever it is delivered. The Association of Chiropractic Colleges “Paradigm of Chiropractic,” adopted by the profession internationally at the World Federation of Chiropractic’s Paris Congress in 2001, contains principles and goals. The “Paradigm” emphasizes an approach to the health and well-being of patients by adjustment and manipulation to address vertebral subluxation and joint dysfunction and the effect of spinal problems on biomechanical and neurologic integrity and health.⁵⁴

For a perspective from outside the profession, Wardwell,⁹ a noted chiropractic scholar and sociologist, has offered five possible outcomes for the chiropractic profession. The first option envisions the chiropractic profession disappearing altogether, with other professions (e.g., physical therapy and medicine) providing manual therapy. A second outcome for chiropractic places the profession in an ancillary position to medicine in a status similar to the role physical therapy provides today. Third, chiropractic could follow the path of osteopathy toward fusion with medicine. In a fourth possibility, the profession could evolve to a limited medical status comparable to dentistry, podiatry, optometry, or psychology. Finally, the profession may simply remain in the position it occupies today, a position of increasing recognition and public acceptance and use, but outside mainstream medical care.

Wardwell⁹ favors the fourth scenario, in which chiropractic evolves into a limited medical profession specializing in the treatment of musculoskeletal disorders. This should place the profession as an accepted member of the health care team, cooperating with medicine rather than in an adversarial position.⁵⁵

Although the profession faces some significant challenges and competition for its services, it appears unlikely that the profession will be supplanted by physical therapy or follow the path of osteopathy into medical absorption. Whether chiropractic will eventually become a limited medical profession is for the future to tell, but this also seems unlikely based on the public’s increased use of chiropractic and other CAM professions and therapies.

The chiropractic profession has survived its first century against great odds and seems destined to grow as it receives increasing acceptance from the public and the health care community. However, along with increased awareness and acceptance comes increased scrutiny. The future holds the chance for opportunity and advancement and the chance to lose some hard-gained privileges. To ensure a bright future, the profession needs to remain committed to critical self-evaluation and investigation while placing the needs of the patient above its own economic self-interests.

A challenge for the future is to classify and place all chiropractic techniques into a framework that allows the profession to determine which ones have a basis in fact. Such work has indeed begun.⁴⁴⁻⁴⁹ The profession can then begin to weed out unacceptable procedures that are promoted largely on the strength of the cult of personality that surrounds the founder of the system. The profession can appreciate the effort and drive that led so many chiropractic pioneers to devise their systems, but to allow those systems to flourish solely because of those efforts is to do a grave disservice to those who follow. Serious investigation into many of these systems is underway.

The techniques in this book are not those of any particular system, but represent a collection of procedures from many different systems, thus providing information about adjusting a wide range of areas in the body. Taken as a whole, they are a fair cross-representation of what the chiropractic profession has to offer. This book represents but one effort to ensure that credible, rational methods of chiropractic technique are available.

CONCLUSIONS

The science of chiropractic is moving forward in the investigation of the art of chiropractic. The need to continue and expand scientific research is paramount to maintaining chiropractic practice rights. The process initiated by the profession's consensus conferences, research efforts, and standard of care clinical guidelines development must be ongoing. Phillips would posit that scientific inquiry in chiropractic has created a "new soul" that is willing to search for truth, to challenge the "status quo" in the hope of making it better, and to be self-reflective of its internal values.⁵⁶

The chiropractic profession is rapidly gaining acceptance. It now has a body of credible research supporting significant elements of its patient care. The profession's research capacity and clinical research literature have expanded significantly. Several fine scientific journals (at least one of which is indexed worldwide) exist and the profession has an increasing number of high-quality textbooks. The early signs of incorporating an EBP approach to patient management are emerging. An increasing number of chiropractic colleges have been awarded EBP curriculum development grants, and most chiropractic colleges promote and support the inclusion of EBP within their curricula and patient clinics. A number of postgraduate offerings in EBP are available, and chiropractic EBP resources are available and expanding.

Meeker and Haldeman⁵⁷ have noted, despite some major health care advances during the preceding 20 years, that the chiropractic profession is still in a "transitional phase" with its future role in the overall health care system remaining unclear. They suggest that this is because the profession has yet to resolve "questions of professional and social identity."⁵⁷

Whatever identity members of the profession might prefer, any effective identity chosen must reflect not only chiropractic education, competencies, and legal scope of practice, but also the realities and dictates of the health care marketplace. At this time it appears that the majority of the general public perceives the profession as a specialist for back pain much as a dentist is viewed as a specialist for teeth.

In some countries such as Canada, Denmark, and the United Kingdom, that process has advanced significantly. A number of chiropractic schools outside the United States have affiliated with universities, and chiropractic services are covered within the national health care systems in a number of these countries. The identity of chiropractic in these markets is evolving into a limited-practice model focused on expertise, evaluation, and treatment of a narrow range of musculoskeletal disorders, especially spinal problems.⁵⁸

In a survey conducted at the Institute of Social Research at Ohio Northern University, important issues for the chiropractic profession were addressed including the appropriateness of various services, attitudes toward prescription drugs and immunization, and opinions on whether specific or general visceral health problems may be related to subluxation or its correction.⁵⁹ The results of the survey found that the North American chiropractic profession has largely outgrown its historical stereotype of being defensive, divided, and isolated from mainstream health care. The survey concluded that "North American chiropractors are less defensive, less absolutist and less polemic than the stereotype. The data also indicate that chiropractors know they offer patients a valuable service. The picture emerging from their survey is of a confident, pragmatic and discerning profession, more capable than ever of participating in an interdisciplinary health care environment."⁵⁹

JOINT ANATOMY AND BASIC BIOMECHANICS

OUTLINE

FUNDAMENTAL CONCEPTS, PRINCIPLES, AND TERMS

Levers	11
Body Planes	12
Axes of Movement	13
Joint Motion	13
Synovial Joints	15
Bony Elements	15
Articular Cartilage	16

Fibrocartilage	16
Ligamentous Elements	17
Synovial Fluid	17
Articular Neurology	18
JOINT FUNCTION	20
MECHANICAL FORCES ACTING ON	
CONNECTIVE TISSUE	23
Tension Forces	24
Compression Forces	24
Shear Forces	24

Torque Forces	25
Newton's Laws of Motion	25
PROPERTIES OF CONNECTIVE	
TISSUE	26
Muscle	27
Ligaments	28
Facet Joints	29
Intervertebral Discs	30
MODELS OF SPINE FUNCTION	33

This chapter provides an academic picture of the applied anatomy and clinical biomechanics of the musculoskeletal system. The human body may be viewed as a machine formed of many different parts that allow motion. These motions occur at the many joints formed by the specific parts that compose the body's musculoskeletal system. Although there is some controversy and speculation among those who study these complex activities, the information presented here is considered essential for understanding clinical correlations and applications. Clinical biomechanics and applied anatomy encompass the body of knowledge that employs mechanical facts, concepts, principles, terms, methodologies, and mathematics to interpret and analyze normal and abnormal human anatomy and physiology. Discussions of these concepts require specific nomenclature, which enables people working in a wide variety of disciplines to communicate (see glossary). Biomechanics is often overwhelming because of its mathematical and engineering emphasis. This chapter presents a nonmathematical approach to defining clinically useful biomechanical concepts necessary to describe and interpret changes in joint function. Thorough explanations of biomechanical concepts are discussed in other works.¹⁻³

FUNDAMENTAL CONCEPTS, PRINCIPLES, AND TERMS

Mechanics is the study of forces and their effects. *Biomechanics* is the application of mechanical laws to living structures, specifically to the locomotor system of the human body. Therefore biomechanics concerns the interrelations of the skeleton, muscles, and joints. The bones form the levers, the ligaments surrounding the joints form hinges, and the muscles provide the forces for moving the levers about the joints. *Force* is an action exerted on a body that causes it to deform or to move. The most important forces involved with musculoskeletal levers are those produced by muscle, gravity, and physical contacts within the environment.

Kinematics is a branch of mechanics that deals with the geometry of the motion of objects, including displacement, velocity, and acceleration, without taking into account the forces that produce

the motion. *Kinetics*, however, is the study of the relationships between the force system acting on a body and the changes it produces in body motion.

Knowledge of joint mechanics and structure, as well as the effects that forces produce on the body, has important implications for the use of manipulative procedures and, specifically, chiropractic adjustments. Forces have vector characteristics whereby specific directions are delineated at the points of application. Moreover, forces can vary in magnitude, which affects the acceleration of the object to which the force is applied.

LEVERS

A lever is a rigid bar that pivots about a fixed point, called the *axis* or *fulcrum*, when a force is applied to it. A force in the body is applied by muscles at some point along a lever to move a body part to overcome some form of *resistance*. The lever is one of the simplest of all mechanical devices that can be called a *machine*. The relationship of fulcrum to force and to resistance distinguishes the different classes of levers.

In a first-class lever, the axis (fulcrum) is located between the force and resistance; in a second-class lever, the resistance is between the axis and the force; and in a third-class lever, the force is between the axis and the resistance (Figure 2-1). Every movable bone in the body acts alone or in combination, forcing a network of lever systems characteristic of first- and third-class levers. There are virtually no second-class levers in the body, although opening the mouth against resistance is an example.

With a first-class lever, the longer the lever arm, the less force required to overcome the resistance. The force arm may be longer, shorter, or equal to the resistance arm, but the axis is always between these two points. An example of a first-class lever in the human body is the forearm moving from a position of flexion into extension at the elbow through contraction of the triceps muscle.

Third-class levers are the most common types in the body because they allow the muscle to be inserted near the joint and can thereby produce increased speed of movement, although at a sacrifice of force. The force must be smaller than the resistance arm,

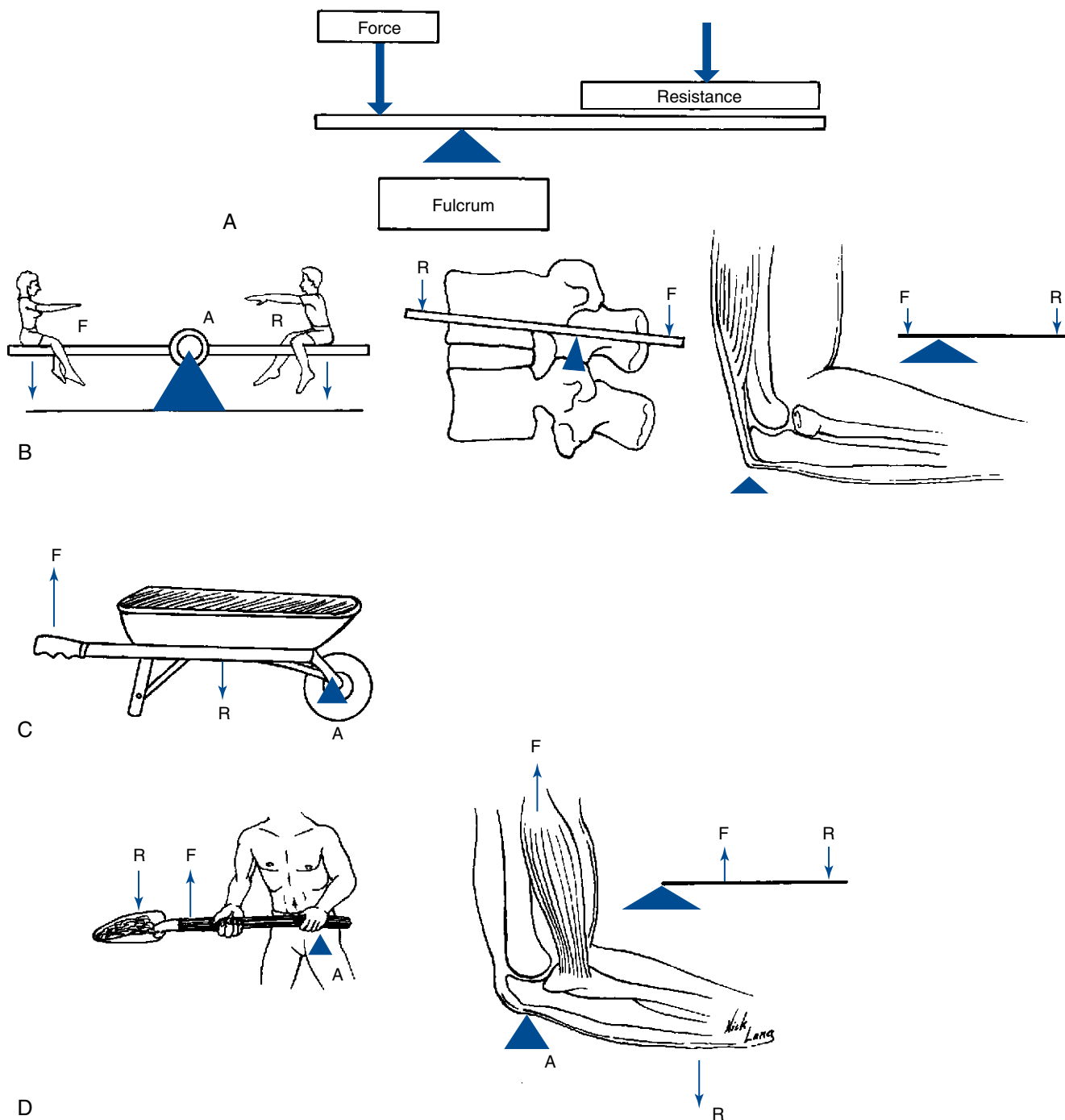


Figure 2-1 A, Lever system showing components. B, First-class lever system. C, Second-class lever system. D, Third-class lever system. A, Axis (fulcrum); F, force; R, resistance.

and the applied force lies closer to the axis than the resistance force. An example of a third-class lever is flexion of the elbow joint through contraction of the biceps muscle.

BODY PLANES

It is also necessary to delineate the specific body planes of reference, because they are used to describe structural position and directions of functional movement. The standard position of

reference, or anatomic position, has the body facing forward, the hands at the sides of the body, with the palms facing forward, and the feet pointing straight ahead. The body planes are derived from dimensions in space and are oriented at right angles to one another. The *sagittal plane* is vertical and extends from front to back, or from anterior to posterior. Its name is derived from the direction of the human sagittal suture in the cranium. The *median sagittal plane*, also called the *midsagittal plane*, divides the body into right and left halves (Figure 2-2, A, Table 2-1). The *coronal*

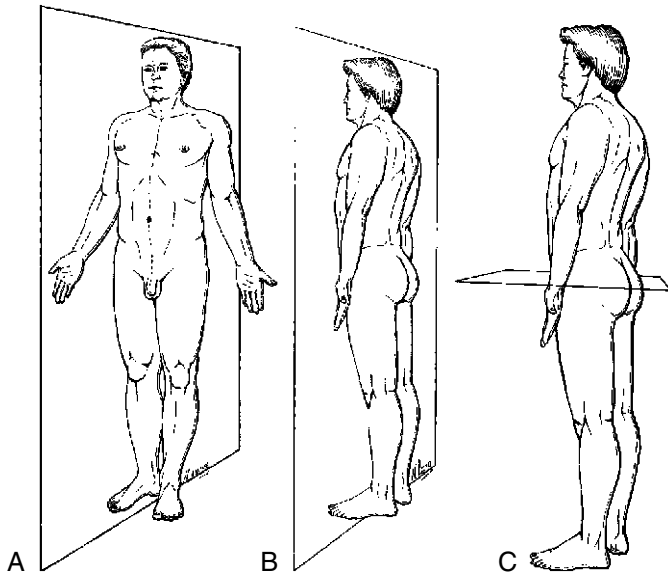


Figure 2-2 A, Midsagittal plane. Movements of flexion and extension take place about an axis in the sagittal plane. B, Coronal plane. Movements of abduction and adduction (lateral flexion) take place about an axis in the coronal plane. C, Transverse plane. Movements of medial and lateral rotation take place about an axis in the transverse plane.

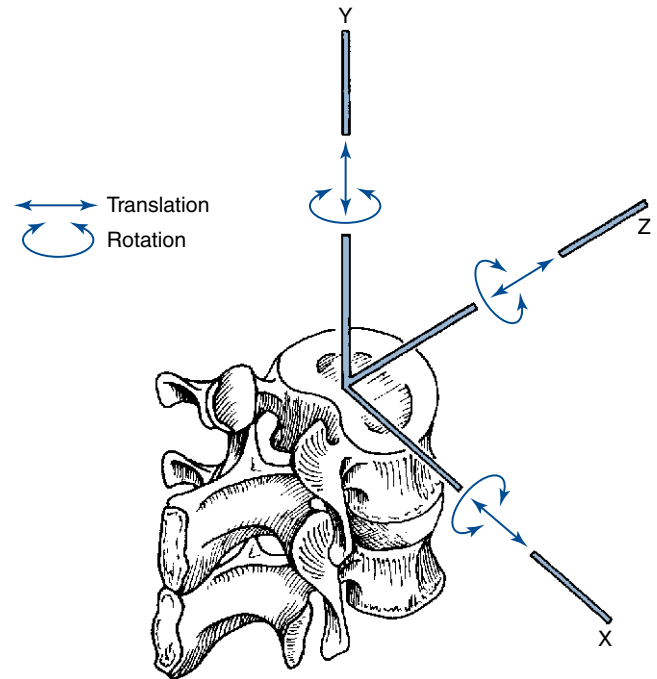


Figure 2-3 Three-dimensional coordinate system identifying the translational and rotational movements along or around the three axes to produce 6 degrees of freedom.

TABLE 2-1 Body Planes of Movement

Plane of Movement	Axis	Joint Movement
Sagittal	x	Flexion and extension; Lateral to Medial, and Medial to Lateral Glide
Coronal	z	Abduction and adduction (lateral flexion); Anterior to Posterior, and Posterior to Anterior Glide
Transverse	y	Medial and lateral rotation (axial rotation) Inferior to Superior, and Superior to Inferior Glide (compression, distraction)

plane is vertical and extends from side to side. Its name is derived from the orientation of the human coronal suture of the cranium. It may also be referred to as the *frontal plane*, and it divides the body into anterior and posterior components (Figure 2-2B). The transverse plane is a horizontal plane and divides a structure into upper and lower components (Figure 2-2C).

AXES OF MOVEMENT

An axis is a line around which motion occurs. Axes are related to planes of reference, and the cardinal axes are oriented at right angles to one another. This is expressed as a three-dimensional coordinate system with X, Y, and Z used to mark the axes (Figure 2-3). The significance of this coordinate system is in defining or locating the extent of the types of movement possible at each joint—rotation, translation, and curvilinear motion. All movements that occur about an axis are considered *rotational*, whereas linear movements along an axis and through a plane are called

translational. *Curvilinear* motion occurs when a translational movement accompanies rotational movements. The load that produces a rotational movement is called *torque*; a force that produces a translational movement is called an *axial* or *shear force*.

JOINT MOTION

Motion can be defined as a continuous change in position of an object and can be described as rotational, translational or curvilinear. Rotational motion takes place around an axis. Translational movements are linear movements or, simply, movement in a straight line. The terms *slide* and *glide* have been used to refer to translational movements between joint surfaces. Curvilinear motion combines both rotational and translational movements and is the most common motion produced by the joints of the body (Figure 2-4).

The three axes of motion (x, y, and z) are formed by the junction of two planes. The x-axis is formed by the junction of the coronal and transverse planes. The y-axis is formed by the junction of the

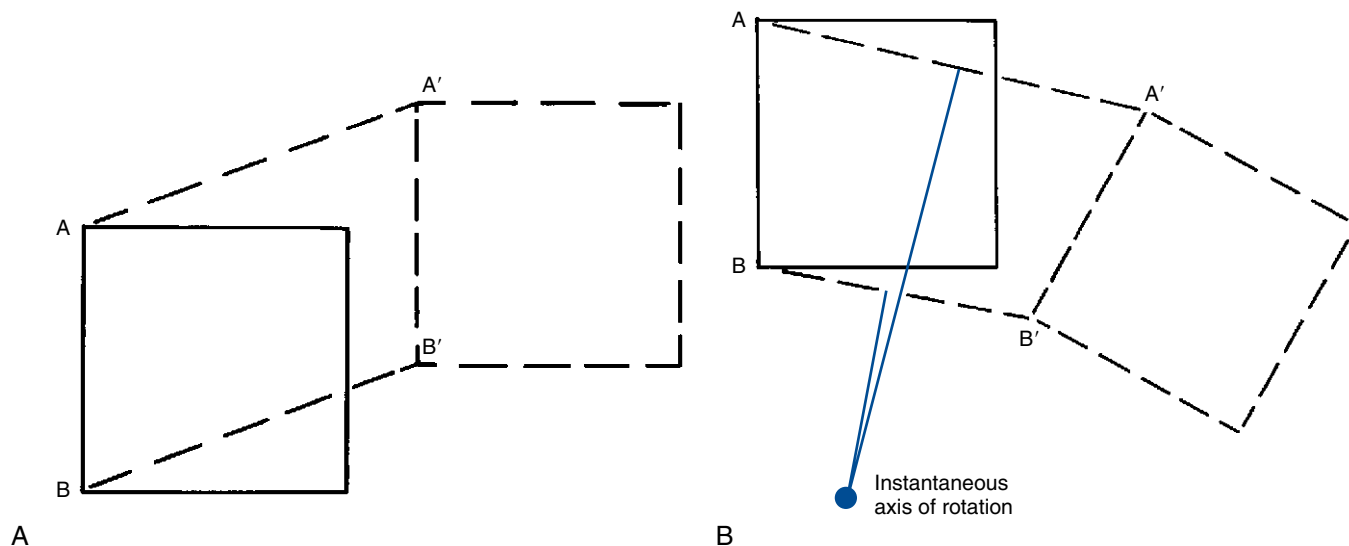


Figure 2-4 A, Translational movement. B, Curvilinear movement: a combination of translation and rotation movements.

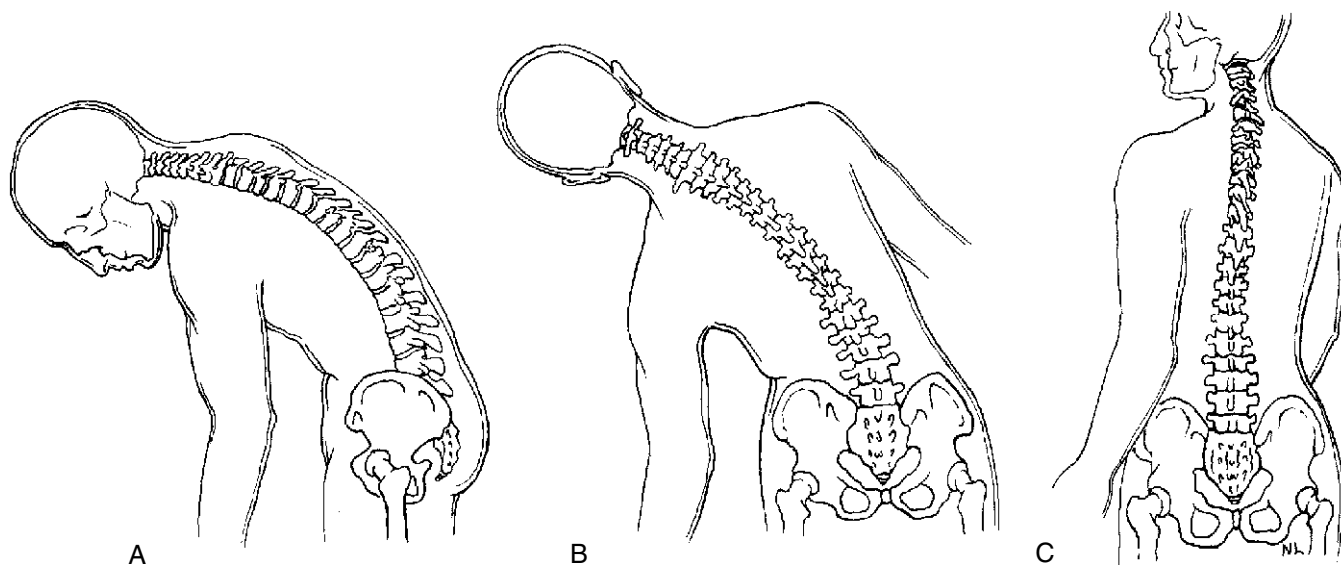


Figure 2-5 A, Sagittal plane movement of flexion. B, Coronal plane movement of lateral flexion. C, Transverse plane movement of axial rotation.

coronal and sagittal planes. The z-axis is formed by the junction of the sagittal and transverse planes. The potential exists for each joint to exhibit three translational movements and three rotational movements, constituting 6 degrees of freedom. The axis around or along which movement takes place and the plane through which movement occurs define specific motions or resultant positions.

The x-axis extends from one side of the body to the other. The motions of flexion and extension occur about this axis and through the sagittal plane. Flexion is motion in the anterior direction for joints of the head, neck, trunk, upper extremity, and hips. Flexion of the knee, ankle, foot, and toes is movement in the posterior direction. Extension is motion in the direct opposite manner from flexion (Figure 2-5, A). Lateral to medial glide and medial to lateral glide (laterolisthesis) translate through the coronal plane and along the x-axis.

The z-axis extends horizontally from anterior to posterior. Movements of abduction and adduction of the extremities, as

well as lateral flexion of the spine, occur around this axis and through the coronal plane. Lateral flexion is a rotational movement and is used to denote lateral movements of the head, neck, and trunk in the coronal plane (see Figure 2-5, B). In the human, lateral flexion is usually combined with some element of rotation. Abduction and adduction are also motions in a coronal plane. Abduction is movement away from the body, and adduction is movement toward the body; the reference here is to the midsagittal plane of the body. This would be true for all parts of the extremities, excluding the thumb, fingers, and toes. For these structures, reference points are found within that particular extremity. Anterior to posterior glide (anterolisthesis) and posterior to anterior glide (retrolisthesis) are translational movements through the sagittal plane and along the z-axis.

The longitudinal axis (y-axis) is vertical, extending in a head-to-toe direction. Movements of medial (internal) and lateral

(external) rotation in the extremities, as well as axial rotation in the spine, occur around it and through the transverse plane. Axial rotation is used to describe this type of movement for all areas of the body except the scapula and clavicle. Rotation occurs about an anatomic axis, except in the case of the femur, which rotates around a mechanical axis.⁴ In the human extremity, the anterior surface of the extremity is used as a reference area. Rotation of the anterior surface toward the midsagittal plane of the body is medial (internal) rotation, and rotation away from the midsagittal plane is lateral (external) rotation (see Figure 2-5, C). Supination and pronation are rotation movements of the forearm. Distraction and compression (altered interosseous spacing or superior or inferior glide) translate through the transverse plane along the y-axis.

Because the head, neck, thorax, and pelvis rotate about longitudinal axes in the midsagittal area, rotation cannot be named in reference to the midsagittal plane. Rotation of the head, spine, and pelvis is described as rotation of the anterior surface posteriorly toward the right or left. Rotation of the scapula is movement about a sagittal axis, rather than about a longitudinal axis. The terms *clockwise* or *counterclockwise* are used.

The extent of each movement is based more or less on the joint anatomy and, specifically, the plane of the joint surface. This is especially important in the spinal joints. Each articulation in the body should then exhibit, to some degree, flexion, extension, right and left lateral flexion, right and left axial rotation, anteroposterior glide, posteroanterior glide, lateromedial glide, mediolateral glide, compression, and distraction.

Joints are classified first by their functional capabilities and then are subdivided by their structural characteristics. Synarthroses allow very little, if any, movement; an amphiarthrodial (symphysis) joint allows motion by virtue of its structural components; diarthroses, or true synovial joints, allow significant movement. The structural characteristics of these joints are detailed in Table 2-2.

SYNOVIAL JOINTS

Synovial joints are the most common joints of the human appendicular skeleton, representing highly evolved, movable joints. Although these joints are considered freely movable, the degree of possible motion varies according to the individual structural design, facet planes, and primary function (motion vs. stability). The components of a typical synovial joint include the bony elements, articular cartilage, fibrocartilage, synovial membrane, fibroligamentous joint capsule, and articular joint receptors. An understanding of the basic anatomy of a synovial joint forms the foundation for appreciation of clinically significant changes in the joint that lead to joint dysfunction.

BONY ELEMENTS

The bony elements provide the supporting structure that gives the joint its capabilities and individual characteristics by forming lever arms to which intrinsic and extrinsic forces are applied. Bone is actually a form of connective tissue that has an inorganic constituent (lime salts). A hard outer shell of cortical bone provides structural support and surrounds the cancellous bone, which contains marrow and blood vessels that provide nutrition. Trabecular patterns develop in the cancellous bone, corresponding to mechanical stress applied to and required by the bone (Figure 2-6). Bone also has the important role of hemopoiesis (formation of blood cells). Furthermore, bone stores calcium and phosphorus, which it exchanges with blood and tissue fluids. Finally, bone has the unique characteristic of repairing itself with its own tissue as opposed to fibrous scar tissue, which all other body tissues use. Bone is a very dynamic tissue, constantly remodeling in response to forces from physical activity and in response to hormonal influences that regulate systemic calcium balance. Bone, by far, has the best capacity for remodeling, repair, and regeneration of all the tissues making up joint struc-

TABLE 2-2 Joint Classification

Joint Type	Structure	Example
Synarthrotic		
Fibrous	Suture—nearly no movement	Cranial sutures
	Syndesmosis—some movement	Distal tibia-fibula
Cartilaginous	Synchondrosis—temporary	Epiphyseal plates
	Symphysis—fibrocartilage	Pubes
		Intervertebral discs
Diarthrotic		
Uniaxial	Ginglymus (hinge)	Elbow
	Trochoid (pivot)	Atlantoaxial joint
	Condylar	Metacarpophalangeal joint
Biaxial	Ellipsoid	Radiocarpal joint
	Sellar (saddle)	Carpometacarpal joint of the thumb
Multiaxial	Triaxial	Shoulder
	Spheroid (ball and socket)	Hip
Plane (nonaxial)		Intercarpal joints
		Posterior facet joints in the spine

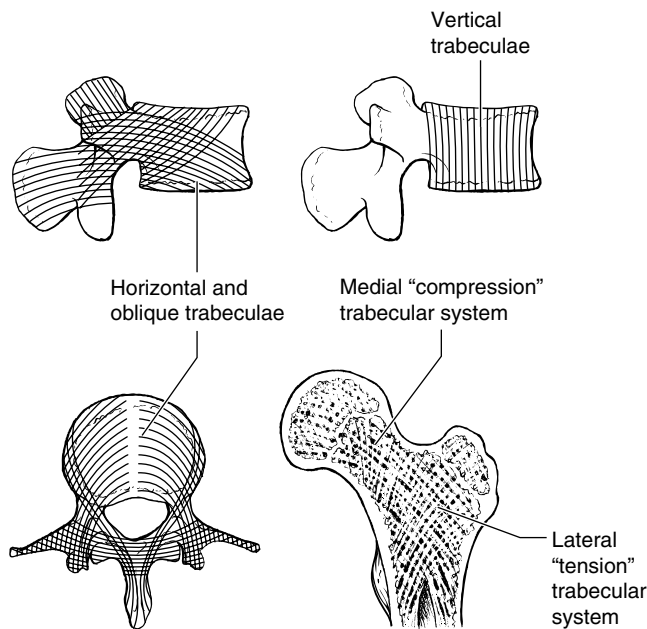


Figure 2-6 Trabecular patterns corresponding to mechanical stresses in the hip joint and vertebra. (Modified from Hertling D, Kessler RM: *Management of common musculoskeletal disorders: Physical therapy principles and methods*, ed 2, Philadelphia, 1990, JB Lippincott.)

tures. The bony elements of the spine are the vertebral body and neural arch. The cortical shell (compact bone) and cancellous core (spongy bone) play a significant role in weight-bearing and the absorption of compressive loads. The compressive strength of the vertebrae increases from C1 to L5.

ARTICULAR CARTILAGE

Articular cartilage, a specialized form of hyaline cartilage, covers the articulating surfaces in synovial joints and helps to transmit loads and reduce friction. It is bonded tightly to the subchondral bone through the zone of calcification, which is the end of bone visible on x-ray film. The joint space visible on x-ray film is composed of the synovial cavity and noncalcified articular cartilage. In its normal composition, articular cartilage has four histologic areas or zones (Figure 2-7). These zones have been further studied and refined so that a wealth of newer information regarding cartilage has developed.

The outermost layer of cartilage is known as the *gliding zone*, which itself contains a superficial layer (outer) and a tangential layer (inner). The outer segment is made up solely of collagen randomly oriented into flat bundles. The tangential layer consists of densely packed layers of collagen, which are oriented parallel to the surface of the joint.⁵ This orientation is along the lines of the joint motion, which implies that the outer layers of collagen are stronger when forces are applied parallel to the joint motion rather than perpendicular to it.⁶ This particular orientation of fibers provides a great deal of strength to the joint in normal motion. The gliding zone also has a role in protecting the deeper elastic cartilage.

The *transitional zone* lies beneath the gliding zone. It represents an area where the orientation of the fibers begins to change from the parallel orientation of the gliding zone to the more

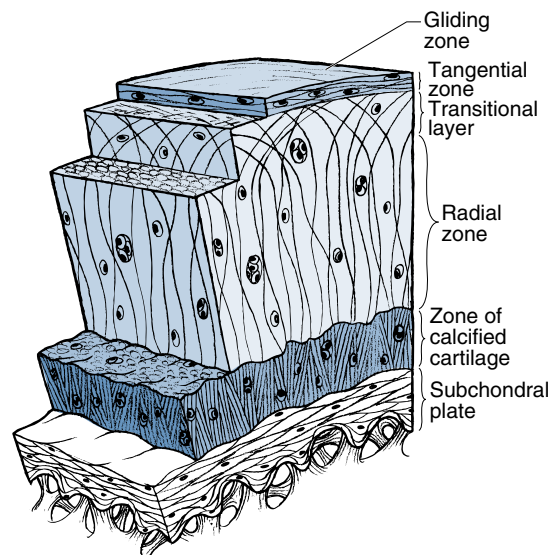


Figure 2-7 Microscopic anatomy of articular cartilage.

perpendicular orientation of the radial zone. Therefore fiber orientation is more or less oblique and, in varying angles, formed from glucuronic acid and *N*-acetylgalactosamine with a sulfate on either the fourth or sixth position. The keratin compound is formed with galactose and *N*-acetylgalactosamine. All of this occurs in linked, repeating units (Figure 2-8).

Articular cartilage is considered mostly avascular and lacks a perichondrium, eliminating a source of fibroblastic cells for repair. Articular cartilage must rely on other sources for nutrition, removal of waste products, and the process of repair. Therefore intermittent compression (loading) and distraction (unloading) are necessary for adequate exchange of nutrients and waste products. The highly vascularized synovium is believed to be a critical source of nutrition for the articular cartilage it covers. The avascular nature of articular cartilage limits the potential for cartilage repair by limiting the availability of the repair products on which healing depends. Chondrocytes, the basic cells of cartilage that maintain and synthesize the matrix, are contained within a mesh of collagen and proteoglycan that does not allow them to migrate to the injury site from adjacent healthy cartilage.⁷ Moreover, the articular cartilage matrix may contain substances that inhibit vascular and macrophage invasion and clot formation that are also necessary for healing.⁸ After an injury to the articular cartilage, the joint can return to an asymptomatic state after the transient synovitis subsides. Degeneration of the articular cartilage depends on the size and depth of the lesion, the integrity of the surrounding articular surface, the age and weight of the patient, associated meniscal and ligamentous lesions, and a variety of other biomechanical factors.⁷ Continuous passive motion has increased the ability of full-thickness defects in articular cartilage to heal, producing tissue that closely resembles hyaline cartilage.⁹

FIBROCARTILAGE

Fibrocartilage has a higher fiber content than other types of cartilage. It has the properties of both dense irregular connective tissue and articular cartilage. Fibrocartilage forms much of the substance

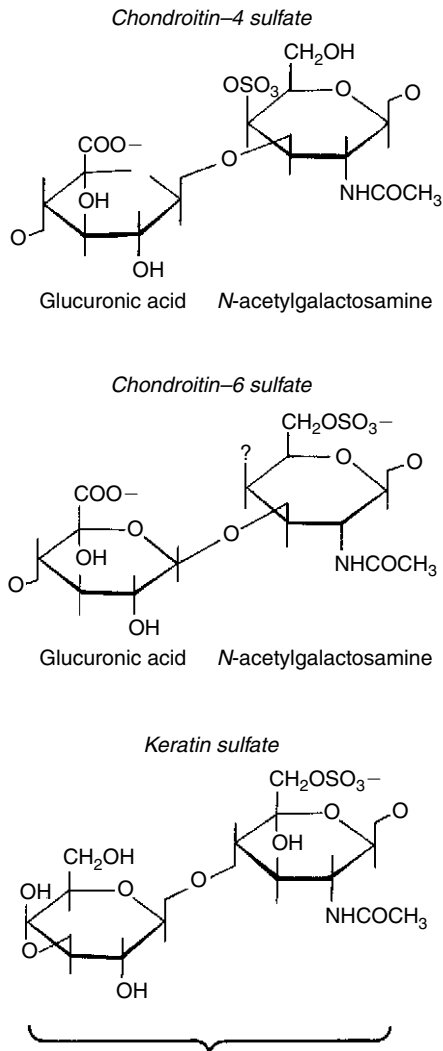


Figure 2-8 Structure of chondroitin and keratin compounds.

of the intervertebral discs and the discs located within the pubic symphysis and other joints of the extremities (e.g., knee). The role of fibrocartilage is to support and stabilize the joints as well as dissipate compressive forces. Fibrocartilage largely depends on diffusion of nutrients contained in the adjacent trabecular bone. Therefore, it depends on a “load-unload” mechanism to help the diffusion of nutrients and removal of metabolic wastes.

LIGAMENTOUS ELEMENTS

The primary ligamentous structure of a synovial joint is the joint capsule. Throughout the vertebral column the joint capsules are thin and loose. The capsules are attached to the opposed superior and inferior articular facets of adjacent vertebrae. Joint capsules in the spine have three layers.¹⁰ The outer layer is composed of dense fibroelastic connective tissue made up of parallel bundles of collagen fibers. The middle layer is composed of loose connective tissue and areolar tissue containing vascular structures. The inner layer consists of the synovial membrane. The fibers are generally oriented in a direction perpendicular to the plane of the facet joints. This joint capsule covers the posterior and lateral aspects of the

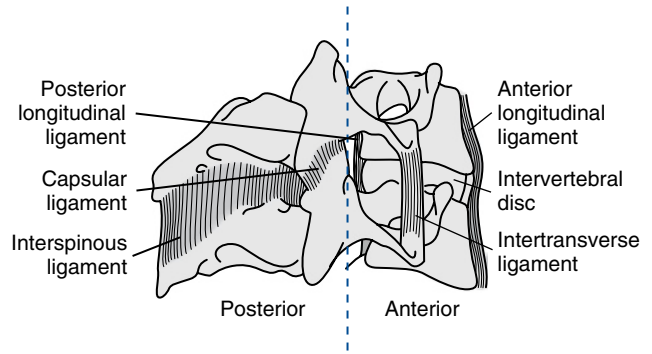


Figure 2-9 Lateral view of a cervical motion segment identifying the ligamentous structures.

zygapophyseal joint. The capsular ligaments provide flexion stability in the cervical spine.¹ The ligamentum flavum covers the joint capsules anteriorly and medially and connects the borders of adjacent laminae from the second cervical vertebra to the first sacral vertebra. These ligaments, referred to as *yellow ligaments*, are composed of a large amount of elastic fibers. This allows for a significant amount of tension to the ligament without permanent deformation. Clinically this is an important characteristic for the spine if it suddenly goes from full flexion to full extension. The high elasticity of the ligamentum flavum minimizes the chances of any impingement of the spinal cord. The anterior longitudinal ligament (ALL) is a fibrous tissue structure that is attached to the anterior surfaces of the vertebral bodies, including part of the sacrum. The ALL attaches firmly to the edges of the vertebral bodies but is not firmly attached to the annular fibers of the disc. It is narrowed at the level of the disc. The posterior longitudinal ligament (PLL) runs over the posterior surfaces of all of the vertebral bodies down to the coccyx. It has an interwoven connection with the intervertebral disc and is wider at the disc level but narrower at the vertebral body level. Both the ALL and PLL deform with separation and approximation between the two adjacent vertebrae and with disc bulging. The ALL has been found to be twice as strong as the PLL.¹ The intertransverse ligaments attach between the transverse processes. They are fairly substantial in the thoracic spine, but quite small in the lumbar spine. The interspinous and supraspinatus ligaments attach between the spinous processes (Figure 2-9).

SYNOVIAL FLUID

Synovial fluid is an ultrafiltrate of blood with additives produced by the synovium to provide nourishment for the avascular articular cartilage and contribute to the lubrication and protection of the articular cartilage surfaces.¹¹ The identity of the significant active ingredient within synovial fluid that provides the near frictionless performance of diarthrodial joints, has been the quest of researchers for many years. Initially, hyaluronic acid was thought to be the lubricant, but it has not demonstrated the load-bearing properties required within the physiologic joint. Currently lubricin is being investigated as the possible substance within the synovial fluid with the necessary attributes. Lubricin is the glycoprotein fraction of synovial fluid that is secreted by surface chondrocytes and synovial

cells. It has been shown to have the same lubricating ability because of the surface-active phospholipids present in lubricin.^{12,13}

Although the exact role of synovial fluid is still unknown, it is thought to serve as a joint lubricant or at least to interact with the articular cartilage to decrease friction between joint surfaces. This is of clinical relevance because immobilized joints have been shown to undergo degeneration of the articular cartilage.¹⁴ Synovial fluid is similar in composition to plasma, with the addition of mucin (hyaluronic acid), which gives it a high molecular weight and its characteristic viscosity. Three models of joint lubrication exist. The controversy lies in the fact that no one model of joint lubrication applies to all joints under all circumstances.

According to the hydrodynamic model, synovial fluid fills in spaces left by the incongruent joint surfaces. During joint movement, synovial fluid is attracted to the area of contact between the joint surfaces, resulting in the maintenance of a fluid film between moving surfaces. This model was the first to be described and works well with quick movement, but it would not provide adequate lubrication for slow movements and movement under increased loads.

The elastohydrodynamic model is a modification of the hydrodynamic model that considers the viscoelastic properties of articular cartilage whereby deformation of joint surfaces occurs with loading, creating increased contact between surfaces. This would effectively reduce the compression stress to the lubrication fluid. Although this model allows for loading forces, it does not explain lubrication at the initiation of movement or the period of relative zero velocity during reciprocating movements.¹⁵

In the boundary lubrication model, the lubricant is adsorbed on the joint surface, which would reduce the roughness of the surface by filling the irregularities and effectively coating the joint surface. This model allows for initial movement and zero velocity movements. Moreover, boundary lubrication combined with the elastohydrodynamic model, creating a mixed model, meets the demands of the human synovial joint (Figure 2-10).

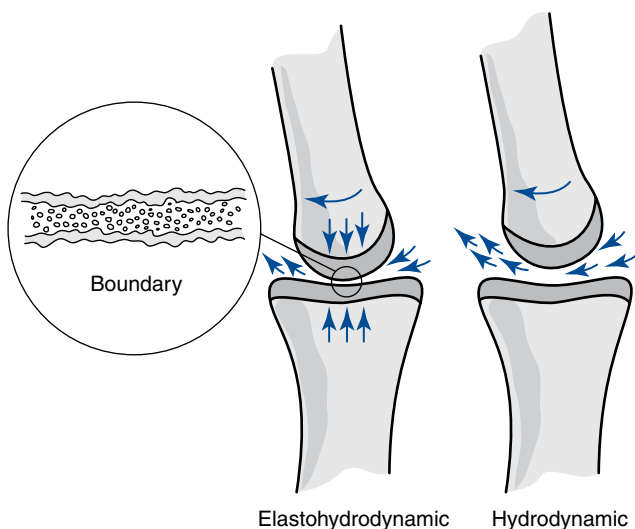


Figure 2-10 Lubrication models for synovial joints. (Modified from Hertling D, Kessler RM: *Management of common musculoskeletal disorders: Physical therapy principles and methods*, ed 2, Philadelphia, 1990, JB Lippincott.)

ARTICULAR NEUROLOGY

Articular neurology provides information on the nature of joint pain, the relationship of joint pain to joint dysfunction, and the role of manipulative procedures in affecting joint pain. The spinal viscoelastic structures, including disk, capsule, and ligaments, were found to have abundant afferents capable of monitoring proprioceptive and kinesthetic information.¹⁶ Therefore, spinal structures are well suited to monitor sensory information and provide kinesthetic perception for coordinated motor control and movement.

Synovial joints are innervated by three or four varieties of neuroreceptors, each with a wide variety of parent neurons. The parent neurons differ in diameter and conduction velocity, representing a continuum from the largest heavily myelinated A α -fibers to the smallest unmyelinated C fibers. All are derived from the dorsal and ventral rami, as well as the recurrent meningeal nerve of each segmental spinal nerve (Figure 2-11). Information from these receptors spreads among many segmental levels because of multilevel ascending and descending primary afferents. The receptors are divided into the four groups according to their neurohistologic properties, which include three corpuscular mechanoreceptors and one nociceptor.¹⁷

Type I receptors are confined to the outer layers of the joint capsule and are stimulated by active or passive joint motions. Their firing rate is inhibited with joint end approximation, and they have a low threshold, making them very sensitive to movement. Some are considered static receptors because they fire continually, even with no joint movement. Because they are slow-adapting, the effects of movement are long lasting. Stimulation of type I receptors is involved with the following:

1. Reflex modulation of posture, as well as movement (kinesthetic sensations), through constant monitoring of outer joint tension
2. Perception of posture and movement
3. Inhibition of centripetal flow from pain receptors via an enkephalin synaptic interneuron transmitter
4. Tonic effects on lower motor neuron pools involved in the neck, limbs, jaw, and eye muscles

Type II mechanoreceptors are found within the deeper layers of the joint capsule. They are also low-threshold and again are stimulated with even minor changes in tension within the inner joint. Unlike type I receptors, however, type II receptors adapt very rapidly and quickly cease firing when the joint stops moving. Type II receptors are completely inactive in immobilized joints. Functions of the type II receptors are likely to include the following:

1. Movement monitoring for reflex actions and perhaps perceptual sensations
2. Inhibition of centripetal flow from pain receptors via an enkephalin synaptic interneuron neutral transmitter
3. Phasic effects on lower motor neuron pools involved in the neck, limbs, jaw, and eye muscles

Type III mechanoreceptors are found in the intrinsic and extrinsic ligaments of the peripheral joints, but they had been previously thought to be absent from all of the synovial spinal joints. However, McLain¹⁸ examined 21 cervical facet capsules from three normal human subjects and found type III receptors, although they were less abundant than either type I or type II. These recep-

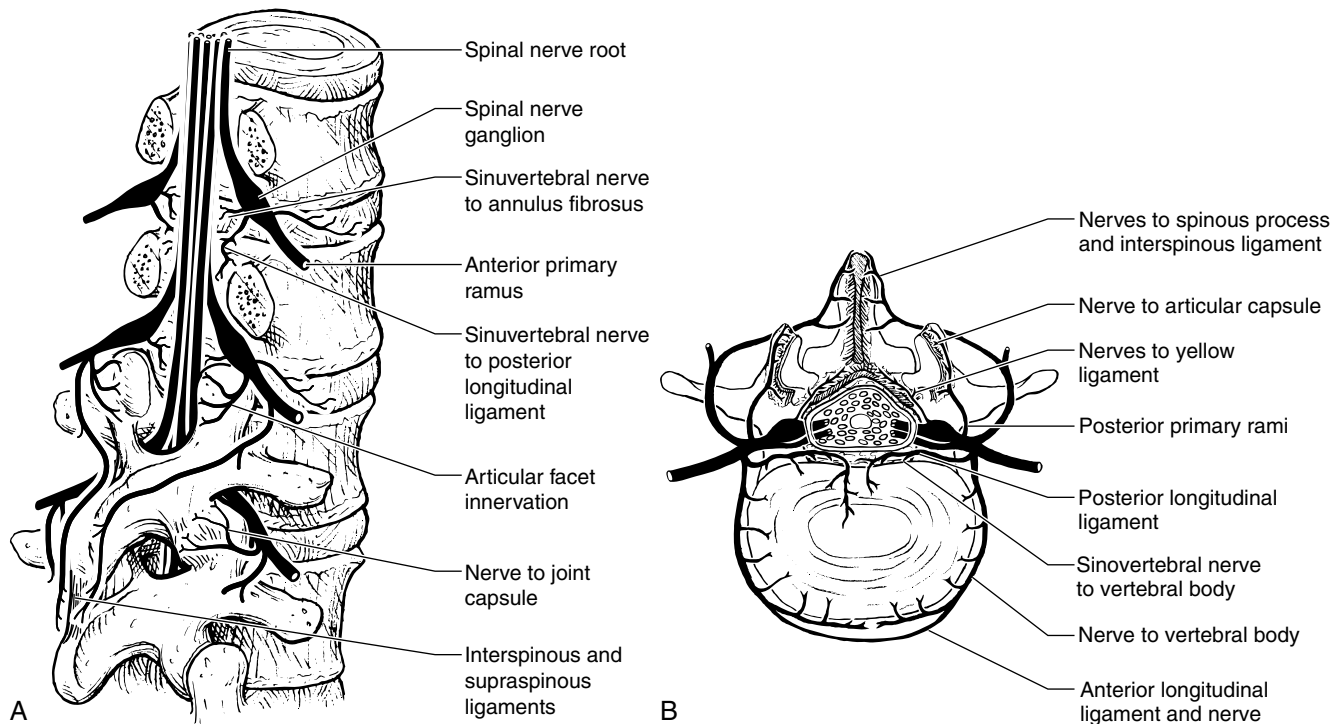


Figure 2-11 Innervation of the outer fibers of the disc and facet joint capsule by the sinuvertebral nerve. **A**, Oblique posterior view. **B**, Top view. (Modified from White AA, Panjabi MM: *Clinical biomechanics of the spine*, Philadelphia, 1978, JB Lippincott.)

tors are very slow adapters with a very high threshold because they are innervated by large myelinated fibers. They seem to be the joint version of the Golgi tendon organ in that they impose an inhibitory effect on motor neurons. Although the functions of type III receptors are not completely understood, it is likely that they achieve the following:

1. Monitor direction of movement
2. Create a reflex effect on segmental muscle tone, providing a “braking mechanism” against movement that over-displaces the joint
3. Recognize potentially harmful movements

Type IV receptors are composed of a network of free nerve endings, as well as unmyelinated fibers. They are associated with pain perception and include many different varieties with large ranges of sensations, including itch and tickle. They possess an intimate physical relationship to the mechanoreceptors and are present throughout the fibrous portions of the joint capsule and ligaments. They are absent from articular cartilage and synovial linings, although they have been found in synovial folds.^{19,20} They are very high-threshold receptors and are completely inactive in the physiologic joint. Joint capsule pressure, narrowing of the intervertebral disc, fracture of a vertebral body, dislocation of the zygapophyseal joints, chemical irritation, and interstitial edema associated with acute or chronic inflammation may all activate the nociceptive system. The basic functions of the nociceptors include the following:

1. Evocation of pain
2. Tonic effects on neck, limb, jaw, and eye muscles
3. Central reflex connections for pain inhibition
4. Central reflex connections for myriad autonomic effects

Postural control represents a complex interplay between the sensory and motor systems and involves perceiving environmental stimuli, responding to alterations in the body’s orientation within the environment, and maintaining the body’s center of gravity within the base of support.^{21,22} Sensory information about the status of the body within the environment emanates primarily from the proprioceptive, cutaneous, visual, and vestibular systems. Researchers²³⁻²⁵ have concluded that individuals rely primarily on proprioceptive and cutaneous input to maintain normal quiet stance and to safely accomplish the majority of activities of daily living, but must integrate information from multiple sensory systems as task complexity and challenge to postural stability increase.

A relationship exists between mechanoreceptors and nociceptors such that when the mechanoreceptors function correctly, an inhibition of nociceptor activity occurs.¹⁷ The converse also holds true; when the mechanoreceptors fail to function correctly, inhibition of nociceptors will occur less, and pain will be perceived.¹⁷

Discharges from the articular mechanoreceptors are polysynaptic and produce coordinated facilitatory and inhibitory reflex changes in the spinal musculature. This provides a significant contribution to the reflex control of these muscles.¹⁷ Gillette¹⁹ suggests that a chiropractic adjustment produces sufficient force to coactivate a wide variety of mechanically sensitive receptor types in the paraspinal tissues. The A- δ -mechanoreceptors and C-polymodal nociceptors, which can generate impulses during and after stimulation, may well be the most physiologically interesting component of the afferent bombardment initiated by high-velocity, low-amplitude manipulations. For normal function of the joint structures, an integration of proprioception, kinesthetic perception, and reflex regulation is absolutely essential.

Pain-sensitive fibers also exist within the annulus fibrosus of the disc. Malinsky²⁰ demonstrated the presence of a variety of free and complex nerve endings in the outer one third of the annulus. The disc is innervated posteriorly by the recurrent meningeal nerve (sinuvertebral nerve) and laterally by branches of the gray rami communicantes. During evaluation of disc material surgically removed before spinal fusion, Bogduk²⁶ found abundant nerve endings with various morphologies. The varieties of nerve endings included free terminals, complex sprays, and convoluted tangles. Furthermore, many of these endings contained substance P, a putative transmitter substance involved in nociception.

Shinohara²⁷ reported the presence of such nerve fibers accompanying granulation tissue as deep as the nucleus in degenerated discs. Freemont and associates²⁸ examined discs from individuals free of back pain and from those with back pain. They identified nerve fibers in the outer one third of the annulus in pain-free disc samples, but they found nerve fibers extending into the inner one third of the annulus and into the nucleus pulposus of the discs from the pain sample. They suggest that their findings of isolated nerve fibers that express substance P deep within diseased intervertebral discs may impart an important role in the pathogenesis of chronic low back pain. Abundant evidence shows that the disc can be painful, supporting the ascribed nociceptive function of the free nerve endings.^{20,26-36}

Because structure and function are interdependent, the study of joint characteristics should not isolate structure from function. The structural attributes of a joint are defined as the *anatomic joint*, consisting of the articular surfaces with the surrounding joint capsule and ligaments, as well as any intraarticular structures. The functional attributes are defined as the *physiologic joint*, consisting of the anatomic joint plus the surrounding soft tissues, including the muscles, connective tissue, nerves, and blood vessels (Figure 2-12).

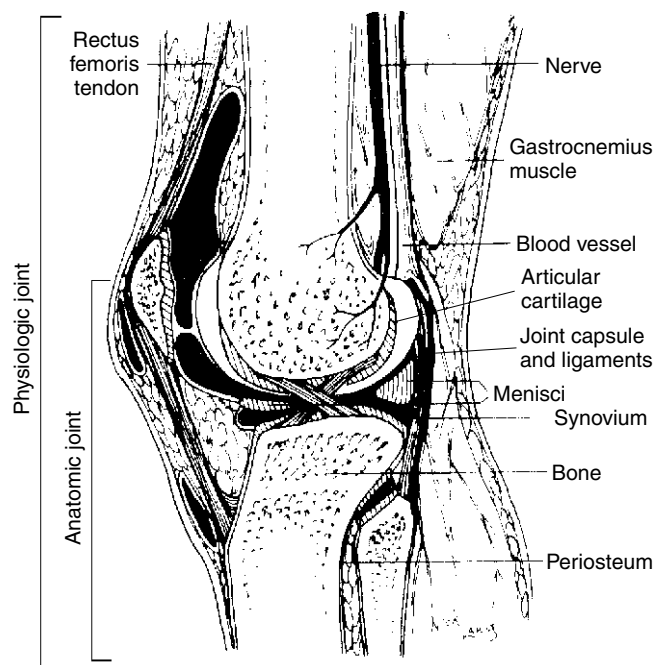


Figure 2-12 Structures that make up the anatomic joint and the physiologic joint.

JOINT FUNCTION

The physiologic movement possible at each joint occurs when muscles contract or when gravity acts on bone to move it. This motion is termed *osteokinematic movement*. Osteokinematic movement describes how each bony joint partner moves relative to the others. Movement at a joint can be considered from two perspectives: the proximal segment can rotate against the relatively fixed distal segment or the distal segment can rotate against the relatively fixed proximal segment. For example, knee flexion can occur with the foot fixed on the ground during a deep-knee bend or while sitting with the foot off the ground. A series of articulated segmental links, such as the connected shoulder girdle, arm, forearm, wrist, and hand of the upper extremity, is considered a kinematic chain. A kinematic chain can be either open or closed. An opened kinematic chain describes a situation in which the distal segment, such as the hand in the upper extremity, is not fixed to an immovable object and is free to move. A closed kinematic chain describes a situation in which the distal segment is fixed to an immovable object, leaving the proximal segment free to move.

The specific movements that occur at the articulating joint surfaces are referred to as *arthrokinematic movement*. Consideration of the motion between bones alone or osteokinematic movement is insufficient, because no concern is given to what occurs at the joint and because movement commonly involves coupling of motion around different axes. Furthermore, arthrokinematic movements consider the forces applied to the joint and include the accessory motion present in a particular articulation.

It is therefore important to relate osteokinematic movement to arthrokinematic movement when evaluating joint motion (Figure 2-13). This involves determining the movement of the mechanical axis of the moving bone relative to the stationary joint surface. The *mechanical axis of a joint* is defined as a line that passes through the moving bone to which it is perpendicular while contacting the center of the stationary joint surface (Figure 2-14).

When one joint surface moves relative to the other, spin, roll, slide, or combinations occur. MacConnail and Basmajian³⁷ use the term *spin* to describe rotational movement around the mechanical axis, which is possible as a pure movement only in the hip, shoulder, and proximal radius. *Roll* occurs when points on the surface of one bone contact points at the same interval of the other bone. *Slide* occurs when only one point on the moving joint surface contacts various points on the opposing joint surface (Figure 2-15).

In most joints of the human body, these motions occur simultaneously. The concave-convex rule relates to this expected coupling of rotational (roll) and translational (slide) movements. When a concave surface moves on a convex surface, roll and slide movements should occur in the same direction. When a convex surface moves on a concave surface, however, roll and slide should occur in opposite directions (Figure 2-16). Pure roll movement tends to result in joint dislocation, whereas pure slide movement causes joint surface impingement. Moreover, coupling of roll and slide is important anatomically because less articular cartilage is

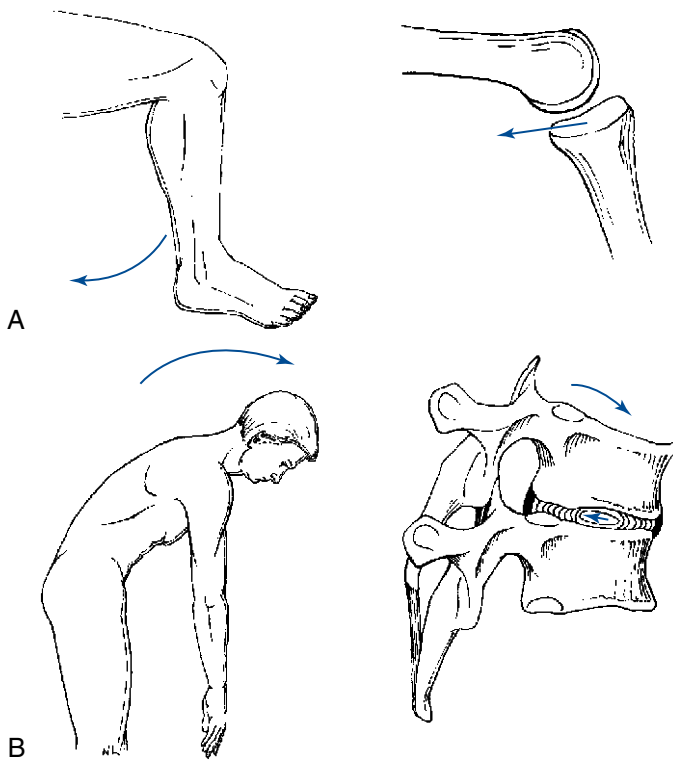


Figure 2-13 A, Osteokinematic movement of knee and trunk flexion. B, Arthrokinematic movements of tibiofemoral and T6–T7 joint flexion.

necessary in a joint to allow for movement and may decrease wear on the joint (Figure 2-17).

These concepts are instrumental in clinical decision-making regarding the restoration of restricted joint motion. Roll and spin can be restored with passive range-of-motion procedures that induce the arthrokinematic movements of the dysfunctional joint. Manipulative (thrust) techniques are needed to restore slide movements and can also be used for roll and spin problems.³⁸

In addition, when an object moves, the axis around which the movement occurs can vary in placement from one instant to another. The term *instantaneous axis of rotation (IAR)* is used to denote this location point. Asymmetric forces applied to the joint can cause a shift in the normal IAR. Furthermore, vertebral movement may be more easily analyzed as the IAR becomes more completely understood (Figure 2-18). White and Panjabi¹ point out that the value of this concept is that any kind of plane motion can be described relative to the IAR. Complex motions are simply regarded as many very small movements with many changing IARs.¹ This concept is designed to describe plane movement, or movement in two dimensions.

When three-dimensional motion occurs between objects, a unique axis in space is defined called the *helical axis of motion (HAM)*, or screw axis of motion (Figure 2-19). HAM is the most precise way to describe motion occurring between irregularly shaped objects, such as anatomic structures, because it is difficult to consistently and accurately identify reference points for such objects.

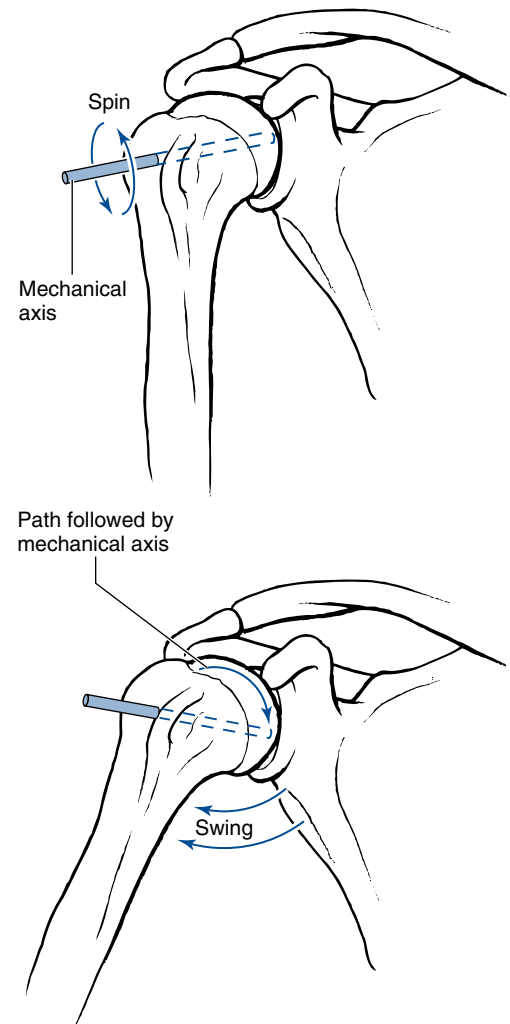


Figure 2-14 Mechanical axis of a joint and MacConnail and Basmajian's concept of spin and swing.

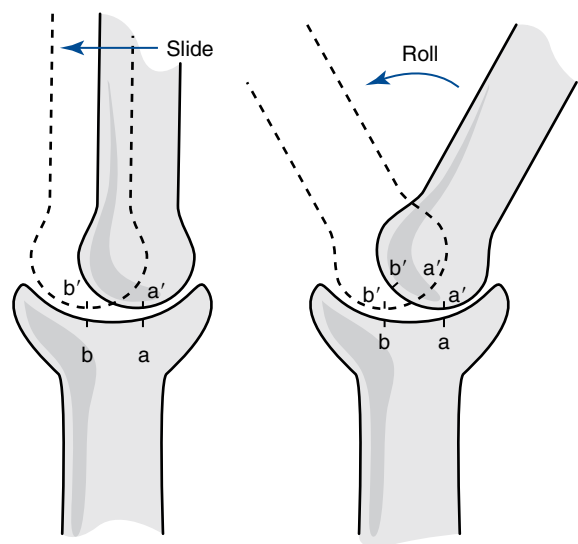


Figure 2-15 Arthrokinematic movements of roll and slide. (Modified from Hertling D, Kessler RM: *Management of common musculoskeletal disorders: Physical therapy principles and methods*, ed 2, Philadelphia, 1990, JB Lippincott.)

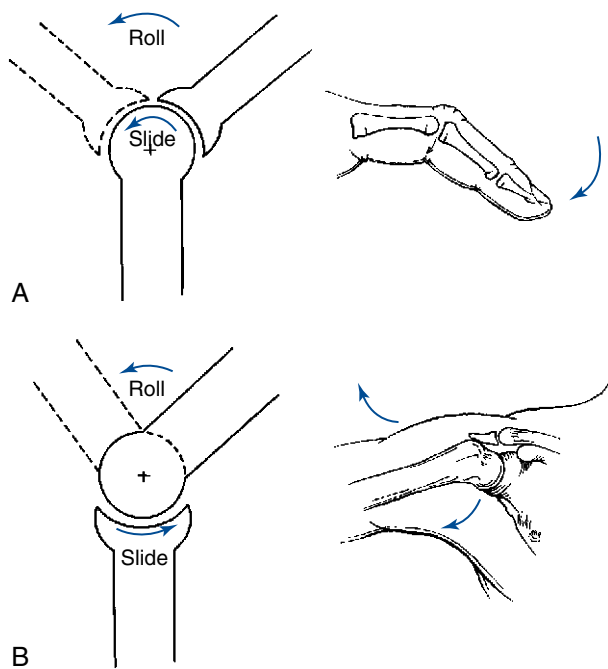


Figure 2-16 Concave-convex rule. **A**, Movement of concave surface on a convex surface. **B**, Movement of a convex surface on a concave surface.

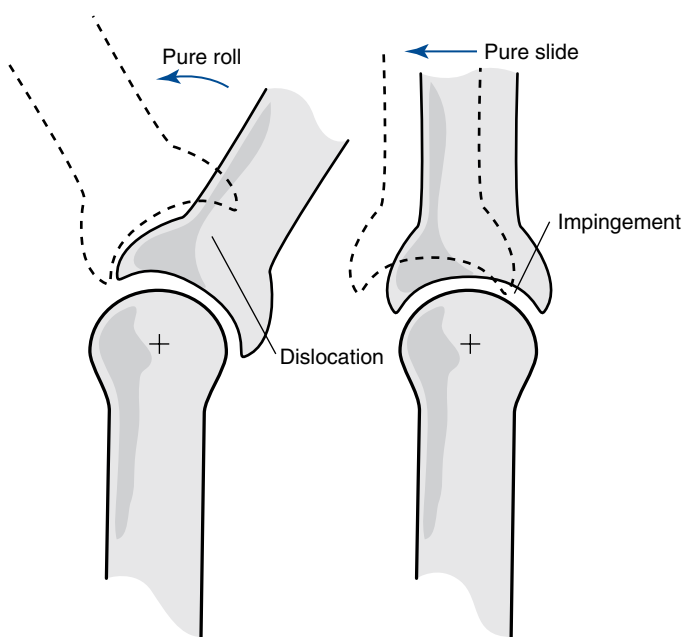


Figure 2-17 Consequences of pure roll or pure slide movements. (Modified from Hertling D, Kessler RM: *Management of common musculoskeletal disorders: Physical therapy principles and methods*, ed 2, Philadelphia, 1990, JB Lippincott.)

Clearly, most movements occur around and through several axes simultaneously, so pure movements in the human frame rarely occur. The nature and extent of individual joint motion are determined by the joint structure and, specifically, by the shape and direction of the joint surfaces. No two opposing joint surfaces are perfectly matched, nor are they perfectly geometric. All joint surfaces have some degree of curvature that is not constant but changing from point to point. Because of the incongruence between joint

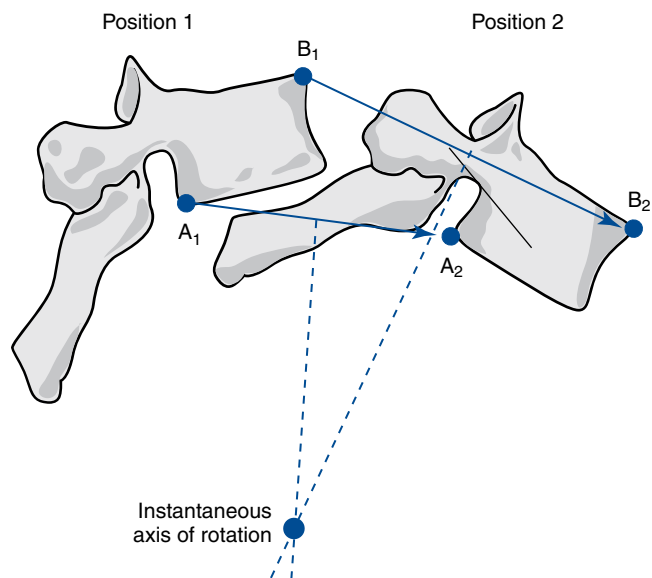


Figure 2-18 Instantaneous axis of rotation. (Modified from White AA, Panjabi MM: *Clinical biomechanics of the spine*, Philadelphia, 1978, JB Lippincott.)

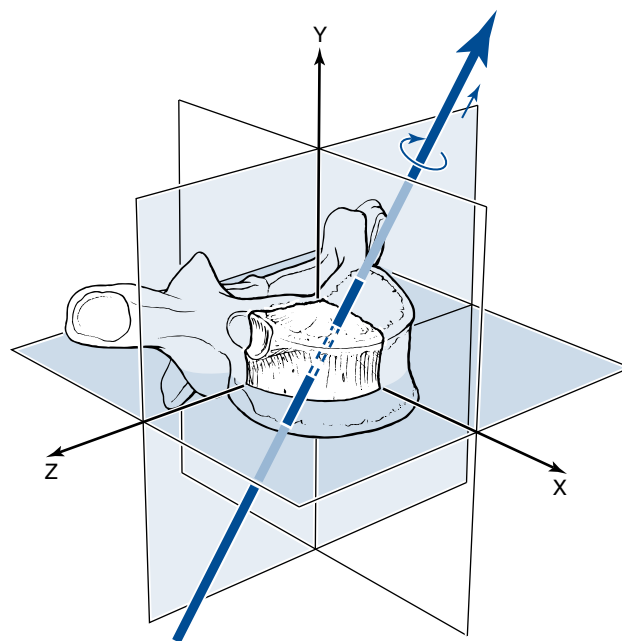


Figure 2-19 Helical axis of motion. (Modified from White AA, Panjabi MM: *Clinical biomechanics of the spine*, Philadelphia, 1978, JB Lippincott.)

surfaces, some joint space and “play” must be present to allow free movement. This joint play is an accessory movement of the joint that is essential for normal functioning of the joint.

For most synovial joints there is only one position, typically at or near the end range of motion, in which the joint surfaces fit together with the most congruency. The position of maximal joint congruency is referred to as the joint’s *close-packed position*. In this position most ligaments are taut and there is maximal

contact between the articular surfaces, making the joint very stable and difficult to move or separate. Any other position of the joint is referred to as the *loose-packed position*. The joint surfaces are generally less congruent and the ligaments and capsule are relatively slackened. For most synovial joints, the loose-packed position is toward flexion. The resting (maximal loose-packed) position of a joint, or its neutral position, occurs when the joint capsule is most relaxed and the greatest amount of play is possible. When injured, a joint often seeks this maximum loose-packed position to allow for swelling.

Joint surfaces will approximate or separate as the joint goes through a range of motion. This is the motion of compression and distraction. A joint moving toward its close-packed position is undergoing *compression*, and a joint moving toward its loose-packed position is undergoing *distraction*³⁷ (Table 2-3).

Joint motion consists of five qualities of movement that must be present for normal joint function. These five qualities are joint play, active range of motion, passive range of motion, end feel or play, and paraphysiologic movement. From the neutral close-packed position, joint play should be present. This is followed by a range of active movement under the control of the musculature. The passive range of motion is produced by the examiner and includes the active range, plus a small degree of movement into the elastic range. The elastic barrier of resistance is then encountered, which exhibits the characteristic movement of end feel. The small amount of movement available past the elastic barrier typically occurs postcavitation and has been classified as paraphysiologic movement. Movement of the joint beyond the

paraphysiologic barrier takes the joint beyond its limit of anatomic integrity and into a pathologic zone of movement. Should a joint enter the pathologic zone, there will be damage to the joint structures, including the osseous and soft tissue components (see Figures 3-22 and 3-23).

Both joint play and end-feel movements are thought to be necessary for the normal functioning of the joint. A loss of either movement can result in a restriction of motion, pain, and most likely, both. Active movements can be influenced by exercise and mobilization, and passive movements can be influenced by traction and some forms of mobilization, but end-feel movements are affected when the joint is taken through the elastic barrier, creating a sudden yielding of the joint and a characteristic cracking noise (cavitation). This action can be accomplished with deep mobilization and a high-velocity, low-amplitude manipulative thrust.

MECHANICAL FORCES ACTING ON CONNECTIVE TISSUE

Whereas an understanding of structure is needed to form a foundation, an understanding of the dynamics of the various forces affecting joints aids in the explanation of joint injury and repair. Functionally, the most important properties of bone are its strength and stiffness, which become significant qualities when loads are applied (Figure 2-20). Living tissue is subjected to many different combinations of loading force throughout the requirements of daily living. Although each type of loading force is described individually, most activities produce varying amounts and combinations of all of them.

TABLE 2-3 Close-Packed Positions for Each Joint

Region	Specific Joint	Close-Packed Position
Fingers	Distal interphalangeal joints	Maximal extension
	Proximal interphalangeal joints	Maximal extension
	Metacarpophalangeal joints	Maximal flexion
Hand	Intermetacarpal joints	Maximal opposition
Wrist	Intercarpal joints	Maximal dorsiflexion
Forearm	Radioulnar joints	5 degrees of supination
Elbow	Ulnohumeral joint	Extension in supination
	Radiohumeral joint	Flexion in supination
Shoulder	Glenohumeral joint	Abduction and external rotation
	Acromioclavicular joint	90 degrees of abduction
	Sternoclavicular joint	Maximal elevation
Toes	Distal interphalangeal joints	Maximal extension
	Proximal interphalangeal joints	Maximal extension
	Metatarsophalangeal joints	Maximal extension
Foot	Intermetatarsal joints	Maximal opposition
Ankle	Tarsometatarsal joints	Maximal inversion
	Tibiotalar joint	Maximal dorsiflexion
Knee	Tibiofemoral joint	Maximal extension and external rotation
Hip	Coxofemoral joint	Maximal extension, internal rotation, and abduction
Spine	Three-joint complex	Maximal extension

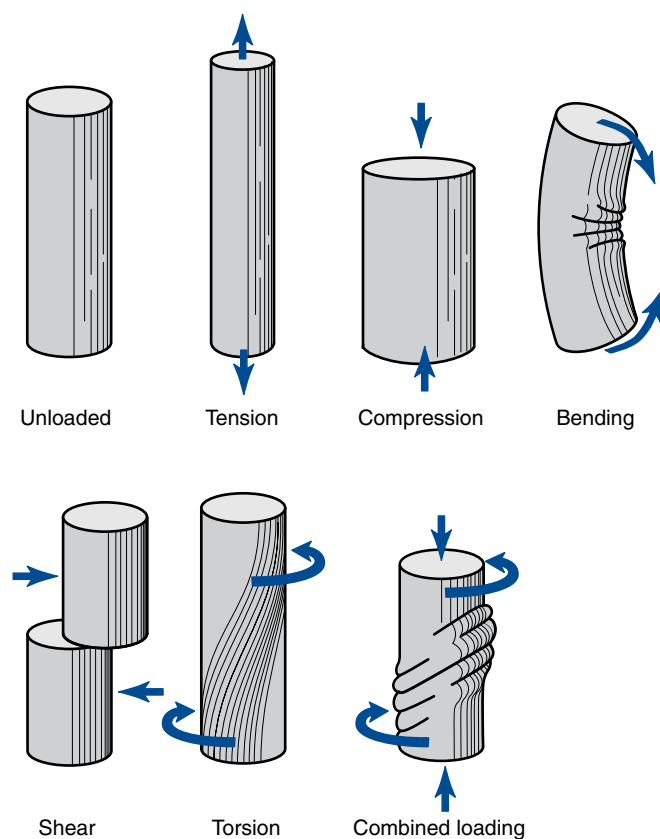


Figure 2-20 Loads to which bone may be subjected. (Modified from Soderberg GL: *Kinesiology: Application to pathological motion*, Baltimore, 1986, Williams & Wilkins.)

TENSION FORCES

The force known as *tension* occurs when a structure is stretched longitudinally. Tensile loading is a stretching action that creates equal and opposite loads outward from the surface and tensile stress and strain inward. Therefore, a tension force tends to pull a structure apart, causing the cross-sectional area of the structure to decrease. When a material is stretched in the direction of the pull, it contracts in the other two directions. If the primary stress is tensile, there will be secondary stresses that are compressive and vice versa.

The tension elements of the body are the soft tissues (fascia, muscles, ligaments, and connective tissue) and have largely been ignored as construction members of the body frame. The tension elements are an integral part of the construction and not just a secondary support. In the spine, the ligaments are loaded in tension.³⁹ Tensile forces also occur in the intervertebral disc during the rotational movements of flexion, extension, axial rotation, and lateral flexion. The nucleus tends to bear the compressive load, and the annular fibers tend to bear the tensile loads.

COMPRESSION FORCES

Compression occurs when a load produces forces that push the material together, creating a deforming stress. The behavior of a structure in compression depends a great deal on its length and how far or long the load is applied.

Compressive forces are transmitted to the vertebral body and intervertebral disc in the spine. The nucleus pulposus is a semi-liquid or gel that has the characteristics of a fluid or hydraulic structure. It is incompressible and must therefore distort under compressive loads. The nucleus pulposus dissipates the compressive force by redirecting it radially.

It is important clinically to note that mechanical failure occurs first in the cartilaginous endplate when compressive forces applied alone are too great. The result is nuclear herniation into the vertebral body, called a *Schmorl's node*. However, failure may be modified when the spine is loaded in either flexion or extension. Compressive loads applied in flexion tend to cause anterior collapse of the endplate or vertebral body, where the bony structure is weaker. With compressive loads applied in extension, a significant percentage of the compressive load is transmitted through the facets, leading to capsular injuries.

Compressive loads applied with torque around the long axis can produce circumferential tears in the disc annulus. Compression loading (axial loading) on bone creates equal and opposite loads toward the surface and compressive stress and strain inward, causing the structure to become shorter and wider. Compression fractures of the vertebral bodies are examples of failure to withstand compressive forces.

Bending loads are a combination of tensile and compressive loads. The magnitude depends on the distance of the forces from the neutral axis. Fractures to long bones frequently occur through this mechanism.

SHEAR FORCES

The biomechanical effects on living things would be a great deal easier to understand if the loads, stresses, and strains were all either tensile or compressive ones. However, living things are also subjected to shear forces. A *shear* force creates sliding or, more specifically, resistance to sliding. Shear loading causes the structure to deform internally in an angular manner as a result of loads applied parallel to the surface of the structure.

Primarily, the facet joints and the fibers of the annulus fibrosus resist shear forces in the spinal motion segment. Under normal physiologic conditions, the facets can resist shear forces when they are in contact. If, however, the disc space is narrowed by degeneration with subsequent thinning of the disc, abnormally high stresses may be placed on the facet joints, and the limit of resistance to such forces is not well documented.^{40,41}

Because there is no significant provision for resisting shear stress, the risk of disc failure is greater with tensile loading than with compression loading.¹ However, the studies available demonstrating the effects of shear forces have been performed mostly on cadavers in which the posterior elements have been removed. The lumbar facets are aligned mostly in the sagittal plane with an interlocking mechanism that only allows a few degrees of rotation. Therefore, at least in the lower lumbar segments, the facet joints do provide resistance to shear stress. Cancellous bone is most prone to fracture from shear loading, with the femoral condyles and tibial plateaus often falling victim.

TORQUE FORCES

Torsion occurs when an object twists, and the force that causes the twisting is referred to as *torque*. Torque is a load produced by parallel forces in opposite directions about the long axis of a structure. In a curved structure, such as the spine, bending also occurs when a torque load is applied.

Farfan and co-workers⁴² estimate that approximately 90% of the resistance to torque of a motion segment is provided by its disc. They further state that the annulus provides the majority of the torsional resistance in the lumbar spine and speculate that with torsional injury, annular layers will tear, leading to disc degeneration.⁴² This concept is developed around the idea that when torsional forces are created in the spine, the annular fibers oriented in one direction will stretch, whereas those oriented in the other direction will relax. The result is that only half of the fibers are available to resist the force.

However, Adams and Hutton⁴³ disagreed with Farfan and co-workers and demonstrated that primarily the facets resist the torsion of the lumbar spine and that the compressed facet was the first structure to yield at the limit of torsion. Others have performed experiments that further suggest and support that the posterior elements of the spine, including the facet joints and ligaments, play a significant role in resisting torsion.^{44,45} In deference to Farfan and co-worker's conclusions, these authors suggest that torsion alone is unimportant as a causal factor of disc degeneration and prolapse, because rotation is produced by voluntary muscle activity and the intervertebral disc experiences relatively small

stresses and strains. Bogduk and Twomey⁴⁶ state that axial rotation can strain the annulus in torsion, but ordinarily the zygapophyseal joints protect it. Normal rotation in the lumbar spine produces impaction of the facet joints, preventing more than 3% strain to the annulus. With further rotation force, the impacted facet joint can serve as a new axis of rotation, allowing some additional lateral shear exerted on the annulus. Excessive rotational force can result in failure of any of the elements that resist rotation.⁴⁶ Fracture can occur in the impacted facet joint; the pars interarticularis can also fracture; capsular tears can occur in the nonimpacted facet joint; and circumferential tears can occur in the annulus (Figure 2-21). Spiral fractures are another example of the results of torsional loads applied to long bones.

NEWTON'S LAWS OF MOTION

The outcome of movement is determined by the forces applied to the body being moved. Sir Isaac Newton, based on the teachings of Galileo, observed that forces were related to mass and motion in a predictable fashion. His "laws of motion" form the framework for describing the relationship between forces applied to the body and the consequences of those forces on human motion. Newton's laws of motion are the law of inertia, the law of acceleration, and the law of action-reaction.

Law of Inertia

The first law of motion states that a body remains at rest or in constant velocity except when compelled by an external force to change its state. Therefore, a force of some kind is required to start, stop, or alter linear motion. Inertia is related to the amount of energy required to alter the velocity of the body or overcome its resistance. Each body has a point about which its mass is evenly distributed. This point, called the *center of mass*, can be considered where the acceleration of gravity acts on the body. For the entire upright human body, the center of mass lies just anterior to the second sacral vertebra.

Law of Acceleration

The second law of motion states that the acceleration of the body is directly proportional to the force causing it, takes place in the same direction in which the force acts, and is inversely proportional to the mass of the body. It is from this law that the equation force (F) is equal to mass (m) times acceleration (a) is derived. Newton's second law can also be used to provide a work-energy relationship. Work is equal to the product of the force applied to an object and the distance the object moves. Furthermore, power can then be defined by work divided by time.

Law of Action-Reaction

The third law of motion states that for every action there is an equal and opposite reaction. This means that in every interaction, there is a pair of forces acting on the two interacting objects. The size of the forces on the first object equals the size of the force on the second object. The direction of the force on the first object is opposite to the direction of the force on the second object. Forces always come in pairs—equal and opposite action-reaction force pairs. When the two equal and opposite forces act on the same

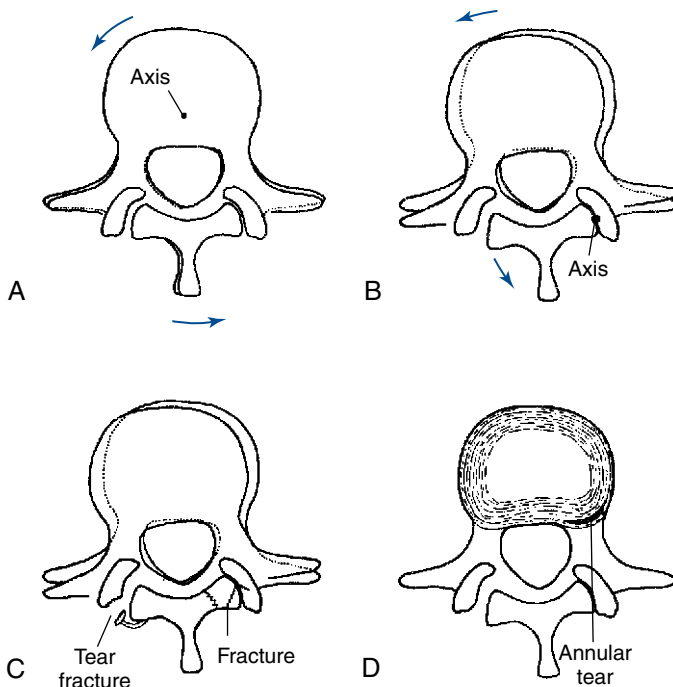


Figure 2-21 Effects of rotation on lumbar segments. **A**, Rotation is limited by impaction of facet joint. **B**, Further rotation causes a shift in the axis of rotation. **C**, The impacted facet is exposed to fracture, and the distracted facet is exposed to avulsion or capsular tear. **D**, The disc is exposed to lateral shear that can lead to circumferential tears in the annulus. (Modified from Bogduk N, Twomey LT: *Clinical anatomy of the lumbar spine*, ed 2, Melbourne, Australia 1991, Churchill Livingstone.)

object, they cancel each other so that no acceleration (or even no motion) occurs. This is not an example of the third law, but of *equilibrium* between forces. Newton's third law is one of the fundamental symmetry principles of the universe.

PROPERTIES OF CONNECTIVE TISSUE

The response of connective tissue to various stress loads contributes significantly to the soft tissue component of joint dysfunction. Within the past several decades, a great deal of scientific investigation has been directed to defining the physical properties of connective tissue. The composition, proportion, and arrangement of biologic materials that compose the connective tissues associated with joints strongly influence the mechanical performance of the joints. The biologic materials are fibers, ground substance, and cells blended in various proportions based on the mechanical demands of the joint.⁴⁷

Connective tissue contributes to kinetic joint stability and integrity by resisting rotatory moments of force. When these rotatory moments of force are large, considerable connective tissue power is required to produce the needed joint stability and integrity. Connective tissue is made up of various densities and spatial arrangements of collagen fibers embedded in a protein-polysaccharide matrix, which is commonly called *ground substance*. Collagen is a fibrous protein that has a very high tensile strength. Collagenous tissue is organized into many different higher-order structures, including tendons, ligaments, joint capsules, aponeuroses, and fascial sheaths. The principal sources of passive resistance at the normal extremes of joint motion include ligaments, tendons, and muscles. Therefore, under normal and pathologic conditions, the range of motion in most body joints is predominantly limited by one or more connective tissue structures. The relative contribution of each to the total resistance varies with the specific area of the body.

All connective tissue has a combination of two qualities—elastic stretch and plastic (viscous) stretch (Figure 2-22). The term *stretch* refers to elongation in a linear direction and increase in length. Stretching, then, is the process of elongation. *Elastic stretch* represents springlike behavior, with the elongation produced by tensile loading being recovered after the load is removed. It is therefore also described as temporary, or recoverable, elongation. *Plastic (viscous) stretch* refers to putty-like behavior; the linear deformation produced by tensile stress remains even after the stress is removed. This is described as *nonrecoverable*, or *permanent, elongation*.

The term *viscoelastic* is used to describe tissue that represents both viscous and elastic properties. Most biologic tissues, including tendons and ligaments, are viscoelastic materials. Viscoelastic

materials possess time-dependent or rate-sensitive stress-strain relationships.⁴⁸ The viscous properties permit time-dependent plastic or permanent deformation. Elastic properties, on the other hand, result in elastic or recoverable deformation. This allows it to rebound to the previous size, shape, and length.

Different factors influence whether the plastic or elastic component of connective tissue is predominantly affected. These include the amount of applied force and the duration of the applied force. Therefore, the major factors affecting connective tissue deformation are force and time. With a force great enough to overcome joint resistance and applied over a short period, elastic deformation occurs. However when the same force is applied over a long period, plastic deformation occurs.

When connective tissue is stretched, the relative proportion of elastic and plastic deformation can vary widely, depending on how and under what conditions the stretching is performed. When tensile forces are continuously applied to connective tissue, the time required to stretch the tissue a specific amount varies inversely with the force used. Therefore, a low-force stretching method requires more time to produce the same amount of elongation as a higher-force method. However, the proportion of tissue lengthening that remains after the tensile stress is removed is greater for the low-force, long-duration method. Of course, high force and long duration also cause stretch and possibly rupture of the connective tissue.

When connective tissue structures are permanently elongated, some degree of mechanical weakening occurs, even though outright rupture has not occurred. The amount of weakening depends on the way the tissue is stretched, as well as how much it is stretched. For the same amount of tissue elongation, however, a high-force stretching method produces more structural weakening than a slower, lower-force method.

Because plastic deformation involves permanent changes in connective tissue, it is important to know when plastic deformity is most likely to occur. The greatest effect occurs when positions of stress are maintained for long periods. Awkward sleep postures and stationary standing for extended periods can create plastic changes that have the potential for skeletal misalignment, joint dysfunction, and instability.

After trauma or surgery, the connective tissue involved in the body's reparative process frequently impedes function; it may abnormally limit the joint's range of motion as a result of fibrotic tissue replacing elastic tissue. Scar tissue, adhesions, and fibrotic contractures are common types of pathologic connective tissue that must be dealt with during chiropractic manipulative procedures.

Connective tissue elements can lose their extensibility when their related joints are immobilized.⁴⁹ With immobilization, water is released from the proteoglycan molecule, allowing connective tissue fibers to contact one another and encouraging abnormal cross-linking that results in a loss of extensibility.⁵⁰ It is hypothesized that manual therapy can break the cross-linking and any intraarticular capsular fiber fatty adhesions, thereby providing free motion and allowing water inhibition to occur. Furthermore, procedures can stretch segmental muscles, stimulating spindle reflexes that may decrease the state of hypertonicity.⁵¹

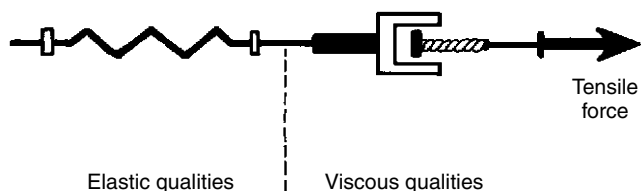


Figure 2-22 Model of connective tissue properties.

The response of connective tissue to various stress loads contributes significantly to the soft tissue component of joint dysfunction. Within the past several decades, a great deal of scientific investigation has been directed to defining the physical properties of connective tissue.

MUSCLE

The role of muscles is to move bone and allow the human body to perform work. In the normal man, muscle accounts for approximately 40% to 50% of body weight. For the woman, this falls to approximately 30% of total body weight. Three types of muscle are in the body: striated skeletal muscle, nonstriated smooth involuntary muscle, and cardiac muscle. Only the skeletal muscle is under voluntary control.

There are three gross morphologic muscle types in striated muscle (Figure 2-23). Parallel muscles have fibers that run parallel throughout the length of the muscle and end in a tendon. This type of muscle is essentially designed to rapidly contract, although it typically cannot generate a great deal of power. Pennate muscles are those in which the fibers converge onto a central tendon. A muscle of this type is unipennate if the fibers attach to only one side of a central tendon, and it is bipennate if the muscle attaches to both sides of a central tendon. Finally, there is a multipennate muscle in which the muscle fibers insert on the tendon from a variety of differing directions. This form of muscle can generate large amounts of power, although it performs work more slowly than a parallel muscle.

Muscle comprises three layers (Figure 2-24). An epimysium formed of connective tissue surrounds the muscle; a perimysium separates the muscle cells into various bundles; and an endomysium surrounds the individual muscle cells. The muscle fibers also have three layers. The outermost layer is formed of collagen fibers. A basement membrane layer comprises polysaccharides and

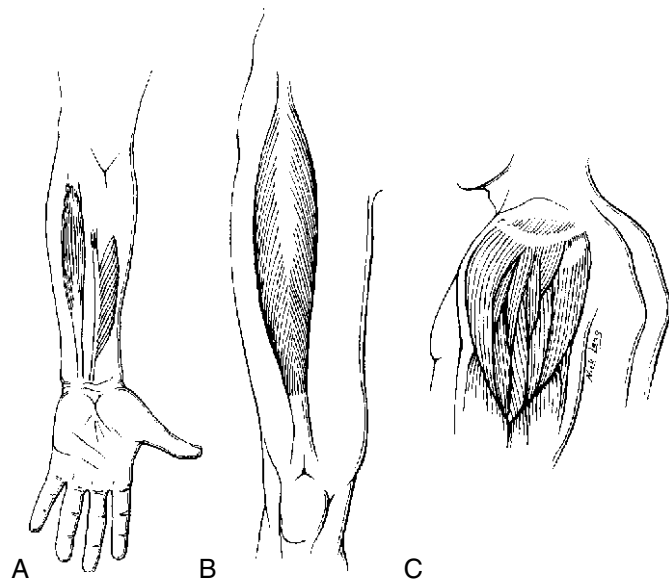


Figure 2-23 Morphologic muscle types. **A**, Unipennate. **B**, Bipennate. **C**, Multipennate.

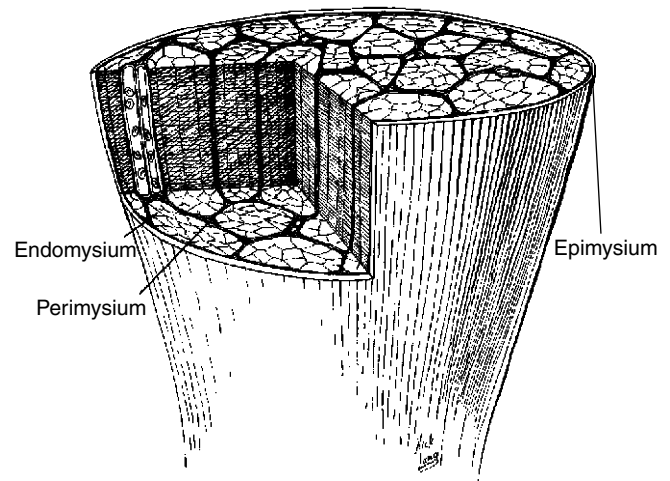


Figure 2-24 Connective Tissue Layers.

protein and is approximately 500 Å thick. The innermost layer, the sarcolemma, forms the excitable membrane of a muscle.

Muscle fibers contain columns of filaments of contractile proteins. In striated muscle, these molecules are interrelated layers of actin and myosin molecules. These myofibrils are suspended in a matrix called *sarcoplasm*, composed of the usual intracellular components. The fluid of the sarcoplasm is rich with potassium, magnesium, phosphate, and protein enzymes. Numerous mitochondria lie close to the actin filaments of the I bands, suggesting that the actin filaments play a major role in using adenosine triphosphate formed by the mitochondria.⁵² The sarcoplasmic reticulum functions in a calcium ion equilibrium. A transverse tubular system transmits membrane depolarization from the muscle cell to the protein. Also located within the sarcoplasm is the protein myoglobin that is necessary for oxygen binding and oxygen transfer.

Skeletal muscle occurs in two forms, originally known as *white* and *red* muscle. The white muscle is a fast-twitch, or phasic, muscle. It has a rapid contraction time and contains a large amount of glycolytic enzyme. Essentially, this muscle allows for rapid function necessary for quick contractions for short periods. Red muscle is a slow-twitch, or tonic, muscle. It contracts much more slowly than does white muscle and contains a great deal more myoglobin and oxidative enzymes. Red muscle is more important in static activities that require sustained effort over longer periods. Standing is a good example of this. In the human body, each individual muscle is composed of a mix of both types of muscle.

When a stimulus is delivered to a muscle from a motor nerve, all fibers in the muscle contract at once.⁵³ Two types of muscle contractions have been defined. During an *isotonic* contraction, a muscle shortens its fibers under a constant load. This allows work to occur. During an *isometric* contraction, the length of the muscle does not change. This produces tension, but no work. No muscle can perform a purely isotonic contraction, because each isotonic contraction must be initiated by an isometric contraction.

Muscle contraction refers to the development of tension within the muscle, not necessarily creating a shortening of the muscle. When a muscle develops enough tension to overcome a resistance so that the muscle visibly shortens and moves the body

part, *concentric contraction* is said to occur. *Acceleration* is thus the ability of a muscle to exert a force (concentric contraction) on the bony lever to produce movement around the fulcrum to the extent intended.

When a given resistance overcomes the muscle tension so that the muscle actually lengthens, the movement is termed an *eccentric contraction*. Deceleration is the property of a muscle being able to relax (eccentric contraction) at a controlled rate. There are numerous clinical applications of the eccentric contraction of muscles, particularly in posture.

Muscles can perform various functions because of their ability to contract and relax. One property is that of shock absorption, another is acceleration, and a third is deceleration. Each is very important to the overall understanding of the biomechanics of the body and is discussed separately. The predominant responsibility for the dissipation of axial compression shocks rests with the musculotendon system. As a result, shock causes many musculoskeletal complaints. Shin splints, plantar fasciitis, Achilles tendinitis, lateral epicondylitis, as well as some forms of back pain, can result from the body's inability to absorb and dissipate shock adequately.

Although the muscular system is the primary stabilizer of the joint, if the muscle breaks down, the ligaments take up the stress. This is often seen in an ankle sprain, when the muscles cannot respond quickly enough to protect the joint and the ligaments become sprained or torn. If the ligaments are stretched but not torn completely through, this can lead to a chronic instability of the joint, especially if the surrounding musculature is not adequately rehabilitated. When the muscles fail and the ligaments do not maintain adequate joint stability, the stress cannot be fully absorbed by those tissues, and the bone and its architecture take up the stress.

Forces applied to joints in any position may cause damage to the bony structure, ligaments, and muscles. Tensile forces generated by muscle contractions can pull apart the cement from the osteons, resulting in fractures (the most common of which is at the base of the fifth metatarsal from the pull of the peroneus brevis). Calcaneal fractures from the pull of the Achilles tendon also occur through this mechanism. Because the closed-packed position has the joint surfaces approximated and capsular structures tight, an improperly applied force may cause fracture of the bone, dislocation of the joint, or tearing of the ligaments. Kaltenborn⁵⁴ states that it is important to know the closed-packed position for each joint because testing of joint movements and manipulative procedures should not be done to the joint in its closed-packed position (see Table 2-3). When an improperly applied force is applied in the open-packed position, the joint laxity and loss of stability may allow damage to the ligaments and supporting musculature.

One of the signs of segmental dysfunction is the presence of muscle hypertonicity. Localized increased paraspinal muscle tone can be detected with palpation, and in some cases with electromyography. Janda⁵⁵ recognizes five different types of increased muscle tone: limbic dysfunction, segmental spasm, reflex spasm, trigger points, and muscle tightness. Liebensohn⁵⁶ has discussed the treatment of these five types using active muscle contraction and relaxation procedures.

Acute traumatic injury to muscle is generally considered to result from a large force of short duration, influencing primarily the elastic deformation of the connective tissue. If the force is

beyond the elastic range of the connective tissue, it enters the plastic range. If the force is beyond the plastic range, tissue rupture occurs. More commonly encountered by the chiropractor is the microtrauma seen in postural distortions, repetitive minor trauma occurring in occupational and daily living activities, and joint dysfunction as a result of low gravitational forces occurring over a long period, thus creating plastic deformation.

Immobilization is often associated with a decrease in muscle elasticity. This condition is called *muscle contracture*, but the mechanism is not yet clear. Muscle immobilized in a shortened position develops less force and tears at a shorter length than freely mobile muscle with a normal resting length.⁵⁷ For this reason, vigorous muscle stretching has been recommended for muscle tightness.⁵⁵ However, for the stretch to be effective, the underlying joints should be freely mobile. Patients therefore often require manipulation that specifically moves associated joints before muscle stretching. Selective atrophy of fast-twitch type 2 fibers has also been identified in pain-related immobilization of a joint,⁵⁸ further supporting the importance of proper joint function.

LIGAMENTS

Ligaments are usually cordlike or bandlike structures made of dense collagenous connective tissue similar to that of a tendon. Ligaments are composed of type I and type III collagen, with intervening rows of fibrocytes. Also interwoven with the collagen bundles are elastin fibers that provide extensibility. The amount of elastin varies from ligament to ligament. Ligaments exhibit a mechanical property called *crimping* that provides a shock-absorbing mechanism and contributes to the flexibility of the ligament.

Spinal ligaments serve two roles, allowing smooth motion within the spine's normal range of motion and protecting the spinal cord by limiting excessive motion and absorbing loads.⁵⁹ Jiang⁶⁰ identified that stretching of spinal ligaments results in "a barrage of sensory feedback from several spinal cord levels on both sides of the spinal cord." This sensory information has been found to ascend to many higher (cortical) centers. Such findings provide provocative evidence that the spinal ligaments, along with the Z joint capsules and the small muscles of the spine (interspinales, intertransversarii, and transversospinalis muscles), play an important role in mechanisms related to spinal proprioception (joint position sense) and may play a role in the neural activity related to spinal adjusting.⁶¹

Large loads are capable of overcoming the tensile resistance of ligaments, resulting in complete- or partial-tear injuries. Ligament healing occurs through the basic mechanisms of inflammation, repair, and remodeling. Immobilization of ligamentous tissue results in a diminished number of small-diameter fibers⁶² that presumably lead to joint stiffness. However, the precise mechanism by which immobilization leads to joint stiffness has not been determined. It likely results from a combination of intraarticular adhesion formation and active contraction of ligaments by fibroblasts.⁶³⁻⁶⁵ Using a cat model, deformation or stress in the supraspinous ligament, and possibly in other spinal ligaments, recruits multifidus muscle force to stiffen one to three lumbar motion segments and prevent instability.⁶⁶ Strong muscular activity is seen when loads that can cause permanent damage to the ligament are applied, indicating that spastic muscle activity and possibly pain can be caused by ligament overloading.

FACET JOINTS

The common factor in all of the spinal segments from the atlantooccipital joint to the pelvis is the fact that each has two posterior spinal articulations. These paired components have been referred to as the *zygapophyseal* (meaning an “oval offshoot”) *joints* and are enveloped in a somewhat baggy capsule, which has some degree of elasticity. Each of the facet facings is lined with articular cartilage, as is the case with all contact-bearing joint surfaces, with the exception of the temporomandibular joint and the sternoclavicular joint. These joints have intracapsular fibrocartilaginous discs that separate the joint surfaces.

Compared with intervertebral discs, facet joints have been the focus of very little biomechanical research. Yet these structures must control patterns of motion, protect discs from shear forces, and provide support for the spinal column. The orientation of the joint surface varies with each spinal region, largely governing the degree of freedom each region can accomplish (Figure 2-25).

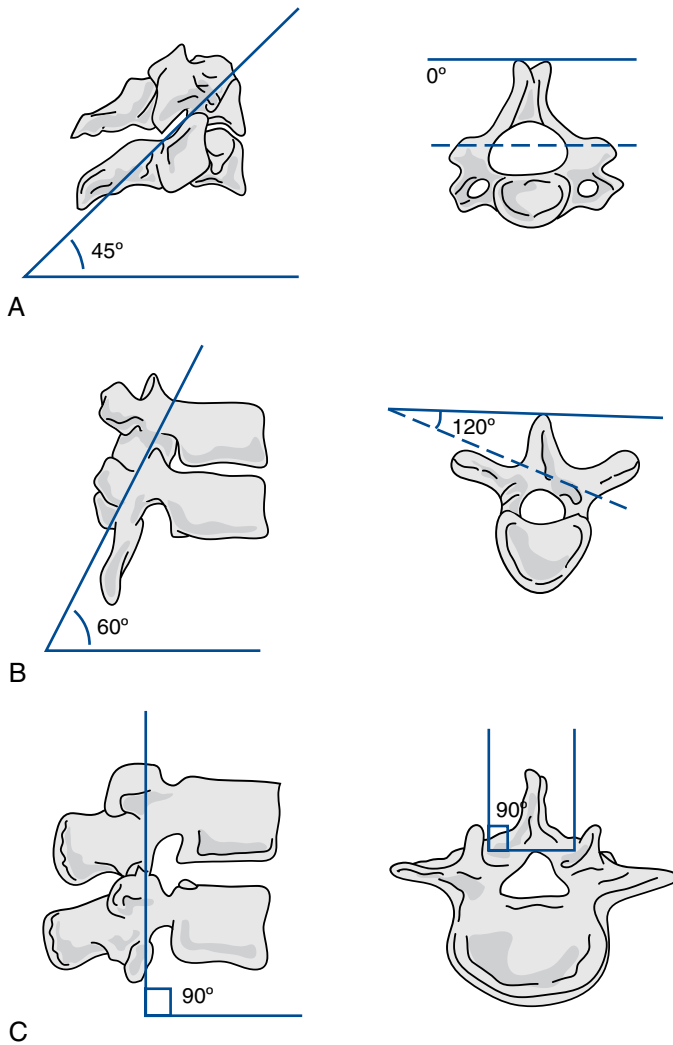


Figure 2-25 Facet planes in each spinal region viewed from the side and above. **A**, Cervical (C3–C7). **B**, Thoracic. **C**, Lumbar. (Modified from White AA, Panjabi MM: *Clinical biomechanics of the spine*, ed2, Philadelphia, JB Lippincott, 1990.)

Because these joints are true diarthrodial (synovial) articulations, each has a synovial membrane that supplies the joint surfaces with synovial fluid. The exact role of synovial fluid is still unknown, although it is thought to serve as a joint lubricant or, at least, to interact with the articular cartilage to decrease friction between joint surfaces. In addition, the synovium may be a source of nutrition for the avascular articular cartilage. Intermittent compression and distraction of the joint surfaces must occur for an adequate exchange of nutrients and waste products to occur.² Furthermore, as mentioned, immobilized joints have been shown to undergo degeneration of the articular cartilage.¹⁴ Certainly, the nature of synovial joint function and lubrication is of interest because there is evidence that the facet joints sustain considerable stress and undergo degenerative changes.

The capsule is richly innervated with nociceptors (pain) and mechanoreceptors (proprioception), allowing the supporting structures to react to many combinations of tension and compression movements imposed by different postures and physical activity. Each movement of the joint must first overcome the surface tension of the capsule, but must then be able to return to its original position maintaining joint apposition. The lateral portions of the capsule are much more lax and contain fewer elastic fibers.⁶⁷ Creep during sustained lumbar flexion occurs significantly faster than creep during repetitive lumbar flexion, suggesting that both result in immediate and residual laxity of the joint and stretch of the facet joint capsule, which could increase the potential for joint pain.⁶⁸

Although the posterior joints were not designed to bear much weight, they can share up to about one third of this function with the intervertebral disc. Moreover, as a part of the three-joint complex, if the disc undergoes degeneration and loses height, more weight-bearing function will fall on the facets. During long periods of axial loading, the disc loses height through fluid loss, thereby creating more weight-bearing on the facets on a daily basis.

The posterior joints also have been found to contain fibroadipose meniscoids that apparently function to adapt to the incongruity of the articular surfaces, but the clinical significance of which remains controversial. Bogduk and Engel⁶⁹ provide an excellent review of the meniscoids of the lumbar zygapophyseal joints. Although the genesis of their article was as a literature review to support the contention that the meniscoids could be the cause of an acute locking of the low back because of entrapment, the article also provided a comprehensive review of the anatomic consideration of lumbar meniscoids.

The meniscoids appear to be synovial folds continuous with the periarticular tissues and with both intracapsular and extracapsular components. Microscopically, the tissue consisted of loose connective and adipose tissue, mixed with many blood vessels (Figure 2-26). The meniscoids could present in various shapes, including annular menisci found in the thoracic region, with linguiform menisci and filiform menisci commonly found in the lumbar region.⁷⁰

These meniscoid structures can project into the joint space when the joint surfaces of articular cartilage are not in contact. Bogduk and Engel⁶⁹ noted two groups; one is located along the dorsal and ventral margins of the joint and one is located at the superior and inferior aspects of the joint. In their view, only the ones located along the dorsal and ventral borders of the joint

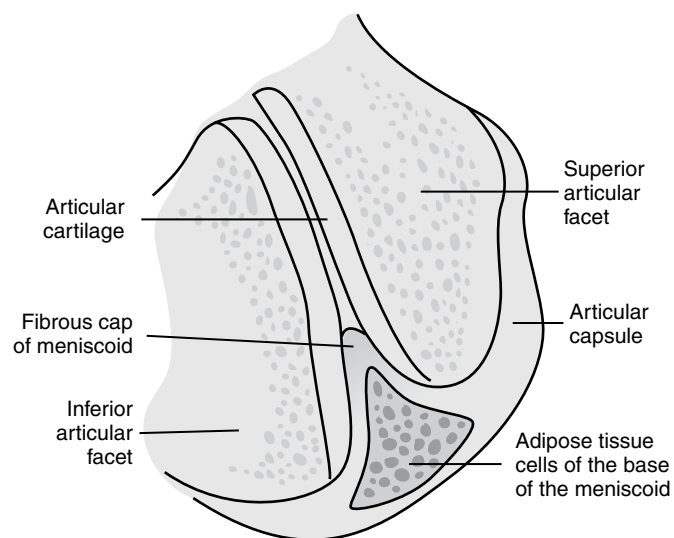


Figure 2-26 Fibroadipose meniscoid in a lumbar facet joint. (Modified from Bogduk N, Engel R: The menisci of the lumbar zygapophyseal joints: A review of their anatomy and clinical significance, *Spine* 9:454, 1984.)

represent true meniscoids. Functionally, Bogduk and Engel believe these structures may help to provide greater stability to a lumbar zygapophyseal joint by helping to distribute the load over a wider area. In their words, meniscoids play a “space-filling” role.⁶⁹

Clinically and theoretically these meniscoids may become entrapped or extrapped.⁷¹ Entrapment of the meniscoid between the joint surfaces itself is not believed to be painful, although pain can be created by traction on the joint capsule through the base of the meniscoid. This could, through a cascade of events, lead to more pain and reflex muscle spasm, known as *acute locked low back*, which is amenable to manipulative therapy. Extrapment of the meniscoid may occur when the joint is in a flexed position and the meniscoid is drawn out of the joint but fails to reenter the joint space on attempted extension. It gets stuck against the edge of the bony lip or articular cartilage, causing a buckling of the capsule that serves as a space-occupying lesion. Pain is produced through capsular distention.⁷²

Giles and Taylor^{67,73} examined the innervation of meniscoids (synovial folds) in the lumbar zygapophyseal joints, using both light microscopy and transmission electron microscopy. The authors removed part of the posteromedial joint capsule along with the adjacent ligamentum flavum and synovial folds after a laminectomy, fixed these specimens in various solutions, and prepared them for microscopy. They demonstrated that neurologic structures were located in the areas studied. Nerves seen in the synovial fold were 0.6 to 12 μm in diameter. These neurologic structures may give rise to pain.

Taylor and Twomey⁷⁴ suggest that because of their rich blood supply, spinal joint meniscoids do not undergo degeneration with age as do the intervertebral disc and articular cartilage. However, with degenerative changes to disc and especially articular cartilage, the meniscoid inclusions are exposed to abnormal biomechanical forces that may result in their demise.

Adams and Hutton⁷⁵ examined the mechanical function of the lumbar apophyseal joints on spines taken from cadavers. The

authors wanted to examine various loading regimens on the function of these joints. They found that the lumbar zygapophyseal joints can resist most of the intervertebral shear force only when the spine is in a lordotic posture. These joints also can aid in resisting the intervertebral compressive force and can prevent excessive movement from damaging the intervertebral discs. The facet surfaces protect the posterior annulus, whereas the capsular ligament helps to resist the motion of flexion. The authors noted that in full flexion the capsular ligaments provide nearly 40% of the joint's resistance. They conclude that “the function of the lumbar apophyseal joints is to allow limited movement between vertebrae and to protect the discs from shear forces, excessive flexion and axial rotation.”⁷⁵

Taylor and Twomey⁷⁴ studied how age affected the structure and function of the zygapophyseal joints. They took transverse sections of the lumbar spine from cadavers ranging in age from fetus to 84 years and prepared them in staining media. They noted that fetal and infant lumbar zygapophyseal joints are coronally oriented, which only later (in early childhood) become curved or biplanar joints. In the adult, the joint has a coronal component in the anterior third of the joint and a sagittal component in the posterior two thirds of the joint. The joint is generally hemicylindrical.

The structures located in the anterior third of the joint, primarily articular cartilage and subchondral bone, tend to show changes that are related to loading the joint in flexion. The posterior part of the joint shows a variety of different changes related to age. There may be changes from shearing forces. The subchondral bone thickens as it ages and is wedge-shaped. These changes occur because of loading stresses from flexion.⁷⁴

Taylor and Twomey⁷⁴ are careful to note that they could make no clinical correlation with their findings, which is one of the problems with cadaveric studies of this sort. They believe that this work has biomechanical implications; they believe that the lumbar zygapophyseal joints limit the forward translational component of flexion to only a very small displacement. Indeed, they believe this fact may be the most important component limiting forward flexion. Although the lumbar facet joints are oriented in the sagittal plane, they are not purely sagittal, and flexion with anterior translation will result in impaction of the facets limiting this movement.⁵⁷

INTERVERTEBRAL DISCS

The intervertebral discs are fibrocartilaginous mucopolysaccharide structures that lie between adjoining vertebral bodies. In the adult there are 23 discs, each given a numeric name based on the segment above. Thus the L5 disc lies between the fifth lumbar segment and the sacrum, and the L4 disc lies between the fourth and fifth lumbar segments. In the early years of life, the discs between the sacral segments are replaced with osseous tissue, but remain as rudimentary structures; they are generally regarded as having no clinical significance.

The unique and resilient structure of the disc allows for its function in weight-bearing and motion. The anterior junction of two vertebrae is an amphiarthrodial symphysis articulation formed by the two vertebral endplates and the intervertebral disc. The discs

are responsible for approximately one fourth of the entire height of the vertebral column. The greater the height of the intervertebral disc as compared to the height of the vertebral body, the greater the disc to vertebral body ratio and the greater the spinal segmental mobility. The ratio is greatest in the cervical spine (2:5) and least in the thoracic spine (1:5), with the lumbar region (1:3) in between. A disc has three distinct components: the annulus fibrosus, the nucleus pulposus, and the cartilaginous endplates.

The cartilaginous endplates are composed of hyaline cartilage that separates but also helps attach the disc to the vertebral bodies. There is no closure of cortical bone between the hyaline cartilage and the vascular cancellous bone of the vertebral body. The functions of the endplates are to anchor the disc, to form a growth zone for the immature vertebral body, and to provide a permeable barrier between the disc and body. This role allows the avascular disc material to receive nutrients and repair products.

The annulus fibrosus is a fibrocartilage ring that encloses and retains the nucleus pulposus, although the transition is gradual, with no clear distinction between the innermost layers of the annulus and outer aspect of the nucleus. The fibrous tissue of the annulus is arranged in concentric, laminated bands, which appear to cross one another obliquely, each forming an angle of about 30 degrees to the vertebral body (Figure 2-27). The annular fibers of the inner layers are attached to the cartilaginous endplates, and the outer layers are attached directly to the osseous tissue of the vertebral body by means of Sharpey fibers.⁷⁶

Superficially, the ALL and the PLL reinforce the fibers. The PLL is clinically significant in that as it courses caudally, its width narrows until it covers only approximately 50% of the central portion of the lower lumbar discs. The weakest area of the annulus, and hence the area most likely to be injured, is the posterolateral aspect. This is the most likely spot for a disc herniation in the lumbar spine.⁷⁷

The annulus fibrosus contains little elastic tissue, and the amount of stretch is limited to only 1.04 times its original length, with further stretch resulting in a tearing of fibers. The functions of the annulus fibrosus include enclosing and retaining the nucleus pulposus, absorbing compressive shocks, forming a structural unit between vertebral bodies, and allowing and restricting motion.

The nucleus pulposus is the central portion of the disc and is the embryologic derivative of the notochord. It accounts for approximately 40% of the disc and is a semifluid gel that deforms easily, but is considered incompressible. The nucleus is composed of a loose network of fine fibrous strands that lie in a mucoprotein matrix containing mucopolysaccharides, chondroitin sulfate, hyaluronic acid, and keratin sulfate. These large molecules are strongly hydrophilic, capable of binding nearly nine times their volume of water, and are therefore responsible for the high water content of the disc. In young adults, the water content of a disc approaches 90% and maintains an internal pressure of approximately 30 pounds per square inch.¹ The water content, however, steadily decreases with age. The composition of the nucleus produces a resilient spacer that allows motion between segments, and although it does not truly function as a shock absorber, it does serve as a means to distribute compressive forces.

The image of the nucleus as a round ball between two hard surfaces must be abandoned. This gives the impression that the nucleus can roll around between the two endplates. The only means for significant nuclear migration is through a tear in the annular fibers, allowing the nucleus to change shape but not actually shift position. The result of nuclear migration is a potential change in the instantaneous axis of movement and potential aberrant segmental motion.

The intervertebral disc is a vital component for the optimal, efficient functioning of the spinal column. In conjunction with the vertebral bodies, the discs form the anterior portion of the functional unit responsible for bearing weight and dissipating shock. In so doing, it distributes loads, acts as a flexible buffer between the rigid vertebrae, permits adequate motion at low loads, and provides stability at higher loads.

The simple compression test of the disc has been one of the most popular experiments because of the importance of the disc as a major load-carrying element of the spine. Axial compression forces continually affect the disc during upright posture. The nucleus bears 75% of this force initially, but redistributes some to the annulus.

Furthermore, the ability of the disc to imbibe water causes it to “swell” within its inextensible casing. Thus the pressure in the nucleus is never zero in a healthy disc. This is termed a *preloaded*

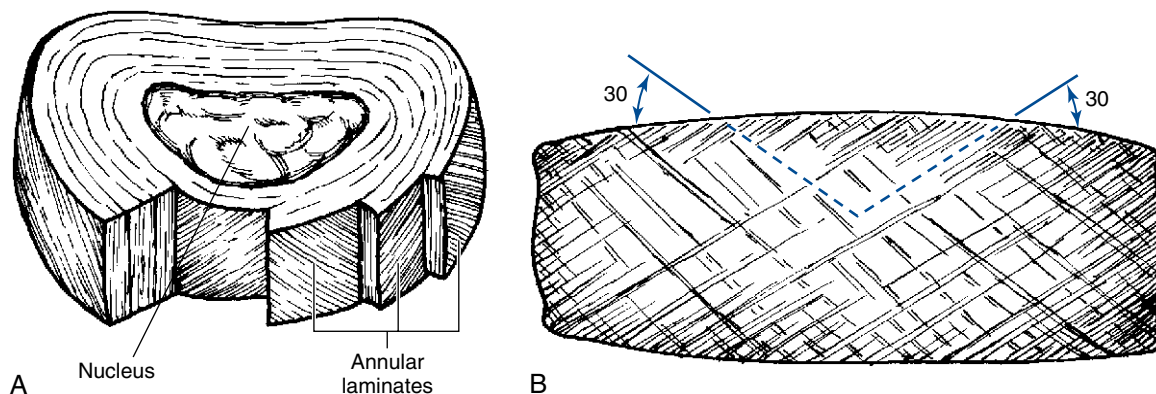


Figure 2-27 Intervertebral disc. **A**, Nucleus pulposus and annulus fibrosus. **B**, Orientation of annular fibers. (Modified from White AA, Panjabi MM: *Clinical biomechanics of the spine*, Philadelphia, 1978, JB Lippincott.)

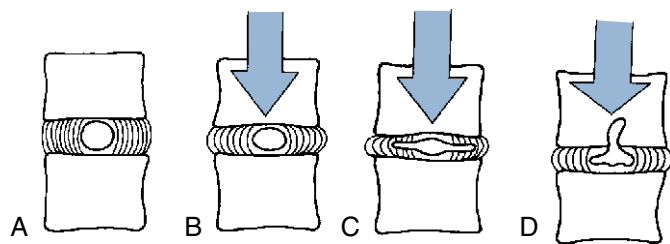


Figure 2-28 Effects of axial loads on vertebral body and disc. **A**, Normal disc height. **B**, Normal disc under mild to moderate axial load, showing slight approximation of bodies. **C**, Diseased disc under same axial load, showing significant loss of disc height. **D**, Endplate fracture from significant axial load causing a Schmorl node.

state. The preloaded state gives the disc a greater resistance to forces of compression.

With age and exposure to biomechanical stresses, the chemical nature of the disc changes and becomes more fibrous. This reduces the imbibition effect and, in turn, the preloaded state. As a result, flexibility is diminished and more pressure is exerted on the annulus and peripheral areas of the endplate. A disc that has been injured deforms more than a healthy one.

The preloaded state also explains the elastic properties of the disc. When the disc is subjected to a force, the disc exhibits dampened oscillations over time. If the force is too great, however, the intensity of the oscillations can destroy the annulus, thus accounting for the deterioration of intervertebral discs that have been exposed to repeated stresses.

Compressive forces are transmitted from endplate to endplate by both the annulus and the nucleus. When compressed, the disc bulges in the horizontal plane. A diseased disc compresses more, and, as this occurs, stress is distributed differently to other parts of the functional unit, notably the apophyseal articulations. Because the disc is prepared for axial compression, it should be noted that under large loads, the endplate will fracture (Schmorl node) (Figure 2-28) or the anterior vertebral body will collapse.

Axial tensile stresses are also produced in the annulus during the movements of flexion, extension, and lateral flexion. The motions create compression stresses ipsilaterally and tensile stresses contral-

aterally. This causes a bulging (buckling) on the concave side and a contraction on the convex side of the disc (Figure 2-29).

Axial rotation of the spine also produces tensile stresses in the disc. Studies have shown that the greatest tensile capabilities of the disc are in the anterior and posterior regions; the center portion of the disc is the weakest. When the disc is subjected to torsion, shear stresses are produced in the horizontal and axial planes. Shear stresses act in the horizontal plane, perpendicular to the long axis of the spine. It has been found that torsional forces, and hence shear forces, can be the injury-causing load factors. During normal movements, the disc is protected from excessive torsion and shear forces by the lumbar facet joints.

All viscoelastic structures, which include the disc, exhibit hysteresis and creep. Cadaveric studies allowed Twomey and Taylor⁷⁸ to study creep and hysteresis in the lumbar spine. *Hysteresis* refers to the loss of energy when the disc or other viscoelastic structures are subjected to repetitive cycles of loading and unloading. It is the absorption or dissipation of energy by a distorted structure. For example, when a person jumps up and down, the shock energy is absorbed by the discs on its way from the feet to the head. The larger the load, the greater the hysteresis.¹ When the load is applied a second time, the hysteresis decreases, meaning there is less capacity to absorb the shock energy (load). This implies that the discs are less protected against repetitive loads.

Creep is the progressive deformation of a structure under constant load. When a load is applied to a viscoelastic structure, it immediately deforms under the load. If the load is maintained, there will be continued deformation over time. As might be expected, the creep and hysteresis created in differing types of load forces (e.g., flexion loading vs. extension loading) may differ, but this has not been quantified for the lumbar spine.

Because the disc is under the influence of the preloaded state of the nucleus, movements have specific effects on the behavior of the nucleus and annular fibers. When a distraction force is applied, the tension on the annular fibers increases and the internal pressure of the nucleus decreases. When an axial compression force is applied symmetrically, the internal pressure of the nucleus increases and transmits this force to the annular fibers. The vertical force is transformed into a lateral force, applying pressure outward.

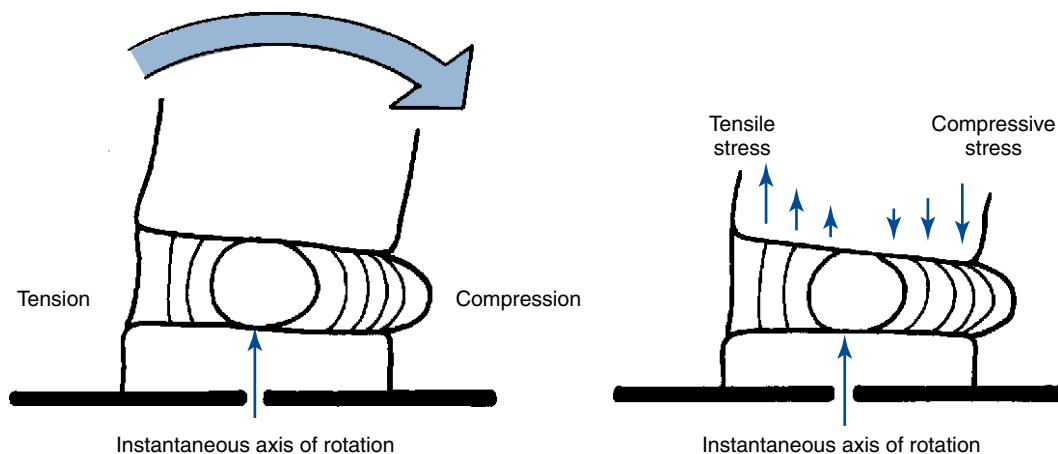


Figure 2-29 Disc stresses with bending movements of flexion, extension, and lateral flexion. Tension is produced on the convex side, whereas compression and buckling occur on the concave side.

During the asymmetric movements of flexion, extension, and lateral flexion, a compressive force is applied to the side of movement, and a tensile force occurs on the opposite side. The tension transmitted from the nucleus to the annular fibers helps to restore the functional unit to its original position by producing a “bow-string–like” tension on the annular fibers.

During axial rotation, some layers of the annulus are stretched and others are compressed (slackened). Tension forces reach a maximum within the internal layers of the annulus. This has a strong compressive force on the nucleus and causes an increased internal pressure proportional to the degree of rotation.

Kurowski and Kubo⁷⁹ investigated how degeneration of the intervertebral disc influences the loading conditions on the lumbar spine. Because disc degeneration is common, it will almost inevitably contribute to low back dysfunction by influencing motion and load bearing at each individual level. Kurowski and Kubo⁷⁹ examined load transmission through the lumbar spine with differing amounts of disc degeneration and used fine element analysis to study stress transmission. In a healthy disc, they found the highest effective stresses in the center of the endplate of the vertebra, but in an unhealthy and degenerated disc, they found these stresses in the lateral aspects of the endplates, as well as in the cortical wall and vertebral body rims.

MODELS OF SPINE FUNCTION

Understanding the overall function of the human spine has proved to be difficult and frustrating. It is important to view the spine as an integrated functioning unit. It must be remembered, however, that the spine is also a part of the larger locomotor system. If consideration is not given to the whole locomotor system, the potential for clinical failure results.

The spine is a mechanical structure characterized by the vertebrae articulating with each other in a controlled manner through a complex of levers (vertebrae), pivots (facets and discs), passive restraints (ligaments), and activations (muscles).¹ There are three important and fundamental biomechanical functions of the spine.¹ First and foremost, the spine must house and protect the spinal cord, yet allow for transmission of neurologic impulses to and from the periphery. Second, it must provide support for the upright posture by being able to absorb shock and bear and transfer weight from the resultant bending moments of the head and trunk to the pelvis. Finally, it must allow for sufficient physiologic motions between the body parts in the six degrees of freedom. The vertebral column is a flexible axis composed of the articulated vertebrae. The spine must be rigid for it to maintain upright bipedal posture, yet it has to deform its shape to allow for mobility. In addition, it houses and protects the spinal cord and provides a means for neurologic transmission to and from the periphery.

Many models of spine function have been developed,^{80–84} each attempting to define spine function according to new and different parameters. However, each of these models fails in some way to consider all of the characteristics and requirements of the spine’s complex and integrated structural and functional relationship. A spine model has been proposed that considers the struc-

tural integrity of the spine as a whole, providing an interesting look at how adaptation to upright biped posture places specific demands on the spinal components. A *structure* is defined as any assemblage of materials that is intended to sustain loads. Each life form needs to be contained by a structure. Even the most primitive unicellular organism has to be enclosed and protected by cell membranes that are both flexible and strong, yet capable of accommodating cell division during reproduction. With advancement of and competition in evolving life forms, the structure requirements need to become more sophisticated. The majority of living tissues have to carry mechanical loads of one kind or another. Muscles also have to apply loads, changing shape as they do so. By making use of contractile muscles as tension members and strong bones as compression members, highly developed vertebrate animals have been able to withstand necessary loads and still allow for mobility, growth, and evolution.

Parallels have been drawn between the spine and the mast of a ship. Compressive loads are concentrated in the vertebrae of the spine and the wooden mast of the ship. Tension loads are diffused into tendons, skin, and other soft tissues of the body and into the ropes and sails of the ship to maintain an upright position. However, a ship mast is immobile, rigidly hinged, vertically oriented, and dependent on gravity. These rigid columns require a heavy base to support the incumbent load. In contrast, the biologic structure of the spine must be a mobile, flexibly hinged, low-energy-consuming, omnidirectional structure that can function in a gravity-free environment.⁸⁵

Comparisons have also been made between the spine and a bridge (or truss). The musculoskeletal configuration of a large, four-legged animal (e.g., a horse) is capable of bearing a substantial load in addition to its own weight, rests on four slender compression members (leg bones), and is supported efficiently by an assortment of tension members (tendons, muscles, and skin). Trusses have flexible, even frictionless, hinges, with no bending moments about the joint. The support elements are either in tension or compression only. Loads applied at any point are distributed about the truss as tension or compression.⁸⁵

Although this model sounds quite plausible for the spine, it is not a complete explanation. Most trusses are constructed with tension members oriented in one direction. This means that they function in only one direction and can therefore not function as the mobile, omnidirectional structure necessary for describing the spine functions. Moreover, bridges do not have to move, whereas vertebrate animals do. Furthermore, the comparison cannot be directly applied to the human skeleton, because the human skeleton is upright and the forces are applied in the long axis rather than along it.

Levin⁸⁵ identifies another class of truss called *tensegrity structures* that are omnidirectional so that the tension elements always function in tension regardless of the direction of the applied force. The structure that fits the requirements of an integrated tensegrity model has been described and constructed as the tensegrity icosahedron. In this structure, the outer shell is under tension, and the vertices are held apart by internal compression struts that seem to float in the tension network (Figure 2-30). In architecture, stable form is generated through an equilibrium between many interdependent structures, each of which is independently in a state of disequilibrium. Complex architecture cannot be

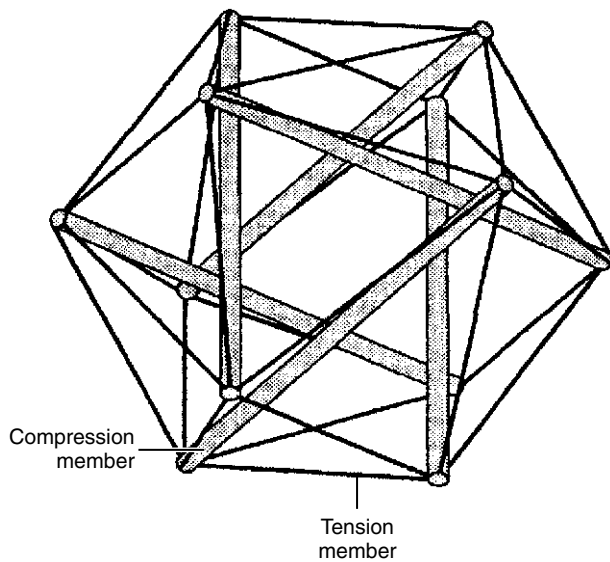


Figure 2-30 Tensegrity icosahedron with rigid compression members and elastic tension members. Multiple units sharing a compression member form a structural model of the spine. (Modified from Bergmann TE, Davis PT: *Mechanically assisted manual techniques: Distraction procedures*, St Louis, 1998, Mosby.)

broken up into isolated pieces without losing qualities that are inherent to the structural whole. This is extremely important in biologic systems in which every functional unit is literally more than the sum of its constituent parts.⁸⁶

Many architectural structures depend on compressive forces for structural integrity. Compression-dependent structures are inherently rigid and poorly adapted for a rapidly changing environment. Most naturally occurring structures depend on natural forces for their integrity.⁸⁷ The human body can be described as a

tensile structure in which tensional integrity (tensegrity) is maintained by muscles suspended across compression-resistant bones.

Fuller⁸⁸ spoke for many years of a universal system of structural organization of the highest efficiency based on a continuum of tensegrity. Fuller's theory of tensegrity developed out of the discovery of the geodesic dome, the most efficient of architectural forms, and through study of the distribution of stress forces over its structural elements. A *tensegrity system* is defined as an architectural construction that is composed of an array of compression-resistant struts (bones) that do not physically touch one another but are interconnected by a continuous series of tension elements (muscles and ligaments).⁸⁶ Because action and reaction are equal and opposite, the tension forces have to be compensated by equal and opposite compressive forces and vice versa.

Gravitational force is a constant and greatly underestimated stressor to the somatic system. The most obvious effect of gravitational stress can be evaluated by careful observation of posture, which is both static and dynamic. The static alignment of body mass with respect to gravity is constantly adjusted by dynamic neuromuscular coordination as the individual changes position. Over time, individual static postural alignment conforms to inherent connective tissue structure, as well as the cumulative functional demands of both static and dynamic postural conditions.

Musculoligamentous function is also significantly influenced by, as well as responsible for, static and dynamic postural alignment.⁸⁹ The development of asymmetric functional barriers in the spine likely has more than one cause. A unifying factor, however, is the transfer of forces within the soft tissues that creates altered and asymmetric tension, namely the *tensegrity mechanism*.

When the various principles and research noted here are combined, a more complete picture of spinal biomechanics is developed in which pathologic changes may ultimately be better studied as well.

JOINT ASSESSMENT PRINCIPLES AND PROCEDURES

OUTLINE			
THE MANIPULABLE LESION	36	Examination Procedures and	Reliability of Palpation
SUBLUXATION	36	Diagnostic Criteria	Procedures
VERTEBRAL SUBLUXATION		History	Validity of Palpation
COMPLEX	37	Physical Examination	Procedures
Mechanical Components	38	Pain and Tenderness	Sacroiliac Articulation
Joint Malposition	38	Asymmetry	Bony Palpation
Joint Fixation (Hypomobility)	39	Range-of-Motion	Soft Tissue Palpation
Clinical Joint Instability and		Abnormality	Motion Palpation
Hypermobility	41	Tone, Texture, and Temperature	Accessory Joint Motion
Mechanical Models of Spinal		Abnormality	Joint Challenge
Dysfunction and		Special Tests	(Provocation)
Degeneration	42	Clinical Usefulness of Joint	Percussion
Neurobiologic Components	43	Assessment Procedures	Muscle Testing
Theory of Intervertebral		Reliability	Provocative (Orthopedic) Tests
Encroachment and Nerve		Validity	Radiographic Analysis
Root Compression	43	Responsiveness	Spinal X-ray
Theory of Altered Somatic and		Utility	Examinations
Visceral Reflexes	45	Outcome Assessment	Functional X-ray
Inflammatory and Vascular		Procedures	Examination
Components	46	Symptoms of Joint Subluxation/	Videofluoroscopy
Vascular Congestion	46	Dysfunction Syndrome	Clinical Use of X-ray
Inflammatory Reactions	46	Patient Observation	Examination
JOINT SUBLUXATION/		Gait Evaluation	Instrumentation
DYSFUNCTION SYNDROME	47	Postural Evaluation	Algometry
SPINAL LISTINGS	47	Leg Length Evaluation	Thermography
CLINICAL EVALUATION OF JOINT		Range-of-Motion	Galvanic Skin Resistance
SUBLUXATION/DYSFUNCTION		Assessment	Surface Electromyography
SYNDROME	47	Measurement Procedures	CLINICAL DOCUMENTATION
		Palpation	82

The doctor of chiropractic views the human being as a dynamic, integrated, and complex living thing who has an innate capacity for self-healing.¹⁻⁶ Chiropractic health care focuses on the evaluation and treatment of neuromusculoskeletal (NMS)-based disorders, but does not disregard the multiple potential causes of ill health and the complex nature of health maintenance.^{7,8}

In keeping with this philosophy and the responsibility as “portal-of-entry” health care providers, chiropractors must maintain broad and thorough diagnostic skills. The Council on Chiropractic Education (CCE) defines the primary care chiropractic physician as an “individual who serves as a point for direct access to health care delivery; the doctor of chiropractic’s responsibilities include (1) patient’s history; (2) completion and/or interpretation of physical examination and specialized diagnostic procedures; (3) assessment of the patient’s general health status and resulting diagnosis; (4) provision of chiropractic care and/or consultation with continuity in the co-management, or referral to other health care providers; and (5) development of sustained health care partnership with patients.”⁹

Before applying therapy, the chiropractor must first ascertain if there is a clinical basis for treatment. The chiropractic physician who chooses to limit therapeutic alternatives must still possess the skills necessary to determine if patients seeking their care have a health problem responsive to the specific treatments they provide.¹⁰ This dictates that chiropractors be trained to screen and evaluate a broad range of complaints if they wish to maintain their primary contact privileges. Diagnostic skills must have sufficient depth to screen all organ systems of the body for those conditions that are and are not amenable to chiropractic treatment. The social expectation and regulatory requirement of a primary contact provider are to provide a suitable health status assessment and initial clinical impression regardless of the patient presentation or the health care professional’s particular discipline, philosophy, or theories.

A core area of focus and expertise for the chiropractic physician is the evaluation of the NMS system. This chapter focuses on the knowledge, principles, and evaluation procedures central to the process of determining whether a patient is a candidate for adjustive therapy.

THE MANIPULABLE LESION

Manual therapy has been proposed as an effective treatment for a wide variety of conditions, but it is most commonly associated with disorders that have their origins in pathomechanical or pathophysiologic alterations of the locomotor system and its synovial joints. As a result, manual therapy is based on assessment procedures that take into consideration both functional and structural alteration of the NMS system. Haldeman¹¹ has referred to this process as the *identification of a manipulable lesion*. Spinal manipulation is thought to act on this manipulable or functional joint lesion, but given the historical presumption of this entity, it is somewhat surprising that there is not more information on its pathomechanical properties.¹² The lesion is viewed as a set of possible individual maladies responsible for the patient's symptoms.^{13,14}

The identification of the common functional and structural components of the manipulable lesion is critical to the management of this condition, but it has also contributed to the misconception that all manipulable disorders have the same pathologic basis. The overwhelming majority of disorders effectively treated with chiropractic adjustments do display joint and somatic functional alterations, but many pathologic processes can induce joint dysfunction.

A diagnosis of joint dysfunction syndrome identifies local altered mechanics, but it does not identify the underlying nature of the dysfunction. Although joint derangements may present as independent clinical syndromes, they are more commonly associated with other identifiable disorders and injuries of the NMS system.¹⁵⁻²³

If chiropractors limit their examination to the identification of structural or functional signs of joint dysfunction, they may minimize the extent of the disorder and the effectiveness of their treatment. For example, both the patient with acute disc herniation and the patient with acute facet syndrome present with clinical signs of joint dysfunction. An evaluation confined to the detection of joint dysfunction might not uncover the underlying pathomechanical and pathophysiologic differences between these two conditions and the distinctions in therapy that might be necessary. Furthermore, other disease states or traumatic events that would contraindicate adjustive therapy may induce spinal malpositions or fixations.

A singular diagnosis of *joint dysfunction* or *subluxation syndrome* should be reserved for instances when it is determined to be the sole identifiable lesion; the terms should not be used as a category for all conditions treated with adjustive therapy. When joint dysfunction is perceived as the sole cause of the disorder being considered for treatment, adjustive therapy may be the only treatment necessary. However, when joint dysfunction is secondary to other disorders that are not responsive to adjustive treatments, other effective treatments should be provided or made available to the patient by referral.

Determination of the appropriateness of adjustive therapy should not be based on the presence of a fixation, malposition, or spinal listing alone. The cause of the altered mechanics indicates whether adjustive therapy or some other form of therapy is in order.²³

SUBLUXATION

Within the chiropractic profession, the manipulable lesion has been equated primarily with the term *joint subluxation*. The concept of subluxation is a central defining clinical principle and the source of contentious debate and disagreement within the profession.²⁴ Mootz suggests that the chiropractic profession's attention to subluxation (pro and con) is found in virtually every dimension of the profession's existence, be it clinical, scientific, philosophical, or political.²⁵ He identifies four distinct ways that subluxation is used by the profession, each with merits and liabilities. They are²⁵:

- Subluxation as chiropractic theory: Subluxation is used as an explanatory mechanism for physical effects of chiropractic intervention.
- Subluxation as professional identity: Subluxation forms the entire basis of and for chiropractic practice.
- Subluxation as a clinical finding: Subluxation serves as target for localizing manipulative and adjustive intervention.
- Subluxation as a clinical diagnosis: Subluxation represents a distinct clinical condition or syndrome.

Historically, *joint subluxation* was defined predominantly in structural terms.^{1,2,23,26-30} The founder of chiropractic, D.D. Palmer, defined *joint subluxation* as a "partial or incomplete separation, one in which the articulating surfaces remain in partial contact."³¹ Central to Palmer's original subluxation hypothesis was the concept that vertebral subluxations could impinge on the spinal nerve roots (NRs) as they exit through the intervertebral foramina. This was postulated to obstruct the flow of vital nerve impulses from the central nervous system to the periphery and to induce lowered tissue resistance and potential disease in the segmentally innervated tissues.^{1,2,8,29,31-35} Palmer went so far as to suggest that the primary cause of all disease could be related to subluxations and interruption of normal "tone—nerves too tense or too slack."^{1,8}

The most impassioned supporter of this concept was D.D. Palmer's son, B.J. Palmer. Throughout his career, B.J. Palmer ardently promoted a monocausal concept of disease,^{8,27,28,36,37} specifically stating that chiropractic is "a science with provable knowledge of one cause of one disease being an internal interference of the internal flow of abstract mental impulses or nerve force flow supply, from above down, inside out."³⁶

Although the profession today emphasizes the important relationship between health and the structure and function of the NMS system,^{4-7,32-35,38,39} it does not promote a monocausal concept of subluxation-induced disease.^{7-10,37-40} The monocausal concept runs contrary to much of the profession's recent literature^{24,34,35,37-39} and to the view held by the overwhelming majority of practicing chiropractors.⁸ Although a small minority of chiropractors still promotes this extreme view, both the profession's national associations and the CCE have disavowed it.^{9,39}

Beginning with the published work of Gillet,⁴¹⁻⁴⁶ Illi,⁴⁷ and Menell,^{48,49} and later through the writings of Sandoz^{23,30,50,51} and Faye,^{52,53} the importance of the dynamic characteristics of joint subluxation moved to the forefront. As a result, *joint integrity* was defined not only in structural terms but also in functional terms.^{23,30,34,35,42-56} Within this context, *joint subluxation* took on a

broader definition, and joint malposition became a possible sign of disturbed joint function, not absolute confirmation.

This view provides a more dynamic perspective and suggests that minor joint misalignment does not necessarily predict the presence or absence of joint dysfunction or the direction of possible restricted movement.^{23,30,50-54} From this perspective, joints do not have to be malpositioned to be dysfunctional. Joint fixation can occur with the joint fixed in a neutral position, or it can have multiple planes of joint restriction.^{23,30,50,57,58} Consequently, treatment decisions concerning adjustive therapy and adjustive vectors, once based predominantly on the direction of malposition, grew to incorporate an assessment of the functional status of the patient including an assessment of joint mobility.⁴¹⁻⁵⁵ Today, consideration is given to both the static and dynamic components of spinal dysfunction, including presence or absence of joint pain with loading (joint provocation/challenge).^{23,32,34}

Other health care providers within the field of manual medicine also struggle with multiple definitions and explanations for manipulable lesions.⁵⁹⁻⁶³ Box 3-1 contains a list of terms and definitions commonly used to describe functional or structural disorders of the synovial joints. A common principle behind all of these concepts is that there is a somatic component to disease and that dysfunction of the NMS system can affect a

patient's overall health status as well as the ability to recover from injury and disease.

VERTEBRAL SUBLUXATION COMPLEX

Because of continued professional debate and increasing scientific inquiry, a trend toward viewing subluxations as complex clinical phenomena has unfolded.* Rather than a condition definable by one or two characteristics, subluxation is more commonly presented as a complex, multifaceted pathologic entity, known as the *vertebral subluxation complex* (VSC) (see Box 3-1). The VSC is a conceptual model and should not be confused with the vertebral subluxation syndrome. The *vertebral subluxation/dysfunction syndrome* defines a clinical disorder identified by its presenting symptoms and physical signs.

Gitelman, and later Faye, were the first to promote this broader model and its theoretic components.^{51,56,65,66} More recently, Lantz⁶⁷ and Gatterman^{60,64} have championed this cause. In 1994, a consensus⁶⁰ presented broader definitions for the VSC that seems to be growing in recognition and acceptance.

* References 23, 26, 30, 31-35, 39, 55, 56, 60, 64.

BOX 3-1 Terms Describing Functional or Structural Disorders of the Synovial Joints

ORTHOPEDIC SUBLUXATION

A partial or incomplete dislocation.⁵⁹

SUBLUXATION

The alteration of the normal dynamic, anatomic, or physiologic relationships of contiguous articular structures⁵⁶; a motion segment in which alignment, movement integrity, or physiologic function is altered, although the contact between the joint surfaces remains intact⁶⁰; an aberrant relationship between two adjacent articular structures that may have functional or pathologic sequelae, causing an alteration in the biomechanical or neurophysiologic reflections of these articular structures or body systems that may be directly or indirectly affected by them.¹⁰

SUBLUXATION SYNDROME

An aggregate of signs and symptoms that relate to pathophysiology or dysfunction of spinal and pelvic motion segments or to peripheral joints.⁶⁰

SUBLUXATION COMPLEX

A theoretic model of motion segment dysfunction (subluxation) that incorporates the complex interaction of pathologic changes in nerve, muscle, ligamentous, vascular, and connective tissues.¹⁰

JOINT DYSFUNCTION

Joint mechanics showing area disturbances of function without structural change—subtle joint dysfunctions affecting quality and range of joint motion. Definition embodies disturbances in function that can be represented by decreased motion, increased motion, or aberrant motion.⁶¹

Joint hypomobility: decreased angular or linear joint movement

Joint hypermobility: increased angular or linear joint movement; aberrant joint movements are typically not present.

Clinical joint instability: increased linear and aberrant joint movement; the instantaneous axes of rotation (centroids) and patterns of movement are disturbed.

SOMATIC DYSFUNCTION

Impaired or altered function of related components of the somatic (body framework) system; skeletal, arthrodial, and myofascial structures; and related vascular, lymphatic, and neural elements.⁶²

OSTEOPATHIC LESION

A disturbance in musculoskeletal structure or function, as well as accompanying disturbances of other biologic mechanisms. A term used to describe local stress or trauma and subsequent effects on other biologic systems (e.g., effects mediated through reflex nerve pathways, including autonomic supply of segmentally related organs).⁶³

JOINT FIXATION

The state whereby an articulation has become temporarily immobilized in a position that it may normally occupy during any phase of physiologic movement; the immobilization of an articulation in a position of movement when the joint is at rest or in a position of rest when the joint is in movement.³⁰

Although the trend toward a broader perspective of subluxation has helped move the profession from a simplistic and reductionistic model of spinal health, it has not necessarily advanced the investigation into its existence and nature. Reaching consensus on subluxation theory and expanding the number of clinical spinal disorders that are supposedly subluxation-related does not provide proof of their presence as the primary “lesion” treated by chiropractors. Faye suggests that the subluxation complex is a conceptualization for organizing the essential information relevant to treatment, allowing a chiropractor to examine a person in both a classic orthoneurologic manner and using a biomechanical approach to arrive at a double diagnosis.⁶⁸ The first assesses the state of the pathologic tissue changes and also aids in determining the prognosis. The second determines the therapeutic procedures to be used and the treatment schedule.⁶⁸

Nelson²⁴ states that subluxation theory lacks several necessary properties that would allow it to serve as a vehicle for research. First, a theory should attempt to explain existing phenomena and observations; the VSC theory has not been used to explain any specific clinical phenomena. Lantz⁶⁷ adds that the VSC does not identify any single event or process as the sole causative element in the complex process of subluxation development. Second, a theory should make predictions; the VSC theory makes none. It does not lead in any particular direction or draw any distinction or specific conclusions. The VSC theory suggests that any number of pathologic conditions affecting tissue are possible, with none being more important than any other.⁶⁷ Third, a theory should be testable and falsifiable so that a study may provide results or observations that either confirm or refute the theory. The VSC theory is so encompassing, allowing for a wide range of mitigating and changing circumstances, that it is difficult to evaluate. Nelson²⁴ points out that this circular type of argument and reasoning (tautology) validates itself simply by renaming accepted principles as a new theory or principle. A tautology has the virtue of being irrefutable, but the deficiency of being useless. It explains nothing, makes no predications, draws no distinctions, and is untestable.

There is value in reaching consensus on the theoretic pathophysiologic and pathomechanical components of functional disorders of spinal motion segments, but mainly for purposes of dialogue and research. The VSC therefore remains a theoretic model in need of investigation. The VSC theory should not be considered as one grand theory, but rather a series of interlocking and interdependent principles. The principles that form a basis for considering the existence and significance of the subluxation should be consistent with current basic science precepts. They must reflect current practice and educational standards, be clinically meaningful, and present a distinct and unique point of view. Unfortunately, the available research data tell us little about the presumed clinical meaningfulness of the traditional chiropractic lesion. Clinical meaningfulness refers to the practical value of a concept in directing the clinician to successful resolution of the health problem the patient has presented. Unfortunately, no one has systematically addressed the predictive power (if any) of subluxation correction for any specific disease or “condition.” None of the controlled clinical trials of the effects of spinal manipulative therapy has, to date, included a subluxation element.^{69,70}

Keating and colleagues point out that the concept of chiropractic subluxation stands pretty much today as it did at the dawn of

the 20th century: It is an interesting notion without validation.⁷¹ Although there is a strong intraprofessional commitment to the subluxation construct and there are reimbursement strategies that are legally based on subluxation, there is no scientific “gold standard” for detecting these clinical entities.⁷² The term *chiropractic subluxation* continues to have as much or more political than scientific meaning.⁷³

Subluxation is still the most common term chiropractors use to describe the spinal joint disorders they treat.⁷⁴ However, chiropractors are much more likely to view subluxations as disorders that have either structural or functional components rather than simply malpositioned joints. Furthermore, the VSC has been described using theoretic pathologic components broadly divided into mechanical, inflammatory-vascular, and neurobiologic categories. Although these divisions are modeled after those proposed by previous authors, they do not represent an established professional convention. Instead the categories and topics presented here represent an overview of the theoretic effects of the VSC and are not intended to be an all-inclusive or exhaustive treatise on the subject. While these categories are discussed separately, it must be emphasized that although these characteristics may occur in isolation, they can also occur in varying combinations. Some are emphasized more than others, depending on the mode of onset, rate of repair, and length of treatment time.

MECHANICAL COMPONENTS

The mechanical category of the VSC includes derangements or disorders of the somatic structures of the body that lead to altered joint structure and function. Derangement of the articular soft tissues and mechanical joint dysfunction may result from acute injury, repetitive-use injury, faulty posture or coordination, aging, immobilization, static overstress, congenital or developmental defects, or other primary disease states.*

Joint Malposition

Historically, the basis for subluxation was founded on the concept that traumatic events could lead to altered joint position and that this malposition would interfere with neurologic impulses. Both the chiropractic profession (through D.D. Palmer) and the osteopathic profession (through A.T. Still) have stressed joint position as an important quality for normal joint function.^{1,94}

One of the oldest concepts from the literature on manipulation is the interdependence of structure and function. In other words, structure determines function and function determines structure. When there is a change in structure, there will be a change in function. Therefore, if a structural alteration is identified, a functional change should also be perceived. When a spinal joint is either acutely traumatized or undergoes chronic repetitive stresses, it is assumed that asymmetric muscle tension is likely to develop and hold the joint in a position away from its neutral alignment. The central idea is that misaligned positions of skeletal components can result in movement limitations, associated inflammatory changes, and irritation of nociceptors leading

*References 15-23, 26, 30, 34, 50-54, 56, 75-93.

to pain. From a historical perspective, the chiropractic profession primarily viewed spinal subluxations as a structural failure that alters body function.⁹⁵

The concept of static vertebral misalignment is difficult to support, however. Triano cites evidence that there is no “normal position” between vertebrae in the sense of the historic subluxation argument.⁹⁵ The spine and the component parts are not perfectly symmetric in their development. Spinous processes in particular are quite prone to asymmetric growth. It is also very unlikely that one could palpate a displacement of a few millimeters or degrees based on the location of the spinous processes. Identification of joint malposition is typically through static palpation or radiographic mensuration. Both of these procedures have only fair to poor inter- and intraobserver agreement. Furthermore, there is no evidence that supports a change in alignment following manipulative intervention. Clearly the “bone-out-of-place” concept is not likely to be the sole explanation for subluxation.^{25,96}

Joint Fixation (Hypomobility)

A more biologically plausible model of spinal joint pain incorporates abnormal joint mechanics and postulates that vertebral hypomobility can cause pain and abnormal spinal mechanics because of changes in sensory input from spinal and paraspinal tissues. Work by Henderson and associates provide the first preliminary anatomic evidence that altered spinal mechanics may produce neuroplastic changes in the dorsal horn of the spinal cord.⁹⁷⁻⁹⁹ Their preliminary data suggest that chronic vertebral hypomobility (fixation) at L4 through L6 in the rat affects synaptic density and morphology in the superficial dorsal horn of the L2 spinal cord level.⁹⁹

Soft Tissue Injury and Repair. A commonly proposed source of joint fixation (hypomobility) and dysfunction is periarticular soft tissue injury with its resultant fibrosis and loss of elasticity and strength.^{15-22,54,56,57,75-77} Soft tissue injury and fibrosis may result from acute or repetitive trauma to muscular, tendinous, myofascial, or ligamentous tissue. Regardless of the mechanism of injury, an ensuing inflammatory response is triggered⁵⁷ resulting in extracellular accumulation of exudates and blood. Platelets then release thrombin-converting fibrinogen into fibrin, which organizes into collagenous scar tissue, resulting in a variety of soft tissue and articular adhesions. This process is considered to be nonspecific and often excessive in the case of traumatic NMS injuries.^{15,79} As a consequence, early conservative management is often directed at limiting the extent of the inflammatory response. Therapies directed at minimizing the extent of associated inflammatory exudates are helpful in reducing pain and muscle spasm and in promoting early pain-free mobilization and flexible repair.^{79,83-85,93,100-113} Aggressive early care and mobilization provide the best opportunity for optimal healing and an early return to work for the patient. Bed rest and prolonged inactivity increase the chances of long-term disability and lost work time.^{103,105,114,115}

The exudates that form as a byproduct of injury and inflammation set the stage for the next step in the process of connective tissue repair. They provide the matrix for the development of granulation tissue and scar formation. The formation of granulation tissue is predominantly carried out by the proliferation of fibroblasts and the synthesis and deposit of collagen tissue. The collagen is initially very poorly organized and must add additional collagen cross-linkages and reorganize along planes of stress

to improve the tensile strength of the injured area. This process of repair and remodeling may take months and may result in less than optimal restoration and extensibility of the involved tissue. Immobilization slows the process of recovery, leading to loss of strength and flexibility and potential intra-articular fatty adhesions.^{75,76,83-93} Immobilization also leads to dehydration, causing proteoglycans to approximate and stick together.^{83,84,88} If injury or immobilization leads to decreased flexibility, therapies such as articular adjustments or joint mobilization should be directed toward the restoration of motion.^{15,79,82,102}

Myofascial Cycle. Painful conditions capable of triggering persistent muscle hypotonicity are additional sources of restricted joint motion (Figure 3-1). Muscle contraction, once initiated, may become

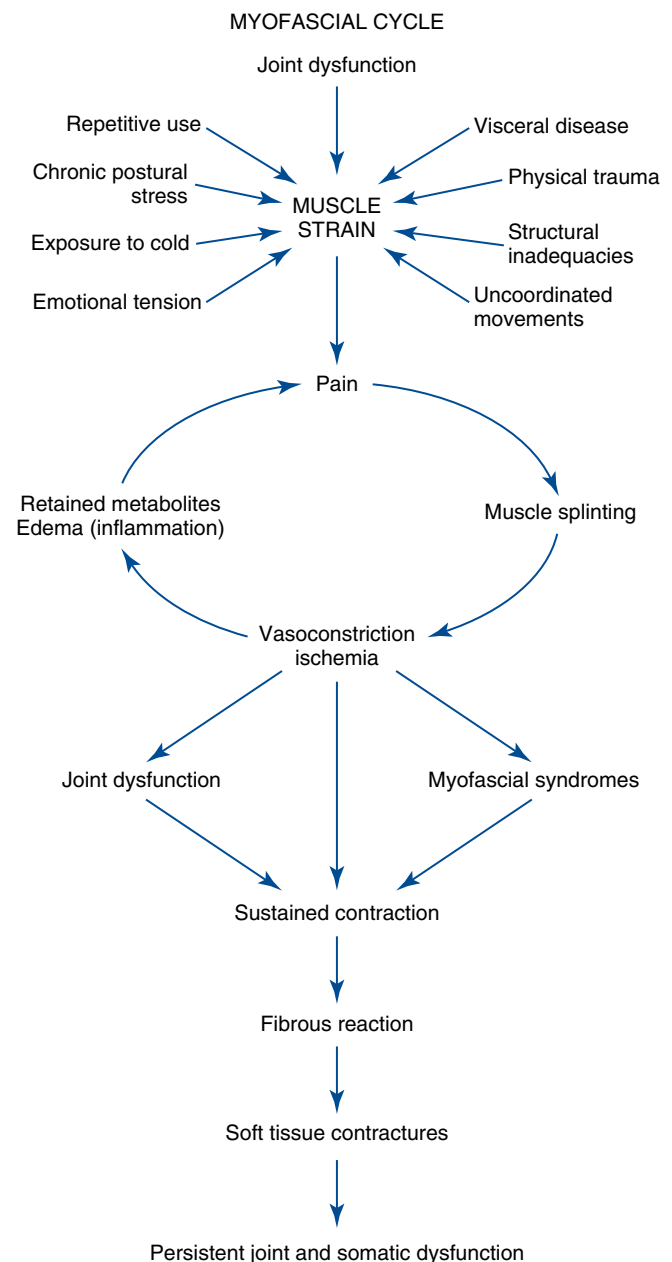


Figure 3-1 Myofascial conditions are triggered by many causes and can become self-perpetuating sources of pain, muscle spasm, and joint dysfunction.

a self-perpetuating source of pain and muscle hypotonicity.* Reactive splinting in the joint's intrinsic muscles may further accentuate this process by blocking passive joint movement and the pain-inhibiting qualities of joint mechanoreceptor stimulation.¹²⁰ Persistent contractions over time may develop into muscle contractures as a result of adaptational shortening and loss of elasticity from disuse or under-use. Although there is little direct evidence to support the belief that sustained muscle contraction is a feature of intervertebral dysfunction, the concept of protective muscle splinting appears plausible.¹²¹ Maladies capable of producing acute muscle contraction are wide ranging; they include trauma, structural inadequacies, visceral disease, emotional distress, and exposure to cold.^{122,123}

Interarticular Derangements. A number of internal joint derangements have also been submitted as probable causes of joint locking and back pain. They include internal derangements of the intervertebral disc (IVD; intradiscal block), derangements of the posterior spinal joints (interarticular, intermeniscoid block),^{50,51,77,78,130-146} and compressive buckling injuries.^{12,13} They are hypothesized to induce mechanical blockage to movement and unleveling of the motion segment, with resultant tension on the joint capsule, annulus, or both. The joint capsule and posterior annulus are pain-sensitive structures, and tension on these elements may induce additional painful muscle splinting, further accentuating the mechanical blockage and joint restriction. Mechanical joint dysfunction is therefore considered to be a significant and frequent cause of spinal pain and a potential source of spinal degeneration.

Interarticular Block. One source of derangement of the posterior joints is speculated to result from entrapment (Figure 3-2) or extrapment (Figure 3-3) of joint meniscoids or synovial folds.¹³¹⁻¹⁴¹ The intra-articular meniscoids are leaflike fibroadipose folds of synovium that are attached to the inner surface of the joint capsule and project into the joint cavity. These meniscoids have been found to be present in all of the posterior joints of the spine.

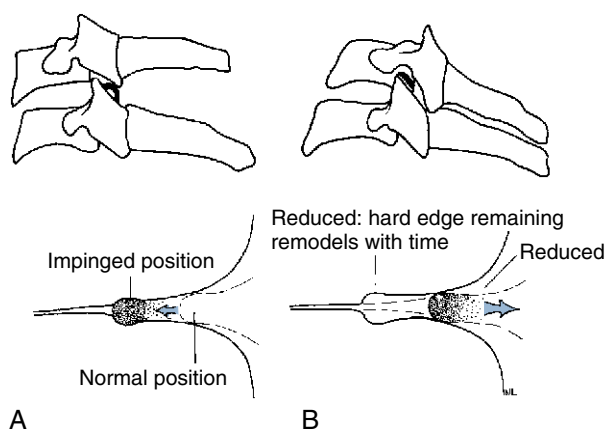


Figure 3-2 Theory of meniscoid entrapment. **A**, Diagrammatic representation of meniscoid entrapment inducing flexion and extension malpositions, capsular tension, pain, and subsequent restrictions in spinal mobility. **B**, Manipulation of the joint separates the joint surfaces, allowing the meniscoid to return to a neutral position.

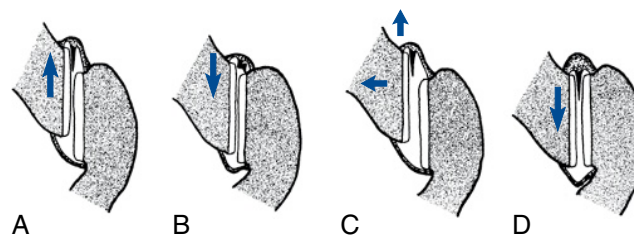


Figure 3-3 Theory of meniscoid extrapment. **A**, On flexion, the inferior articular process of a zygapophyseal joint moves upward, taking a meniscoid with it. **B**, On attempted extension, the inferior articular process returns toward its neutral position, but the meniscoid, instead of reentering the joint cavity, buckles against the edge of the articular cartilage, forming a space-occupying lesion under the capsule. **C**, Manipulation gaps the joint and allowing the meniscoid to return to its neutral resting position (**D**).

Bogduk and Jull¹⁴⁰ have suggested that extrapment of these meniscoids may be one cause of restricted joint motion. They speculate that the meniscoid may occasionally be pulled out of its resting position by the inferior articular process of a zygapophyseal joint as it moves upward during flexion. On attempted extension, the inferior articular process returns toward its neutral position, but the meniscoid, instead of re-entering the joint cavity, impacts against the edge of the articular cartilage and buckles, representing a space-occupying lesion under the capsule. Pain occurs as a result of capsular tension, and extension motion is restricted. The use of a distractive or joint gapping adjustive procedure may function to separate the articular surfaces and release the extrapped meniscoid (see Figure 3-3).^{140,147}

Maigne⁷⁸ and others^{77,116,137,148-152} have proposed a model of interapophysary meniscus entrapment rather than extrapment. In this model the menisci are purportedly drawn into a position between the joint margins during poorly coordinated spinal movements or sustained stressful postures. With resumption of normal postures, pain resulting from impaction of the menisci or traction of the articular capsule induces reactive muscle splinting and joint locking. The development of a painful myofascial cycle is initiated as prolonged muscle contraction leads to muscle fatigue, ischemia, and more pain. If spasm and locking persist, the articular cartilage may mold around the capsular meniscus, causing it to become more rigidly incarcerated within the joint.¹¹⁶⁻¹¹⁸ To interrupt the cycle of pain, muscle cramping, and joint locking, distractive adjustments have also been presented as a viable therapy capable of inducing joint separation, cavitation, and liberation of the entrapped menisci (see Figure 3-2).¹¹⁸ It is important to note that meniscoid derangement is only one hypothetical cause of joint dysfunction. Meniscoid derangement is postulated to be a more likely source of joint dysfunction in circumstances in which trivial trauma leads to acute joint irritation or locking and associated muscle spasm.¹³⁹

Interdiscal Block. The mechanical derangements of the IVD that may lead to joint dysfunction are postulated to result from pathophysiologic changes associated with aging, degenerative disc disease, and trauma. Farfan¹⁵³ has proposed a model of progressive disc derangement based on repetitive rotational stress to the motion segment. He postulates that repetitive torsional loads of sufficient number and duration may, over time, lead to a fatigue injury in the outer annular fibers. The process would begin with circumferential distortion and separation in the outer annular fibers, followed by progression to radial fissuring and outward

*References 34, 51, 54, 56, 76, 78, 116-119.

migration of nuclear material. Another view postulates that disc derangement, fissuring, and herniation begin in the innermost annular rings and progresses outward.¹⁵⁴

The rate of fatigue and injury depends on the duration and magnitude of the force applied. In the individual with disrupted segmental biomechanics, the process is potentially accelerated as an altered axis of movement leads to increased rotational strain on the IVD. Postmortem dissection studies of degenerated discs have indeed identified radial fissures in the annulus fibrosus. Cyriax¹⁵⁵ believes that displaced nuclear material along an incomplete fissure is the source of joint fixation. Nuclear migration along these radial fissures has also been demonstrated by computed tomography (CT) discography and correlated with patient pain.¹⁵⁶

Interwoven in the natural history of degenerative disc disease may be episodes of acute mechanical back pain and joint locking. Maigne⁷⁸ and others^{23,129-131} have postulated that incidents of blockage may occur during efforts of trunk flexion as nuclear fragments become lodged in fissures in the posterior annulus (interdiscal block) (Figure 3-4). Consequently, tension on the posterior annulus and other mobile elements of the involved motion segment are produced, initiating local muscle guarding and joint locking. Cyriax¹²⁶ proposes that these lesions may induce tension on the dura mater, inducing lower back pain (LBP) and muscle splinting. Once local pain and muscle splinting are initiated, a self-perpetuating cycle of pain, cramping, and joint locking may result.

Adjustive therapy has been proposed as a viable treatment for interrupting this cycle of acute back pain and joint locking. In addition to the distractive effect on the posterior joints, adjustive therapy is thought to have a potential direct effect on the IVD, either by directing the fragmented nuclear material back toward a more central position or by forcing the nuclear fragment toward a less mechanically and neurologically insulting position (see Figures 4-18 and 4-19). Of course there are spinal joints (atlanto-occipital and atlantoaxial articulations) that do not have IVDs, and they are common sites of dysfunction. This clearly indicates that IVD derangement is not the sole source of spinal joint subluxation or dysfunction.

Compressive Buckling Injury. Triano suggests that a causal factor for a manipulable lesion may be a compressive buckling injury.^{12,13} Intersegmental buckling is likely the result of some error in neuromuscular control that fails either to provide adequate pre-stability to the segment or to respond appropriately with muscle

activation to a perturbation.¹⁵⁷ When a mechanical overload to spinal functional units occurs, either as a single traumatic event or cumulative events, a critical buckling load may be reached. Individual structural elements (disc, facet, ligament, nerve, muscle) may experience concentration of local stresses with reduced functional limits and symptom production specific to the tissue affected. The result is a state of dysfunction that may lead to local inflammatory or biomechanical changes.^{158,159}

Each joint possesses some inherent stability resulting from the stiffness of the ligaments and joint capsule. Further stability and control are provided by the neuromuscular system and faulty motor control may lead to inappropriate levels of muscle force and stiffness at a given spinal segment. This may compromise segmental stability at that level,¹⁶⁰ leading to transient intersegmental buckling.¹⁶¹ The segment briefly exceeds its safe physiologic motion, which leads to loading of the surrounding soft tissues (ligaments, IVD, etc.).¹⁵⁷ Furthermore, exposure to vibration and previous disc injury may augment the buckling event. The result of intersegmental buckling is asymmetric positioning of the vertebra that is maintained by the intrinsic muscles producing hypomobility of the functional unit.

Clinical Joint Instability and Hypermobility

Joint dysfunction resulting from soft tissue injury or degeneration does not necessarily result in joint hypomobility. Disturbances of function of the vertebral column can also result from a loss of joint stability. Joint derangement and dysfunction resulting from a loss of joint stability are commonly referred to as *joint hypermobility* or *clinical joint instability*. Both terms are often used interchangeably, and there is no standard for defining these terms. Definitions vary among clinicians and authors and between the clinical and biomechanical literature.^{162,163}

Although numerous definitions abound, all seem to incorporate a loss of stiffness or sensorimotor control affecting the joints' stabilizing structures.¹⁶²⁻¹⁶⁵ The loss of stiffness is clinically relevant if excessive or aberrant movements lead to pain, progressive deformity, or compromised neurologic structures. Movement can be abnormal in quality (abnormal coupling) or in quantity (increased movement).

Attempts have been made to distinguish clinical joint instability from hypermobility (Table 3-1). The differences are a reflection of the structures involved and degree of pathologic change in the joints' stabilizing structures. Hypermobility joints are assumed to be stable under normal physiologic loads. Hypermobility joints demonstrate increased segmental mobility, but they maintain normal patterns of movement. Hypermobility may be in one plane and not associated with any abnormal translational movements.^{166,167}

In contrast, patients with clinically unstable joints have been postulated to have ineffective neural motor control or more advanced changes in the joints' stabilizing structures.¹⁶⁸ Damage to these structures leads to abnormal patterns of coupled and translational movements and possible multiple planes of aberrant joint movement. Clinical joint instability should not be equated with gross orthopedic instability resulting from fracture or dislocation.

There is little doubt that clinical spinal joint instability exists, but current methods lack the necessary sensitivity and specificity for clearly identifying its contributions to back pain.¹⁶² Clinical opinion suggests that the typical presentation is one of recurring

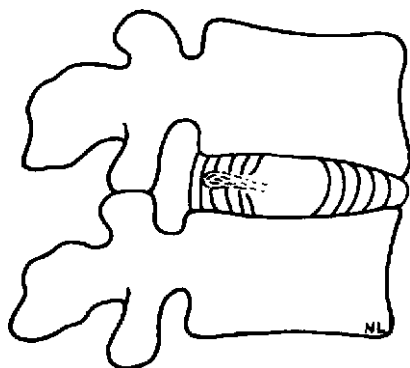


Figure 3-4 Interdiscal block. Illustration of nuclear material migrating into internal annular fissures, producing tension on the posterior annulus.

TABLE 3-1 Joint Hypermobility versus Instability

	Range of Motion	Translational Movements	Coupled Movements
Hypermobility	Increased	Normal ratio	Normal
Instability	Increased or normal	Increased proportion or aberrant	Aberrant

episodes of marked back pain, often initiated by trivial events such as bending or twisting. Global movements are often limited and may demonstrate a painful arc with abnormal patterns of deviation or hitching. Symptoms often resolve within several days, only to recur at a later date.¹⁶⁵

Physical examination tools are limited but increasing.^{162,168} Manual palpation of passive posteroanterior glide has been suggested as one physical means of testing for excessive shear and instability. One recent investigation did demonstrate that prone posterior-to-anterior (P-A) passive joint play (JP) evaluation of the spine can accurately identify abnormal segmental translation as compared with a reference standard of flexion extension radiographs.¹⁶⁹ This test demonstrated good specificity (89%) but poor sensitivity (29%), with a positive likelihood ratio of 2:52. Both the P-A passive segmental mobility assessment and the prone “instability test” were predictive of which patients with low back pain (LBP) would benefit from a lumbar exercise stabilization program.¹⁶⁸ The prone instability test requires the patient to lie in a prone position on an examination table with his or her feet on the floor. The doctor applies segment-passive P-A pressure and, if pain is produced, the patient is asked to raise his or her feet off the floor. If pain is diminished, the test is considered positive and indicative of segmental instability.

Dynamic flexion-extension and lateral bending radiographs are the most commonly used radiographic methods for detecting end-range instability, but they do not provide information about quality of movement during the midrange of segmental motion.¹⁶² Methods using transducers or markers placed over bony landmarks have not demonstrated effective results as a consequence of the skin motion artifact. Methods using pins embedded in the spinous processes to measure movement have adequate accuracy, but these methods are invasive and are not practical for clinical use.¹⁶²

In the absence of gold standard diagnostic tools for detecting spinal joint instability, the chiropractor should pay close attention to the clinical presentation, including history and manual examination, and consider instability in a patient who has recurring episodes of back pain with only temporary relief from manipulation. Suspicion of instability may be reinforced by dynamic x-ray flexion-extension examination, but this procedure may have false-negative results. When instability is still suspected, a conservative treatment trial directed at stabilizing the spine through proprioceptive and specific spinal stabilizing exercises should be applied.¹⁶⁸

Mechanical Models of Spinal Dysfunction and Degeneration

The profession places significant emphasis on the mechanical components of joint dysfunction and subluxation. Mechanical joint dysfunction is considered a significant and frequent cause of spinal pain and a potential source of spinal degeneration.*

The spine is viewed as an interdependent organ system inextricably connected with the rest of the locomotor system. Altered mechanics in one component of the motion segment are perceived to have unavoidable mechanical effects on other functional elements of the motion segment and spine. Several models that outline the proposed sequential dysfunctional and degenerative effects that may ensue subsequent to spinal dysfunction have been developed.

Gillet Model. Gillet^{41-46,53} considers the process of mechanical joint dysfunction developing through three different phases of joint fixation: muscular, ligamentous, and articular. Muscular fixation is considered to be a product of segmental muscle hypertonicity and contraction; ligamentous fixations, the product of contracture and shortening in the joint capsule and its periarticular ligaments; and articular fixations, the product of fibrous interarticular adhesions between articular surfaces. The end stage of articular adhesions is the potential progression to full bony ankylosis and irreversible fixation.

Muscular fixations are identified by the palpation of taut and tender muscle fibers and restricted joint mobility. The end play (EP) is restricted, but has a rubbery and giving quality. Ligamentous fixations demonstrate restricted joint movement and a hard, abrupt, leathery end feel. Articular fixations demonstrate the same quality of restriction, but in all planes of motion.

Gillet maintains that ligamentous or articular fixations are the most significant. He considers muscular fixations as secondary compensations to marked fixations at other levels. As a result, he presents an approach that stresses the identification and treatment of the patient's major fixations. Gillet classifies major fixations as those demonstrating the most dramatic blockages to movement. He contends that the major fixations are frequently not the most symptomatic sites, but are the key to inhibiting pain-free spinal function. Although his ideas are intriguing and have had a profound effect on the profession, they have not been experimentally confirmed.

Kirkaldy-Willis' Model. Kirkaldy-Willis^{169,170} presents a pattern of spinal degeneration founded on the principle that spinal degeneration often begins with local mechanical derangement in the absence of structural alteration. He postulates that

*References 15, 26, 33-35, 39, 45, 50-54, 75, 76.

*References 26, 34, 39, 50-54, 56, 75, 167

the process is often initiated with the development of individual motion segment dysfunction secondary to alteration in segmental muscle tone and function. Although the disorders that are postulated to initiate dysfunction are extensive, most share as a consequence the potential to induce joint hypomobility.²⁶ Joint hypomobility is speculated to initiate the degenerative cycle through the development of altered segmental biomechanics.*

If mechanical derangement persists, repetitive abnormal loading eventually leads to fatigue and attenuation of the articular soft tissues. Local joint instability develops as a result of capsular laxity and internal disruption of the IVD.^{26,170,171} Consequently, if the derangement is of sufficient magnitude, osseous structural alteration will result, and degenerative joint disease becomes radiographically visible (Figure 3-5).¹⁷⁰

The final effect of this degenerative cycle is the restabilization of the joint through soft tissue fibrosis and bony exostosis.^{26,170} As a consequence, the incidence of spinal pain may decrease during the later stages of stabilization. However, bony entrapment of the NRs or stenosis of the spinal canal are of increasing frequency, which may lead to an increased frequency of leg pain and neurologic deficits.^{170,171}

The presented models of motion segment degeneration and the compensational adaptations initiated are not necessarily limited to the involved joint. Not only is it possible for joint hypomobility, instability, and degenerative joint disease all to occur at the same motion segment, but it is also possible for compensatory dysfunction and degenerative changes to develop at other spinal levels or other joints within the locomotor system.^{26,34,46,51-53,75}

Certainly not all joint dysfunction fits this pattern of progression. A large percentage of dysfunction is self-limiting or so minor that an individual adapts and compensates to the change with limited structural or functional alteration. If dysfunction persists, the processes of local and distant joint degeneration may ensue. A point of emphasis and concern for the chiropractic profession is therefore to detect persistent mechanical dysfunction at an early stage of alteration and strive to eliminate it before it develops into irreversible or permanent disorders.

* References 26, 34, 39, 50-54, 56, 75, 167

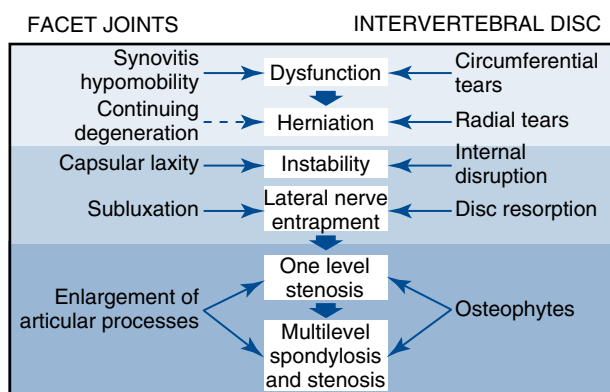


Figure 3-5 The proposed sequence of pathologic changes in the facet joints and disc as a consequence of biomechanical derangement. (From Kirkaldy-Willis WH, Bernard TN Jr: *Managing low back pain*, ed 4, New York, 1999, Churchill Livingstone.)

NEUROBIOLOGIC COMPONENTS

Theory of Intervertebral Encroachment and Nerve Root Compression

Historically, the profession has emphasized spinal NR compression as the significant neurologic disorder accompanying vertebral subluxations.^{1-3,27-38} Spinal subluxations were hypothesized to induce NR compression as a result of direct anatomic compression of neural elements (non-impulse-based model) within the intervertebral foramen (IVF) (Figure 3-6). The resulting NR dysfunction was subsequently hypothesized to induce dysfunction of the somatic or visceral tissues they supplied. Marked or prolonged compression was hypothesized to induce loss of function. More moderate compression was hypothesized to lead to increased neural activity and increased pain, paresthesias, and hypertonic muscles.^{2,3,27-38}

The initial model of direct bony compression of NRs has produced considerable skepticism outside the profession and less than universal endorsement within the profession.^{35,38,172} In 1973, Crelin¹⁷² challenged the anatomic plausibility of subluxation-induced NR compression. He conducted cadaveric lumbar dissections, measuring the lateral borders of the IVF, and concluded that the bony borders of the lateral IVF provided for a minimum of 4 mm of space around each exiting NR. In addition, the NRs gain a dural covering at their point of entry to the IVF, further reducing their vulnerability to compression.^{173,174} He concluded that in the absence of degenerative joint or disc disease, it was unlikely that joint subluxation could produce enough narrowing of the IVF to produce direct anatomic compression of spinal NRs.

In 1994, Giles¹⁷⁵ revisited Crelin's criticism of the chiropractic model of subluxation-induced NR compression. Lumbar cadaveric dissections were again performed, but this time they included dissections at the level of the interpedicular zone, not just at the lateral borders of the IVF as Crelin had performed. Measurements made at the interpedicular zone demonstrate an average of 0.4 to 0.8 mm of space around each NR and the

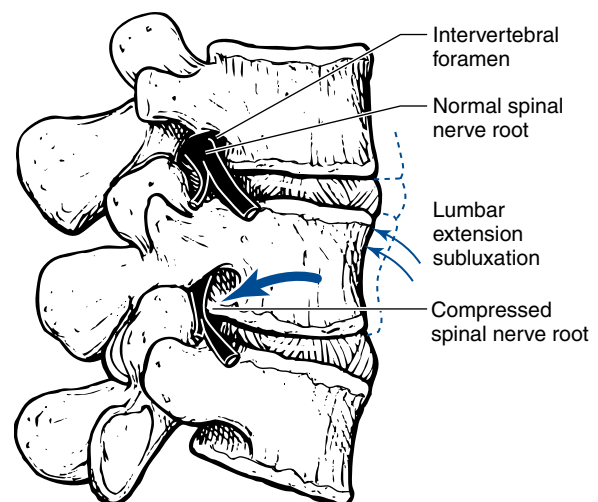


Figure 3-6 Diagram illustrating lumbar extension subluxation and theory of subluxation-induced compression of spinal nerve roots as they exit the intervertebral foramen.

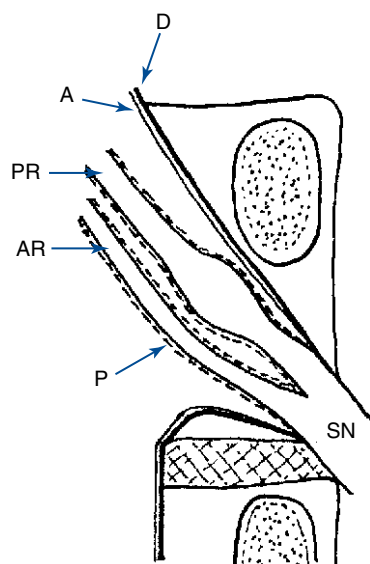


Figure 3-7 Diagram showing the interpedicular zone in a lumbar motion segment. Contained within the zone are dura mater (*D*), arachnoid mater (*A*), the anterior root (*AR*), pia mater (*P*), the posterior root (*PR*), ganglion, and the spinal nerve (*SN*). Note the proximity of the neural structures to the cephalad pedicle. (Modified from Giles LGF: *J Manipulative Physiol Ther* 17:4, 1994.)

dorsal root (DR) ganglion (Figure 3-7). These margins are only a small percentage (10% to 20%) of the space originally described by Crelin and theoretically small enough to be affected by joint dysfunction and subluxation. Moreover, his methodology and conclusions did not account for structural variants such as the transforaminal ligaments or the presence of functional alterations such as edema.¹⁷⁶

The spinal nerve rootlets at this level lack the epineural covering of the NRs as they exit the IVF and are more susceptible to pressure, inflammation, and ischemia. The DR ganglia (DRG), which lie within this space, are especially susceptible to compressive forces,¹⁷⁷ and chronically injured dorsal NRs respond more vigorously to mechanical deformation.¹⁷⁸

Furthermore, it is not necessary for spinal NRs to be directly compressed by bony structures to develop pathologic dysfunction. There are other structures within the IVF (e.g., arteries, veins, recurrent meningeal nerve, lymphatics, fat, areolar connective tissue) that occupy space, making it possible that other kinds of mechanical stresses may affect the nerve tissue.²⁵ Sustained misalignment or inflammation of the spinal motion segment may stretch or compress the local vascular structures, leading to disruption of neural blood supply and neuroischemia.^{35,179} It has also been demonstrated that mechanical pressures and tensions applied to the spinal segments may create myriad subclinical neurophysiologic alterations ranging from changes in intraneural protein composition to altered nerve conduction characteristics.¹⁸⁰⁻¹⁸³

The density of sodium ion channels in the soma and initial segment of DRG cells is relatively high, suggesting these regions may be unusually excitable.¹⁸⁴ These properties may render neural tissue within the IVF vulnerable to effects of mechanical compression and the chemical environment produced by changes

in the IVD or facet joints.¹⁸⁵ Substantial evidence demonstrates that the DRs and DRG are more susceptible to the effects of mechanical compression than are the axons of peripheral nerves because impaired or altered function is produced at substantially lower pressures.^{185,186} Whether spinal manipulation can alter neural function by mechanically changing compressional pressures or reducing the concentration of metabolites in the IVF is unknown.¹⁸⁷

Compression studies investigating how herniated IVDs affect NR function have been performed. The mechanism by which a herniated disc could directly compress the DRs or DRG is well understood and straightforward.¹⁸⁷ However, a herniated IVD could also affect NR function through indirect effects mediated by the release of neuroactive chemicals.¹⁸⁸ This explains the common observation that in the absence of compression, herniated discs can produce neurologic findings. Recent studies demonstrate that the application of nucleus pulposus to a lumbar NR causes mechanical hyperalgesia in the distal limb and causes swelling in and decreased blood flow to the DRG.^{189,190} In addition, phospholipase A₂, an inflammatory mediator associated with disc herniation^{188,191} is neurotoxic in high doses to afferent nerves.¹⁹² In moderate doses it increases mechanical sensitivity of the DRs, producing long-lasting discharge, and it increases the discharge of previously silent DRG cells.^{192,193}

The intervertebral canal and each of its motion segments have a vascular supply composed of spinal arteries and veins. The spinal arteries provide oxygenated blood to the spinal cord and dorsal and ventral NRs. Blood vessels are softer and more susceptible to the effects of stretch and compression than are the nerves they supply, making localized neuroischemia, without direct compression, a possible result of spinal joint dysfunction.

If joint malposition does contribute to dysfunction of the spinal NRs, it is more likely to occur by narrowing the more vulnerable interpedicular zone. Furthermore, joint subluxation has a greater potential to affect NR function if it is secondary to other disorders that have already led to narrowing of the lateral recess, such as disc herniation or other space-occupying lesions, degenerative disc and joint disease, and joint instability.

Although recent anatomic investigations have provided a plausible mechanism by which joint subluxation may contribute to NR dysfunction, it still remains a tenuous theory. The clinical literature has established that encroachment of neural structures within the IVF may produce NR dysfunction, but it has not established whether spinal subluxations alone (i.e., without other neurocompressive sources) can cause encroachment and altered neural activity. Furthermore, it must be appreciated that a subluxation occurs within the normal ROM for the segment. The IVFs of each segment change size and shape with movements. Extension combined with rotation and lateral flexion to the same side maximally decreases the opening of the IVF, yet no NR compression occurs. Therefore, something in addition to a sustained malposition has to occur to produce clinical signs of NR compression, such as inflammation, disc deformation, or vascular changes.

In conclusion, it appears that the early "foot-on-the-hose" model of joint subluxation and NR compression is not biologically plausible. Joint subluxations alone are extremely unlikely to "pinch" the spinal NRs at the margins of the IVF.

Theory of Altered Somatic and Visceral Reflexes

Somatosomatic and Somatovisceral Reflexes. In the absence of evidence to confirm the NR compression hypothesis, the profession has assembled an alternative model of subluxation syndrome–induced neurologic alterations (impulse-based model). The impulse-based paradigm of neurodysfunction has been developed from the work of Homewood and Korr.^{32-35,37-39,194}

A somatoautonomic reflex is elicited when stimulation of somatic tissue (the musculoskeletal system and the dermis of the skin) is manifested as an alteration in autonomic nervous system function. A spinal visceral reflex is a type of somatoautonomic reflex in which stimulation of the spinal column alters visceral function.¹⁹⁵

This hypothesis envisions vertebral joint dysfunctions as lesions capable of inducing chronically altered nociceptive and proprioceptive input. This persistent afferent input, driven by mechanical alteration, pain, and potential local inflammation, triggers a segmental cord response, which in turn induces the development of pathologic somatosomatic or somatovisceral reflexes. The persistent altered afferent input is then theorized to produce sensitization of local spinal neuron pools and the establishment of abnormal somatosomatic or somatovisceral reflexes. The reflexes, once established, become the potential driving source of altered somatic or visceral function. If these reflexes persist, they are hypothesized to induce altered function in segmentally supplied somatic or visceral structures.^{32-35,37-39,56,196-198}

Thus joint subluxation/dysfunction syndrome (JSDS) may initiate secondary dysfunction in tissues with shared segmental innervation. Indeed, clinical investigations have demonstrated that altered muscle tone, deep tendon reflexes, and altered sympathetic activity may accompany joint derangement and dysfunction.¹⁹⁹⁻²⁰³ Many of these findings had been previously assumed to be associated with NR dysfunction only. The segmental muscle hypertonicity that may be associated with joint dysfunction illustrates a clinical example of a somatosomatic reflex; cervical disequilibrium secondary to cervical joint dysfunction illustrates an example of a somatovisceral disorder (Figure 3-8).²⁰¹

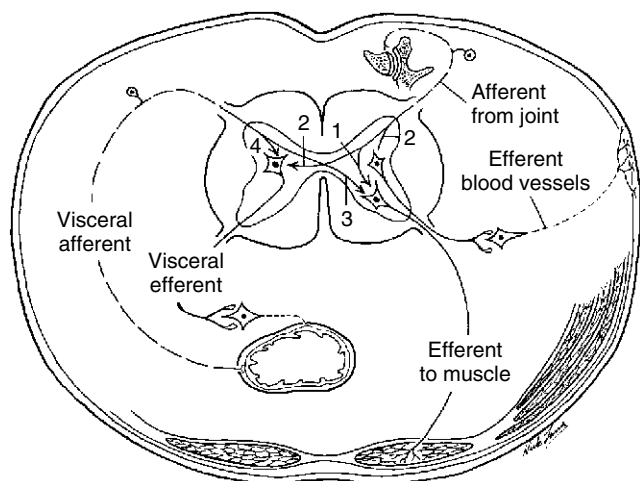


Figure 3-8 Afferent pathways from the somatic and visceral structures can produce somatosomatic (1), somatovisceral (2), viscerosomatic (3), and viscerovisceral reflex phenomena (4).

The proposed joint subluxation- or dysfunction-induced neurologic phenomena may be clinically manifested by the presence of referred pain, hypertonicity, hyperesthesia, or altered sympathetic activity, such as altered temperature regulation and skin conductance.¹⁹⁹⁻²⁰³ Manual therapy, including soft tissue techniques and other forms of adjustive therapy, would have the potential for arresting both the local and distant somatic and visceral effects by terminating the altered neurogenic reflexes that are associated with somatic or joint dysfunction.

Viscerosomatic (Autonomic) Reflexes. Persistent pathologic conditions in visceral structures also have the theoretic potential to induce reflexive dysfunction in other somatic or visceral structures. Visceral disease or dysfunction may activate the autonomic nervous system through connections with the lateral horn cells in the cord to produce vasomotor, trophic, visceral, or metabolic changes (see Figure 3-8). Numerous conditions have been linked to hyperactivity of the sympathetic nervous system; these include various types of cardiovascular, gastrointestinal, and genitourinary disorders, and certain musculoskeletal disorders such as complex regional pain syndrome.

It has been suggested that the body wall manifestations of visceral disease are an integral part of the disease process, rather than just physical signs and symptoms,²⁰⁴ although the definitive causal factors and the characteristic response of the individual are still unknown. Early signs of most disease states are manifested as symptoms and signs that are part of a common reaction pattern to injury or stress. Pain in the somatic tissues is a frequent presenting symptom in acute conditions related to visceral dysfunction. Palpatory cues of transient muscle hypertonicity and irritation or subcutaneous edema may be accompaniments of ill-defined subclinical states.²⁰⁵ Moreover, subtle changes in tissue texture, joint position, and joint mobility identified by discerning palpatory skills may at times be latent manifestations of the somatic component of visceral disease.

In a study performed on cardiac patients in an intensive care unit,²⁰⁶ autonomic spinal reference changes were noted for the involved viscera (Box 3-2). In studies by Kelso,²⁰⁷ it was noted that as the visceral condition progresses, the somatic stress pattern subsides, and a typical visceral reflex pattern is seen. Therefore, the chronic phase of reflex activity is characterized by trophic changes in the skin and subcutaneous tissues, as well as local muscle contraction. This may result in a joint misalignment and decreased segmental mobility. However, it is not known whether the continuation of reflex somatic dysfunction is related to the initial effect of the visceral disease or whether it is a result of long-term segmental facilitation.

BOX 3-2

Autonomic Changes in Soft Tissues Identified in Patients with Viscera Problems

Vasomotor reaction—increase in skin temperature
Sudomotor reaction—increase in skin moisture
Increase in muscle tone and contraction
Skin texture changes—thickening
Increased subcutaneous fluid

In a blind study of 25 patients, Beal²⁰⁸ was able to differentiate patients with cardiac disease from those with gastrointestinal disease with a reported accuracy of 76% using a compression test to examine for soft tissue texture changes and resistance to segmental motion. Similarly, Beal and Dvorak²⁰⁹ examined 50 patients in a physician-blind format and were able to identify characteristics specific for patients with cardiovascular, pulmonary, gastrointestinal, or musculoskeletal diseases.

In summation, it is apparent that spinal dysfunction has the potential to both produce and be the product of visceral or somatic dysfunction or disease. The literature supports the existence of somatovisceral and viscerosomatic reflexes,²¹⁰⁻²¹² but there is little or no evidence to support the notion that the VSC can cause prolonged aberrant discharge of these reflexes. Also unsupported in the literature is the notion that the prolonged activation of these reflexes can induce pathologic change and visceral disease. Nor does the literature support the position that spinal manipulative therapy can alter the prolonged reflex discharge to an extent that induces a reversal of the pathologic degeneration of the affected tissues.^{213,214} Although there have been investigations using animal models on the effects of mechanical stimulation of the spine on blood pressure, heart rate, and renal sympathetic nerve activity,²¹⁵⁻²²⁰ unfortunately there is almost no physiologic research concerning responses in humans to either spinal pain or innocuous mechanical stimulation. Furthermore, most of the data obtained were elicited with noxious stimulation. There is still little support for the contention that painless spinal dysfunction can affect organ function, which is not surprising, considering that all the basic physiologic work cited was performed on anesthetized animals. The evidence does suggest that muscle spindles in cervical paraspinal muscles may in fact be capable of eliciting somatoautonomic reflexes.²²¹

The complex interrelationship of the NMS system demands that chiropractors and other manual therapists be open to the numerous potential sources of their patients' complaints. Spinal pain and dysfunction may be secondary to a disorder that is not amenable to manual therapy. In circumstances in which JSDs are secondary to active visceral or somatic disease, manipulative treatment alone would be inappropriate.

INFLAMMATORY AND VASCULAR COMPONENTS

Joint injury, chronic mechanical joint derangement, or joint immobilization may initiate the inflammatory and vascular components of the VSC.^{34,54,56,75} These components include vascular congestion, ischemia, and inflammation.

Vascular Congestion

It is unclear at this time what role, if any, spinal segmental function or dysfunction plays in local vascular congestion. Speculation has centered on the potential for motion segment dysfunction or associated inflammation to impede blood flow through segmental venous structures. Venous pressure is very low and depends on gravity in the spinal veins, making them quite susceptible to compression and venous congestion. Lantz suggests that immobilization may lead to localized venous stasis, creating a negative pressure and lack of proper venous drainage that may lead to inflammation.²²²

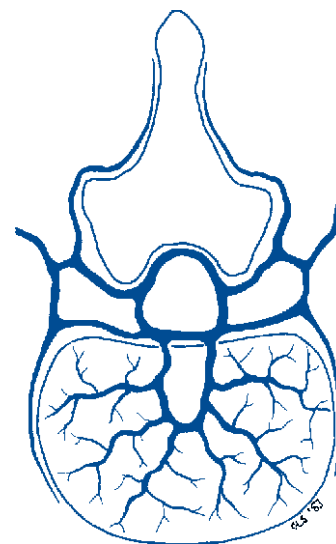


Figure 3-9 The internal and external systems for segmental venous drainage. (From Kirkaldy-Willis WH, Bernard Jr TN: *Managing low back pain*, ed 4, New York, 1999, Churchill Livingstone.)

A segmental vein drains each motion segment and related spinal canal. Each segmental vein receives venous blood from an extensive internal venous plexus (Batson), which in turn receives blood from a basivertebral vein that drains each individual vertebra (Figure 3-9). The intraspinal venous plexus is located within the epidural space and basically consists of two paired columns that are united via a transverse communication vein. A lack of venous drainage in these structures is speculated to lead to increased capillary pressure, diminished arterial blood flow, and the production of local ischemia, inflammation, and potential associated joint stiffness.²²³ In addition, a tear in a fragile vein may occur, possibly as a result of repetitive increases in intra-abdominal pressure, producing a hematoma that serves as a space-occupying lesion. Because the veins course vertically at the posterolateral aspect of each disc bilaterally, they can produce the same clinical picture as a disc herniation as they expand. The only differentiating test is magnetic resonance imaging (MRI), using T₂-weighted images to visualize more water content in the blood-filled hematoma.

Inflammatory Reactions

Inflammatory reactions are largely mediated by the vascular system and accompanied by cellular and humoral components that act as an intrinsic source of pain and vasodilation.²²⁴ The inflammatory reaction initiated by musculoskeletal injury or dysfunction is identical to that initiated by a foreign object or infection. Although it is a normal protective response, it may accentuate the pain response, slow the recovery time, and perpetuate joint dysfunction. Pain accompanying inflammation may initiate local reflex muscle contraction, which, over time, may lead to local ischemia and potentially more pain and muscle splinting. The result, as described previously, is a self-perpetuating cycle of pain and continued muscle spasm.* If the muscle contraction persists, it may eventually develop into a muscle contracture as the

*References 34, 51, 54, 56, 76, 78, 116-119.

myofascial structures become shortened and infiltrated with fibrotic tissue.^{170,225} The resulting soft tissue derangements and contractions that develop must be dealt with therapeutically, or they will serve as a source of continued pain and recurring joint subluxation and dysfunction.

With persistent inflammation and pain, plastic changes may occur in the peripheral and central nervous systems that lower pain thresholds, giving rise to allodynia (pain in response to a normally innocuous stimulus), hyperalgesia (heightened pain intensity in response to a normally painful stimulus), and sensitization of the central nervous system. Afferent nerve fibers that are quiescent in normal joints may become active and start to send nociceptive information to the central nervous system, which can also become sensitized to perceive what is typically nonpainful stimuli as painful.²²⁶⁻²²⁸

In addition, chronic joint inflammation may lead to synovial tissue hyperplasia and thickening as a result of persistent irritation and secretion of synovial fluid.^{229,230} Synovial tags may develop as a hyperplastic reaction to chronic inflammation, and they in turn may become further impediments to joint movement.^{225,231} Eventually, fibrous invasion of the synovial connective tissue layer may induce an attendant loss of vascularity and subsequent loss of synovial fluid secretion.¹⁴⁸

Some degree of joint or soft tissue inflammation should be suspected when the patient's pain is constant. Clinical signs include muscle splinting, soft tissue swelling, and temperature alteration. Inflammation associated with spinal joint injuries or dysfunction is unlikely to produce palpable swelling at the surface. Some have suggested, however, that joint dysfunction may be associated with a local sympathetic reflex alteration capable of inducing a slight boggy feeling in overlying segmental tissues.

JOINT SUBLUXATION/DYSFUNCTION SYNDROME

A Joint Subluxation/Dysfunction Syndrome (JSDS) diagnosis is a clinical diagnosis defined by an aggregate of signs and symptoms that are assumed to identify dysfunction of spinal, pelvic, or peripheral joints.^{232,233} It is a functional (biomechanical) diagnosis, not a structural (pathoanatomic) diagnosis. When applied to the spine, it implies that the spinal motion segments and their associated soft tissues are the source of the patient's symptoms. Unlike traditional structural diagnoses like disc derangement, sprain or strain, and spinal stenosis, the diagnosis of JSDS does not attempt to identify specific tissue pain generators within the spinal motion segment. This diagnosis typically includes local axial spine pain reproduced or accentuated by static or dynamic palpation. It may be associated with sclerogenic referred pain into the proximal extremity. The diagnosis of JSDS usually denotes to chiropractic physicians that the condition may be amenable to manual therapy; high velocity–low amplitude (HVLA) adjustive therapy is most commonly applied treatment.

Joint dysfunction may occur in isolation, but is commonly associated with other identifiable functional and pathoanatomic disorders and conditions. The individual chiropractor and the profession as a whole should make every attempt to incorporate these diagnoses in assessment and patient management.

SPINAL LISTINGS

As the chiropractic profession has evolved, it has developed various abbreviated descriptions for designating abnormal joint position or movement. The result is a profession laden with redundant nomenclatures (listing systems) that describe spinal subluxations and fixations. As new descriptive terms are introduced, old ones are not replaced. It is not uncommon for each technique to have its own unique listing system. Unique listing methods may be efficient for those performing the associated technique, but many are not commonly understood.

As part of the process to include chiropractic in Medicare, there was an attempt to standardize listing systems at the 1977 American Chiropractic Association (ACA) conference in Houston. Although the parties did succeed in developing a common nomenclature for Medicare claims based on standard kinesiological terms, it unfortunately did not form a basis for larger professional consensus. There is still significant variation among chiropractors and on national board examinations as to the preferred listing systems.

To their students' continual frustration, colleges are left in a position of teaching repetitive and often contradictory methods of describing joint malpositions and fixations. Presently, the common systems used to describe abnormal position are Medicare, Palmer-Gonstead, and National–Diversified systems. In an attempt to reduce the confusion and redundancy, this book emphasizes standard kinesiological terms and the Medicare listing system. When deviations in position are described, the term *malposition* is used, and when limitations to movement are described, the term *restriction* is used.

Spinal joint listing systems should be incorporated only in conjunction with a diagnosis of spinal JSDS. They describe characteristics of subluxation and dysfunction syndromes, but they are not expected to be stand-alone diagnostic terms. Spinal listings should be viewed only as a short-hand method of recording which joint changes were subjected to manipulation (Figure 3-10).

All motion segment malpositions are described with the position of the upper vertebra compared with the lower vertebra. For example, a *flexion malposition* describes a vertebra that has deviated into a position of flexion relative to the vertebra below, and a *flexion restriction* describes a limitation or loss of joint flexion between the two vertebrae.

Trunk and neck movements are described in kinesiological terms. They are based on vertebral body movement, not spinous process movement. Left rotation of the trunk is defined by *left posterior vertebral body rotation*, not by *right rotation of the spinous process*.

CLINICAL EVALUATION OF JOINT SUBLUXATION/DYSFUNCTION SYNDROME

Before adjustive treatments are applied, the chiropractor must evaluate the patient's complaint and determine if the patient is suffering from a condition (manipulable lesion) that is amenable to chiropractic care. As mentioned previously, therapeutic decisions on where and how to apply adjustive therapy are based primarily on the evaluation of the NMS system and a determination that injury, derangement, or disease has led to altered function.

Although the diagnosis of joint dysfunction identifies a painful clinical syndrome that may respond to manual therapy, the nature of the dysfunction must be evaluated before therapy is administered. The mere presence of joint subluxation or dysfunction does not determine the need for adjustive therapy. Joint dysfunction

may result from diseases or disorders that contraindicate treatment or result from disorders that do not respond to adjustive treatments. The ability to thoroughly evaluate and triage disorders of the NMS system and distinguish those conditions that are appropriate for chiropractic care is critical. Differentiating

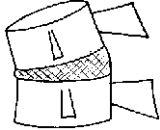
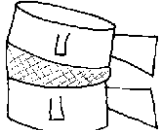
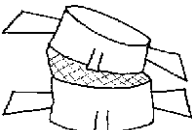
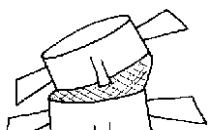
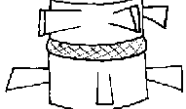

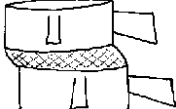
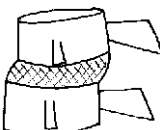
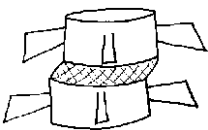
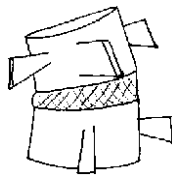
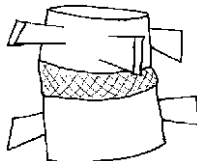
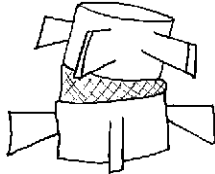
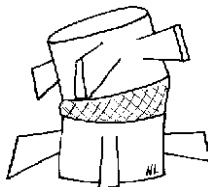
	Medicare (Vertebral body reference)	Palmer-Gonstead (Spinous process reference)	National-Diversified (Vertebral body reference)
	Flexion malposition	None	Anterior inferior
	Extension malposition	Posterior	Posterior inferior
	Right lateral flexion malposition	None	Right inferior
	Left lateral flexion malposition	None	Left inferior
	Left rotational malposition	Posterior spinous right	Left posterior
	Right rotational malposition	Posterior spinous left	Right posterior
	Anterolisthesis	None	Anterior
	Retrolisthesis	Posterior	Posterior
	Right lateral listhesis	None	Right lateral

Figure 3-10 Comparative chart of static listing systems. (Modified from ACA Council on Technic: *J Am Chiropr Assoc* 25[10]:46, 1988.)

	Medicare (Vertebral body reference)	Palmer-Gonstead (Spinous process reference)	National-Diversified (Vertebral body reference)
	Left rotational malposition Left lateral flexion malposition	Posterior right Superior spinous	Left posterior inferior
	Left rotational malposition Right lateral flexion malposition	Posterior right Inferior spinous	Left posterior superior
	Right rotational malposition Right lateral flexion malposition	Posterior left Superior spinous	Right posterior inferior
	Right rotational malposition Left lateral flexion malposition	Posterior left Inferior spinous	Right posterior superior

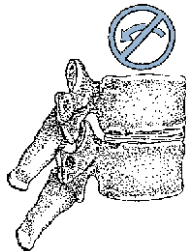
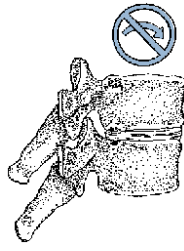

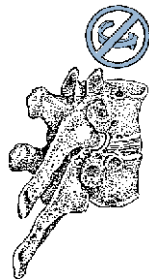

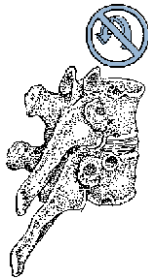
Dynamic (motion) listing: designation of abnormal joint movement Restriction: direction of limited movement in subluxated dysfunctional joints		Dynamic listing nomenclature 1. Flexion restriction 2. Extension restriction 3. Lateral flexion restriction (right or left) 4. Rotational restriction (right or left)			
					
Extension restriction	Flexion restriction	Right rotational restriction	Left rotational restriction	Right lateral flexion restriction	Left lateral flexion restriction

Figure 3-10—Cont'd

mechanical from nonmechanical conditions, assessing the source of the presenting complaint, and understanding the potential pathomechanics and pathophysiology of the disorders being considered for chiropractic care are crucial elements for successful treatment. Therefore, before instituting treatment, the clinician must perform a thorough case history, physical examination,

and any other appropriate imaging or laboratory procedures to rule out any disorders that contraindicate adjustive treatments. The evaluation should assess whether the dysfunction is associated with joint hypermobility or hypomobility and the site, side, and potential directions of immobility, aberrant movement, or hypermobility.

EXAMINATION PROCEDURES AND DIAGNOSTIC CRITERIA

Uncomplicated JSDS is a clinical diagnosis identified by a collection of presenting symptoms and physical findings. It is not independently detectable by laboratory procedures, and a single gold standard for detecting primary joint subluxation or dysfunction does not currently exist. Often it is suspected after the possibilities of other conditions with a similar presentation have been eliminated. A favorable patient response to manipulation or mobilization (decreased pain or improved function) and reduction or normalization of abnormal physical findings indicates the original working diagnosis and application of manual therapy was a clinically sensible and effective approach.

History

JSDS is commonly symptomatic but the diagnosis does not depend on the patient being symptomatic. However, in asymptomatic JSDS, one would expect the physical findings supporting the diagnosis to be pronounced. In the spine, patients with JSDS commonly complain of pain located in the midline to paraspinal region with or without pain referral into the extremities. Although the somatic referred pain does not usually extend below the knee or upper arm, pain may radiate as far as the foot or hand. However, the location, quality, and referral patterns of the patient's pain complaints are not unique to this diagnosis. These symptoms overlap with a number of other axial spine complaints and do not differentiate JSDS from other mechanical spine disorders. The patient's history is also crucial in identifying possible red flags and differentiating nonspecific mechanical back pain from nonmusculoskeletal or nonmechanical NMS disorders. It is also helpful in implicating neurologic involvement and identifying mechanisms of possible injury and load sensitivities pertinent to JSDS.

Physical Examination

With the exception of radiographic evaluation, the majority of the commonly used examination procedures devoted to assessing joint structural and functional integrity are physical examination procedures. They include standard orthopedic, neurologic, and physical examination procedures and a wide array of unique "system technique" diagnostic procedures. Observation and palpation are the most commonly used physical examination procedures and include postural and gait evaluation, soft tissue and bony palpation, global ROM, and segmental ROM testing or what is also referred to as *passive intervertebral motion tests*.^{55,74,234-239} Manual palpation is the primary evaluative tool, necessitating many hours of practice and concentration to develop adequate skill. The application of joint manipulation relies heavily on the clinician's ability to locate and identify landmarks, painful musculoskeletal tissue, painful joint movements, contracted muscles, restrictions of motion, and hard EP resistance.²⁴⁰

Specialized laboratory procedures, such as thermography and electromyography (EMG), are presently not in common clinical use for detection of JSDS. Further research is necessary before their role in clinical practice can be fully ascertained. The classic

physical signs indicative of JSDS are provocation of pain, abnormalities in alignment, abnormal resistance to joint movement, and altered tissue texture. Bergmann,²⁴¹ modifying the acronym *PARTS* from Bourdillon and Day,²⁴² identifies the five diagnostic categories commonly applied by chiropractors for the identification of joint dysfunction: *p*ain and tenderness; *a*symmetry; *R*OM abnormality; *t*one, texture, and temperature abnormality; and *s*pecial tests. Various investigators have suggested that detection of the spinal manipulative lesion should not rely on a single assessment method.

During spinal evaluation, the physical examination should focus on identifying the source of the patient's complaints and differentiating segmental from nonsegmental sources. The examination findings supportive of a spinal JSDS diagnosis can be divided into primary and secondary categories and are listed in Box 3-3. It is recommended that the physical assessment of JSDS focus on reproducing the patient's joint pain with palpation and joint provocation and challenge procedures. Although a number of manual examination findings have historically purported to confirm this disorder, bony and paraspinal soft tissue tenderness or pain reproduced with JP or EP are the most reliable and potentially valid diagnostic tools.²⁴³⁻²⁴⁶

It has been suggested that tests should be considered in groupings leading to a multidimensional approach.²⁴⁷⁻²⁵¹ A 2006 literature review by Stochkendahl and associates concluded that a "global assessment" (i.e., segmental static and motion tenderness, palpatory altered joint motion, and palpable tissue changes) demonstrates reproducible intraexaminer reliability (0.44 kappa). However, there was not enough evidence to calculate pooled results for interexaminer reliability. The significance of a multidimensional approach is further illustrated by the Health Care Financing Administration requirement that the manipulable lesion be supported by physical examination.²⁵² From the initial coverage of chiropractic care in the Medicare program in 1974–1999, Medicare required x-rays to demonstrate subluxation of the spine and therefore the clinical necessity for chiropractic care. Beginning in 2000, Medicare allowed physical examination findings (the pain and tenderness, asymmetry or misalignment, ROM abnormality, and tissue or tone changes [PARTs] multidimensional approach) for the demonstration of subluxation in place of x-rays: To demonstrate a subluxation based on physical examination, two of the four criteria mentioned under "physical examination" are required, one of which must be asymmetry/misalignment or ROM abnormality.²⁵²

Pain and Tenderness

The perception of pain and tenderness is evaluated in terms of *location*, *quality*, and *intensity*. Most primary musculoskeletal disorders manifest by a painful response. The patient's description of the pain and its location is obtained. Furthermore, the location and intensity of tenderness produced by palpation of osseous and soft tissue are noted. Pain and tenderness findings are identified through observation, percussion, palpation, and provocative orthopedic testing. The patient's description and location of pain is obtained verbally, physically, or by a pain drawing. The location and intensity of tenderness produced by palpation of osseous and soft tissues is identified and noted. Changes in pain intensity can be objectified

BOX 3-3 Physical Examination Findings Supportive of Spinal Joint Subluxation/Dysfunction Syndrome Diagnosis**PRIMARY FINDINGS****Palpable segmental bony or soft tissue tenderness/dysesthesia****Painful or altered segmental mobility testing**

Joint motion is traditionally assessed in its open packed position with joint play (JP) procedures, through its segmental range of motion, and with end play (EP) at the end range of motion. All three components of joint motion are evaluated for quantity, quality, and pain response. Clinical studies indicate that JP and EP are more reliable for pain response than range of motion assessment.

Palpable alterations in paraspinal tissue texture or tone

Tissues texture changes are represented by a loss of paraspinal tissue symmetry at the segmental level or between adjacent segments. These changes are characterized by palpable alterations in muscle resting tone (hypo or hypertonicity or spasm) and textural changes characterized by a palpable sense of tissue induration or fibrosis often described as a hardening or thickening of tissue.

SECONDARY FINDINGS**Palpable malposition (e.g., spinous deviation)**

Note: Because of individual variation and the high prevalence of asymmetry many manual therapists do not consider this an indicator of joint dysfunction

Repetitive loading in direction of EP restriction may improve symptoms

Alterations in sectional or global range of motion:

Decreased and painful global active range of motion and various positive pain-provoking orthopedic tests are not primary features of a joint dysfunction diagnosis because of their commonality with multiple painful musculoskeletal disorders. Note: active range of motion may be normal with joint dysfunction syndrome because of the spine's ability to compensate at other segmental levels.

using visual analog scales, algometers, and pain questionnaires. The production of palpatory pain over osseous and soft tissues has been found to have good levels of interexaminer and intraexaminer reliability.^{244,246,253-256} The validity of motion palpation or pain reproduction with palpation to identify painful spinal joints or direct effective treatment is limited. The results have been mixed but encouraging in a few studies.²⁵⁷⁻²⁶² Although assessment of segmental motion has generally scored poorly in terms of reliability, in several studies lumbar P-A mobility assessment did succeed in achieving acceptable predictor scores (likelihood ratios) for classifying and directing various types of therapies (e.g., manual therapy vs exercise).^{263,264} In these studies P-A mobility testing was only one of several presentations or physical findings used to categorize patients, and P-A mobility testing may not be a materially contributing factor in predicting outcome.

Asymmetry

Asymmetric qualities are noted on a sectional or segmental level. This includes observation of posture and gait, as well as palpation for misalignment of vertebral segments and extremity joint structures. Asymmetry is identified through observation (posture and gait analysis), static palpation for misalignment of vertebral segments, and evaluation of static plain-film radiographs for malposition of vertebral segments. The complex structure of the human body, and especially its frame, is never completely or perfectly symmetric. Therefore, focal changes in symmetry may or may not be clinically significant. They must be judged by the degree of deviation and placed within the context of the overall clinical presentation and examination.

Range-of-Motion Abnormality

Changes in active, passive, and accessory joint motions on a segmental and sectional basis are noted. These changes may be reflected

by increased, decreased, or aberrant motion. It is thought that a decrease in motion is a common component of joint dysfunction. Global ROM changes are measured with inclinometers or goniometers. Segmental ROM abnormalities are identified through the procedures of motion palpation and stress x-ray examination.

Tone, Texture, and Temperature Abnormality

Changes in the characteristics of contiguous and associated soft tissues, including skin, fascia, muscle, and ligaments, are noted. Tissue tone, texture, and temperature (vasomotor skin response) changes are identified through observation, palpation, instrumentation, and tests for length and strength.

Special Tests

The category of special tests includes two major subsets. One group incorporates testing procedures that are specific to chiropractic technique systems, such as specific leg length tests (e.g., Derfield) and muscle tests (e.g., arm fossa test). The other group encompasses laboratory procedures such as x-ray examination, EMG, and thermography.

System technique assessment procedures are typically manual examination procedures. They are commonly the products of individual innovation. They are distinguished from other physical examination procedures by their unique use and association with brand-name techniques. Most of these procedures have not been subjected to testing, and their reliability and validity have not been evaluated.

Many of the laboratory procedures that are promoted as potential detectors of JSDS have substantiated value in evaluating disorders of the NMS system. However, most have not been subjected to in-depth evaluation relative to their ability to detect segmental joint dysfunction. In addition, visceral relationships are considered (e.g., evaluation of the upper thoracic spine in cases of asthma) in localizing the spinal segment or segments that might be dysfunctional.

CLINICAL USEFULNESS OF JOINT ASSESSMENT PROCEDURES

Although the effectiveness and appropriateness of chiropractic adjustive therapy for treating mechanical neck and back pain has been demonstrated (see Chapter 4), the clinical value and usefulness of many of the diagnostic procedures used to detect JSDS have not been thoroughly or properly evaluated.^{236,244,253,256,265-272} The clinical usefulness of a diagnostic procedure is measured by its ability to provide accurate information that leads to appropriate and effective management of health care problems. These attributes can be evaluated by assessing a given procedure's reliability, validity, responsiveness, and utility.

Chiropractic is not alone in its need to advance the critical appraisal of its diagnostic and therapeutic procedures.²⁷³⁻²⁷⁷ Other health disciplines also suffer from significant variations in the use of diagnostic tests, and many lack experimental evaluation and confirmation.²⁷⁸ The prudent practitioner should remain skeptical of unsubstantiated and biologically unfeasible claims, but supportive of and open-minded toward investigation of untested procedures. Untested procedures are not necessarily invalid procedures. It is just as wrong to reject a therapeutic procedure because it is untested as it is to accept the same procedure in the absence of supporting evidence.

It is likely, however, that examination procedures that depend on human evaluation will always carry the potential for some error. Furthermore, quantifying a manual art is difficult because of the lack of a standard for comparison.²⁴⁰ The chiropractic doctor must be aware of these limits, yet constructively use the physical evaluation to help gain the patient's confidence and compliance. Physical examination procedures placed within proper clinical perspective still provide a significant cost-effective contribution to the formation of a clinical impression. Within this context, it is important not to rely excessively on any one procedure, but rather to use a combination of diagnostic procedures and allow the weight of evidence to build a clinical impression of the patient's problem.

Reliability

"Reliability is the reproducibility or consistency of measurement or diagnosis. It is the extent to which a test can produce the same result on repeated evaluation of an unchanged characteristic."²⁷¹ Reliability estimates the contribution a given test makes to the clinical decision-making process beyond what would be expected by chance. Reliability measures include evaluation for interexaminer and intraexaminer consistency, and test-retest evaluation to determine if measurements are reproducible and consistent over time. Fortunately, the profession has witnessed a significantly increased interest in evaluating its diagnostic procedures. It is now possible to make some generalizations about the reliability of common chiropractic diagnostic procedures.

In 1991, Haas²⁶⁵ reviewed the literature on the reliability of chiropractic joint assessment procedures and concluded that many of the studies had questionable design and statistical analyses. These same conclusions have been echoed repeatedly since then.^{243,244,256,279-284} In addition, most of the studies evaluated the reliability of only one procedure at a time. This leaves the question of combined reliability in need of further evaluation; the

procedures may demonstrate higher reliability when used in conjunction with each other.^{244,245,253,285,286} Furthermore, combining different assessment methods in a multitest regimen more closely parallels actual clinical practice.^{262,286} There are a number of retrievable studies investigating the utility of multidimensional evaluation procedures.^{256,286-291} Only one primary study showed any reliability approaching acceptable levels, and that varied from marginal to good.²⁵⁶ A systematic review conducted in 2006 implied that multitest regimens did appear to demonstrate acceptable intraexaminer reliability.²⁴⁴ Based on the available research, it has to be concluded that there is insufficient evidence to determine the level of interexaminer reliability of a multidimensional manual examination procedure for detecting manipulable lesions. Certainly further research is warranted to better investigate a multidimensional approach.

Validity

Reliability testing is critical, but it is only one element in the process of assessing the clinical value of diagnostic procedures and it must not be confused with validity. The accuracy, or validity, of a procedure, or the degree to which the test actually evaluates what is intended, is of paramount importance.²⁷¹ Valid health care procedures are those that are useful in helping health care providers make accurate and effective health care decisions.

Although reliability testing for chiropractic joint assessment procedures has expanded significantly in the last several decades, validity testing remains in its infancy. Most chiropractors and manual therapists accept the face validity of common joint assessment²⁹² procedures, but most procedures have not been subjected to rigorous experimental evaluation. Face validity is a measure of a diagnostic procedure's plausibility (biologic reasonableness) to evaluate a known phenomenon.

Experimental evaluation of diagnostic procedures is necessary to establish their true merit in accurately identifying a given disorder. Experimental evaluation of validity can be broken down into construct-based and criterion-based validity assessments. Construct validity attempts to measure the accuracy of a procedure when a reference standard is not available. Construct validity measures "the ability of a test to perform up to the standards predicted from a theoretical model or construct."²⁹³

Hass and colleagues²⁹³ illustrate an example of construct validity evaluation in their assessment of joint motion palpation. Motion palpation theory assumes that EP restriction is a palpable indication for thrust manipulation and that immediate postmanipulative restoration of motion should be palpable in some cases. Therefore, the construct validity of motion palpation for the assessment of EP and manipulable subluxation and dysfunction could be assessed by testing the examiner's ability to identify EP restrictions and discern if improvement in EP restriction occurs after thrust manipulation.

Criterion-based validity testing allows the evaluation of a diagnostic procedure as it compares with a known gold and coworkers standard procedure.²⁹⁴ The glucose tolerance test is an established criterion for substantiating blood glucose levels. This test could therefore be used as the standard for comparing new tests. There is no established gold standard test for identifying JSDSs.

Responsiveness

The responsiveness of a diagnostic procedure measures its ability to respond to changes in the condition or phenomenon it is assessing. For a testing procedure to be effective in this category, it must demonstrate the ability to change with the entity being evaluated. If a given testing procedure is responsive, it has the ability to reflect improvement or worsening in the condition or function it is measuring. Responsive tests are valuable in measuring the effects of treatment and therefore are effective outcome measures.

Utility

Test utility represents the practical usefulness of a diagnostic test. Clinical utility measures the health benefits provided by a given procedure. It represents the value the procedure has in directing effective patient care. A new diagnostic test demonstrates good clinical utility if it leads to fewer adverse reactions, improved patient care, improved patient outcome, or equal outcome at lower costs. A new radiographic procedure that provides the same information as a palpatory procedure has poor utility and no diagnostic value because it provides the same information at a greater cost and risk to the patient.

OUTCOME ASSESSMENT PROCEDURES

The limited understanding of the nature, cause, pathophysiologic condition, and diagnostic criteria for identifying JSDSs has stimulated a search for alternative and more valid outcome measures by which to measure the effectiveness of chiropractic care. Escalating health care costs and the need to document the appropriateness and effectiveness of care further illustrate the need for the profession to develop and use valid outcome measures.²⁹⁵ Instead of relying solely on procedures traditionally used to identify JSDS, chiropractors should also use procedures that measure the effect their treatment is having on the patient's symptoms and function. In this context, the name and nature of the disorder become less of a focus, and more attention is paid to how the patient is functioning and responding to treatment.^{55,296,297}

The disorders commonly treated by chiropractors are painful or have a significant effect on the patient's ability to function. Therefore, the degree of the patient's pain and his or her ability to perform physical maneuvers and activities of daily living are important outcome measures of the efficiency and effectiveness of chiropractic treatment. A number of the procedures presented can be used in this context. However, many are more useful in guiding decisions on where and how to adjust patients than they are as outcome measures. Each examination procedure presented includes a brief discussion on the procedure's clinical usefulness and appropriateness for use as outcome measures.

SYMPTOMS OF JOINT SUBLUXATION/ DYSFUNCTION SYNDROME

Pain is a common and clinically important sign of JSDS, but JSDS cannot be excluded or confirmed by the presence or absence of pain alone. Pain is considered a subjective finding and some contend that subjective findings such as pain reproduction have

less significance than findings that are "objective." However, many so-called objective tests rely on the patient's report of pain. For example, the straight-leg raising test is considered an objective test, yet it is the patient's report of leg pain that constitutes a positive test. This is no different than applying pressure over osseous or soft tissue structures and having the patient report the presence or absence of pain. The use of provocative tests to localize a painful area is therefore a useful means for identifying musculoskeletal problems, including JSDS. These manual physical maneuvers are designed to reproduce the patient's symptoms or verify the location of pain, thereby giving support for the local presence of a dysfunctional process. Typically, these tests stretch, compress, or distract specific anatomic structures with the patient reporting pain characteristics. When patients experience pain caused by one of these mechanical tests, there is likely to be a local mechanical component contributing to the condition.

Joint dysfunction is typically, but not necessarily, symptomatic. The nature and cause of joint pain and dysfunction cannot be determined from the pain pattern alone. Joint pain does not discriminate between joint hypomobility, hypermobility, and clinical instability. Furthermore, not all structures of the synovial joint are sensitive to pain. Some are very poorly innervated, and some are not innervated at all. The articular cartilage, nondisrupted nucleus pulposus, and cartilaginous end plates are devoid of nociceptive innervation.¹³¹ Thus pathologic change within certain articular structures may be insidious and well advanced before it becomes symptomatic.

Spinal or extremity joint pain is often poorly localized, and sites of pain and pathologic conditions may not necessarily correspond. Disorders of the musculoskeletal system are often associated with areas of referred pain and hypalgesia.^{298,299}

Referred pain is sclerogenic, ill-defined, deep, and aching. It is referred from the deep somatic tissues of the involved joint to the corresponding sclerotome. The anatomic sites of referred pain correspond to tissues that share the same segmental innervation (Figure 3-11).

Sites of referred pain may be more painful to palpation and of greater intensity than the site of injury. The common phenomenon of interscapular pain with cervical joint derangement or disc herniation illustrates this point. The body is also more adept at discriminating sensations on the surface than pain originating in deep somatic structures and joints.^{298,299} Ordinarily, the closer the affected tissue is to surface of the body, the better the pain coincides to the site of injury.

Joint pain of mechanical origin characteristically has pain-free intervals, whereas joint pain associated with inflammation is more constant. Joint movement and the activities of daily living often aggravate mechanical joint pain. Although it is often alleviated by decreased activity, total immobilization may accentuate the pain response. Pain diagrams, visual and verbal analog scales (Figure 3-12), and functional capacity questionnaires are very helpful measures in the examination and quantification of painful complaints.³⁰⁰⁻³⁰⁶

Because the character, location, quality, and intensity of pain can vary greatly from individual to individual and from disorder to disorder, it is essential to subject all painful joint disorders to a thorough physical examination and to rule out contraindications before considering adjustive therapy.

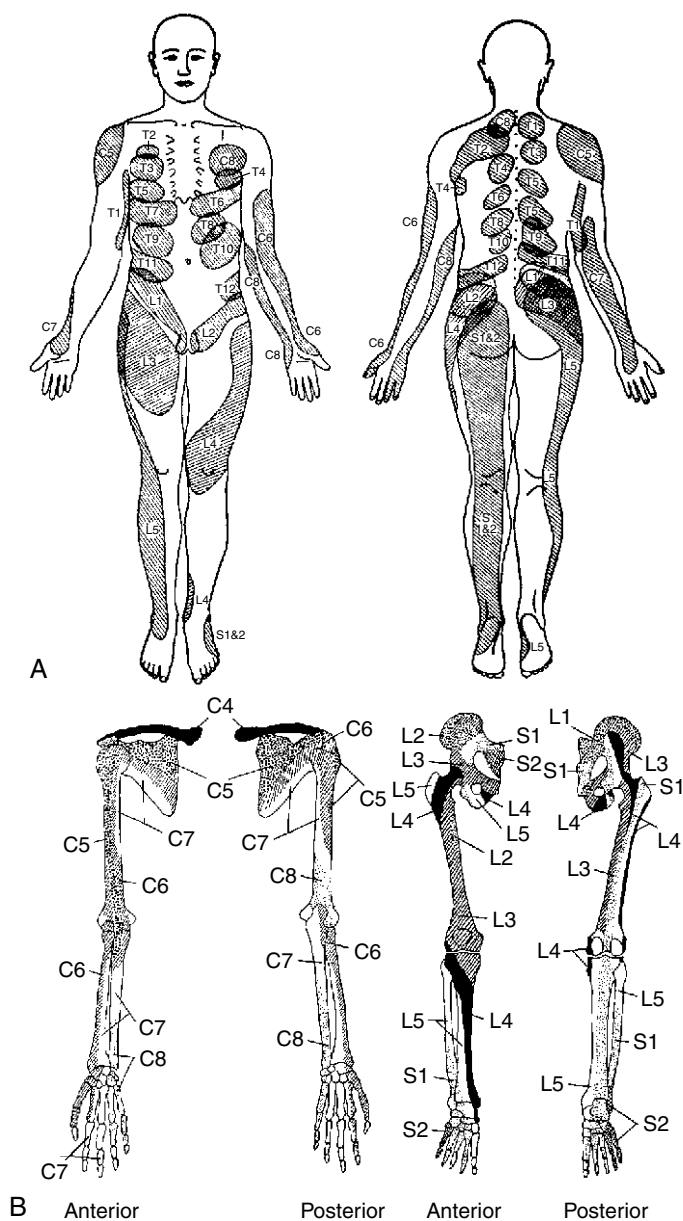
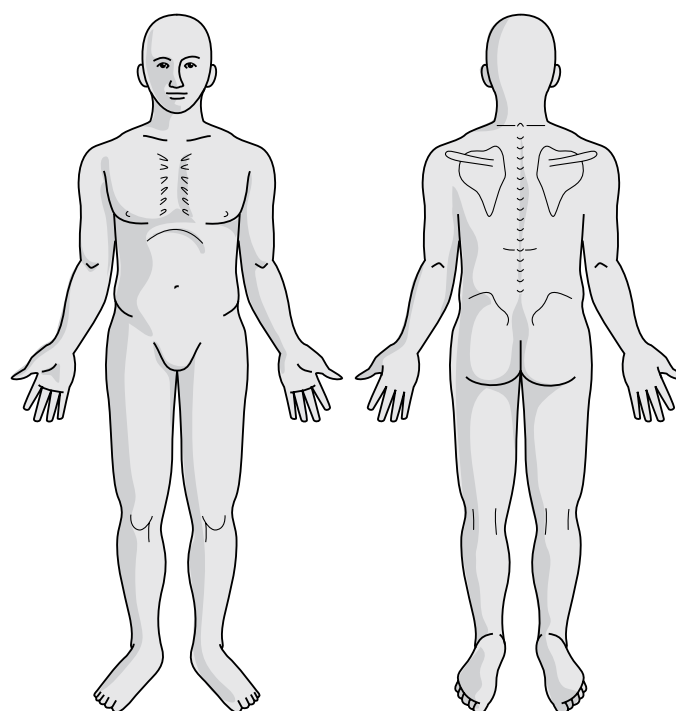


Figure 3-11 Segmental areas of pain of deep somatic origin. **A**, Interspinous ligament injection—Kellgren. **B**, Sclerotomal pain patterns. (A from Lewis T: *Pain*, New York, 1942, Macmillan. B from Grieve GP: *Common vertebral joint problems*, ed 2, Edinburgh, 1988, Churchill Livingstone.)

PATIENT OBSERVATION

The examination of any regional complaint begins with superficial observation and investigation for any signs of trauma or inflammation. These signs include abrasions, lacerations, scars, discoloration, bruises, erythema, pallor, swelling, or misalignment. Acute injury, congenital or developmental defects, and many systemic diseases of the NMS are often represented by abnormalities observed in posture or gait.

The human body uses an ingenious three-dimensional framework of bones, joints, muscles, and ligaments for posture and movement.³⁰⁷ Therefore, the observational evaluation of NMS



Numbness	== ==	Burning	x x x x
Pins/Needles	0 0 0 0	Stabbing	/ / / /
		Aching	a a a a

Scoring sheet for pain drawing

Writing anywhere	1
Unphysiologic pain pattern	1
Unphysiologic sensory change	1
More than one type of pain	1
Both upper and lower areas of the body involved	1
Markings outside the body	1
Unspecified symbols	1

Score: 1 = Normal. 5 or more = Functional overlay.

A

Visual analog pain severity scale

Instructions: Please make a mark on the line provided below that corresponds to how you presently feel.

No pain Worst pain imaginable

B

Figure 3-12 Tools to localize and record pain intensity. **A**, Pain diagram. **B**, Visual analog scale. (A adapted from Mooney V, Robertson J: *Clin Orthop* 115:149, 1976.)

function routinely incorporates an assessment of patient symmetry, posture, and locomotion. The examination is based on the premise that there is a postural ideal that can be used as a comparative standard and that deviations in posture, gait, or movement may identify NMS disease or dysfunction or predispose an individual to NMS disease or dysfunction. Poor posture can be viewed as a faulty relationship of bones, ligaments, and muscles that produces an increased stress on the supporting structures leading to decreased efficiency for maintaining the body's balance over its

base of support. Ample evidence supports the association of painful disorders of the NMS with restrictions to joint motion and abnormalities in posture.^{308–321}

Evidence also suggests that deviations from “ideal posture” may predispose an individual to NMS dysfunction and possible joint degeneration.^{199,322–326} However, the degree of deviation necessary to affect a patient’s health has not been established. Individual biologic variation and adaptability certainly play a role in limiting the development and morbidity of joint dysfunction and degenerative joint disease. Those that would set a narrow standard for posture and ROM ignore the research evidence that suggests a range of normal individual variation.^{23,316,324,327–335}

Gait Evaluation

Gait evaluation is conducted formally during the physical examination, but it begins as the patient walks into the examination room. Locomotion involves integrated activity of numerous components of the motor system and therefore becomes an efficient method for screening NMS function.

The objectives of gait analysis are to identify deviations, to obtain information that may assist in determining the cause of the deviations, and to provide a basis for the use of therapeutic procedures or supportive devices to improve the walking pattern.³³⁶ There are two basic phases of the normal pattern of gait: one involves a weight-bearing period (stance phase) and the other, a non-weight-bearing period (swing period) (Figure 3-13). Disease or dysfunction may affect one phase and not the other, necessitating careful evaluation of both components.

Evaluation begins with a general impression of locomotion. Is it guarded or painful? Is the patient protective of any part or unwilling to put equal weight on each leg?

The movements of the upper and lower extremities are noted. Length of stride, degree of pronation or supination, tilt of pelvis, adaptational movements of the shoulder girdle, and pendulousness of the arms are assessed. Specific components of gait evaluation are listed in Box 3-4, and disorders that may alter gait are listed in Box 3-5.

Apparent abnormal findings or deviations from the expected pattern identified with gait analysis must be supported or validated by other test procedures, including muscle tests for strength, length, tone, and texture, as well as tests for joint function.

BOX 3-4 Components of Gait Evaluation

- Alignment and symmetry of the head, shoulders, and trunk
- Gross movements of the arms and legs, looking for reciprocal and equal amplitude of movement
- Symmetry of stride from side to side for length, timing, and synchronization
- Assessment of body vertical oscillations at an even tempo
- Assessment of pelvic transverse rotation, anteroposterior rotation, lateral tilt, and lateral displacement through the phases of gait
- Assessment to determine if the lower extremities medially rotate, then laterally rotate, going from swing to stance
- Assessment to determine if the knees have two alterations of extension and flexion during a single-gait cycle
- Assessment to determine if the ankles go from dorsiflexion to plantar flexion when going from the stance phase to the swing phase

Postural Evaluation

Like all physical examination skills, postural evaluation must be learned and practiced. Reliable and accurate assessment is founded on attention to proper technique. The room must be appropriately lit to clearly illuminate the body parts being examined and to prevent shadows from projecting false contours. The doctor should be oriented to the patient so that the dominant eye is located in the midline between the landmarks being compared.³³⁷ If observing the patient while he or she is supine or prone, the doctor stands on the side of eye dominance (Box 3-6).

When combining observation and palpation of asymmetry, it is important that the doctor’s hands and eyes are on the same reference plane. For example, when evaluating the relative heights of the iliac crests, the doctor places a hand on each crest and positions the dominant eye in the midline on the same plane as his or her hands.

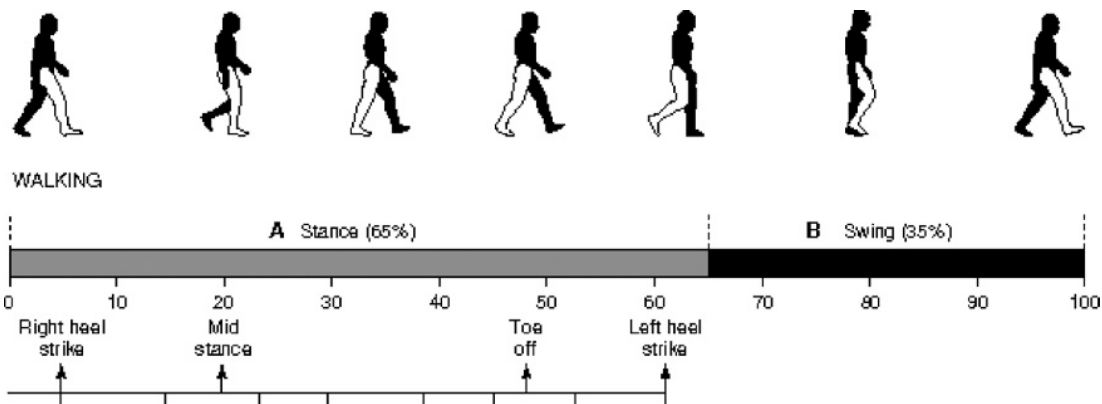


Figure 3-13 The phases of gait. **A**, Stance phase. **B**, Swing phase. (Modified from Adelaar RS: *Am J Sports Med* 14:497, 1986.)

BOX 3-5 Disorders That May Induce Altered Gait

Pain or discomfort during the weight-bearing phase
 Muscle weakness and imbalance
 Limitation of joint motion—active, passive, or accessory
 Incoordination of movement as a result of neurologic condition (e.g., Parkinson syndrome)
 Changes and deformities in bone or soft tissues

BOX 3-6 Determination of Dominant Eye

1. Bring both hands together to form a small circle with the thumbs and index fingers.
2. Straighten both arms out, and with both eyes open, sight through the small circle an object at the other end of the room.
3. Close one eye. If the object is still seen, the open eye is dominant. For example, if the right eye is closed and the object is still seen, the left eye is dominant. If the right eye is closed and the object is no longer seen, the right eye is dominant.

The assessment of symmetry, locomotion, and posture is critical in the evaluation of NMS dysfunction. They are objective signs supportive of NMS disease or injury³¹⁰ and effective outcome measures for monitoring patient progress. Regional asymmetry should trigger further evaluation of that area, but asymmetry alone does not confirm or rule out the presence of segmental subluxation and dysfunction syndromes.

Postural asymmetry is a challenge to homeostatic regulation and does indicate potential areas of muscular imbalance, bony asymmetry, and mechanical stress. Its relationship to initiating, predisposing, or perpetuating segmental dysfunction should not be overlooked. In a rush to find the specific level of spinal JSDS, chiropractors often overlook significant postural decompensations that may predispose the patient to pain and dysfunction. The patient with extremity or spinal complaints may not respond to local therapy until gait and postural stresses are removed.

Spinal Postural Evaluation. Although deviations in spinal posture do not identify the presence or absence of a specific level of spinal dysfunction, deviations do provide evidence of underlying postural syndromes or the presence of painful NMS conditions. Spinal postural assessment has demonstrated satisfactory reliability^{309,338-340} and validity as a screening procedure for distinguishing symptomatic myofascial back pain subjects from normal subjects.³¹⁰ In this capacity, it may function as a useful outcome measure to document changes in painful antalgic postures associated with NMS disease and dysfunction.

During standing postural assessment, the patient is instructed to assume a relaxed stance, looking straight ahead, with feet approximately 4 to 6 inches apart and arms hanging loose at the sides. The patient should be in a gown or undergarments, and should not be wearing shoes. If the patient has orthotics or corrective footwear,

posture is assessed with the patient's shoes both off and on. The evaluation is conducted from the posterior and anterior to determine distribution of weight and symmetry of landmarks in the coronal plane and from the side to evaluate posture and landmarks relative to the center of gravity line. In addition, the upper and lower extremities are surveyed for deformity, pronation or supination, and internal or external rotation.

The examination should include a determination of the carriage of the center of gravity and symmetry of key bony and soft tissue landmarks. Any curvatures, scoliosis, or rib humps should be measured and recorded. The flexibility (Adams test) of the curve should also be determined and noted.

The evaluation of spinal posture may be aided by the use of a plumb line (Figure 3-14) and devices such as the posturimeter, scoliometer, and bilateral weight scales. The plumb line assessment from the posterior should find the gravity line, splitting the body into equal left and right halves. The plumb line should pass from the external occipital protuberance through the center of the spinal column to the center of the sacrum and points equidistant from the knees and ankles. The lateral plumb line assessment has the gravity line splitting the body into equal front and back portions. The plumb line should pass from the external auditory meatus down through the shoulder joint to the greater trochanter of the femur, continuing down to just anterior to the midline of the knee and slightly anterior to the lateral malleolus.

In a patient with suspected scoliosis, an assessment for potential leg length inequality and a screen for anatomic or functional leg length discrepancy should be included. Suspected anatomic discrepancy should be measured and radiographically confirmed if clinically significant. Postural distortions with possible muscle imbalance causes are identified in Table 3-2.

The identification of postural imbalances can be helpful in diagnosing disorders or in guiding clinical treatments. In some cases it may be central to the identification of the underlying disorder, such as idiopathic scoliosis, and in others it can help guide treatment decisions, such as exercise prescription in patients with postural imbalances and LBP. Subsequent evaluations are used to monitor progress and make decisions about treatment changes. The significance and usefulness of these evaluations depend on repeatability sufficient to ensure that ensuing changes are attributable to the prescribed treatment program and not to any naturally occurring variability in posture. This may not be attainable in all clinical situations; Dunk and coworkers³⁴¹ demonstrated that the ability to return to the same starting posture exhibited poor to moderate repeatability. This brings into question the benefit and validity of using small deviations from ideal spinal posture in clinical decision making. Therefore, users of postural analysis tools should interpret small to modest postural deviations from a vertical reference with caution, because there are many inherent factors that can contribute to the variability of these measured postures.³⁴¹ Studies have also demonstrated that visual assessments for an increase or decrease in cervical or lumbar lordosis are not reliable or accurate.^{342,343}

Leg Length Evaluation

The evaluation of leg length inequality incorporates consideration for both anatomic and functional discrepancies. Anatomic inequality results primarily from osseous asymmetry. Functional

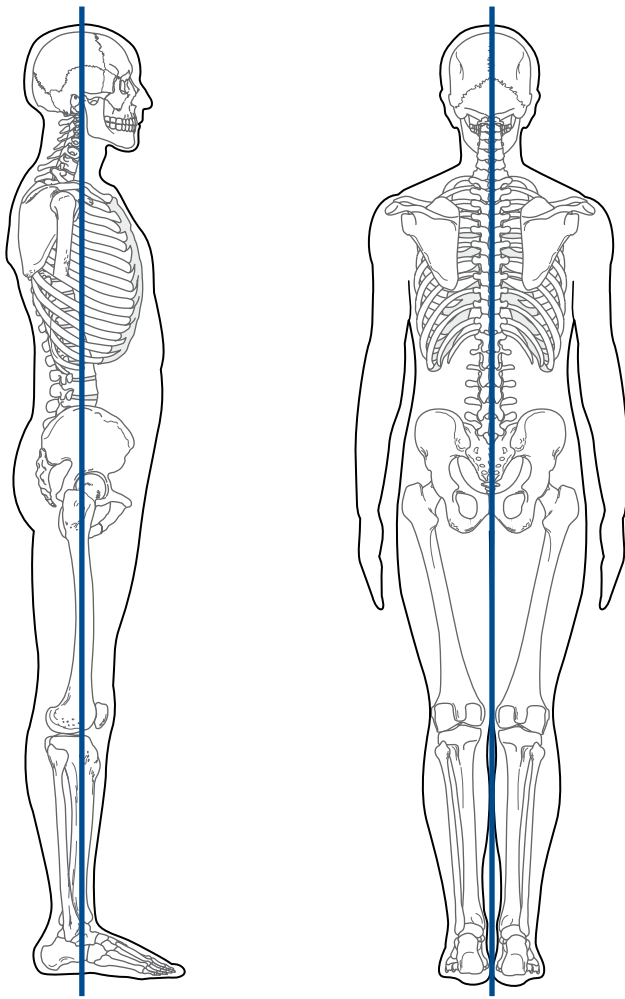


Figure 3-14 Anteroposterior and lateral plumb line evaluation of spinal posture.

Lateral View

The gravitational line should pass:

- Through the earlobe
- Just anterior to the shoulder joint
- Through the midline of the thorax
- Through the center of L3 vertebral body
- Through the greater trochanter
- Just anterior to the midline of the knee joint
- Just anterior to the lateral malleolus

Posterior or Anterior View

- The gravitational line should pass:
- Through the midline of the skull
- Through the spinous processes

Through the gluteal crease

Midway between the knees

Midway between the ankles

The following landmarks should be evaluated for unleveling or asymmetry:

- Gluteal folds
- Gluteal contours
- Iliac crests
- Posterior superior iliac spine
- Rib cage
- Inferior angles of the scapula
- Vertebral borders of the scapula
- Acromioclavicular joints
- Earlobes

leg length inequality implies that the legs are anatomically of equal length but appear unequal as a result of a disorder in the NMS system. Anatomic asymmetry is viewed as a potential source of derangement and dysfunction that is potentially treatable with heel or sole lifts. Functional asymmetry is viewed primarily as a consequence of dysfunction.

TABLE 3-2 Common Postural Findings With Possible Muscular Imbalances

A-P or P-A Postural	Examination
Head tilt	Neck extensors and/or scalenes
Head rotation/tilt	SCM
Shoulder tilt	Trapezius (upper, lower) latissimus
Scapular winging	Serratus anterior
Scapular heights	Rhomboids
Arm rotation	Subscapularis, teres minor, infraspinatus
Pelvic unleveling	TFL, adductors, psoas, quadratus lumborum, gluteals
Genu valgus/varus	TFL, sartorius, gracilis
Leg rotation	Hamstrings, tibialis anterior, peroneus, piriformis, psoas
Lateral Postural	Examination
Forward head carriage	Neck extensors, longus coli, scalenes
Thoracic kyphosis	Trapezius (middle and lower)
Lumbar lordosis	Psoas
Pelvic tilt	Quadriceps, hamstrings, sartorius, gracilis, abdominals, gluteus maximus
Forward lean	Gastrocnemius, soleus
Knee hyperextension	Quadriceps, popliteus, gastrocnemius, soleus

A-P, Anterior-to-posterior; P-A, posterior-to-anterior; SCM, sternocleidomastoid; TFL, tensor fascia lata.

Leg length evaluation has a long history of affiliation with chiropractic, and functional leg length inequality is considered an important sign of spinal or pelvic subluxation/dysfunction syndromes.³⁴⁴ Spinal joint dysfunction is hypothesized to potentially affect leg length by inducing reflex alterations in spinal muscle balance and unleveling of the pelvis and legs.³⁴⁴⁻³⁴⁶

Disturbances in sacroiliac function and pelvic alignment are theorized to induce torsion between the innominate and affect leg length by creating positional changes in the femoral heads or imbalances in hip flexor and extensor muscle tone.³⁴⁴ A significant percentage of practicing chiropractors and a number of system technique methods emphasize the role of leg length evaluation in detecting spinal subluxations/dysfunction and directing decisions on where, when, and how to adjust patients.³⁴⁵⁻³⁴⁷ Leg alignment change in response to provocative springing (vertebral challenge) is used by some chiropractic techniques to determine the level of spinal dysfunction and direction of therapeutic adjustive thrust.³⁴⁶

Leg length equality can be assessed by physical means or by radiographic evaluation. Evaluation of inequality by physical means includes procedures that assess leg length through direct tape measurement, by visual inspection, or indirectly through combined visual and palpatory assessment of symmetry. Physical measures are appropriately used to screen for leg length inequality, estimate the amount of any noted discrepancy, and participate in differentiating anatomic leg length inequality from functional leg length inequality. Physical assessment procedures cannot determine exact differences in anatomic length.

X-ray evaluation is necessary when precise determination of leg length is required. Radiographic procedures should not be used to screen for possible leg length inequality. X-ray evaluation of leg length should be considered only after suspicion of a significant anatomic leg discrepancy is identified and corrective heel or shoe lifts are being contemplated. Radiographic procedures include both standing and supine methods. Radiographic evaluations are recognized as reliable and valid procedures for determining anatomic leg length discrepancies.³⁴⁵

Standing methods are used to assess the comparative height of the femoral heads and include full spine, lumbar, and femoral head views. Femoral head views afford the most accurate evaluation of femoral head height because they eliminate the false appearance of inequality that can result from rotation of the patient's pelvis during patient positioning. When precise comparison of length is desired, supine methods are usually used. Of these, the scanogram is the method most commonly used.

The most common physical assessment method used in chiropractic practice combines visual assessment and palpation of

symmetric bony landmarks. Evaluation is performed with the patient in a prone or supine position, with his or her shoes on or off, and the doctor standing at the end of the table. The doctor evaluates equality by contacting and comparing the inferior poles of the medial malleoli, the soles of the shoe, or the patient's heels (Figure 3-15). It is advisable to remove the shoes if significant shoe wear is present or if the doctor suspects that the patient's heel and shoe cannot remain in firm contact. When using the soles of the shoe or the patient's heels as the comparative landmark, it is important for the doctor to neutralize the ankles to prevent eversion or inversion from creating a false appearance of inequality.

If leg length inequality is noted in the prone position, the doctor bends the patient's legs to 90 degrees and again observes for inequality. If the inequality remains anatomic, shortening of one tibia is considered. If the heels approach or reach equal height, some degree of functional leg length inequality is suspected. Various interpretations of this process (leg checks) have been developed and are in common use. The Derifield pelvic leg check is foremost in this regard and is commonly affiliated with the Activator and Thompson technique systems.^{348,349} The specifics of this test are presented in Chapter 5 in the section covering pelvic evaluation procedures.

Despite the common use of physical procedures to detect leg length inequality, significant questions concerning the clinical significance and reliability, validity, and responsiveness of these methods remain.^{265,344,345,347,350} Procedures using tape measurement, iliac crest comparative height checks, or visual leg checks to assess comparative leg length have demonstrated mixed results.³⁴⁵ These procedures have demonstrated both poor and

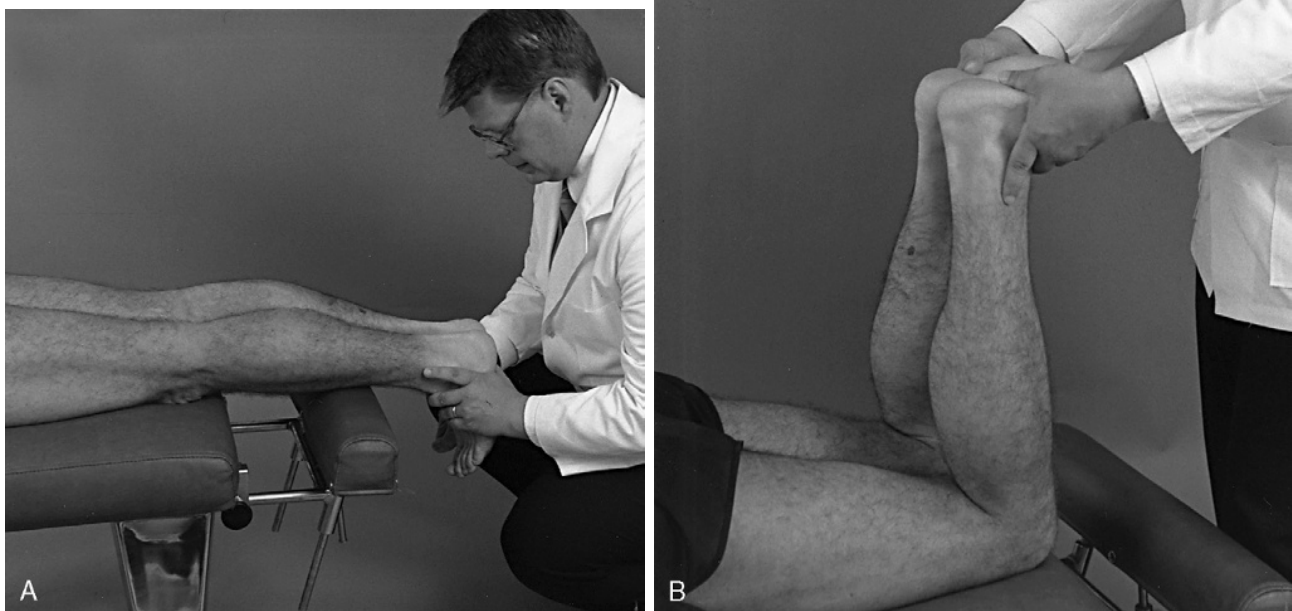


Figure 3-15 Prone evaluation of leg length. **A**, Evaluation for symmetry of leg length by comparing the patient's heels or inferior poles of the malleoli. **B**, Evaluation of tibial length by comparing heel symmetry in a flexed-knee position.

good interexaminer reliability.^{346,347,351-357} A significant number of the studies using visual leg checks have been criticized for poor experimental design or statistical analysis.^{265,344,345} Leg length inequality testing has also failed to respond as predicted to thoracic rotary vertebral challenge and thoracic adjustment.^{346,358} Schneider and associates²⁴⁶ performed an interexaminer reliability evaluation of the prone leg length analysis procedure and found good reliability in determining the side of the short leg in the prone position with knees extended, but found poor reliability when determining the precise amount of that leg length difference. In addition, they found that the head rotation test for assessing changes in leg length was unreliable in this sample of patients, nor did there appear to be any correlation between the side of pain noted by the patient and the side of the short leg. It was interesting to note that all 45 patients in this sample were found by both clinicians to have a short leg.²⁴⁶ The weakest element in the leg check procedure is the second position, with the knees bent to 90 degrees, in which overall agreement is poor, reaching only as high as “fair” 25% of the time.^{345,356}

The validity of visual leg checks for anatomic accuracy or subluxation and dysfunction detection has yet to be evaluated clinically. Cooperstein and colleagues did determine that visual leg checks were accurate in measuring artificially induced leg length inequality,³⁵⁷ but no studies have been done to measure their relationship to level of spinal dysfunction or treatment outcomes. Because of the lack of validity testing, it is difficult to form any definitive conclusions as to the clinical utility of these procedures.³⁴⁵

RANGE-OF-MOTION ASSESSMENT

Measurement of joint mobility is a critical element in the evaluation of NMS function, and qualitative and quantitative evaluation of joint motion is a fundamental component of the examination of the NMS system. Significant limitation and asymmetry of movement is considered to be evidence of NMS impairment,³⁵⁹ and improvement in regional mobility may be a valuable outcome measure for assessing effectiveness of treatment.

Disorders capable of altering individual joint and regional spinal movements are extensive. They include joint subluxation/dysfunction, dislocation, effusion, joint mice, myofibrosis, periarticular fibrosis, muscle hypertrophy, degenerative joint disease, muscle guarding, and fracture. Other nontraumatic disease states with pathologic effects on somatic structures or the nervous system also produce abnormalities in movement.

Although regional ROM assessment has demonstrated the ability to differentiate individuals with low back disorders from those without,^{310,360} spinal abnormalities in GROM are more valuable in identifying and monitoring NMS dysfunction than confirming a specific level of joint subluxation/dysfunction. Regional abnormalities in range of spinal motion are potential signs of dysfunction, but they do not confirm the presence of segmental joint dysfunction. GROM may be falsely positive in situations in which spinal injuries or disease affects the nonsegmental somatic tissues and spares the vertebral joints. In these circumstances, altered regional movements are present, but the loss of mobility is

uniform, and the individual spinal motion segments demonstrate normal JP and EP (feel). Conversely, normal regional ROM may be falsely negative in circumstances in which individual spinal joint restrictions are concealed by compensatory hypermobility at adjacent joints.

Evaluation of repeated regional spinal movements in conjunction with the patient's description of pain and limitations to movement have been promoted as effective tools for diagnostically classifying back pain patients.^{361,362} The information gained about the patient's symptomatic and mechanical responses to loading allows the clinician to determine which specific movements, positions, and activities to either pursue or avoid in the treatment plan.

The McKenzie method of evaluation and treatment is the most widely practiced procedure using repeat movements to classify back pain patients. It is common for chiropractors to use McKenzie diagnostic procedures, but is more commonly used by physical therapists. Donelson, Aprill, and Grant³⁶² demonstrated that the procedures were capable of reliably differentiating discogenic from nondiscogenic pain and a competent from an incompetent annulus. In comparison with MRI, it also demonstrated superior ability in distinguishing painful from nonpainful discs.³⁶² In a later commentary article, Delany and Hubka³⁶³ re-evaluated the data from the original study and concluded that the study demonstrated “informative but not definitive” ability to detect discogenic pain. They concluded that “high sensitivity but low to moderate specificity was demonstrated.”

Early reliability testing of McKenzie procedures demonstrated mixed results.^{364,365} However, recent studies have concluded that the McKenzie method demonstrates good reliability for classifying patients into syndrome categories based on repeated movements and the principle of centralization of pain.³⁶⁶⁻³⁶⁸

Measurement Procedures

Methods for assessing mobility are commonly used and include both visual and instrument-based procedures. They range from goniometric and inclinometric measurements to the more technical approaches of computerized digitation.^{308,369} Visual observation and the fingertip-to-floor method of recording motion have demonstrated mixed reliability³⁷⁰ and are considered to be invalid tests because they cannot effectively differentiate lumbar mobility from hip or thoracic movement. The modified Schober method of measuring lumbar mobility has shown consistent reliability, but it has limited use because it measures only lumbar flexion.³⁷⁰⁻³⁷²

The use of inclinometers for spinal ROM and inclinometers or goniometers for extremity ROM is becoming a minimal standard.³⁵⁹ For the spine, the one- or two-inclinometer method as described by Mayer and associates³⁰⁸ is a reliable, inexpensive, and efficient technique. With the exception of one study,³⁷³ evaluation of motion with hand-held inclinometric measuring devices has demonstrated consistent reproducibility within and between examiners.^{308,370,371,372-376} It can be used in the measurement of all spinal movements, including trunk rotation, when the spine is in a flexed position (Figures 3-16 and 3-17). However, the range of trunk rotation is significantly limited

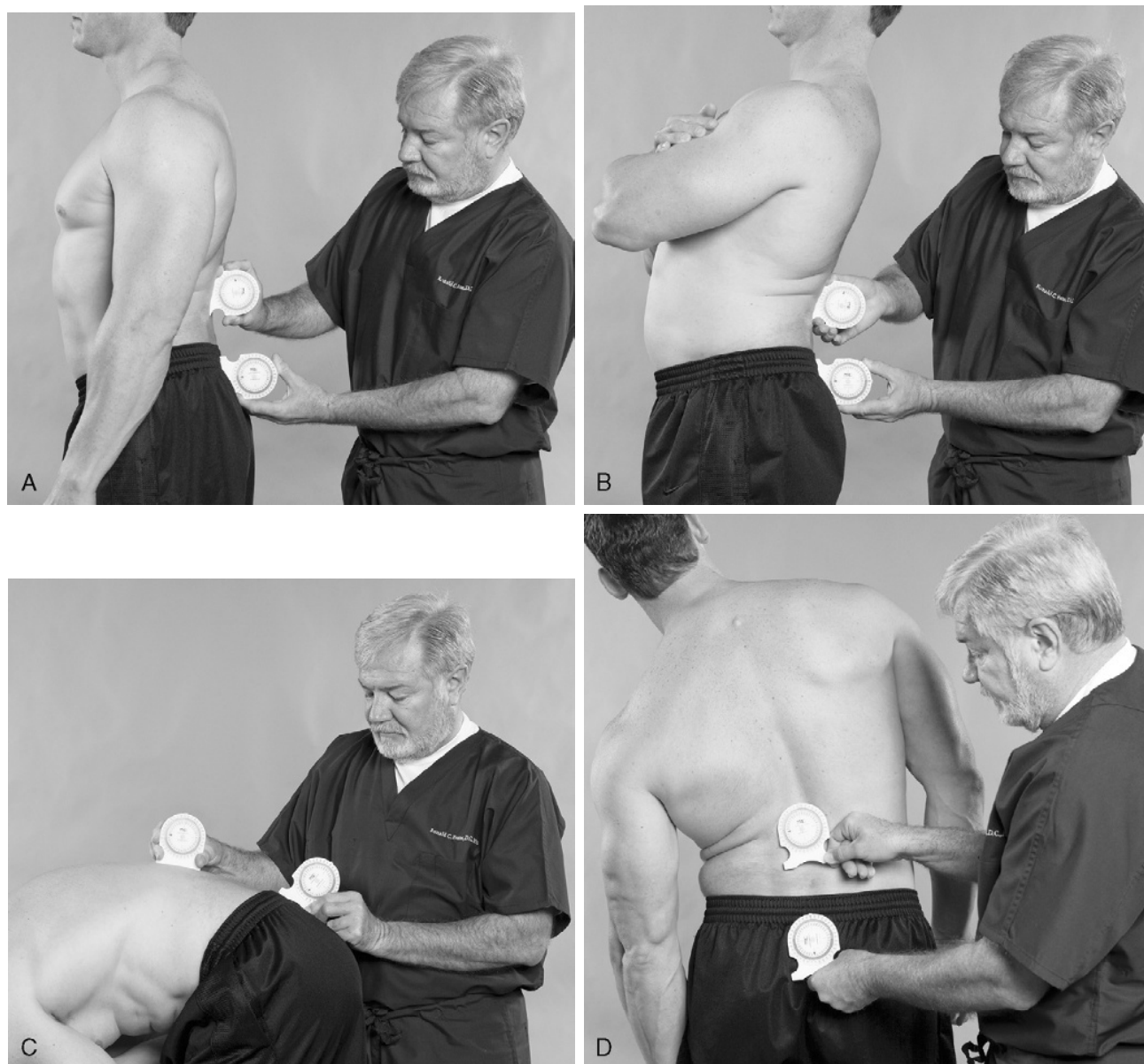


Figure 3-16 Measurement of lumbar range of motion using dual inclinometers. **A**, neutral starting position for evaluating flexion and extension; **B**, measurement of extension; **C**, measurement of flexion; **D**, measurement of left lateral flexion. (Evans, RC: *Illustrated orthopedic physical assessment*, ed 3, Mosby, Elsevier, St. Louis, Mo 2009.)

when it is placed in a flexed position. In the spine, quantitative measures of joint mobility rely primarily on regional ROM measures as a result of joint inaccessibility and limited ROM. Methods for estimating the quality and quantity of individual spinal motion through manual palpation have been developed and are covered regionally in Chapter 5.

Evaluation of spinal and extremity mobility must take into consideration the normal variations that exist between individuals and gender. Alterations in mobility may be a product of occupation, recreation, or aging, and may not be associated with dysfunction or pain. This increases the importance of making a bilateral comparison of joint and spinal mobility.

Spinal and extremity joint motion are measured in degrees relative to the zero or starting position. In a patient demonstrating 50 degrees of cervical extension and 55 degrees of cervical flexion,

the motion is recorded as “EXT/FL 50-0-55.” All physiologic movements, on both sides of the zero position (0 degrees), should be measured and recorded. Table 3-3 outlines the common format for recording spinal and extremity ROM.

PALPATION

Palpation is the application of variable manual pressures, through the surface of the body, to determine tenderness, shape, size, consistency, position, and inherent motility of the tissues beneath.⁶¹ It can also serve as an important doctor-patient communication tool, helping patients understand the significance of their problems because they can feel the provoked pain and resistance as they are palpated.²⁴⁰ Furthermore, palpation is the oldest examination technique used by chiropractors to detect subluxation/dysfunction.^{74,234-237}



Figure 3-17 Measurement of cervical ranges of motion using dual inclinometers. **A**, measurement of extension; **B**, measurement of right lateral flexion; **C**, neutral starting position for measuring rotation; **D**, measurement of right rotation. (Evans RC: *Illustrated orthopedic physical assessment*, ed 3, Mosby, Elsevier, St. Louis, Mo 2009.)

Like observational skills, palpation skills are learned tasks that take hours of devotion and practice. Good palpation skills are the result of both physical abilities and mental concentration. The skillful palpator is one who has developed an improved ability to tactually discriminate and mentally focus.

Palpatory procedures are commonly divided into static and motion components. Static palpation, which is often further subdivided into bony and soft tissue palpation, is performed with the patient in a stationary position. Motion palpation is performed during active or passive joint movement and also involves the evaluation of accessory joint movements. Motion palpation procedures have been an integral part of chiropractic since its inception, but not until the work and cultivation of Gillet⁴¹⁻⁴⁵ and Faye^{52,53} have formalized techniques been widely disseminated.

Reliability of Palpation Procedures

Clinical evaluation of palpation procedures has increased significantly in the last several decades. The majority of tests have been conducted on reliability. Reliability testing for the various manual palpation procedures has demonstrated mixed results. Interexaminer palpation for bony alignment and muscle tension has demonstrated poor results, but palpation for bony and soft tissue tenderness has established good to excellent interexaminer reliability.^{245,255,377-386} Palpation for bony and soft tissue tenderness is frequently cited as one of the most valuable clinical cues for identifying dysfunction and targeting spinal manipulation. Research by Schneider and coworkers³⁸⁶ confirmed the reliability of pain provocation. They tested the reliability of spinal palpation for segmental mobility testing and pain provocation

TABLE 3-3 Assessment of Spinal and Extremity Motion

Spinal Motion		
Extension	0	Flexion
Right lateral bending	0	Left lateral bending
Right rotation	0	Left rotation
Extremity Motion		
Extension	0	Flexion
Abduction	0	Adduction
External rotation	0	Internal rotation
Supination	0	Pronation
Radial deviation	0	Ulnar deviation
Inversion	0	Eversion

in 39 patients with a history of LBP. The resulting kappa values showed generally good reliability for the springing palpation that relied on patient self-reports of pain but poor reliability for assessment of mobility.³⁸⁶

Joint motion palpation, particularly passive EP assessment, is recognized as an essential skill by manual medicine disciplines³⁸⁷ and is used extensively by chiropractors, physical therapists, osteopaths, and medical manipulators in clinical practice as an indicator for spinal manipulation and mobilization. However, despite a few noteworthy exceptions that identified good interexaminer and intraexaminer reliability,^{384,385,388-391} the majority of studies evaluating spinal and sacroiliac joint (SIJ) mobility tests have identified poor interexaminer reliability and good intraexaminer reliability.* Some studies have shown weak or clinically insignificant reliability of motion palpation for certain groups of young, asymptomatic subjects.⁴⁰⁷⁻⁴¹⁰ Most other studies have obtained poor to mixed results,^{247,378,391} with a few demonstrating an acceptable level of reliability for motion palpation testing.^{392,398} A summary of reliability studies conducted by Haneline and Cooperstein for motion palpation, pain provocation, landmark location, and alignment is presented in Appendix 2.

Good intraexaminer reliability in the face of poor interexaminer reliability should be viewed with caution. Good intraexaminer reliability does allow the examiner to evaluate how a test responds to treatment and may be helpful in directing individual patient treatments, but the ability to show internal consistency has limited value if different examiners cannot agree on their findings.²⁷¹

The poor interexaminer reliability demonstrated in the majority of studies evaluating motion palpation is likely the product of several factors. One may be the very small qualitative and quantitative changes it attempts to measure. The line between normal and abnormal segmental joint movement has not been clearly established.⁴¹¹ In this environment, each examiner develops his or her own standard of what constitutes pathologic movement. Each examiner must develop his or her own “feel” for what is

abnormal, with no common basis for comparison. The wide range of techniques taught and individual modifications and idiosyncrasies that each practitioner develops further complicate reliability. Interexaminer reliability is also likely negatively affected by the inability of examiners to precisely or repeatedly identify spinal bony landmarks and segmental joint level of palpation.^{391,412} Interexaminer reliability for motion palpation may demonstrate poor results not because examiners are inaccurate at sensing movement changes but because they are mislabeling and disagreeing on the joints that each is palpating. Furthermore, there is growing evidence that the biomechanical effects of spinal manipulation may not be as joint-specific as previously thought,^{413,414} and if the biomechanical effects are more widespread, we may be focusing on the wrong clinical question. A more appropriate clinical research topic might be the reliability of motion palpation assessment within a spinal region or two or three spinal motion segments.

The question of whether a multidimensional diagnostic approach to the physical examination of mechanical spine pain might lead to more reliable outcomes has had limited investigation. A study designed to assess the intraexaminer and interexaminer reliability of such an approach demonstrated moderate levels of intraexaminer reliability for the decision to manipulate a certain spinal segmental level and fair interexaminer agreement pooled across all spinal joints. However, the conclusion of the study was that the common diagnostic methods, including visual postural analysis, pain description by the patient, plain static erect x-ray film of the lumbar spine, leg length discrepancy, neurologic tests, motion palpation, static palpation, and orthopedic tests used on patients with chronic mechanical LBP are not reproducible. They further suggest that the implementation of these examination techniques alone cannot provide reliable information concerning where to direct a manipulative procedure in patients with chronic mechanical LBP.²⁹¹

Based on available information, it seems reasonable to conclude the following: Palpation for bony and soft tissue pain and movement-induced joint pain (joint provocation or challenge) are reliable spinal manual examination procedures. Intraexaminer reliability for spinal motion palpation is mixed and interexaminer reliability for spinal motion palpation is generally poor. It is difficult to evaluate the interexaminer reliability of motion palpation because of inherent difficulties related to the standardization of the procedure, the inherent problems with identifying a specific segmental level of palpation, and the subtlety of functional spinal joint lesions. The following list provides a more detailed summary of the present status of spinal palpation reliability:

1. Interexaminer reliability for segmental ROM palpation and EP (end feel) is generally poor.
2. Interexaminer and intraexaminer reliability for EP evaluation is slightly better than segmental ROM palpation.
3. Intraexaminer reliability for EP motion palpation is good.
4. Interexaminer and intraexaminer reliability for joint pain provocation (challenge) tests is fair to good.
5. Interexaminer and intraexaminer reliability for palpation of bony or soft tissue pain is good.
6. Interexaminer and intraexaminer reliability for palpation for soft tissue textural changes is poor.
7. Interexaminer and intraexaminer reliability for regional ROM is good.

*References 253, 268, 269, 369, 378, 380, 383, 385, 391-406

8. Interexaminer and intraexaminer reliability for leg length evaluation is good for the prone, extended knee position and poor (less than chance agreement) in the prone flexed knee position.

If intercollege standards for identification of abnormal spinal segmental motion can be developed, and agreement reached on the degree of joint specificity that is needed, spinal motion palpation may have the potential to develop improved interexaminer reliability. Insight into how this may be educationally accomplished is illustrated by an experiment conducted by Harvey and Byfield.⁴¹⁵ They constructed a mechanical spinal model that was covered with leather to simulate skin and equipped with devices for artificially fixating segmental motion. Good interexaminer agreement was demonstrated by 8 graduate chiropractors and 19 final-year chiropractic students when given a choice between movement and the absence of movement. If mechanical models can economically be designed to simulate varying degrees of reduced movement, rather than complete absence of movement, they might have a valuable role to play in teaching and improving palpation reliability.

Validity of Palpation Procedures

Although the reliability of spinal motion palpation procedures has been extensively evaluated, there is a limited body of literature on the validity of motion palpation. A summary of validity studies is in Appendix 3. One of the early promising validity studies on spinal joint assessment was conducted by Jull, Bogduk, and Marsland.²⁹⁰ They investigated the accuracy of manual examination procedures in locating painful cervical joints confirmed by diagnostic nerve blocks. Using a combination of pain response and accessory and physiologic joint movements, a group of therapists identified the appropriate individuals and levels of abnormal painful cervical joints with 100% sensitivity and specificity. In another study,⁴¹⁶ physiotherapists were able to identify 24 of 26 painful vertebral levels that correlated to a single level of unilateral multifidus muscle wasting confirmed by ultrasound examination. The examination consisted of segmental motion palpation methods with an evaluation for pain reproduction and abnormal resistance to movement.

Although these results are quite dramatic, the results are somewhat suspect. The procedures used during injections did not control for placebo effects and possible false-positive responses. The possibility of false-positive responses bring into question the accuracy of using uncontrolled injections as a gold-standard comparison.⁴¹⁷ Furthermore, both studies included evaluations of mobility and pain. This makes it impossible to discern which procedure or combination of procedures is responsible for identifying the painful joint. Some have suggested that it is the provocation of pain and the patient's verbal pain cues that are responsible for identifying symptomatic spinal joint dysfunction.^{394,418}

In a subsequent single-blind study, Jull, Treleaven, and Versace⁴¹⁹ were able to demonstrate that "cervical symptomatic joint dysfunction could be identified without reference to specific vertebral reports of pain by the subject." The study did not rely solely on the evaluation of abnormal motion, but also allowed the examiner to determine the level of symptomatic joint dysfunction by the presence of "tissue stiffness and associated muscle reactivity or increased resistance through range of motion." The

authors concluded that "mechanical variables in segmental tissue stiffness, which are related to symptoms, can be detected."⁴¹⁹

King and associates²⁵⁸ replicated the study of Jull and colleagues²⁵⁷ with placebo-controlled facet blocks to determine the sensitivity, specificity, and likelihood ratio of manual examination for the diagnosis of cervical zygapophyseal joint pain. Manual joint motion examination demonstrated high sensitivity for cervical zygapophyseal palpated joint pain at the segmental levels that were commonly symptomatic, but its specificity was poor. Likelihood ratios were barely greater than 1:0, indicating that manual palpation for joint pain lacked validity. However, the study did have some significant methodologic limitations that affect its value. The manual examinations were not conducted by chiropractors and were performed by only one examiner with limited manual examination training. The reference standards (facet blocks) were applied only to subjects with positive manual examination findings and not those who had negative findings.

Humphreys and coworkers⁴²⁰ studied the validity of motion palpation using the presence of a congenital block vertebra as a gold standard. Twenty fourth-year chiropractic students had to identify the hypomobile segments in three subjects with a congenital block vertebra. They found a sensitivity of 74% and a specificity of 98% for the general detection of all blocks and a kappa value of 0.67, which is considered good. Assuming that block vertebra are a fair representation of marked spinal joint hypomobility, they concluded that their substantial demonstrated agreement lends support to the validity of motion palpation in detecting major spinal fixations in the cervical spine.

A study evaluating the prevalence of positive motion-palpation findings (so-called fixations and spontaneous pain response) in relation to self-reported LBP status was performed to determine the sensitivity and specificity of the motion-palpation technique on the sacroiliac and lumbar joints.⁴²¹ No logical pattern of fixations and spontaneous pain reactions were found in relation to the LBP status of the patients. The sensitivity was low for fixations and pain, but the specificity was significantly higher for pain in the mid-lumbar area. However, there was no strong association found between fixations and the examiners' interpretation of a pain reaction in response to motion palpation, leading to the conclusion that motion palpation does not appear to be a good method to differentiate persons with or without LBP.⁴²¹ However, it was concluded that it was possible to dissociate the findings of fixations and those of pain reactions.⁴²¹

The identification of hypomobility with prone P-A mobility testing has demonstrated validity in identifying patients who are more likely to benefit from manipulative therapy.^{260,261} This procedure was combined with pain of less than 16 days' duration, no radiating pain below the knee and low fear avoidance belief scores as criteria to select patients for a short trial of manipulative therapy. Subsequent evaluations have indicated that the other criteria may be more predictive of outcome and that the P-A mobility testing may not add significant value.

Another validity study using dynamic x-ray as a reference standard compared lower cervical lateral glide motion palpation to lateral flexion radiographs in patients presenting with mechanical neck pain.⁴²² The lateral gliding test for the cervical spine was as good as the radiologic assessment for the diagnosis of intervertebral joint dysfunction in the lower cervical spine in this small group of

patients. These results indicate that the lateral gliding test for the cervical spine is as accurate as lateral flexion radiographs in identifying restricted intervertebral mobility in the lower cervical spine.⁴²² However, lateral flexion radiographs have not been validated as a reliable and valid tool for identifying cervical dysfunction.

The clinical value of diagnostic procedures can also be assessed relative to their ability to change in response to treatment. Investigations into the responsiveness of motion palpation to adjustive treatments are limited to one randomized, controlled study conducted on thoracic rotational adjustments.²⁹¹ Patients were evaluated for thoracic rotational EP restrictions and randomly assigned to treatment and control groups. When re-examined by the blinded evaluators, those patients receiving treatments did show significant postmanipulative reductions in EP restrictions. Segmental EP palpation was thus found to have “utility as a postmanipulative evaluative test for patients who are symptomatic or mildly symptomatic in the thoracic spine.”²⁹³ This provides encouraging evidence that chiropractors may be able to manually palpate postmanipulative segmental EP restriction changes in human subjects.

The question also arises as to whether or not reliability and validity of motion palpation may be demonstrating less than optimal results because chiropractors are asking the wrong questions and not structuring the experiments accordingly. The majority of procedures evaluated to date have been based on the premise that a precise level of spinal dysfunction needs to be ascertained before effective treatment can be rendered. Hass and Panzer²⁷¹ question if this is an accurate assumption. They wonder if specific localization “might not be necessary for correction of the ‘true’ underlying manipulable subluxation syndrome.” They pose the possibility that identification of regional dysfunction might be sufficient for effective treatment of dysfunction.

As discussed previously, if identification of regional dysfunction were sufficient to establish effective treatment, it would likely be more reliable than procedures used to identify a specific level of dysfunction.²⁷¹ Although this may be a valid point, Hass and Panzer²⁷¹ correctly point out that it needs to be examined. It would be premature to abandon the specificity model without an established alternative “biomechanical model or clinical evidence to suggest how big the zone of agreement might be or how it might vary in different regions.”²⁷¹

To address this issue, Hass and associates⁴²³ conducted a preliminary investigation to evaluate the efficacy of a specific diagnostic indicator (segmental EP) to see if it improved spinal manipulative outcome. The study evaluated patients with neck pain who were randomized to receive cervical spine manipulation at restricted levels identified by motion palpation versus manipulation at levels randomly generated by a computer. The results show that both groups had similar, and in some cases dramatic, improvements in symptoms directly after receiving one HVLA cervical adjustment. The results of this study indicate that cervical EP-directed manipulation does not improve same-day outcomes in pain or stiffness. The outcome lends support to the hypothesis that spinal manipulation may have a more generalized, nonspecific mechanism of action in relieving symptoms. It also implies that the mechanical effects associated with manipulation may lack spatial specificity and specificity of adjustive contacts and adjustive vector may not be as important as generally thought.

Although the evidence from this study indicates that using EP to identify the level of dysfunction does not improve the measured

outcome, it would be inappropriate to draw conclusions from this study alone. It is the only study to clinically investigate this topic and it has a number of limitations that significantly affect its clinical implications. First, it measures the effects of only one adjustment on immediate and same-day pain and stiffness reduction. It is likely that manipulation has a dose-dependent therapeutic effect,⁴²⁴ and this trial does not come close to approximating the typical course of adjustive treatments. Adjustive treatments for a cervical mechanical pain syndrome average 6 to 12 treatments over the course of a few weeks. EP assessment also may not be a valid indicator for same-day postmanipulative pain and yet valid in directing therapy that has an effect on other clinical outcomes such as pain and function over time. The immediate pain and stiffness relief noted by both groups may also be attributable to placebo or nonspecific effects associated with assessment and treatment, concealing differences between groups that might have developed over time.

Despite the controversy surrounding motion palpation techniques and a call to abandon motion palpation techniques,⁴²⁵ the majority of chiropractors and other manipulating professions continue to use these procedures and consider them to be reliable and valuable methods.^{74,283,292,387} Although chiropractors may not be as informed and questioning of motion palpation procedures as they should be, it is clear that the evaluation of motion palpation procedures is incomplete. Although evidence is building that spinal intersegmental ROM palpation has poor interexaminer reliability, evidence does imply that it may have clinical value in context with other manual examination procedures, especially when incorporated with pain provocation. The results concerning the segmental motion palpation tools are mixed and, in a number of cases, are inconclusive. It is premature to discard a safe, low-cost, and potentially useful procedure. There is not enough evidence to draw firm conclusions on the validity of a number of manual examination procedures at this time. Further research into various palpatory regimens is necessary to evaluate and differentiate clinically relevant and useful palpation procedures. Within this context, it is important to remain informed and not rely excessively on any one procedure, but to use a combination of diagnostic procedures and allow the weight of evidence to build a clinical impression of the patient's problem.

It is also important to stress that all clinical procedures have imperfect diagnostic reliability and validity. Saal⁴²⁶ reviewed the literature on invasive spinal diagnostic tests (imaging studies, facet joint diagnostic blockade, anesthetic blocks, lumbar discography, NR blockade, sciatic nerve block, posterior ramus block, and subcutaneous injection) and concluded that there are inherent limitations in the accuracy of the diagnostic tests they evaluated. Although the reliability of all palpatory procedures is not at the same level as the reading of a thermometer or the taking of blood pressure (sphygmomanometry), it is comparable to cardiac auscultation.⁴²⁷ The agreement between observers and the phonocardiographic gold standard in the correct identification of S_4 and S_3 heart sounds was poor and the lack of agreement did not appear to be a function of the experience of the observers. The overall interobserver agreement for the detection of either S_4 or S_3 was little better than chance alone.⁴²⁷ Yet cardiac auscultation continues to be taught and used because of the low-risk clinical information it yields. The key is for each clinician to understand the strengths and limitations of the procedures they may use. Clinical decisions concerning the application and interpretation of diagnostic and therapeutic procedures

should be based on the best available evidence. For each procedure it is important to understand its comparative advantages, limitations, and costs. When available, knowing a diagnostic test's specificity, sensitivity, predictive value, and likelihood ratios can only lead to better risk-benefit assessments.

Sacroiliac Articulation

A separate and focused discussion of the SIJ is warranted based on the many specialized manual examination procedures that have been developed to evaluate its function.⁴²⁸ Dysfunction of the SIJ is defined as a state of relative hypomobility associated with possible altered positional relationships between the sacrum and the ilium.^{429,430} Motion palpation and pain provocation tests have been used in various forms and advocated by a number of professions employing manual therapies in the assessment and treatment of SIJ dysfunction.⁴³¹⁻⁴³⁵ However, the results of the reliability studies for mobility tests and pain provocation tests of the SIJ have been mixed. Laslett and Williams⁴³⁶ reported in 1994 that pain-provocation SIJ tests are reliable if performed in a highly standardized manner, using sufficient force to stress the SIJ. The results of a review of SIJ tests by van der Wurff and associates⁴³⁷ could not demonstrate reliable outcomes and concluded that there is no evidence on which to base acceptance of mobility tests of the SIJ into daily clinical practice. Hungerford and coworkers⁴³⁸ demonstrated that an altered pattern of intrapelvic motion could be reliably palpated and recognized during the Stork test (a modified interpretation of the Gillet test), and that the practitioner could distinguish between no relative movement and anterior rotation of the innominate during a load-bearing task.⁴³⁸

Tests designed to provoke a patient's pain appear to have more support for use in identifying patients who may have SIJ region dysfunction than do tests presumed to measure SIJ alignment or movement.⁴³⁹ Provocation SIJ tests are more frequently positive in back pain patients than the accepted prevalence of SIJ pain.⁴⁴⁰ This indicates that individual tests may be confounded by a number of false-positive responses. Laslett and colleagues⁴⁴¹ tested provocation tests and found that any two of four positive tests (distraction, compression, thigh thrust, or sacral thrust) or three or more of the full set (distraction, compression, thigh thrust, sacral thrust, and Gaenslen sign) were the best predictors of reducing or abolishing a patient's pain by intra-articular SIJ anesthetic injection. They further concluded that when all of the SIJ provocation tests are negative, painful SIJ pathologic conditions may be ruled out, suggesting that provocation SIJ tests have significant diagnostic utility.

Arab and coworkers⁴⁴² evaluated intraexaminer and interexaminer reliability of individual motion tests and pain provocation tests for the SIJ and found both to have fair to substantial reliability. They also looked at "clusters" of motion palpation or provocation tests and found moderate to excellent reliability. Intraexaminer and interexaminer reliability of composites of motion palpation and provocation tests were also considered substantial to excellent. They therefore concluded that composites of motion palpation and provocation tests together have reliability sufficiently high for use in clinical assessment of the SIJ.⁴⁴²

Manipulative treatment methods for the SIJ are based explicitly or implicitly on the presumption that some biomechanical dysfunction causes the SIJ or its associated soft tissues to become painful. This hypothesis may be questioned because the means for identifying dysfunction are based on an evidential base with disputed or conflicting results concerning reliability and validity of SIJ dysfunction tests.⁴⁴³

BONY PALPATION

The major goal of bony palpation is to locate bony landmarks and assess bony contour for any joint malpositions, anomalies, or tenderness. Typically, the palmar surfaces of the fingers or thumbs are used because they are richly endowed with sensory receptors. Light pressure is used for superficial structures, gently increasing pressure for deeper landmarks.

During spinal palpation, the pelvis, lumbar, and thoracic regions are customarily evaluated while the patient is in the prone position and the patient's cervical spine is evaluated in the sitting or supine position. The spinous processes in the entire spine—the articular pillars in the cervical spine, the transverse processes in the thoracic spine, and the mammillary processes in the lumbar spine—are palpated for tenderness and compared for contour and alignment (Figure 3-18). The cervical articular pillars and thoracic

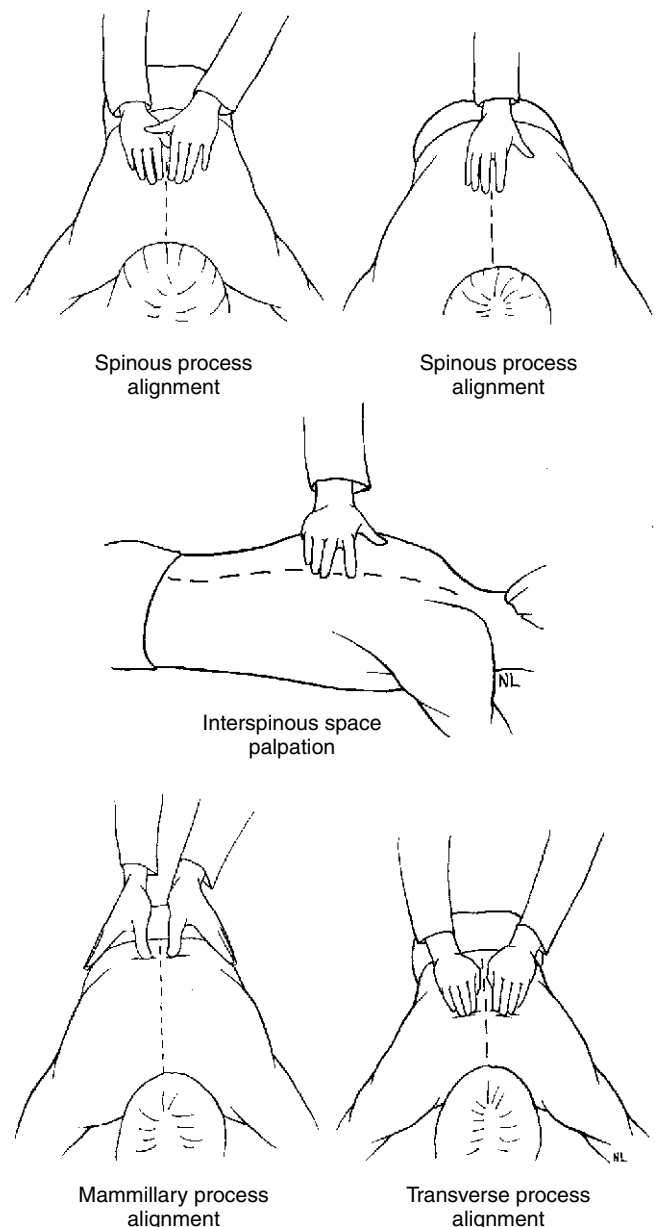


Figure 3-18 Palpation for bony tenderness and alignment of segmental spinal landmarks.

transverse process are both palpated through overlying muscular layers, and tenderness in these structures must be differentiated from tenderness in overlying soft tissues. The lumbar mammillary processes are not directly palpable in most individuals. They are located by a sense of deep resistance palpated through the overlying muscular layer. Individual motion segments are often located relative to these bony landmarks, and it is important to appreciate the anatomic relationship of the transverse processes to the corresponding spinous processes (Figure 3-19).

Tenderness over articular landmarks is an important potential sign of JSDSs. Of all the diagnostic signs of JSDS, palpation for tenderness appears to be the most reliable.^{244,255,256,382,444-446} However, joint dysfunction is not always synonymous with joint pain. Dysfunction may or may not directly cause joint pain. Although JSDS is commonly associated with pain, chronic dysfunctions may be nonpainful, but potentially create a region of altered mobility that can predispose to joint strain and pain elsewhere.

Misaligned articular structures may implicate the presence of joint subluxation/dysfunction, but apparent joint malpositions may result from anomaly or compensation without dysfunction. Spinal landmarks, especially the spinous processes, are prone to congenital or developmental malformation. Disrelationship between adjacent spinous processes can be falsely positive and cannot be relied on to represent true misalignment. Furthermore, the spine functions as a kinetic chain, and disease or dysfunction at one level may force adaptational alterations in neutral alignment at adjacent levels. These sites of compensational change may palpate as being malpositioned (out of ideal neutral alignment), yet have normal pain-free function. Static bony palpation does not ascertain joint mobility or the full extensibility of the articular soft tissues and cannot distinguish normal compensation from joint subluxation/dysfunction.

In the spine, the spinous process and interspinous spaces are commonly palpated for tenderness to screen for a possible level of segmental pathology or dysfunction. The relationship between spinous and interspinous tenderness and dysfunction is speculated to result from reflex sensitivity in tissues with shared segmental innervation (allodynia) or from mechanical deformation in structures attaching at these bony sites.

Remember that bony tenderness may result from many different pathologic processes such as bone infection, neoplasia, osteo-

porosis, and fractures. In addition, the spinous process may be tender whether the joint is hypomobile, hypermobile, or unstable. For the previously outlined reasons, suspected malpositions or bony tenderness must be associated with other clinical signs before an impression of joint subluxation/dysfunction is formed.

SOFT TISSUE PALPATION

One of the commonly stated diagnostic characteristics of the manipulable spinal lesion is altered segmental tissue tone and texture. The major function of soft tissue palpation is to determine the contour, consistency, quality, and presence or absence of pain in the dermal, subdermal, and deeper “functional” tissue layers. The dermal layer incorporates the skin; the subdermal layer incorporates subcutaneous adipose, fasciae, nerves, and blood vessels. The functional layer consists of the muscles, tendons, tendon sheaths, bursae, ligaments, fasciae, blood vessels, and nerves.

Palpation of the dermal layer is directed toward the assessment of temperature, moisture, motility, consistency, and tissue sensitivity (e.g., hyperesthesia and tenderness). Palpation techniques involve light, gentle exploration of the skin with the palmar surfaces of the fingers or thumbs. When manually assessing temperature of superficial tissues, the dorsum of the hands is typically used (Figure 3-20). Motility and sensitivity of the dermal layer may also be assessed by the technique of skin rolling (see Figure 3-20).

The subcutaneous and deeper functional layers are explored for internal arrangement, contour, consistency, flexibility, and response to pressure. The deeper soft tissues are usually investigated with the fingertips or thumbs (Figure 3-21). Palpation of paraspinal soft tissues is customarily performed immediately after bony palpation. The cervical spine is customarily examined with the patient in the supine or sitting position and the lumbopelvic and thoracic regions in the prone position.

The palpatory investigation of the functional layer is the decisive element in the soft tissue investigation for signs of joint dysfunction. Suppleness and flexibility of muscle and connective tissues are important and necessary for proper functioning of the joint systems of the body. Muscular and myofascial dysfunction are considered to be common factors in the pathogenesis of somatic and joint pain syndromes.^{118,447} Segmental tissue texture

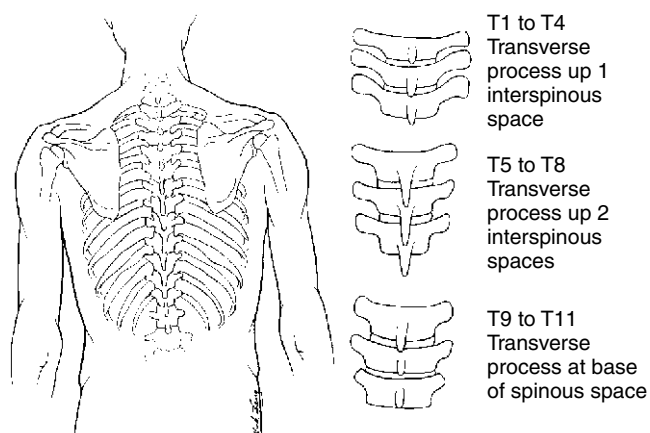


Figure 3-19 The structural relationship between thoracic spinous processes and transverse processes.

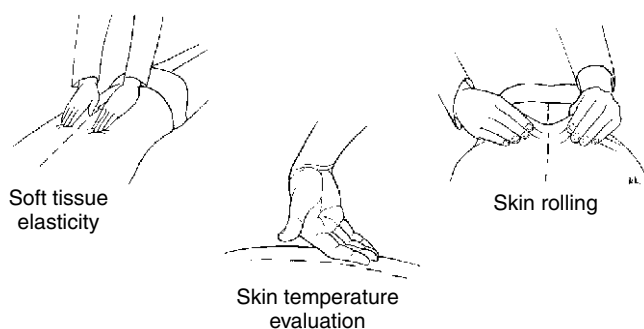


Figure 3-20 Assessment techniques for evaluating alterations in temperature, tenderness, tone, and texture of the superficial layer of the soft tissues.

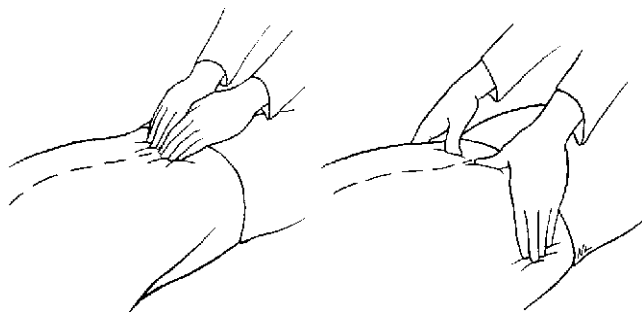


Figure 3-21 Assessment techniques for evaluating tone and texture in the deep paraspinal soft tissues using fingertips.

changes may include abnormal hardness, bogginess, or ropiness of the underlying paraspinal muscles.⁴⁴⁸ The reliability and accuracy of palpation to detect muscle dysfunction are not well established in the scientific literature.^{121,449}

The presence of soft tissue pain and asymmetric tone is regarded as an important indicator of joint dysfunction.⁴¹⁹ Grieve⁴⁴⁹ suggests that it may be the objective findings of muscle abnormality (palpable nodules, bands, or stringiness) and the presence of muscle tenderness that represent external evidence of changes in peripheral tissues related to joint problems. Furthermore, muscle pain is sometimes acute and surprisingly quite unknown to the asymptomatic patient until made manifest by careful localized palpation. Nilsson³⁸² found acceptable reliability of palpation for cervical erector spinae muscle tenderness using a grading pain scale of 0 to 3 that incorporated both verbal and nonverbal responses from the patient. Christensen and colleagues⁴⁵⁰ also reported good interexaminer reliability for thoracic paraspinal tenderness. The interexaminer agreement for the detection of tissue texture changes within muscle tissue appears to be less reliable than the detection of tenderness.¹²¹

In health, normal neuromuscular coordination is accepted as unremarkable; only in dysfunction does the underlying complexity of movement become apparent and the disturbance of reciprocal muscle action become manifest.⁴⁴⁹ Moreover, abnormal soft tissues patterns and presentations may persist after joint function has been restored. Although chronic muscle imbalance has a role in initiating and perpetuating joint problems and somatic pain, it may be secondary to stresses imposed by ligamentous failure, denervation, or reflex inhibition from pain. Adjustments of the joint without attention to the supporting and controlling effects of the soft tissues will likely result in recurrence of joint dysfunction.

Soft tissue asymmetries may also result from congenital or developmental variations or be the product of nonmanipulable disorders. Accordingly, any noted soft tissue abnormalities must be assessed within the context of a broader examination to be clinically significant. Instructions and tips on the use of static bony and soft tissue palpation are included in Boxes 3-7 and 3-8.

MOTION PALPATION

Motion palpation is a procedure in which the hands are used to assess mobility of joints. It is a skill that depends not only on psychomotor training but also on an understanding of the local functional anatomy, biomechanics, and pathomechanics. Each

BOX 3-7 How to Use Palpation Tools

Use the least pressure possible. Your touch receptors are designed to respond only when not pressed too firmly. Experiment with decreasing pressure instead of increasing pressure, and your tactile perception may improve. Try not to cause excessive pain if possible. Pain may induce protective muscle splinting and make palpation more difficult. Try not to lose skin contact before finishing palpation of the area. Use broad contacts whenever possible. For deep palpation, use broad contacts to reach the desired tissue, then palpate with your palpation finger, keeping the overlying tissue from expanding with the other fingers of your palpation hand. Close your eyes to increase palpatory perception.

BOX 3-8 Palpation Hints and Comments

Concentrate on the area or structure you want to palpate; do not palpate casually. Do not let your attention be carried away by unrelated sensations. Concentrate on your fingers; do not feel what you see or expect to feel. Keep an open mind and do not deceive yourself; never let your mind “out palpate” your fingers. Establish a palpation routine and stay with it. Take every opportunity to add to your tactile “vocabulary” through comparative experiences.

individual extremity joint and spinal region has its unique patterns and ROMs that must be learned if the chiropractic student is to master the art of motion palpation.

Motion palpation covers a collection of manual examination procedures that are customarily divided into techniques designed to assess active, passive, and accessory joint movements. Active movements are internally driven and are the result of voluntary muscle contraction. During active movement assessment, the doctor may help guide the patient through a given motion, but the patient provides the muscular effort necessary to induce joint movement. The range of active joint movement is determined by the joint's articular design and the inherent tension and resilience in its associated muscular, myofascial, and ligamentous structures. Greenman³³⁷ has labeled the end point of active joint movement as the *physiologic barrier*. (Figure 3-22).

In contrast, passive joint movements are involuntary movements. With the patient in a relaxed position, the examiner carries the joint through its arc of available motion. The range for passive joint movement is somewhat greater than the range for active joint movement because of decreased muscle activity (see Figure 3-22). The range of passive joint movement also depends on articular design and flexibility of related articular soft tissues.

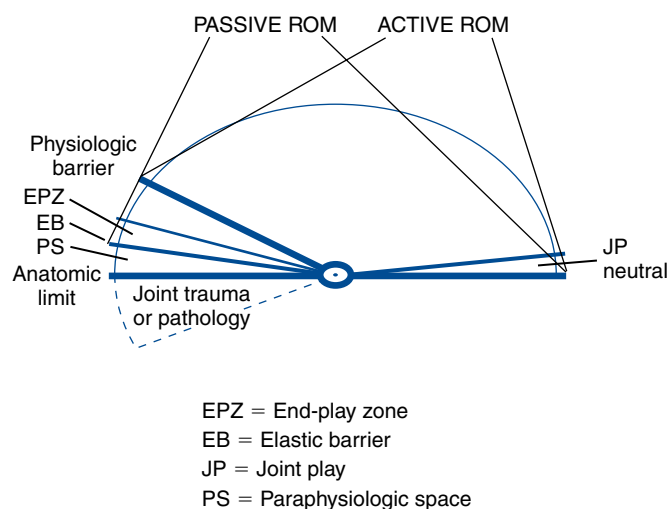


Figure 3-22 Joint motion starting from a neutral position. The first motion evaluated from a neutral starting position is joint play (JP). JP is a component of active and passive joint motion. It is induced by the examiner and represents the give and flexibility of the joint capsule. Active range of motion (ROM) represents the movement that is actively produced by the patient. Passive ROM represents the motion produced by the examiner. It is usually slightly greater in range because the patient's muscles are not active but relaxed. Toward the end of passive movement the end-play zone (EPZ) is encountered. The EPZ represents the increased resistance that is felt as the joint's elastic limits are reached. The elastic barrier represents the end point of the joint's elastic limits and the point at which additional movement is only possible after joint surface separation. Joint surface separation at this point usually occurs only after joint cavitation. After cavitation, the paraphysiologic space (PS) extends the passive ROM. At the end of the PS, the joint's anatomic limits are encountered. If the joint is carried beyond its anatomic limit, injury results.

As the limits of passive joint movement are approached, additional resistance is encountered as the joint's elastic limits are challenged. Movement into this space, the EP zone (EPZ) (see Figure 3-22), may be induced by forced muscular effort by the patient or by additional overpressure (EP) applied by the examiner. If the forces applied at this point are removed, the joint springs back from its elastic limits. Movements into this region are valuable in assessing the elastic properties of the joint capsule and its periarticular soft tissues.

Movement beyond the EPZ is possible, but usually only after the fluid tension between synovial surfaces has been overcome. This process is typically associated with an articular crack (cavitation). Sandoz⁵⁰ has labeled this as the *zone of paraphysiologic movement* and identified its boundaries as the *elastic and anatomic barriers* (see Figure 3-22). In circumstances in which the joint capsule is especially flexible, joint separation may occur without cavitation. The loose capsule allows for separation without fluid tension build-up between articular surfaces.⁴⁵¹

The labeling of the postcavitation increase in joint movement as *paraphysiologic* can be misleading. Although the paraphysiologic space (PS) aptly identifies an area of increased movement, it is still within the joint's elastic range and anatomic limits. Movement into this space does not induce joint injury. However, if the outer boundaries (anatomic limits) of the PS are breached, then plastic deformation and joint injury may occur⁵⁰ (Figure 3-23).

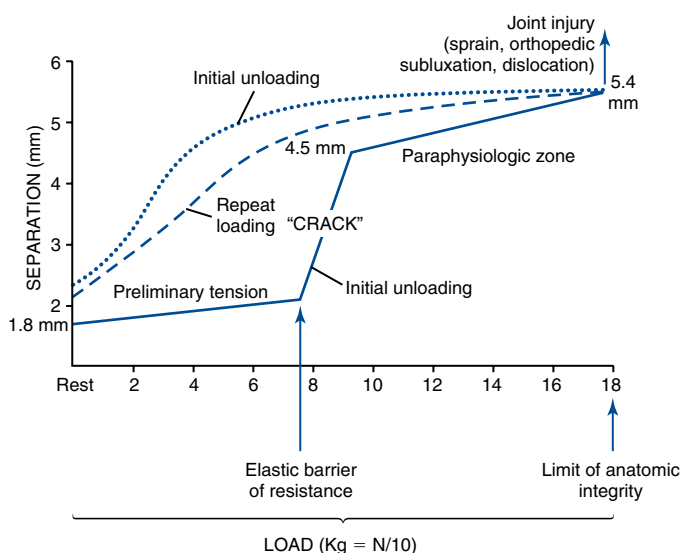


Figure 3-23 Increased movement that occurs after joint cavitation. The solid line represents the initial loading of the joint and the increased joint separation and movement that occurs only with cavitation. The broken line illustrates that repeated loading of the joint will induce the same amount of joint separation without joint cavitation.

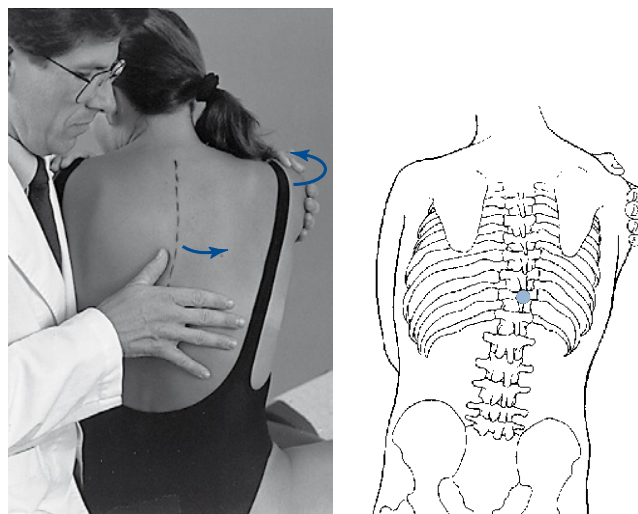


Figure 3-24 Assessment of segmental range of motion (e.g., midthoracic left axial rotation). The small circle located on the skeletal drawing represents the location of the thumb contact traversing the left side of the T10 and T11 spinous processes.

Restrictions of joint motion may occur at any point within the joint's ROM. They may be minor or major in nature and encountered within the joint's active or passive range. Restrictive barriers encountered within the joint's active ROM are primarily a result of myofascial shortening.³³⁷ This may be a product of muscle splinting, hypertrophy, aging, or contracture. Restrictive barriers to movement at the end range of passive motion are more indicative of shortening in the joint capsule and periarticular soft tissues.

During the performance of motion palpation, the examiner characteristically uses one hand to palpate joint movement (palpation hand) while the other hand (indifferent hand) produces or guides movement. The palpation hand establishes bony or soft tissue contacts over the joint as attention is directed to the assessment of joint range, pattern, and quality of movement (Figure 3-24).

When assessing joint motion, the palpator is evaluating the quality and quantity of movement from the starting or zero point to the end range of passive movement. In spinal evaluation, the landmarks commonly used are the spinous processes, articular pillars, transverse process, rib angles, and mammillary processes. During spinal palpation the examiner can attempt to assess the ROM of a single spinal motion segment or take broader contacts to assess a spinal region and several joints at a time.

Accessory Joint Motion

Accessory joint movements are necessary for normal function. They are small, involuntary movements made possible by the give within the articular soft tissues of each synovial joint. Joint surfaces do not form true geometric shapes with matching articular surfaces. As a result, movement occurs around a shifting axis, and the joint capsule must allow sufficient play and separation between articular surfaces to avoid abnormal joint friction.

Accessory joint movements are evaluated by the procedures of JP and EP.^{48,53} EP evaluation is the qualitative assessment of resistance at the end point of passive joint movement, and JP is the assessment of resistance from a neutral or loose-packed joint position.⁶¹ Both motions depend on the flexibility (play) of the articular soft tissue and are not distinguished by some authors.^{48,53} Rather, EP is considered to be JP delivered at the end range of joint motion.

Joint Play. JP assessment is the qualitative evaluation of the joint's resistance to movement when it is in a neutral or loose-packed position. The loose-packed position allows for the greatest possible play between the joint surfaces and the best opportunity to isolate the joint capsule from the periarticular muscles (see Figure 3-22). JP assessment therefore is helpful in the isolation and differentiation of articular-based pain and dysfunction from nonarticular soft tissue disorders. It has also been proposed as an evaluative procedure for the clinical assessment of joint instability; it has demonstrated some validity in detecting excessive translational movements that may result from derangement of the joint's stabilizing structures.^{167,169}

JP is assessed by placing the tested joint in its loose-packed position, establishing palpating contacts over the joint, and inducing gentle shallow springing movements (Figure 3-25).

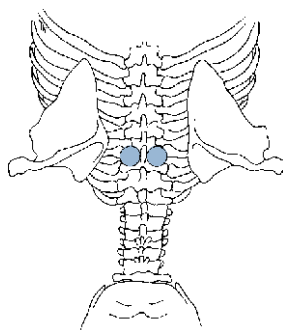
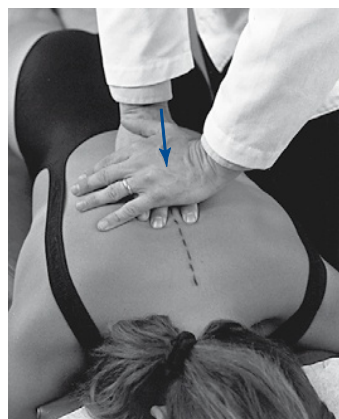


Figure 3-25 Assessment of joint play movement: posteroanterior glide, midthoracic segment. Circles indicate location of fingers over joints to be assessed.

This is most commonly done in the spine by placing the patient in a prone position and applying a P-A force. The true loose-packed position may not be achievable in the acutely injured or pathologic joint, and attempts to force a loose-packed position should be avoided. In such circumstances, the chiropractor should attempt to find the loosest possible pain-free position. JP movements are small in magnitude and vary by spinal region or extremity joint. It is therefore essential that the examiner, through practice, develop an appreciation for the regional and specific qualitative differences. As mentioned previously, this procedure has demonstrated good reliability for reproduction of pain (joint provocation and challenge) but poor reliability of determining hypomobility.²⁴⁶

JP procedures include methods in which the palpatory contacts are established over the joints to be assessed. Methods that involve contacts on both sides of the spinous process can be applied with opposing springing movements in attempts to specifically isolate a particular level of pain or dysfunction (Figure 3-26). During the performance of JP, the chiropractor should check for the presence or absence of pain, the degree of encountered resistance, and the quality of movement. JP should not induce pain; some resistance to movement should be encountered, but the joint should yield to pressure and spring back, producing short-range movements. Production of pain or increased resistance to JP movements suggests that the joint and its articular soft tissue may be the source of the patient's local spine complaint.

End Play. During EP assessment, the chiropractor is concerned with the symptomatic and qualitative assessment of motion through the EPZ (Figure 3-27). The EPZ is characterized by a sense of increasing resistance as it is approached (first stop) and a second firmer resistance (second stop) as its limits are approached (see Figure 3-27). In a healthy joint, it should be pain-free.

EP is assessed by applying additional overpressure to the specified joint at the end range of passive movement. During spinal EP assessment, a gentle springing force is typically induced through the palpation and indifferent hand contacts (see Figure 3-27). To execute end feel, the chiropractor should evaluate the point at which resistance is encountered, the quality of that resistance, and whether there is any associated tenderness.

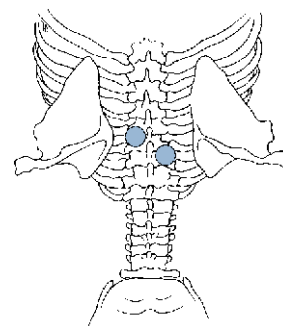
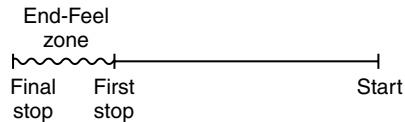
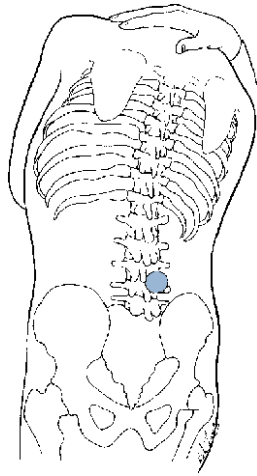


Figure 3-26 Assessment of joint play movement: counter-rotation between T4 and T5. Circles indicate placement of thumb contacts on adjacent spinous processes. Figure illustrates challenging the joint into left rotation.



3-27

Figure 3-27 Assessment of lateral flexion end play motion. The circle between L3 and L4 on the skeletal drawing represents the location of the thumb contact against the lateral surface of the spinous process.

EP evaluation is an important element in the assessment of joint function. In spinal joints, it has been reasoned that EP may be more informative than procedures designed to assess the ROM of individual spinal joints. This is based on the premise that qualitative changes in movement may be more reliably evaluated than quantitative changes, especially in the spine where the joints are deep and less accessible to palpation and the segmental ROM is normally small.⁴⁵² A recent qualitative literature review evaluated this question and did not confirm a significant advantage for EP over segmental ROM. The authors did note an advantage to EP over segmental ROM, but it did not rise to the level of being statistically significant.³⁹¹

However, the number of quality studies is very limited and further evaluation of EP reliability and validity as compared with segmental ROM is needed before conclusive statements are made.³⁹¹

Each spinal region or extremity joint has characteristic EP qualities that are determined by the local bony and soft tissue anatomy (physiologic end feel). For example, elbow extension has a hard, bony end feel produced by the bony impact of the olecranon on the humerus, and elbow flexion has a soft springy end feel produced by the impact, or compression, of soft tissues on the arm and forearm. What may be a normal EP at one joint may be a pathologic EP at another. A hard, bony EP to elbow flexion might indicate a fracture or an intra-articular blockage, and a soft, springing EP to elbow extension might indicate joint effusion. Physiologic and pathologic EPs have been tabulated for the spine and extremity joints and are outlined in Box 3-9.

BOX 3-9

Normal and Abnormal End Feels

CAPSULAR

Firm but giving; resistance builds with lengthening, like stretching a piece of leather

Example: lateral flexion of spine; external rotation of shoulder

Abnormal example: capsular fibrosis or adhesions leading to a capsular pattern of abnormal end feels, see Table 3-5

LIGAMENTOUS

Like capsular, but may have a slightly firmer quality

Example: knee extension

Abnormal example: noncapsular pattern of abnormal resistance as a result of ligamentous shortening

SOFT TISSUE APPROXIMATION

Giving, squeezing quality; results from the approximation of soft tissues; typically painless

Example: elbow flexion

Abnormal example: muscle hypertrophy, soft tissue swelling

BONY

Hard, nongiving abrupt stop

Example: elbow extension

Abnormal example: bony exostosis, articular hypertrophic changes

MUSCULAR

Firm but giving, builds with elongation; not as stiff as capsular or ligamentous

Normal example: hip flexion

MUSCLE SPASM

Guarded, resisted by muscle contraction; muscle reaction should be felt. The end feel cannot be assessed because of pain or guarding

Abnormal example: protective muscle splinting that is a result of joint or soft tissue disease or injury

INTERARTICULAR

Bouncy, springy quality

Abnormal example: meniscal tear, joint mice

EMPTY

Normal end feel resistance is missing; end feel is not encountered at normal point, or the joint demonstrates unusual give and deformation

Abnormal example: joint injury or disease leading to hypermobility or instability

Loss of normal EP elasticity is thought to be indicative of disorders within the joint, its capsule, or periarticular soft tissue. Abnormal EP resistance or increased pain is considered a significant finding in the determination of JSDs and directing adjustive vector. Adjustive therapy is commonly applied in the direction of encountered resistance in an attempt to restore normal mobility.

Cyriax²² has suggested that EP assessment is particularly valuable in isolating the integrity of the joint capsule. He has proposed that injuries or disorders that lead to contractures of the joint capsule will lead to predictable patterns of JP or EP restrictions in multiple ranges. Each joint purportedly has its own characteristic capsular pattern of restricted movement that indicates capsular involvement (Table 3-4). Injuries or contractures in only one aspect of the capsule do not necessarily follow this typical pattern and may affect movement in only one direction.

Loss of normal EP resistance (empty end feel) is also clinically significant because it is a potential manifestation of joint hypermobility or instability. Injuries or disorders that lead to elongation of the joint's stabilizing structures may lead to a loss of normal end-range resistance. Although an empty EP is indicative of possible clinical joint instability, segmental muscle splinting in the symptomatic patient may mask its presence.

TABLE 3-4 Capsular Patterns

Joint	Pattern*
Spine	Ipsilateral rotation and contralateral lateral flexion
Hip	Internal rotation-abduction, flexion-extension, adduction-external rotation
Knee	Flexion (great)-extension (slight)
Ankle	Dorsiflexion-plantar flexion
Metatarsophalangeal joint	Flexion-extension
Interphalangeal joint	Flexion-extension
Shoulder	External rotation-abduction-internal rotation-flexion
Elbow	Flexion-extension (pronation and supination, full range)
Distal radioulnar joint	Pronation-supination
Radioulnar carpal joint	Flexion-extension
Midcarpal joint	Extension-flexion
Thumb carpometacarpal joint	Abduction-extension
Metacarpophalangeal joint	Flexion-extension

*Patterns are in order of decreasing stiffness, except the spine, in which either is possible.

Joint Challenge (Provocation)

The assessment of pain during the application of JP and EP is often referred to as *joint challenging* or *joint provocation*. It is commonly used to isolate joint pain and to determine which segmental tissues placed under tension may be sensitive to mechanical deformation and responsible for the patient's pain. It often involves methods that attempt to isolate a given joint by applying counter-pressure across the joint.

In the spine, the counter-opposing pressures are commonly applied against the spinous processes. During this procedure, the vertebrae are stressed in different directions from their neutral positions, and directions of increased and decreased pain are noted (see Figure 3-26). Pain during movement is theorized to result from increased tension on injured or inflamed articular tissue. The absence of pain during movement indicates that tissues tractioned (challenged) in the direction of movement are not injured.

Provocation of joint pain during movement assessment in combination with tests for mobility have demonstrated promising results.^{389,416,453} Recent studies have indicated that P-A springing of the spine has good interexaminer reliability for the reproduction of pain, but mixed reliability for hypomobility. Some have suggested that the provocation of pain during joint movement assessments is the element responsible for reliably and accurately identifying symptomatic joint dysfunction.^{394,418} Others have suggested that pain provocation is an important tool, but they are concerned that reliance on this procedure would lead to an increased incidence of false-positive results.⁴¹⁹ Spinal pain is often poorly localized and commonly associated with sites of referred pain. The site of maximal tenderness is not always the source of the pathology or JSDs. Chiropractic theory implies that joint restrictions (fixations) are not necessarily symptomatic. Marked reduction in movement at one spinal level may induce increased compensatory hypermobility at other joints that may be more symptomatic than the restricted joints.

In addition, this procedure has been proposed as a method for determining the alignment of joint subluxations and direction of appropriate adjustment. The assumption is that pain is increased when subluxated vertebrae are pushed in directions that increase the misalignment (into lesion) and that pain is decreased in the direction that reduces the misalignment (out of lesion). For example, pressure exerted toward the right against the left side of a right-rotated T4 spinous process (left rotating the joint) purportedly would increase the misalignment and induce pain (see Figure 3-26). Pressure exerted toward the left, against the right side of the T4 spinous process would decrease the misalignment and not elicit discomfort. This approach has value in the evaluation and treatment of the acutely injured patient when the clinician is trying to determine how to induce joint distraction or reduce a traumatic subluxation without causing more tissue damage. However, whether this principle applies equally in all cases of joint subluxation/dysfunction is questionable.

If the rule of pain-free manipulation were applied in the case of post-traumatic joint dysfunction resulting from periarticular soft tissue contractures, would it accurately determine the appropriate direction of adjustment? Manual therapy applied in this scenario would logically be directed to stretch the shortened and contracted tissue. Tensile stretch applied to contracted and

inelastic tissue commonly induces some discomfort. Applying the rule of pain-free manipulation in this scenario would lead to an adjustment in the direction opposite the restriction. In this circumstance, the adjustment should be made in the direction of encountered joint restrictions, even if it is associated with some tenderness. Without attention to patient history and directions of encountered abnormal resistance, proper adjustive care may be missed.

From this discussion, the following generalizations about adjustive treatment for established joint dysfunction can be made:

- Adjustments should never be applied in directions of marked pain and splinting.
- Adjustments should not be applied in the direction of prestress that causes a peripheralization (radiation) of pain.
- Adjustments may be applied in directions of increased tenderness if associated with abnormal increased resistance.
- Adjustments may be applied in the nonpainful direction if directed to reduce joint subluxation or induce pain relief.

The procedures of segmental motion palpation have focused on the detection of joint pain and mobility, and although restricted joint and accessory joint motion may be indicative of joint dysfunction and sufficient evidence for joint manipulation, clinicians must guard against perceiving it as a diagnostic panacea. Isolation of a painful joint does not determine the cause of the pain or possible disease. Motion palpation cannot be used in all clinical situations (e.g., acute joint pain or injury), and certain disease states capable of producing joint restrictions may produce pathophysiologic change that contraindicates adjustive therapy.

As mentioned previously, segmental motion palpation is also subject to error and therefore should not be applied in isolation. However, Phillips and Twomey⁴⁵⁴ did find that motion palpation was highly sensitive and specific for detecting a symptomatic lumbar segment when they incorporated a subjective pain response from the patient. Nonetheless, the determination of joint dysfunction should be made in conjunction with other clinical findings. No one evaluative tool should be the sole source for therapeutic decisions. Goals, principles, and tips for conducting motion palpation are outlined in Boxes 3-10, 3-11, and 3-12.

PERCUSSION

Percussion plays a secondary role in the assessment of joint dysfunction. The area of greatest application is probably the spine, where a positive response may help localize a painful motion segment. Spinal percussion may be applied by the hypothenar of the clinician's hand or with a reflex hammer (Figure 3-28). In both circumstances, the clinician should apply a gentle percussive force sequentially to the spinous processes. A marked or persistent pain response to percussion may indicate an underlying fracture or a nonmechanical pathologic condition, whereas a mild pain response may indicate local irritation and dysfunction. When the response indicates a potentially serious disease, additional radiographic or laboratory procedures are necessary to differentiate a manipulable lesion from a nonmanipulable one.

BOX 3-10 Goals of Motion Palpation

To assess the following:

Quantity: How much does the joint move?

Quality: How does the joint move through its range of motion?

End feel: At what point is end feel encountered, what is the quality of resistance, and at what point does the motion stop?

Joint play: What is the quality of resistance? Is there too much or too little?

Symptoms: Are there changes in the amount or the location of pain during assessment and motion?

BOX 3-11 Principles of Motion Palpation

Joint movement is tested by assessing how two bony joint partners and their soft tissues move in relation to each other.

When evaluating segmental movement, test one movement at one joint around one axis in one plane on one side of neutral whenever possible.

Develop a pattern and test each motion segment being evaluated in sequence.

Move through the entire available range of motion; start and end at neutral. The singular assessment of end feel is an exception to this principle.

Motion must be performed slowly and smoothly with the minimal force necessary.

Compare mobility with the contralateral side and adjacent segments.

BOX 3-12 Motion Palpation Tips

Do not let soft tissue movement and tension changes fool you. They are important indicators of the amount of underlying joint movement, but it takes experience to evaluate them.

Concentrate and be alert from the beginning; valuable information is often gained early in the range of motion.

Where possible, contact both joint partners of the joint being evaluated. This can be done by using two fingers of the same hand, one finger of each hand, or one finger palpating both joint partners simultaneously, thereby crossing the joint space.

Your patient has to feel comfortable, relaxed, and safe.

Do not produce too much movement with your palpation hand. It helps focus your palpation forces, but it must also be free to palpate.

Your palpating finger applies minimal pressure, applies enough pressure so as not to lose firm contact with the bony prominence on the moving joint partner, and is an impartial observer.



Figure 3-28 Percussion of the spinous processes with a reflex hammer.

MUSCLE TESTING

Motor changes are characteristic of many neuromuscular conditions, making tests for muscle length and strength an integral part of the examination process. The testing of muscle structure and function requires knowledge of joint motion, origin and insertion of muscles, agonistic and antagonistic actions, and the ability to palpate the muscle or its tendinous attachments for tone and texture changes.

Muscle strength testing incorporates tests for strength and endurance. Endurance can be evaluated by the patient's ability to perform repeated movements or maintain static postures. Normative values for repetitive squatting, sit-ups, prone arch-ups, and sustained prone back extension have been established and are valid measures for measuring spinal fitness and treatment outcomes (Figure 3-29). Strength testing can be evaluated manually or with the aid of specialized equipment, such as computer-aided dynamometry (e.g., Biodex, Cybex, Med-X, and Promotron).

Manual muscle testing procedures have been extensively described for isolating specific muscle function.⁴⁵⁵ Manual resisted muscle tests are performed to assess the strength and sensitivity of muscle and its tendinous attachments (Figure 3-30). Any noted muscle weakness should be recorded and graded on a five-point system (Box 3-13). Muscle testing procedures are numerous, and descriptions of individual procedures are beyond the scope of this text. The reader is encouraged to refer to any number of excellent texts for detailed descriptions on how to perform individual muscle tests. Pain with muscle contraction may indicate a muscle injury, a joint injury, or a combined muscle and joint injury. Pain with isometric contraction generally indicates a muscle injury rather than a capsular injury.²² Isometric muscle contraction, however, may still produce some degree of joint compression and capsular tension. To differentiate a purely muscular injury from a capsular injury, passive joint movement and compression must be performed and their results compared with the response elicited during isometric muscle contraction.^{19,22}

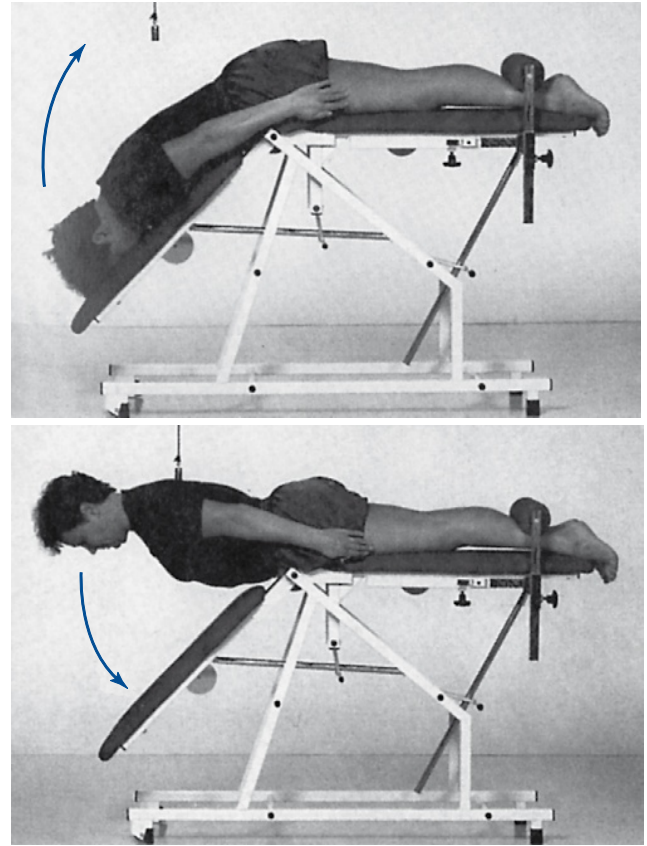


Figure 3-29 Repetitive arch-ups from a flexed position.

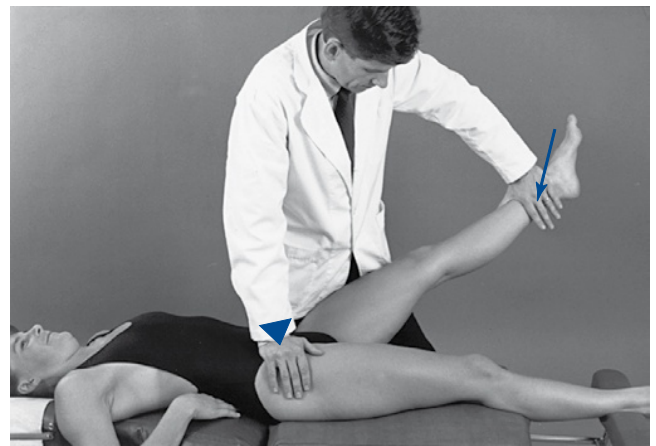


Figure 3-30 Resisted muscle tests evaluate strength and sensitivity at the tendinous attachments (e.g., left psoas muscle test.)

A capsular injury produces pain with passive and active movements as the capsule is elongated. A purely muscular injury produces pain with muscle contraction and muscle elongation, but passive shortening of myofascial tissue should not be painful.

The small segmental muscles of the back are not independently accessible to palpation or specific muscle tests. Injuries to the deep segmental muscle of the back cannot be easily differentiated from other injury or dysfunction of the spinal joints. Therefore, muscle testing in the back is used primarily to differentiate injuries to the large, nonsegmental back muscles from dysfunction or injury to the spinal joints and their associated soft tissues.

BOX 3-13 Five-Point Grading System for Muscle Weakness

- 5—Patient can maintain position against gravity and maximum examiner-applied resistance.
- 4—Patient can maintain position against gravity and minimal examiner-applied resistance.
- 3—Patient can maintain position against gravity.
- 2—Patient can move through arc of motion when gravity is lessened.
- 1—Muscle contraction is visible or palpable, but there is no movement of part.
- 0—There is no palpable or perceivable muscle contraction.

Within the chiropractic profession, manual muscle testing has also been used to evaluate spinal joint function and the health and function of other organ systems of the body. Central to the use of these procedures is the premise that changes in muscle strength can be affected by functional and pathologic changes in other tissues of the body.⁴⁵⁶ Muscle testing in this capacity is controversial and typically is used by professionals who use procedures associated with the technique of applied kinesiology.

The applied kinesiology technique proposes the use of manual muscle tests coupled with the use of applied vertebral pressures to determine the level and direction of spinal subluxation. This procedure is commonly referred to as the *vertebral challenge*.⁴⁵⁶

The vertebral challenge involves digital pressure applied by the examiner against spinal bony landmarks as the examiner simultaneously evaluates the strength of a selected muscle. The tested muscle is evaluated for its ability to resist torque and maintain a locked position. If the muscle gives way under pressure, the test is considered positive and the level of spinal contact is considered a site of dysfunction.

During testing, the contacted vertebra is pushed (challenged) in different directions. If the muscle gets weak, the assumption is that the segment is being directed more into a subluxated position. If the muscle remains strong or gets stronger, the assumption is that the vertebra is being pushed out of its subluxated position. It is believed that all muscles of the body are temporarily inhibited by stimulation of subluxated segments. Therefore, any muscle of the body can be selected as the “indicator muscle” during the use of this procedure.

This procedure is often performed as a rebound challenge. This procedure is based on the premise that subluxation syndromes are associated with hyperactivity in segmental muscles. The rebound challenge is performed by the application and quick release of spinal pressure.⁴⁵⁶ During testing the contacted vertebra is pushed (challenged) in different directions. The rebound phase is represented by the quick release of applied pressure.

If this method is used in a direction that stretches hyperactive muscle, it is assumed the hyperactive muscle will contract against a quick stretch and pull the vertebra farther into its malpositioned state during the rebound phase. If the vertebra is pulled farther in the direction of malposition, a weak muscle response is predicted. Therefore, a weak muscle response during the rebound phase indicates that a segment should be adjusted in the direction of the applied testing force.

The vertebral challenge and rebound vertebral challenge have not been extensively evaluated. The few studies that have been con-

ducted have demonstrated poor intraexaminer and interexaminer reliability and no responsiveness to adjustive treatments.^{457,458}

PROVOCATIVE (ORTHOPEDIC) TESTS

Provocative testing covers a wide range of manual testing procedures, many of which have already been discussed. Provocative procedures are tests that are conducted to reproduce a specific sign or accentuate pain. The major purpose of testing is to locate the anatomic site responsible for producing the patient's pain. Provocative orthopedic tests represent a separate category of named procedures designed to use movements or positions to localize the source and nature of the patient's disorder. The procedures are commonly labeled with the name of the original innovator (e.g., Kemp test) or carry a descriptive label (e.g., straight leg raise test).

Named provocative orthopedic procedures are not commonly cited because many procedures are not applicable to the identification of spinal subluxation/dysfunction syndromes. They are helpful in identifying the anatomic location of painful complaints and discriminating between mechanical, nonmechanical, and NR pain. They have demonstrated less value in discriminating between conditions.²⁹⁷ Orthopedic tests are also helpful in identifying possible contraindications to adjustive therapy and monitoring patients' response to treatment.⁴⁵⁹ Evans provides an excellent description of spine and extremity provocative orthopedic tests, including how they are performed and interpreted.⁴⁵⁹

RADIOGRAPHIC ANALYSIS

Radiographic assessment and determination of joint subluxation have been an integral part of chiropractic evaluation since the early 1900s.^{335,460,461} Ever since Sausser first made a full-spine exposure, the chiropractic profession has desired and sought out methods for marking x-ray films to identify manipulable lesions. The history of chiropractic marking procedures dates back to 1910⁴⁶² when it was first introduced in the Palmer School curriculum.

The early use of diagnostic x-ray examinations in the chiropractic profession centered on the assessment of biomechanical relationships and the measurement and description (listing) of spinal joint malpositions. To that end, the profession and many of its individual technique innovators have developed specific radiographic measurement techniques (spinography) designed to quantify and classify spinal malpositions and subluxations (Figure 3-31).^{32,128,461,463-467}

Although many “systems” to detect static subluxations on x-ray films have emerged over the years, these procedures remain controversial. Criticism and failure of the static x-ray marking systems come from trying to use quantitative measures on landmarks that vary and that are subject to geometric distortion.⁴⁶⁸ Moreover, the spine and its functional units are living, moving, and dynamic structures that depend on complex relationships among bones, ligaments, and muscles. Plain-film x-ray examination does not evaluate movement of the spine, nor does it directly assess the soft tissues.

Although the limitations of radiographic marking systems are well established⁴⁶⁹ (Box 3-14), static alignment abnormalities can have some significance when taken in context with other clinical, historical, and laboratory findings. In recent years, more emphasis has been placed on the dynamic concepts of the subluxation

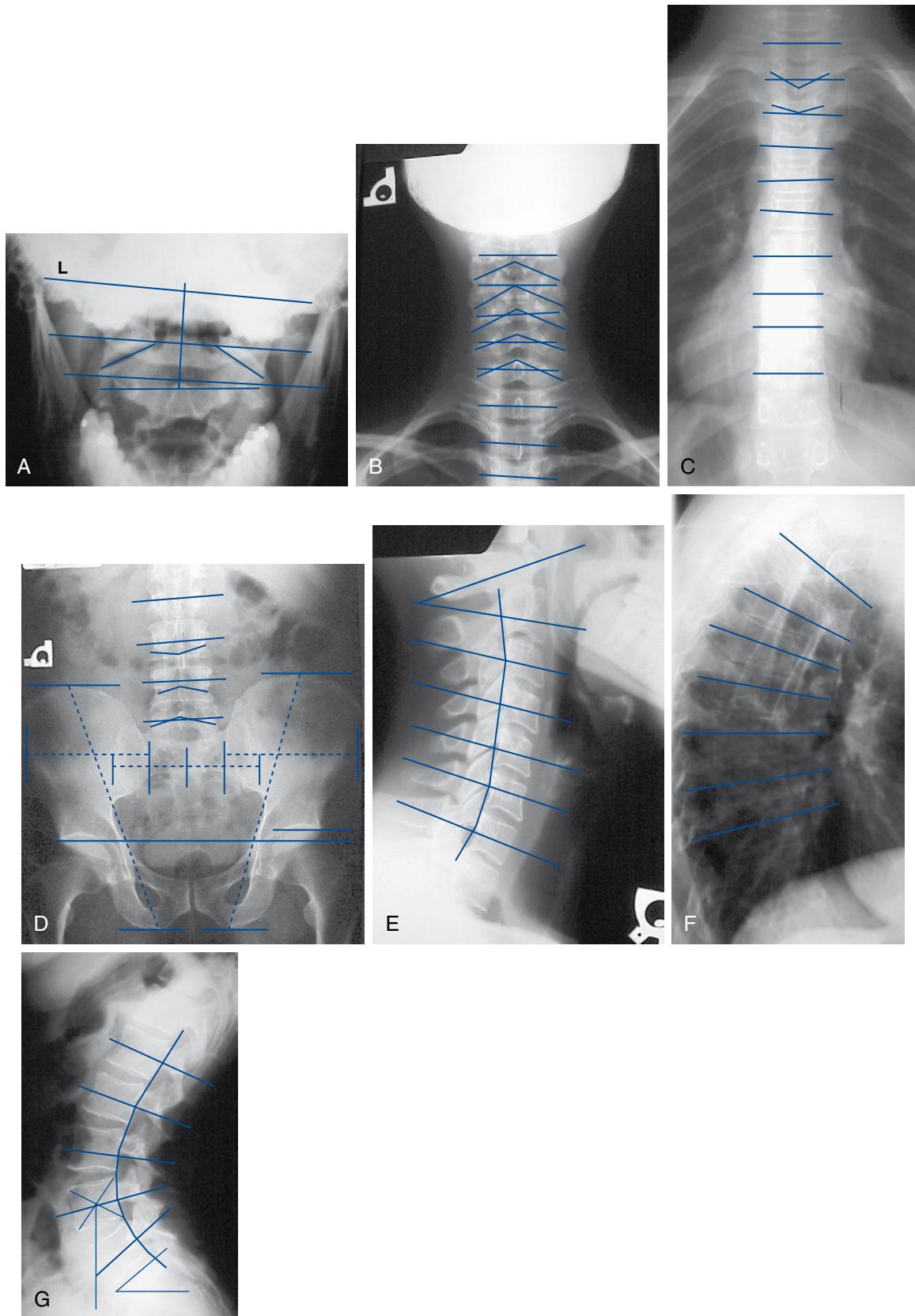


Figure 3-31 Static spinographic measures. **A**, Anteroposterior open mouth. **B**, Anteroposterior lower cervical. **C**, Anteroposterior thoracic and **D**, anteroposterior lumbar and pelvis. **E**, Lateral cervical neutral. **F**, Lateral thoracic and **G**, lateral lumbosacral.

BOX 3-14 Limitations of Radiographic Marking Systems

Anatomic asymmetry
Radiographic magnification
Radiographic distortion
Radiographic malpositioning
Static analysis of dynamic motion segments
Inaccuracy of instruments
Insignificant findings

BOX 3-15 Rationale for Radiography in Chiropractic Practice to Establish a Clinical Diagnosis

To evaluate biomechanics and posture
To identify anomalies
To screen for contraindications
To monitor degenerative processes

complex, in some cases totally disregarding static biomechanical relationships. Sandoz⁴⁷⁰ feels that this shift of emphasis is counterproductive. He suggests considering the mechanical, static, and dynamic concepts in harmony with the neurologic and reflex elements of spinal subluxation/dysfunction.

Over the years, the role of x-ray examination has been modified according to scientific and technical developments, as well as to philosophic tenets and beliefs. Sherman⁴⁶² summarized the clinical rationale for the use of x-ray examination in chiropractic (Box 3-15). Evidence-based diagnostic imaging practice guidelines have been developed.⁴⁷¹⁻⁴⁷⁴ They are intended to assist primary care providers, interns, and residents in determining the appropriate use of diagnostic imaging for specific clinical presentations. In all cases, the guidelines are intended to be used in conjunction with sound clinical judgment and experience. The goal of these guidelines is to avoid unnecessary radiographs, increase examination precision, and decrease health care cost without compromising the quality of care.⁴⁷¹ Ammendolia and co workers⁴⁷⁵ surveyed chiropractic colleges around the world to evaluate whether imaging guidelines were being taught and adhered to. The results of this study suggest that instruction provided at most chiropractic schools appears to adhere to evidence-based guidelines for LBP with respect to the use of routine radiography, full-spine radiography, and oblique views, but there appears to be some disparity between instruction and existing evidence for the use of radiography in acute LBP.⁴⁷⁵

Spinal X-ray Examinations

Historically, the use of spinal radiography examinations in chiropractic centered on the detection and quantification of the intervertebral misalignment.⁴⁷⁶ Proponents of radiographic evaluation for the detection of spinal subluxations claim that x-ray examinations are the best method for accurately determining the level and direction of vertebral malposition.⁴⁶⁰ They contend that chiropractors who do not use radiography to evaluate spinal subluxations

are at a disadvantage in determining and delivering indicated and safe adjustments. In the 1970s, this view produced a policy requiring chiropractors to demonstrate radiographically the presence of spinal subluxations to treat and receive reimbursement for Medicare patients. This policy has been recently modified and was rescinded in 2000 in favor of the PART multidimensional index for joint dysfunction.

Preadjustive x-ray examinations are also rationalized as necessary because the treatment incorporates the use of force. It is reasoned that the integrity and mechanical characteristics of the spine should first be screened radiographically before adjustments are made.⁴⁷⁶ This position is controversial and unsubstantiated. Screening x-ray examinations taken without clear clinical guidelines have not correlated with improved diagnosis or patient outcome.^{477,478} Furthermore, thrusting forms of manipulation have been used safely for centuries without the aid of x-ray examinations.

Spinal x-ray examinations are usually taken with the patient in an upright, weight-bearing position and should consist of two views, typically an anteroposterior and lateral projection. Traditionally, the alignment of the upper vertebrae is compared with that of the lower vertebrae, and any malpositions are recorded.^{32,128,460}

Full-spine radiographs are used primarily for biomechanical evaluation, including the assessment of individual motion segment alignment. Full-spine evaluations provide an integrated view of spinal biomechanics and are the method of choice in the evaluation of spinal scoliosis. Full-spine radiographs, however, compromise bony detail and should not be used as a routine procedure for the assessment of suspected local pathologic conditions.^{469,479,480}

“The clinical justification for the full-spine radiograph must insure that the benefit to the patient is greater than the radiation hazard. The film must be of such quality that the presence or absence of pathology can be determined.”⁴⁸¹ When indicated, consideration should be given to full-spine posteroanterior projections to improve visualization of the lumbar IVD spaces and to minimize exposure to the ovaries and breasts.^{31,461}

Although the majority of the profession uses some form of radiographic measurement and assessment of spinal subluxation, there is considerable controversy as to whether radiographic evaluation should play a significant role in the diagnosis of spinal subluxation syndromes.* Claims of accuracy in detecting minor joint malpositions may not be supportable against the technical limitations of radiography.[†] Inherent radiographic magnification and distortion, patient positional errors, and the exactness of the marking procedures are common concerns.

The lack of a consensus on the definition, pathophysiology, and pathomechanics of spinal subluxations further complicates the debate and analysis of x-ray marking procedures. Therefore, the clinical significance of these measurements is controversial and suspect. Radiographic measures should not be the primary criteria used to perform chiropractic care.⁴⁸⁹ However, if there were a clinical indication for taking a radiograph, it would be imprudent not to evaluate the x-ray examinations for biomechanical relationships and look for correlations to other clinical findings.

*References 335, 461, 463, 469, 479, 480, 482, 483.

†References 335, 461, 463, 479, 481, 484-488.

The process of critically evaluating radiographic marking procedures has only begun in the last several decades.^{335,478,490-495} The process is in its infancy, and a limited number of studies have been conducted. A significant number of procedures have yet to be evaluated. Although it is difficult to draw firm conclusions, it is possible to briefly summarize the present state of affairs.

First, many of the radiographic marking procedures used to evaluate segmental spinal alignment can be reliably performed.* However, most of the reliability studies do not include a full evaluation of all the steps involved in performing and determining segmental alignment. Many of the studies did not include patient positioning. Consequently, at this time it is difficult to conclude whether x-ray marking procedures are or are not reliable for identifying spinal motion segment subluxations.⁴⁶⁹

Although recent attempts have been made to address issues of spinographic reliability, very little has been done to investigate the validity of radiographic measurement in diagnosing and treating spinal dysfunction.^{338,470,476-478,496,497}

Spinal displacement analysis has not demonstrated the ability to identify an established clinical entity nor demonstrated its value as an independent outcome measure. The validity and clinical usefulness of static marking procedures for identifying treatable motion segment misalignment have not been demonstrated.⁴⁸⁸

A retrospective case analysis performed in 1990 identified only one postmanipulation segmental spinographic change, that being a reduction in retrolisthesis. There was no identified change in cervical lordosis, sacral base angle, lumbar lordosis, scapular angle, or Cobb angle.⁴⁸⁹ Yi-Kai and coworkers⁵⁰⁴ investigated the relationship between radiographic signs of subluxation in the cervical spine and their clinical diagnostic value. They concluded that there was little evidence to support the contention that signs of subluxation in the cervical vertebrae are diagnostically significant in identifying individuals with cervical pain. In addition, static marking procedures have not been found to discriminate between those with back pain and those without back pain.^{338,505}

Harrison et al⁴⁸³ reviewed the literature on the reliability and clinical value of spinal displacement analysis in plain-film x-ray examinations, concluding that x-ray line drawing is a reliable and effective outcome measure. The conclusion is based on their assertion that there is an ideal normal spinal configuration based on a mathematical model and that radiographic marking procedures can identify real spinal displacements. However, the vast majority of cited reliability studies were on curve measurements, not spinal segment position.

Haas and colleagues⁴⁶⁹ challenged Harrison and colleagues' conclusions⁴⁸³ by questioning the biologic plausibility of an ideal spine model and the authors' failure "to present any credible evidence for the validity, clinical utility and appropriateness for using these procedures." Haas and colleagues⁴⁶⁹ conclude that there is currently no justification for the routine use of radiographic spinal displacement analysis in clinical practice.

Functional X-ray Examination

The potential limitations of static radiographs in determining joint dysfunction has led to increased use of functional x-ray

studies.^{51,506-510} The principal attraction of functional x-ray examinations is the ability to assess joint mobility and identify disturbances in function that might not be represented by static films. Functional x-ray studies involve the evaluation of regional and segmental spinal movements by comparing range and pattern of movement at each segmental level. A series of three views are typically taken for each plane of movement evaluated: an end-range view in each direction and a neutral view. These views are then used to measure and evaluate restricted or aberrant segmental movements.

Although the use of dynamic x-ray examinations overcomes concerns about the inability to functionally assess the spine with static x-ray examinations, there remains considerable controversy as to their contribution in predicting back pain or differentiating those individuals with back pain from those without. Methods for measuring and classifying segmental motion abnormalities in the lumbar spine and cervical spine are in common use.^{51,164,506-508,511-513} Taylor⁴⁷⁸ suggests that functional radiography should be used to establish the presence of the following:

1. Segmental or global hypomobility
2. Segmental or global hypermobility
3. Segmental instability
4. Aberrant segmental or global motion
5. Paradoxical motion
6. Postsurgical arthrodesis

Flexion-Extension Radiographs. Those investigating the relationship between spinal pathologic conditions and segmental movement have demonstrated supportive evidence for the use of flexion-extension studies in the detection of spinal instability.^{167,478,506,514} Flexion-extension studies are used to identify excessive angular or translational movements between spinal segments (Figure 3-32).^{478,515,516} The amount of translation or angular movement necessary to define instability is not definitively established. Most references classify any flexion-to-extension translation greater than 3 to 5 mm as indicative of instability.⁴⁷⁸ Dvorak and colleagues⁵¹⁷ suggest that applying global overpressure at the end ROM during a functional x-ray examination may aid in identifying translational movement characteristics of instability.

Clinical validity studies have been done for flexion-extension radiographs of the lumbar and cervical spine.^{518,519} The conclusions were that the functional studies did show a tendency for the presence of hypomobility in patients with clinical problems, but they were not sufficient to aid in differentiating the underlying pathologic conditions.

In the cervical spine, flexion-extension studies are used most commonly to ascertain if a traumatic injury has resulted in

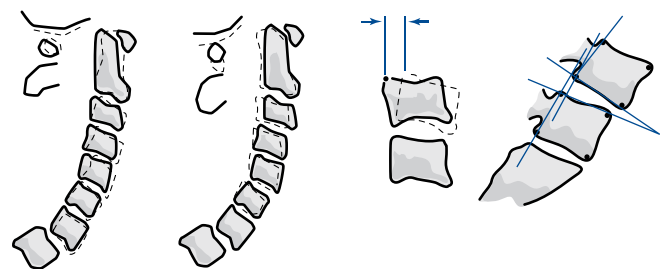


Figure 3-32 Evaluation of flexion and extension radiographs in the cervical and lumbar spine.

*References 265, 335, 464, 470, 476-478, 483-486, 490, 494-503

instability. Translational movements of more than 3 mm are considered a significant finding for instability of the cervical spine. In the cervical spine, an overlay method may be used for templating flexion and extension (see Figure 3-32).

Lateral-Flexion Radiographs. Lateral-flexion (side-bending) radiographs are used predominantly in the evaluation of the lumbar spine. Interpretation of the films incorporates the use of lines and angles that are drawn on the films for the purpose of quantifying and gauging comparative quality of joint motion.

The total range of regional lateral flexion is determined by extending a line from the superior end plate of the uppermost vertebra and the inferior end plate (or sacral base) of the lowest vertebra in the concavity of the curve. Perpendicular lines are constructed from each of these with the angle formed at their intersection, establishing the limit of lateral flexion (Figure 3-33).

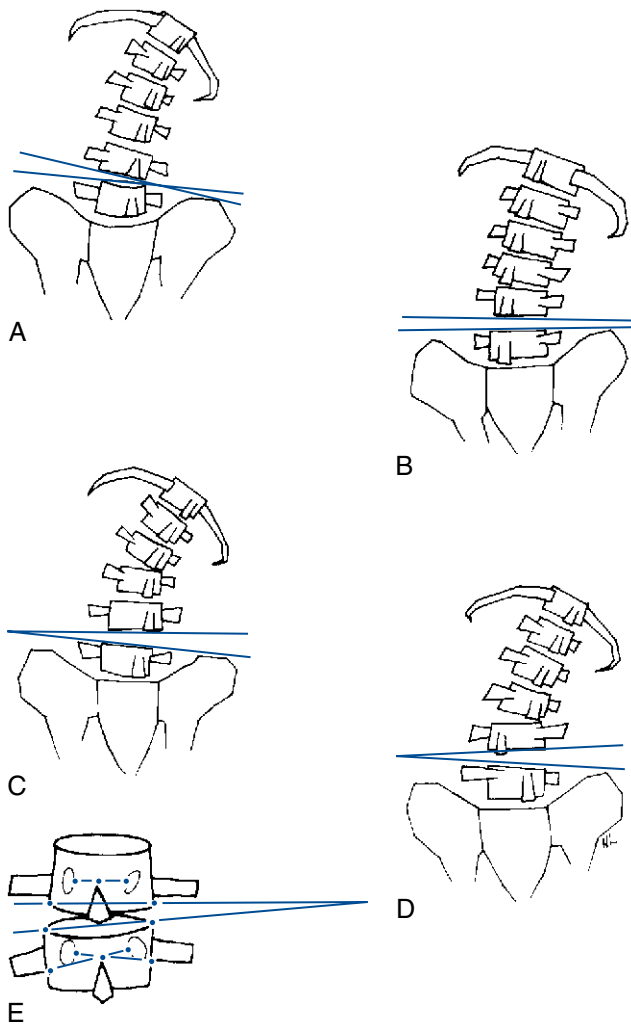


Figure 3-33 Evaluation of functional lateral bending radiographs in the lumbar spine demonstrating movement patterns. **A**, Type I, lateral bending with contralateral rotation. **B**, Type II, lateral bending with ipsilateral rotation. **C**, Type III, contralateral bending with contralateral rotation. **D**, Type IV, contralateral bending with ipsilateral rotation. **E**, Segmental measurement methods for determining rotation in millimeters and lateral flexion in degrees. (**A–D** from Grice A, Cassidy D: *J Manipulative Physiol Ther* 2:18, 1979; **E** from Haas M, Niyendo J, Peterson C: *J Manipulative Physiol Ther* 13[4]:179, 1990.)

The films can then be marked for segmental rotation and lateral flexion. The body-lamina junction is used for rotation and the end plate angulation for lateral disc wedging. Aberrant lateral flexion can be assessed by using superior end plate lines and evaluating if they converge toward the side of lateral bending (see Figure 3-33).

Early investigation into the value of functional radiography did identify its merit in the diagnosis of sciatica,⁵²⁰ although abnormal lumbar motion was also noted in asymptomatic patients. Vernon⁵⁰⁹ concluded that there was a higher prevalence of abnormal lateral bending patterns in symptomatic subjects, but Phillips et al³²⁹ and Haas and colleagues^{332,333} failed to demonstrate a relationship between abnormal spinal motion and patients suffering from LBP.

Although these procedures have demonstrated limited predictive value in differentiating individuals with back pain from those without, their value in managing patients with back pain has not been fully assessed. Using side-bending stress x-ray studies to help ascertain abnormalities of intersegmental motion in individuals with back pain in theory, may affect clinical decision-making in a manner that improves patient outcome. In this context, evaluation would be qualitative and quantitative. The detection of hypomobility, paradoxical motion (reversal of an unexpected motion or aberrant motion), or excessive motion would have precedence over exact measurements.

Identifying levels and directions of decreased movement might affect decisions on where and how to make adjustments in ways that improve patient outcome. This may be particularly applicable in individuals with persistent pain or patients who have not been responsive to treatment. The answers to these questions await further research.

Videofluoroscopy

Videofluoroscopy (VF) of the spine is another radiographic procedure that has been proposed as a potential tool for the assessment of segmental spinal motion. Before the development of VF, cineradiography (CR) was the main radiographic method used to evaluate spinal motion. Fielding⁵²¹ first described its use for the cervical spine, and Illi was the first to use CR in the chiropractic profession to study spinal segmental and sectional motion. He was followed by Rich and Goodrich in the 1960s. Howe^{57,522} performed numerous studies, and this procedure became an experimental procedure at a number of institutions. The major drawback to CR was that it involved taking 16-mm movies during which substantial radiation exposure (often exceeding 10 or 20 radiation absorbed doses) was applied to the spine.

VF development has led to improvements in image intensifiers and digital recording technology that has resulted in far fewer radiation doses and increased interest in recent years.^{523,524} VF has the capabilities to measure the full arc of motion and therefore provides information on the quality of motion in addition to the ROM. This allows the clinician to see aberrations in the mid-ROM, as well as at the extremes. Advocates of VF suggest that these studies provide objective evidence of biomechanical abnormalities not seen with other studies. This technology has made significant advances, and the new techniques of digital VF

(DVF) have dropped the radiation exposure rates considerably below those of the conventional x-ray examination.^{525,526} When appropriate equipment and calibration are used, the procedure has demonstrated promising interobserver and intraobserver reliability, with measurement accuracy between 1 and 2 degrees.⁵²⁵⁻⁵²⁷

Although DVF holds significant promise in the assessment of spinal mechanics, it is presently in the investigational stage, without established clinical protocols for use. It should be stressed that spinal VF is a special test with several limitations and disadvantages (Box 3-16). Much research is necessary to precisely define the role of VF in chiropractic.

In clinical practice, VF should be considered an experimental procedure, and its use should be reserved for complex cases that fail to respond, that respond poorly to a trial of conservative management, or in which suspected ligamentous damage leading to instability has occurred. Growing concern about the inappropriate use of VF has led to the formation of protocols for the use of VF in chiropractic by the American Chiropractic College of Radiology, a branch of the ACA. These protocols should be followed when contemplating the use of VF.⁵²⁸

Clinical Use of X-ray Examination

The clinical utility of static and functional radiographs might be improved if these procedures were placed in a proper clinical perspective and considered a component of evaluation and not a pathognomonic indicator of JSDs. With further refinement, they may eventually parallel a role provided by specialized imaging techniques in the structural detection of IVD derangement.

For example, the presence of IVD derangement on a CT scan or an MRI indicates the presence of anatomic derangement of the IVD, but it does not confirm that the disc derangement is of clinical significance. The incidence of radiographically detected disc derangement in asymptomatic patients is significant (24% to 37%), suggesting that there is a poor correlation between mechanical disc derangement and morbidity.⁵²⁹ Within this context, it becomes apparent that the imaging findings must be matched to the clinical presentation and physical findings before a final impression is established. The role of x-ray examination in the evaluation of spinal subluxation/dysfunction syndromes (JSDS) should play a similar purpose. Radiographic findings alone cannot identify whether a given joint subluxation/dysfunction is clinically relevant and worthy of treatment. They must be placed within the context of the physical examination and patient complaints.

This discussion on the use and application of radiography has been directed toward its relationship to the detection of joint subluxation/dysfunction. This is not meant to imply that chiropractors use x-ray examinations to detect joint subluxation/dysfunction only. They are commonly used to investigate fractures, pathologic conditions, and biomechanical integrity.

A rationale for the use of plain-film imaging in the chiropractic office is (1) to assist in the establishment of a working diagnosis when clinically indicated, (2) to rule out the presence of pathologic conditions that contraindicate manipulative therapy, (3) to identify any anomalies or structural changes that may influence how an adjustment will be made, and (4) to determine static and functional biomechanical relationships that may have clinical relevance to the patient's symptoms or health.

No one tool should be used to make clinical decisions, and x-ray interpretation should not be an exception. The fundamental principles in the use of radiology that are of prime importance and that should be considered before x-ray examinations are ordered are identified in Box 3-17.

INSTRUMENTATION

In the absence of a “gold standard” for the assessment of characteristics associated with joint subluxation/dysfunction syndrome (JSDS), the chiropractic profession has sought an instrument that would objectively measure and quantify its presence.

However, even if such an instrument existed, it would likely be limited to the identification of only a single clinical characteristic or finding. This finding may be associated with joint subluxation/dysfunction or with other clinical entities. Therefore, no single tool should be relied on to make the diagnosis or assessment of subluxation/dysfunction syndrome.

The following tools represent means to identify specific characteristics potentially associated with joint subluxation/dysfunction. Although most have fair to good reliability, their validity has not been adequately determined or tested. Moreover, some proponents of specific instruments have made inappropriate claims about the value of the information gleaned from the instruments.

Algometry

Algometry is the measurement of pain. *Algometers* are force gauges that are used to quantify the amount of pressure necessary to elicit a painful response (Figure 3-34). Algometers are used at both

BOX 3-16 Limitations and Disadvantages of Videofluoroscopy

Expense—equipment ranges from \$60,000 to \$80,000.
Overuse—high costs can lead to extra ordering of tests to offset capital outlay.
Inferior image—image definition is poor in comparison with plain-film studies, and subtle architectural changes are not visualized.
Justification—not enough diagnostic information is provided to warrant the additional radiation exposure.

BOX 3-17 Fundamental Principles in the Use of Radiography

Radiographs should be considered only after an appropriate and thorough history and examination.
 Radiographs should be ordered based only on clinical need. Routine radiographic examination without clinical need is inappropriate.
 When selecting patients for radiographic study, the benefit of the x-ray information must always outweigh the risk of ionizing radiation to the patient's health.

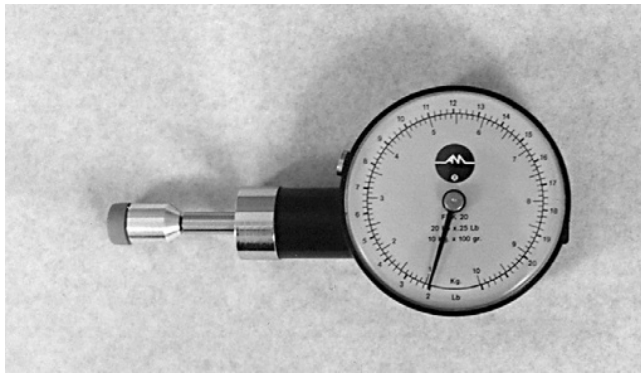


Figure 3-34 Algometer.

bony and soft tissue sites. Algometric measurements represent the point of maximal pressure the patient can tolerate (*pressure tolerance [PTo]*) or the point at which pressure induces pain (*pressure pain threshold [PPT]*).

PPT is alternatively referred to as the *first verbal report of pain*. PTo is alternatively referred to as the *pain reaction point (PRP)*, the point at which the patient reports that it “hurts a lot.” PPT measurements are more commonly used because they are less intrusive.

To evaluate the patient’s PPT, the examiner places the rubber-tipped stylus of the algometer over the site to be evaluated and applies steady, gentle pressure at a rate of approximately 1 kg/sec. Pressure is applied until the patient feels pain and responds by saying “now.” At this point the stylus is removed, and tender sites and their pressure values are recorded.

Normative PPT values for muscle and bone have been established, and repeated measures have demonstrated good reliability.⁵³⁰⁻⁵³⁶ Algometry is presented as an effective tool for evaluating painful musculoskeletal complaints.⁵³⁷ It has demonstrated reliable measurement of pain in temporomandibular joint dysfunction,^{536,538} myofascial trigger point syndromes,⁵³⁴ muscle tension headache,⁵³⁹ and the ability to differentiate patients with inflammatory arthritis from healthy controls.⁵⁴⁰ Algometry is responsive to treatment, effectively documenting increased PTos in disorders treated with adjustive therapy.^{541,542} Algometry has been used most commonly in research settings, but its ease of use, low-tech application, and affordability make it appropriate for use in clinical practice.

Thermography

Medical thermography is the technique of measuring and recording heat emission from the body. The body radiates heat in a symmetric pattern, with only minor variation in temperature from side to side. In a healthy individual, temperature radiation is regionally symmetric to within 0.5 to 1 degree centigrade.^{543,544}

The most common devices used to assess human heat emission are electronic infrared and liquid crystal appliances. Significant asymmetry in heat emission has been postulated as objective evidence of a variety of underlying disorders and painful complaints. In manual medicine, thermography is most frequently discussed as a noninvasive means to detect functional changes that may be associated with disorders of the NMS system.



Figure 3-35 Thermocouple device used to evaluate paraspinal temperature symmetry.

Christiansen,⁵⁴⁵ in a review of the literature, found thermography compared favorably with other diagnostic tests (e.g., myelography, EMG, and CT) for accuracy and sensitivity in determining the level of radiculopathy while demonstrating high correlation with these more invasive tests.⁵⁴⁶ A hand-held device that uses either infrared contact thermistors or thermocouples may be used. This device is used to detect regional variations in midline spinal temperature or variations in segmental paraspinal temperature. The temperature differential is usually displayed on a calibrated galvanometer or plotted on a strip graph.

The thermocouple device is constructed to make physical contact with the surface of the body and measure differences in segmental paraspinal temperature (Figure 3-35). Deflection of the needle will be to the relatively warmer side. Alteration in segmental temperature is speculated to represent objective evidence of spinal subluxation/dysfunction syndromes. It is postulated that spinal subluxation/dysfunction may produce a local inflammatory reaction or reflex alteration in sympathetic tone, which in turn alters the segmental temperature symmetry of the body.⁵⁴⁷⁻⁵⁵¹

Although some techniques advocate using hand-held heat detection instruments for spinal subluxation/dysfunction detection, very little research has been conducted on the reliability or validity of these devices. Normative values for hand-held devices have not been established, and reliability studies are very limited. To date, no studies have been conducted to evaluate their validity in determining spinal dysfunction.⁵⁵² Further research is needed to confirm or refute the theories, indications, and limitations of these devices.⁵⁵³

Galvanic Skin Resistance

Galvanic skin resistance (GSR) is the measure of the skin’s electrical conductivity. Galvanic skin instruments measure the resistance of the skin to a small electrical current. It has been suggested that spinal dysfunction may alter skin conduction by inducing sympathetically mediated changes in sweat gland secretion (somatovisceral reflex).⁵⁵⁴

Conductivity of the skin is significantly affected by its moisture content and sweat gland activity.⁵⁵⁵

Spinal dysfunction is hypothesized to alter conduction by either increasing or decreasing sweat gland activity. Spinal dysfunction that leads to increased peripheral autonomic activity and sweat gland secretion could increase segmental conductance. Spinal dysfunction that leads to inhibitory autonomic activity could result in a decrease in skin conductance. The presence of pain has also been shown to induce a segmental decrease in GSR.^{556,557}

Ease of access to the skin makes GSR an uncomplicated tool for measuring autonomic effects that may be associated with spinal dysfunction. GSR has been used in experimental settings on a very limited basis. Its clinical value remains to be evaluated, and it is seldom, if ever, used in the day-to-day clinical evaluation of joint dysfunction.

Surface Electromyography

A method purported to assess a specific characteristic of spinal dysfunction is paraspinal EMG scanning. Paraspinal muscle dysfunction is considered a clinical manifestation of joint subluxation/dysfunction syndrome, and surface EMG is presented as a method that can provide an objective quantitative evaluation of changes in paraspinal muscle function.

EMG is a technique for recording electrical potentials associated with muscular activity. This is considered important to clinicians because it represents the outflow of motor neurons in the spinal cord to the muscle as a result of voluntary or reflex activation.⁵⁵⁸ Either needle electrodes or surface electrodes can be used to study paraspinal or peripheral muscle function. However, needle electrode and surface electrode EMGs are not interchangeable procedures.⁵⁵⁹ Needle electrode techniques are indicated for the evaluation of specific muscles, innervation potentials, and myopathies. Surface electrode techniques are indicated for kinesiological studies of the global function of groups of muscles.

The muscle activity that is recorded by the various paraspinal scanning machines are relative values, relating to resting and contractile muscle states. Muscle unit action potentials are amplified by the EMG machine. The responses are filtered and rectified by the instrument. These rectified responses are bidirectional waves of depolarization and repolarization along cell membranes. They are integrated over time, and analog signals are converted to digital signals.

Surface electrode EMG with attached electrodes has been shown to exhibit very good to excellent test-retest reliability.⁵⁶⁰⁻⁵⁶⁵ In a comprehensive review article comparing the properties of fixed-surface electrode EMG and intramuscular EMG, Turker⁵⁵⁸ identified that both procedures have indicated uses. Surface electrode EMG was found to be more prone to electrical artifacts, mechanical artifacts, and contamination from the activity of other muscles than intramuscular EMG. However, it is possible to make useful recordings with the surface electrode from large superficial muscles if appropriate precautions are observed.

In a comparison study using needle electrode and fixed-surface electrode EMG, evaluated muscles were electrically silent, during standing and full flexion. The use of fixed-surface electrodes

provided recordings that were similar, although slightly dampened, to those obtained with intramuscular wire electrodes.⁵⁶⁶ The surface measurements were found to accurately document the function of back muscles, permitting the use of fixed-surface electrode EMG in outpatient care. Furthermore, fixed-surface EMG has been shown to provide a very valuable set of data used diagnostically with pain-related disorders.⁵⁶⁷

Scanning-surface EMG should not be confused with surface EMG that uses fixed-surface electrodes. Electromyographic muscle scanning measures 2-second samples of integrated muscle action potentials from individual neck and back muscles using a hand-held scanner with post-style surface electrodes separated by a fixed distance. This scanning technique is used to expeditiously assess muscle activity in the diagnosis of musculoskeletal disorders.^{564,568} The signal recorded by the apparatus is a transient signal. There are a number of potential technical problems with this equipment, including low signal or noise ratios; movement signal artifacts (because the surface electrodes are not fixed to the skin); and other sources of signal artifacts, such as those from the heart and great vessels. Surface-scanning paraspinal EMG gives a rough estimation of transient muscle activity. It cannot give information about specific muscles because the recording electrodes are placed over the skin and not into a muscle.

Thompson and colleagues⁵⁶⁴ report that their study supports hand-held post-style electrodes as providing a satisfactorily stable means of monitoring the surface EMG signal. However, they also identify that the 2-second integration period may be inadequate, favoring instead a 10-second integration, especially in research and treatment outcome studies. When attention is directed to scan preparation, hand-held EMG sensors produce dependable results. The reliability of the surface EMG scanning procedure was investigated using a large clinical sample; the results indicated that with adequate attention to scan preparation, EMG sensors held in place by hand with light pressure produced dependable results. The pattern of reliability was seen to be slightly higher in the lower back on patients in the standing position.⁵⁶⁹

Questions regarding surface electrode EMG's validity and usefulness exist, yet its use is often embraced by many without reserve.⁵⁷⁰ Lehman conducted three experimental studies to evaluate surface electrode EMG for asymmetry and repeatability in populations with LBP and populations without LBP. He concluded that EMG signals during quiet stance show excellent repeatability but segmental differences in asymmetry between problematic and nonproblematic segments were not evident. This suggests that the diagnostic validity of EMG evaluations during simple quiet-stance tasks is highly suspect. Ritvanen et al⁵⁷¹ also found no positive nor statistically significant association between back pain and EMG parameters.

Surface-electrode EMG has the potential to assist the doctor in evaluating the patient's response to treatments.⁵⁵⁹ Myerowitz,⁵⁷² using hand-held scanning electrodes, evaluated the relationship between post-treatment paraspinal surface EMG improvement and improvements in spinal pain or related musculoskeletal symptoms. He treated 42 symptomatic patients with pain and abnormal scanning surface EMG findings. All 42 patients had

post-treatment improvement on scanning-surface EMG readings, and 41 patients (97.6%) reported post-treatment reduction in the pain symptoms. This study raises the possibility of using hand-held scanning surface EMG to correlate EMG activity to symptomatic improvement in common conditions of spinal pain and related musculoskeletal symptoms.

Although scanning-surface EMG has generated some enthusiasm in the profession, the use of scanning surface paraspinal EMG for the detection of spinal subluxation syndromes must be questioned.⁵⁷³ The profession, in attempting to document intersegmental dysfunction, has jumped too quickly onto an unproven application.⁵⁷⁴ Aside from the potential hardware problems presented, chiropractors must question the need for the routine use of an examination procedure that tells the doctor there is some local muscle tone alterations. Chiropractic doctors have sufficient training and palpation skills to assess contracted muscles, and the cost generated by this technology may not be warranted.

Scanning-surface EMG may have potential value as an outcome measure, but its validity in detecting joint subluxation syndromes has not been substantiated. The important clinical questions remain unanswered. Instruments, including scanning-surface EMG, must be evaluated within a clinical context. The interpretation of information derived from these instruments and how it affects clinical decisions and treatment is the determining factor in establishing its validity and clinical utility.⁵⁷⁵

CLINICAL DOCUMENTATION

Practice efficiency is enhanced when manual examination findings are recorded with symbols on charts. One of the less rewarding aspects of practice is the time spent writing reports. Accurate and legible chart notes make that process more efficient and less tedious. A method that is quick and accurate can take the drudgery out of note-taking and free the doctor to concentrate on patient care. Figure 3-36 outlines a set of symbols used to record the location of pain and other bony and soft tissue abnormalities. Figure 3-37 contains examples of methods that can be used to record abnormalities in ROM, JP, and EP. There are many different methods, and each doctor usually makes modifications to fit his or her style. These examples are offered in the hope that they will be of value in the search and development of a method of charting. The total management of the patient includes clinical assessment, application of necessary treatment, and patient education. Clinical assessment procedures are performed to identify appropriate case management—frank acceptance and sole responsibility for care, acceptance with consultation from other health care professionals, and frank referral transferring responsibility for immediate care to another health care professional. Assessment procedures are necessary to identify the nature, extent, and location of the problem, as well as to determine the course of action in treatment. Last, these same procedures must be used to monitor the effects of care. It is an important process to record adequately the various aspects of care.

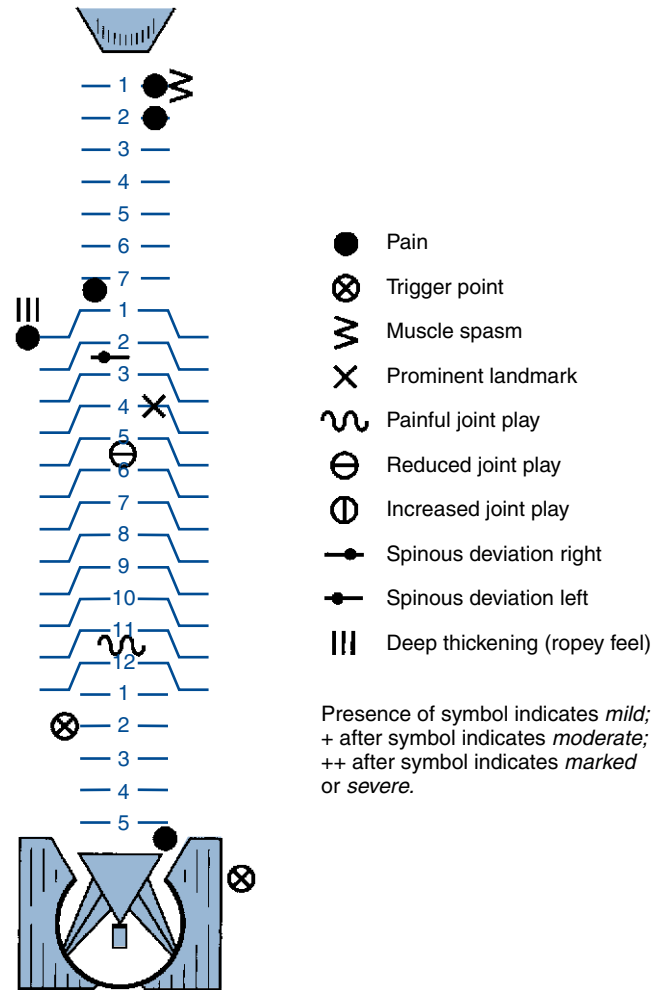


Figure 3-36 Examples of symbols and recording method for charting and tracking joint assessment findings.

Errors in recording that have been identified include failure to record findings altogether, illegible handwriting, obscure abbreviations, improper terminology, and bad grammar. It is imperative that although the clinical record comprises the doctor's personal notations, it must be complete and translatable. If it is not written down, it was not done.

A systematic and accurate record of evaluation facilitates quick reference to salient findings during treatment. When findings either modify or contraindicate some aspect of treatment, this should be noted in a conspicuous location on the patient's record so that it is readily seen before each visit.

It should be noted and emphasized that it is unacceptable to use and assign a diagnosis for convenience. Most clinical entities have specific and expected signs and symptoms. These findings need to be identified and recorded. Although it is nearly impossible to have complete certainty as to the nature and extent of the clinical problem, the compilation of clinical findings is necessary to influence the clinical judgment used in applying interventions in patient care. Furthermore, it must be clearly understood that third-party payers reimburse for problems (they provide "disease" insurance, not "health" insurance). The reporting of problems to

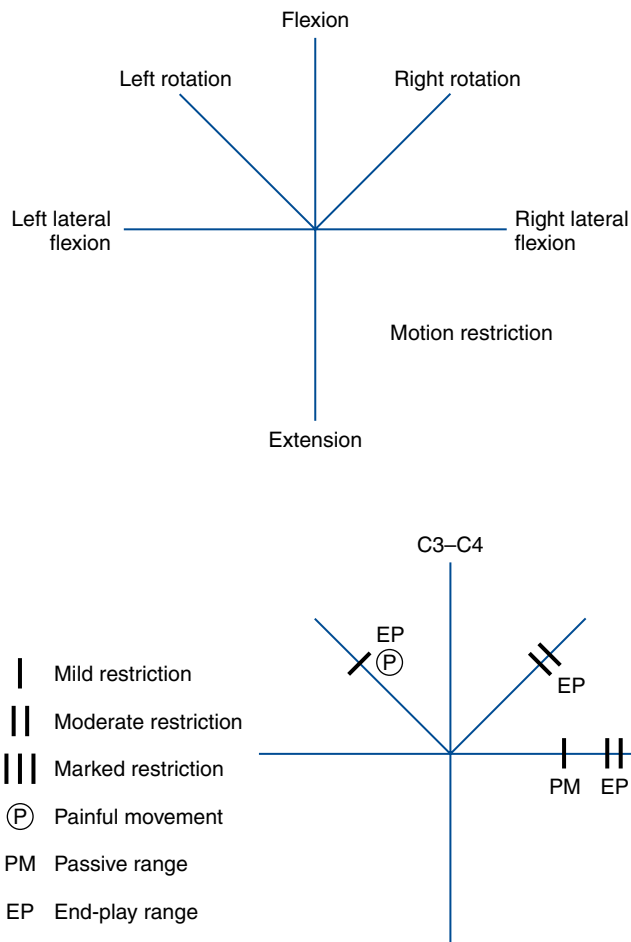


Figure 3-37 Diagram for recording segmental motion palpation findings.

TABLE 3-5 ICD Codes for Subluxation

Version 9	Version 10
739	Nonallopathic lesions including segmental dysfunction
739.0	M99.00 Head region
739.1	M99.01 Cervical region
739.2	M99.02 Thoracic region
739.3	M99.03 Lumbar region
739.4	M99.04 Sacral region
739.5	M99.05 Pelvic region
739.6	M99.06 Lower extremities
739.7	M99.07 Upper extremities
739.8	M99.08 Rib cage
839	Multiple, ill-defined dislocations, closed dislocation
839.00	M99.11 Cervical spine, vertebra unspecified
839.01-07	S13.110A-170A Cervical spine, vertebra specified
839.21	M99.12 Thoracic vertebra
839.20	M99.13 Lumbar vertebra
839.41	S33.2XXA Coccyx
839.42	M99.14 Sacrum

third-party payers must be substantiated in the clinical record. When subluxation/dysfunction syndrome is the primary or reportable component of the diagnosis, an ICD-9CM code can be used. Table 3-5 identifies the codes used to report subluxation/dysfunction syndrome.

OUTLINE			
CLASSIFICATION AND DEFINITION		JOINT SUBLUXATION/DYSFUNCTION	
OF MANUAL THERAPIES	84	SYNDROMES	90
JOINT MANIPULATIVE		Clinical Findings Supportive of	
PROCEDURES	84	Joint Subluxation/Dysfunction	
Adjustment	84	Syndrome	90
Manipulation	88	CONTRAINDICATIONS TO AND	
Joint Mobilization	88	COMPLICATIONS OF	
Manual Traction-Distraction	88	ADJUSTIVE THERAPY	92
SOFT TISSUE MANIPULATIVE		Cervical Spine	94
PROCEDURES	88	Thoracic Spine	102
INDICATIONS FOR ADJUSTIVE		Lumbar Spine	103
THERAPY	89	EFFECTS OF ADJUSTIVE THERAPY	105
MECHANICAL SPINE PAIN	89	Musculoskeletal	105
		Non-musculoskeletal	106
		Mechanical Hypotheses	106
		Joint Fixation	112
		Neurobiologic Hypothesis	115
		Circulatory Hypothesis	120
		APPLICATION OF ADJUSTIVE	
		THERAPY	120
		Joint Anatomy, Arthrokinematics,	
		and Adjustive Movements	121
		Adjustive Localization	123
		Adjustive Psychomotor Skills	128
		Motion-Assisted Thrust	
		Techniques	142

Chiropractors must maintain the necessary diagnostic skills to support their roles as primary contact providers. There is, however, a wide range of choice in the chiropractor's scope of practice. Therapeutic alternatives range from manual therapy and spinal adjustments to physiologic therapeutics and exercise, nutritional and dietary counseling.^{1,2}

Although there is great variation in scope of practice from state to state, nearly all chiropractors use a variety of manual therapies with an emphasis on specific adjustive techniques.^{1,3-8} The preceding chapters focused on the knowledge, principles, examination procedures, and clinical indications for applying adjustive therapy. This chapter focuses on the knowledge, mechanical principles, and psychomotor skills necessary to effectively apply adjustive treatments.

CLASSIFICATION AND DEFINITION OF MANUAL THERAPIES

Manual therapy includes all procedures that use the hands to mobilize, adjust, manipulate, create traction, or massage the somatic or visceral structures of the body.⁹ They may be broadly classified as those procedures directed primarily at the body's joint structures or soft tissue components (Figure 4-1).

JOINT MANIPULATIVE PROCEDURES

Joint manipulative therapies are manual therapies, the primary effect of which is on joint soft tissue structures (Box 4-1). They are physical maneuvers designed to induce joint motion through either nonthrust techniques (mobilization) or thrust techniques (adjustment or thrust manipulation). They are intended to treat disorders of the neuromusculoskeletal (NMS) system by decreasing pain and improving joint range and quality of motion. This leads to their common application in the treatment of NMS disorders that are associated with joint pain or joint hypomobility (subluxation/dysfunction).

When joint dysfunction/subluxation syndrome (hypomobility or malposition) is treated, the adjustive thrust or mobilization is typically delivered in the direction of reduced joint motion to restore normal motion and alignment. For example, if the lumbar spine has a restriction in right rotation, the doctor thrusts to induce more right rotation in the affected region. In some instances, the therapeutic force may be delivered in the relatively nonrestricted and pain-relieving direction. This is most common when acute joint pain and locking limit movement in one direction, but still allow distraction of the joint capsule in another direction.¹⁰⁻¹² Under these circumstances, therapy is most commonly directed at inducing separation of joint surfaces. The goal is to inhibit pain and muscle guarding and to promote flexible healing.

ADJUSTMENT

Adjustments are the most commonly applied chiropractic therapy.³⁻⁵ They are perceived as central to the practice of chiropractic and the most specialized and distinct therapy used by chiropractors.^{3,4,13} Specific reference to adjustive therapy is incorporated in the majority of state practice acts, and it is commonly cited as a key distinguishing feature of chiropractic practice.¹⁴ Although adjustive therapy is central to most chiropractic practices, the authors do not want to impart the impression that chiropractors should limit their clinical care to adjustive treatments. Patient management and treatment plans should be based on the best available evidence, clinical judgment, and patient preferences. There are circumstances in which the best standard of care for a given NMS disorder involves the application of nonadjustive treatments singularly or in combination with adjustive therapy. Other therapies commonly applied by chiropractors include joint mobilization and light-thrust techniques; soft tissue massage and manipulation; physical therapy modalities; and instruction on exercise, ergonomics, lifestyle, and nutrition.

BOX 4-1 Manual Therapy Terminology**MANUAL THERAPY**

Procedures by which the hands directly contact the body to treat the articulations or soft tissues.¹⁶

JOINT MANIPULATION

(1) Joint manipulative therapy broadly defined includes all procedures in which the hands are used to mobilize, adjust, manipulate, apply traction, stimulate, or otherwise influence the joints of the body with the aim of influencing the patient's health; (2) a manual procedure that involves a directed thrust to move a joint past the physiologic ROM without exceeding the anatomic limit;¹⁶ (3) skillful or dexterous treatment by the hand. In physical therapy, the forceful passive movement of a joint beyond its active limit of motion.

ADJUSTMENT

(1) A specific form of joint manipulation using either long- or short-leverage techniques with specific anatomic contacts. It is characterized by a low-amplitude dynamic thrust of controlled velocity, amplitude, and direction. Adjustments are commonly associated with an audible articular crack (cavitation). (2) any chiropractic therapeutic procedure that uses controlled force, leverage, direction, amplitude, and velocity, which is directed at specific joints or anatomic regions. Chiropractors commonly use such procedures to influence joint and neurophysiologic function.¹⁶

DIRECT (SHORT-LEVER)

Specific joint contact; high velocity–low amplitude thrust.

SEMIDIRECT

Combination of specific joint contact and distant long-lever contact; high velocity–low amplitude thrust.

INDIRECT (LONG-LEVER)

Nonspecific contact established at leverage points distant to affected joint.

JOINT MOBILIZATION

(1) Form of nonthrust joint manipulation typically applied within the physiologic range of joint motion. Mobilizations are passive rhythmic graded movements of controlled depth and rate. They may be applied with fast or slow repetitions and various depth. Although joint mobilization is not commonly associated with joint cavitation, deep mobilization (grade 5) may induce cavitation; (2) movement applied singularly or repetitively within or at the physiologic range of joint motion, without imparting a thrust or impulse, with the goal of restoring joint mobility;¹⁶ (3) manual traction-distraction: a form of mobilization producing a tractional or separating force. It may be accomplished manually or with mechanical assistance and can be sustained or intermittent.

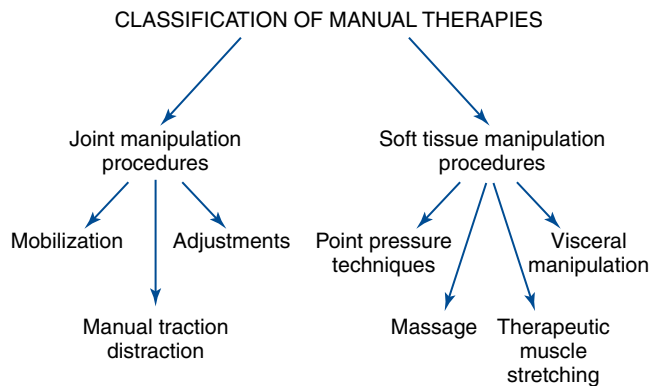


Figure 4-1 Classification of manipulative procedures. (This illustration is not intended to cover all possible manual therapies.)

Unfortunately, the common use of adjustments by chiropractors has not led to a clear and common understanding of the defining characteristics of an adjustment.^{14,15} A mid-1990s consensus process made major strides in reaching consensus on many of the chiropractic profession's unique terms.¹⁹ However, several key terms within this document lack clarity. At issue is whether the definitions presented for *adjustment* and *manipulation* are clear and distinct or so broad that they have limited descriptive value.

Historically, *adjustive therapy* was defined primarily in the context of the doctor's therapeutic intentions. If the doctor applied a

treatment procedure with the intention of reducing a joint subluxation, it was considered an adjustment.^{17,18} Based on this premise, any procedure delivered by a chiropractor and directed at reducing joint subluxation could be considered an adjustment. This approach results in a wide variety of significantly different physical procedures all being classified as *adjustments*.

The 1990s consensus process appropriately moved the focus away from defining adjustments based on therapeutic intention and toward defining an adjustment based on its physical characteristics. However, the definition maintained a very broad and inclusive approach. *Adjustments* were defined "as any chiropractic therapeutic procedure that utilizes controlled force, leverage, direction, amplitude and velocity."¹⁹ The definition did not limit the application of adjustments to the joints of the body, but specified that adjustments could be delivered to any anatomic region (see Box 4-1, Adjustment 2). In this context, it is difficult to perceive a chiropractically applied procedure that would not be classifiable as an adjustment. A wide variety of diverse procedures (thrust and nonthrust joint manipulation, adjustment, massage, manual or motorized traction, etc.) all involve force, leverage, direction, amplitude, and velocity. More than 100 different named technique systems have been identified within the chiropractic profession, and most of them call their treatment procedure an adjustment (see Appendix 1).²⁰ A number of these procedures do not share discrete physical attributes and may not be equivalent in their physical effects and outcomes. The profession needs to

objectively evaluate and compare the effectiveness of chiropractic therapeutic procedures. This cannot be accomplished without physically distinct classifications of commonly employed manual therapies. Until this issue is addressed, it will be difficult for the profession to determine which therapies are most effective and in what clinical conditions.

The basis for distinguishing and classifying adjustive procedures should incorporate their measurable characteristics and should not be based solely on therapeutic intention. Separating the physical components of an adjustment from the rationale for its application does not diminish its significance. As stated by Levine, “It is the reason why techniques are applied and why they are applied in a certain manner that distinguishes chiropractic from other healing disciplines.”²¹

The historically broad perspective on and definitions of what constitutes an adjustment have led to a wide variety of procedures being classified as *adjustive methods*. The assumption that all forms of adjustment, as presently defined, are equivalent must be avoided.²² As discussed previously, many in the profession do not equate an adjustment with a thrust, and a number of chiropractic technique systems do not incorporate thrust procedures.¹⁹ In addition to differences that may exist in the form of applied treatment, many technique systems attempt to distinguish themselves not by the attributes of the adjustment they perform, but rather by what they claim to be their unique underlying biomechanical and physiologic principles and rationale.

Despite the variety of procedures that have been labeled as adjustments, most share the common characteristic of applying a thrust. It is this attribute that we propose as the central defining and distinguishing physical feature of the chiropractic adjustment.^{9,23,24} Although amplitude and velocity of the adjustive thrust may vary, it is a high velocity–low amplitude (HVLA) ballistic force of controlled velocity, depth, and direction. With this in mind, we suggest the following definition: The adjustment is a specific form of direct articular manipulation, using either long- or short-leverage techniques with specific contacts characterized by a dynamic thrust of controlled velocity, amplitude, and direction (see Box 4-1, adjustment 1). Adjustive contacts are usually established close to the joint being treated, and the thrust is delivered within the limits of anatomic joint integrity. Adjustive therapy is commonly associated with an audible articular “crack,” but the presence or absence of joint cracking should not be the test for determining whether or not an adjustment has been performed.

Properly applied adjustments are commonly painless, although the patient may experience some momentary, minimal discomfort. A short-duration mild increase in local soreness after manipulation has been reported in up to 50% of patients treated with manipulation and should not be considered an inappropriate response.²⁵ Adjustments should not be forced when preloading a joint in the direction of intended manipulation induces pain or protective patient guarding and resistance. Adjustive procedures that induce discomfort during application should be considered only if they are directed at increasing joint mobility.

Categorization of Adjustive Procedures

Various proposals have been made to further subclassify adjustive thrust procedures. However, most classification schemes

suffer from the central problem of beginning with an unworkably broad definition of *adjustment*. This creates an unnecessary burden on authors who then try to subclassify adjustments by the very attributes that are commonly used to distinguish adjustments from other forms of manual treatment. One common approach is to distinguish adjustments by the degree of applied velocity. It is not uncommon to see references in the chiropractic literature and trade magazines in which different methods are presented and promoted as *low-force* or *nonforce methods*. This carries an inference that these procedures are different from other adjustive techniques and are associated with less peak force. These descriptions commonly do not explain if the procedures are applied with a thrust, nor do they explain how much actual force is involved or how they truly compare with other adjustive procedures. Furthermore, measurements of adjustive preload, peak force, and amplitude appear to vary within the same adjustive methods. When the same adjustive methods are applied at different anatomic regions or on different patients, the preload, rate of velocity, and peak velocity change significantly.²⁶ These noted differences are no doubt the product of each doctor’s trained ability to note and modify his or her adjustive procedures relative to the encountered joint resistance of each spinal region and patient, rather than a conscious effort to use a different adjustive procedure. It is doubtful that any meaningful distinction can be achieved by trying to subclassify adjustments by moderate differences in applied velocity. How would the velocity be measured in day-to-day practice, and how much of a change would be necessary to distinguish one method from the other? Nothing is gained by redefining a joint mobilization as a low-velocity, moderate-amplitude adjustment simply because it is performed by a chiropractor.

In an attempt to be more precise in the distinction, classification, and validation of chiropractic procedures, Bartol^{15,27} and the Panel of Advisors to the American Chiropractic Association Technique Council proposed an algorithm for the categorization of chiropractic treatment procedures. This scheme includes criteria for velocity, amplitude, and the use of manual or mechanical devices to deliver the adjustment. These models were presented at the Sixth Annual Conference on Research and Education and are commendable attempts to further distinguish adjustive methods.²⁸ However, they too lack any clear criteria for distinguishing various levels of high- and low-velocity or high- and low-amplitude adjustments. The criteria for distinguishing manual from mechanical methods are valuable and easily discernible, but they leave a number of other important qualities and potential distinguishing features unaddressed. The criteria include patient positioning (PP), contact points (CPs), leverage, and type of thrust.

To distinguish one adjustive procedure from the other, we suggest a system that begins with the assumption that adjustments are HVLA thrust procedures, which can be further differentiated and subcategorized by the components listed in Box 4-2. The suggested method incorporates elements used by the National Board of Chiropractic Examiners on Part IV of the Practical Adjustive Examination and avoids the dilemma and technological difficulties encountered in trying to differentiate adjustments by minor changes in velocity and depth of thrust.

SPECIFIC VERSUS GENERAL SPINAL ADJUSTMENTS

Specific adjustments involve procedures used to focus the adjustive force as much as possible to one articulation or joint complex. Specific adjustments typically involve the application of short-lever contacts (Figure 4-2). Specificity is assumed to result from establishing adjustive contacts over or near the targeted joint with precise attention given to adjustive vectors. General adjustments involve procedures that are assumed to have broader sectional contacts and effects, mobilizing more than one joint at a time. They are applied when a regional distraction of a group of articulations is desired and commonly involve longer levers and multiple contact sites (see Figure 4-2). Nwuga²⁹ used the term *nonspecific* in this manner and stated that most of the techniques described by Cyriax³⁰ would fall into this category. Grieve³¹ uses the terms *localized* and *regional* to distinguish between procedures that affect a single joint or a sectional area. Also, the term *general* has been used

to denote the nonspecific, regional, or sectional forms of manipulation.³² Therefore techniques considered to be nonspecific use broad and long-lever contacts taken over multiple sites with the purpose of improving motion or alignment in an area that is generally stiff or distorted. Grice and Vernon³³ suggest that this type of procedure is indicated to free general fixations or reduce general muscle spasms, such as those seen in spinal curvatures.

The chiropractic profession has emphasized short-lever procedures, theorizing that these are more precise in correcting local subluxation/dysfunction without inducing stress or possible injury to adjacent articulations. This may be especially pertinent in circumstances with adjacent joint instability. Recent research investigating some of the biomechanical assumptions of the specificity paradigm has raised some significant challenges to this model.^{34,35} This research does not diminish the demonstrated clinical effectiveness of adjustive therapy,^{36,37,38} but it does bring into question whether precise joint specificity is achievable or essential for adjustive therapy to be clinically effective.³⁴ Further discussion of this topic is presented later in this chapter under the application of adjustive therapy section.

BOX 4-2 Categorization of Adjustive Methods

Manual vs. nonmanual	Contact point (doctor's anatomic contact on patient)
Motion-assisted vs. non-motion-assisted	Segmental contact point (anatomic location of contact on patient)
Anatomic region	Assisted (superior vertebral contact of involved motion segment)
Direct, indirect, or semidirect	Resisted (inferior vertebral contact of involved motion segment)
Patient position	Thrust
Prone	Push
Supine	Pull
Side-posture	Counterthrust (push-pull)
Sitting	
Standing	
Knee-chest	

CHIROPRACTIC TECHNIQUE

Technique refers to a method for accomplishing a desired aim. In chiropractic, the term is generally applied to manual therapeutic procedures directed at treating joint subluxation/dysfunction. Although it is most frequently applied to manual adjustive procedures, it is not unusual to see the term applied to other forms of chiropractic manual and nonmanual therapy.

Many chiropractic diagnostic and therapeutic procedures (techniques) have been developed empirically in the profession by an individual or association of individuals. These techniques are commonly then assembled as a system, incorporating theoretic models of joint dysfunction with procedures of assessment and treatment. Appendix 1 is a list of system techniques.

Chiropractic technique should not be confused with chiropractic therapy or treatment, which includes the application of

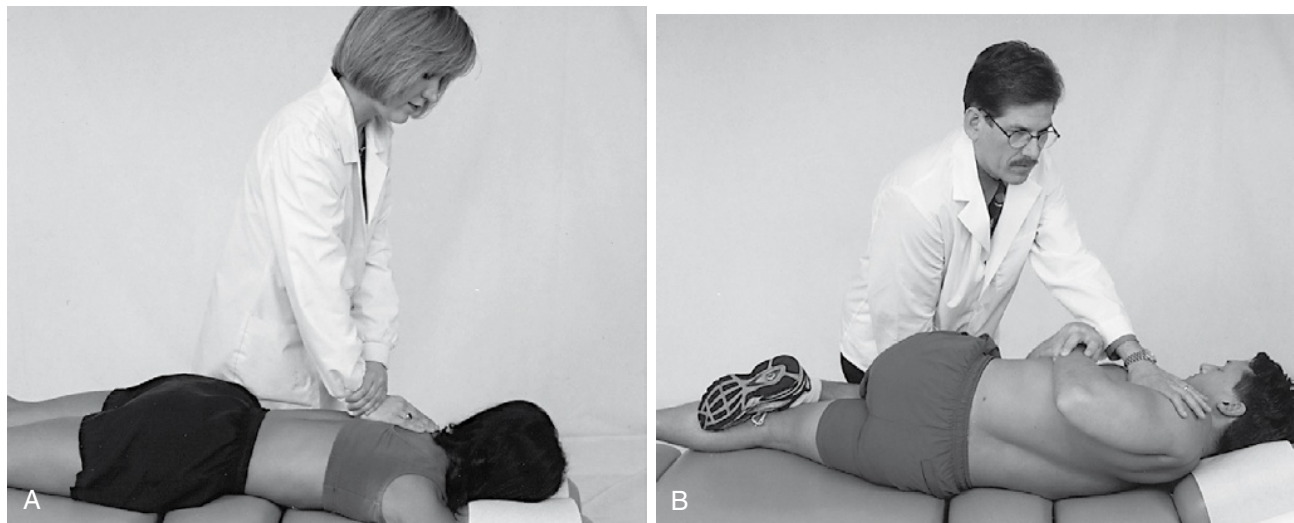


Figure 4-2 A, Prone short-lever thoracic adjustment applied to induce segmental rotation. B, Side-posture long-lever adjustment applied to induce segmental or sectional rotation.

the entire range of primary and ancillary procedures indicated in the management of a given health disorder. These are limited by individual state statutes, but may include such procedures as joint mobilization, therapeutic muscle stretching, soft tissue manipulation, sustained and intermittent traction, meridian therapy, physical therapy modalities, application of heat or cold, dietary and nutritional counseling, therapeutic and rehabilitative exercises, and biofeedback and stress management.

MANIPULATION

In contrast to the broad definition of adjustment, the 1990s consensus project defined *joint manipulation* in more narrow terms and limited its application to joint-thrust procedures (see Box 4-1, joint manipulation 2).¹⁶ This is not uncommon, and it is becoming the norm. However, joint manipulation is also commonly used in a broader context (see Figure 4-1 and Box 4-1, adjustment 1). In this context, *manipulate* means to skillfully use the hands to move, rearrange, and alter objects. When applied to manual therapy and biologic tissue, it has not historically been limited to high-velocity thrust procedures. It frequently had a broader application, which encompassed a number of more specific procedures applied to soft tissues and joints, such as soft tissue manipulation, massage, and joint mobilization (see Box 4-1).

It is not likely that the world of manual therapy will limit use of the word *manipulation* to thrust procedures. Joint manipulation will likely continue to be used in both its broad and narrow contexts. This potentially confusing state could be remedied if the term *joint thrust manipulation* was substituted for *joint manipulation* whenever it is associated with a high-velocity thrust. In the absence of such a convention, the reader must ascertain from the context which specific application is being used. HVLA is being used more commonly in the literature to facilitate a clearer description when thrust manipulation is being referenced.

JOINT MOBILIZATION

Joint mobilization in contrast to adjustive therapy does not use a thrust.^{9,39} Joint mobilization is applied to induce movement through a series of graded movements of controlled depth and rate without a sudden increase in velocity. It is a common mistake to consider mobilization as a procedure that cannot induce movement into the end range of the elastic zone (paraphysiologic space). Deep joint mobilization may be associated with an audible crack (cavitation). Joint cavitations do not occur as frequently with mobilization as they do with thrust procedures, but the presence or absence of joint cavitation during the procedure does not distinguish a mobilization from an adjustment or thrust manipulation. Joint mobilization procedures are detailed in Chapter 7.

MANUAL TRACTION-DISTRACTION

Manual traction-distraction is another form of manual therapy used to mobilize articular tissues. Traction is not a unique and separate form of treatment, but is simply one form of passive mobilization.⁴⁰ Therefore, the distinction between *joint mobilization* and *manual traction-distraction* is not clear, and the separation may be

arbitrary. When the technique is applied to articular tissues, the goal is to develop sustained or intermittent separation of joint surfaces. In the field of manual therapy, traction-distraction is performed through contacts developed by the clinician and is often aided by mechanized devices or tables.

Traction techniques are thought to aid in the application of an adjustment by first allowing physiologic rest to the area, relieving compression that results from weight bearing (axial loading), applying an imbibing action to the synovial joints and discs, and opening the intervertebral foramina. Many of these procedures are also quite useful for elderly patients when an HVLA thrust may be contraindicated. Moreover, traction maneuvers produce long-axis distraction in the joint to which they are applied. There is a long-axis distraction movement of joint play (JP) at every synovial joint in the body.⁴¹ Yet in the spine, the fact that this important joint movement is necessary for normal function of the joint is mostly ignored or forgotten. Perhaps this is because testing for long-axis distraction of the spinal joints can be difficult to elicit manually.

The term *traction* refers to the process of pulling one body in relationship to another, which results in separation of the two bodies.⁴² Traction is a passive translational movement of a joint that occurs at right angles to the plane of the joint, resulting in separation of the joint surfaces. Kaltenborn⁴² divides manual traction into three grades of movement. In the first, there is no appreciable joint separation, because only enough traction force is applied to nullify the compressive forces acting on the joint. The compressive forces are a result of muscle tension, cohesive forces between articular surfaces, and atmospheric pressure. The second effect produces a tightening in the tissue surrounding the joint that is described as “taking up the slack.” The third grade of traction requires more tractive force that produces a stretching effect into the tissues crossing the joint. The principal aim of treatment is restoration of normal, painless range of motion (ROM).

Traction can be applied manually or mechanically, statically or rhythmically, with a fast or slow rate of application. The force applied may be strong or gentle and applied symmetrically or asymmetrically. The effects of traction are not necessarily localized, but may be made more specific by careful positioning. Although traction has focused mostly on the lumbar and cervical spine regions, there are descriptions for the application of rhythmic traction to all regions of the spine and extremities. Furthermore, the indications for traction include changes that are common to most synovial joints in the body. Chapter 7 provides detailed descriptions of traction techniques.

SOFT TISSUE MANIPULATIVE PROCEDURES

Soft tissue manipulative procedures (Box 4-3) are physical procedures using the application of force to improve health. This category includes techniques designed to manipulate, massage, or stimulate the soft tissues of the body.⁹ “It usually involves lateral stretching, linear stretching, deep pressure, traction and/or separation”³⁹ of connective tissue. They may be applied to either articular or nonarticular soft tissues.

Although joint movement may be produced or improved as a result of the application of soft tissue manipulative procedures, the induction of joint movement is not a necessary or common

BOX 4-3 Soft Tissue Manipulative Procedures

Massage: the systematic therapeutic application of friction, stroking, percussion, or kneading to the body.

Effleurage (stroking)

Pétrissage (kneading)

Friction

Pumping

Tapotement (tapping)

Vibration

Roulemont (rolling)

Therapeutic muscle stretching: a manual therapy procedure designed to stretch myofascial tissue, using the principles of postisometric muscular relaxation and reciprocal inhibition

Proprioceptive neuromuscular facilitation (PNF)

Active release (ART)

Postisometric relaxation (PIR)

Contract-relax-antagonist-contract (CRAC)

Proprioceptive rehabilitation

Point pressure techniques: application of sustained or progressively stronger digital pressure; involves stationary contacts or small vibratory or circulatory movements

Nimmo (receptor tonus technique)

Acupressure

Shiatsu

Reflexology

Body wall reflex techniques

Visceral manipulation: a manual method for restoring mobility (movement of the viscera in response to voluntary movement or to movement of the diaphragm in respiration) or motility (inherent motion of the viscera themselves) of an organ, using specific gentle forces.

Modified from Barral JP, Mercier P: *Visceral manipulation*, Seattle, 1988, Eastland Press.

component of soft tissue procedures. The justification for a separate classification is to draw attention to their principal application in the treatment of soft tissue disorders that may be nonarticular.

Soft tissue manipulative procedures are used to alleviate pain; to reduce inflammation, congestion, and muscle spasm; and to improve circulation and soft tissue extensibility.³¹ In addition to their use as primary therapies, they are frequently used as preparatory procedures for chiropractic adjustments. Soft tissue manipulation tends to relax hypertonic muscles so that when other forms of manual therapy are applied, equal tensions are exerted across the joint.

There are numerous named soft tissue manipulative procedures; Box 4-3 provides a list of some of the common methods that are used in manual therapy. Chapter 7 provides detailed descriptions of non-thrust joint mobilization and soft tissue manipulative procedures.

INDICATIONS FOR ADJUSTIVE THERAPY

The assessment and determination of whether a given health care disorder is suitable for a trial of adjustive therapy depends largely on the doctor's clinical examination skills and experience. To deter-

mine if a given health complaint is manageable with chiropractic care and adjustive therapy, the doctor must first form a clinical impression based on the patient's presentation, physical examination, and any indicated laboratory tests. The ability to thoroughly evaluate and triage disorders of the NMS system and distinguish those conditions that are appropriate for chiropractic care is critical. Differentiating mechanical from nonmechanical conditions, assessing the source of the presenting complaint, and understanding the potential pathomechanics and pathophysiology of the disorders being considered for chiropractic care are crucial elements for successful treatment.

Appropriate treatment decisions are founded on an understanding of the natural history of the disorder being considered for treatment and an assessment of the risks versus the benefits of the considered therapy. If it is determined that the patient is suffering from a condition appropriately treated with chiropractic care and other contraindications have been ruled out, the presence of such conditions provides sufficient justification for a trial of adjustive therapy. If care is initiated, monitoring procedures must be maintained to assess whether the patient's condition is responding as expected or is deteriorating. If treatment does not provide results within the expected time, it should be terminated, and other avenues of therapy should be investigated.

MECHANICAL SPINE PAIN

Conditions inducing pain and altered structure or function in the somatic structures of the body are the disorders most frequently associated with the application of manual therapy. The causes and pathophysiologic changes that induce these alterations are likely varied, but are commonly thought to result from nonserious pathologic change commonly lumped under the category of *nonspecific spine pain*. In the low back, 85% to 90% of complaints are estimated to fall within this category.^{43,44} Specific pathologic conditions, such as infection, inflammatory rheumatic disease, or cancer, are estimated to account for approximately 1% of presenting low back pain (LBP) complaints.⁴⁵ Nerve root (NR) pain caused by herniated disc or spinal stenosis is estimated to account for 5% to 7% and referred LBP resulting from visceral pathologic conditions accounts for approximately 2%.⁴⁵

The differentiation of mechanical from nonmechanical spine pain should begin with an evidence-based clinical examination. A "diagnostic triage" process based on a thorough history and brief clinical examination is recommended by numerous national and international guidelines as an efficient first step.⁴⁶⁻⁴⁸ This process is most commonly referenced relative to LBP, but is applicable to any axial spine pain complaint. The triage process is structured to identify any red flags, ensure the problem is of musculoskeletal origin, and classify suspected musculoskeletal problems into three broad categories before beginning treatment. The three major categories are back pain caused by a serious spinal pathologic condition, back pain caused by NR pain or spinal stenosis, or nonspecific (mechanical) LBP. If the history indicates the possibility of a serious spinal pathologic condition or NR syndrome, further physical examination and indicated testing should be conducted before considering treatment.

The chiropractic profession postulates that nonspecific back pain is not homogeneous and a significant percentage of mechanical spine pain results from altered function of spinal motion segments. Recent efforts have been directed toward investigating models of differentiating nonspecific spine pain patients into specific subcategories.^{49,50} Evidence is emerging that categorization and “subgrouping” of nonspecific (mechanical) spine pain patients can lead to improved patient outcomes.^{51,52} Although models for subgrouping nonspecific spine pain patients have been based on both diagnostic and treatment categories,^{50,53} both share the premise that grouping patients by shared collections of signs and symptoms will lead to category-specific treatment and more effective outcomes.

Imbedded in the process of subgrouping spine pain patients is the principle that joint adjustments (HVLA thrust-joint manipulation) are not necessarily the most effective treatment for all mechanical spine pain patients. Patients identified with altered spinal or extremity function are most suitable for manipulation. Other diagnostic categories such as clinical spinal motion segment instability or impaired motor control are examples of treatment categories in which continued joint manipulation may not be appropriate and a trial of rehabilitative or proprioceptive exercise would be more suitable.

JOINT SUBLUXATION/DYSFUNCTION SYNDROMES

The chiropractic profession commonly labels functional alterations of spinal motion segments as *joint subluxation* or *joint dysfunction syndromes*. Furthermore, conditions successfully treated with adjustive therapy are often deemed to incorporate altered joint function as a central, associated, or complicating feature.

This is not to imply that chiropractors treat just joint subluxations or dysfunction. Joint subluxation/dysfunction syndromes are commonly associated with other disorders of the NMS system, and it is crucial that chiropractors accurately identify the complex nature of the conditions they are treating. To simplify and reduce all chiropractic care to the detection and treatment of subluxation syndromes misrepresents the broader range of disorders that are effectively treated by chiropractors. Diagnostic oversimplification runs the risk of boxing chiropractors into a limited role—a role in which chiropractors are perceived as providing limited treatment for a very limited number of NMS disorders.

Although the evaluation of joint function is a critical step in the process of determining whether and how to apply adjustive therapy, the identification of subluxation/dysfunction does not conclude the doctor’s diagnostic responsibility. The doctor must also determine if the dysfunction exists as an independent entity or as a product of other somatic or visceral disease. Joint subluxation/dysfunction may be the product of a given disorder rather than the cause, or it may exist as an independent disorder worthy of treatment and still not be directly related to the patient’s chief complaint. Pain in the somatic tissues is a frequent presenting symptom in acute conditions related to visceral dysfunction, and musculoskeletal manifestations of visceral disease are considered in many instances to be an integral part of the disease process, rather than just physical signs and symptoms.⁵⁴

Before adjustive therapy is applied, the doctor needs to eliminate serious pathologic conditions (red flags), consider whether the identified joint subluxation/dysfunction is negatively affecting the patient’s health, exclude contraindications, and determine if the benefits of adjustive therapy outweigh the risks. If therapeutic procedures outside the doctor’s scope of practice are indicated, referral to another chiropractor or other health care provider must be made.

CLINICAL FINDINGS SUPPORTIVE OF JOINT SUBLUXATION/DYSFUNCTION SYNDROME

Joint Assessment Procedures

The evaluation of primary joint subluxation/dysfunction is a formidable task complicated by the limited understanding of potential underlying pathomechanics and pathophysiologic conditions.⁵⁵ In the early stages of primary joint subluxation/dysfunction, functional change or minor structural alteration may be the only measurable event.^{56,57} Evident structural alteration is often not present, or none is measurable with current technology, and a singular gold standard for detecting primary joint subluxation/dysfunction does not currently exist. Therefore, the diagnosis is based primarily on the presenting symptoms and physical findings without direct confirmation by laboratory procedures.⁵⁵

The physical procedures and findings conventionally associated with the detection of segmental joint subluxation/dysfunction (see Chapter 3 and Box 4-4) include pain, postural alterations, regional ROM alterations, intersegmental motion abnormalities, segmental pain provocation, altered or painful segmental end-range loading, segmental tissue texture changes, altered segmental muscle tone, and hyperesthesia and hypesthesia. Although radiographic evaluation is commonly applied in the evaluation for joint subluxation, it must be incorporated with physical assessment procedures to determine the clinical significance of suspected joint subluxation/dysfunction.

At what point specific physical measures are considered abnormal or indicative of joint dysfunction is controversial and a matter of ongoing investigation.⁵⁸ The profession has speculated about the structural and functional characteristics of the optimal spine, but the degree of, or combination of, abnormal findings that are necessary to identify treatable joint dysfunction has not been confirmed.⁵⁹⁻⁶² Professional consensus on the issue is further clouded by debates on how rigid a standard should be applied in the assessment of somatic and joint dysfunction and whether the standard should be set relative to optimal health or to the presence or absence of symptoms and disease. Until a professional standard of care is

BOX 4-4 Clinical Features of Joint Dysfunction

1. Local pain: commonly changes with activity
2. Local tissue hypersensitivity
3. Decreased, increased, or aberrant joint movement
4. Altered or painful joint play
4. Altered and or painful end-feel resistance
6. Altered alignment
7. Local palpatory muscle hypertonicity/rigidity

established, each practitioner must use reasonable and conservative clinical judgment in the management of subluxation/dysfunction. The decision to treat must be weighed against the presence or absence of pain and the degree of noted structural or functional deviation. Minor structural or functional alteration in the absence of a painful presentation may not warrant adjustive therapy.

The evaluation for and detection of joint restriction should not be the only means for determining the need for adjustive therapy. Patients with acute spinal or extremity pain may be incapable of withstanding the physical examination procedures necessary to definitively establish the nature of the suspected dysfunction, yet they may be suffering from a disorder that would benefit from chiropractic care. A patient with an acute joint sprain or capsulitis (facet syndrome, acute joint dysfunction) may have just such a condition, a disorder that limits the doctor's ability to perform a certain physical examination and joint assessment procedures, yet is potentially responsive to adjustive treatment.⁶³

The patient with an acute facet or dysfunction syndrome typically has marked back pain and limited global movements. Radiographic evaluation is negative for disease and may or may not show segmental malalignment. The diagnostic impression is based on location and quality of palpatory pain, the patient's guarded posture, global movement restrictions and preferences, and elimination of other conditions that could account for a similar presentation.⁶³ The physical findings that are often associated with the presence of local joint dysfunction, painful and restricted segmental motion palpation, and end feel are likely to be nonperformable because of pain and guarding.

The decision to implement treatment in such circumstances must then be based on a determination of whether this is a condition that may respond to adjustive therapy. If this is the case, an evaluation to ensure that manipulation can be delivered without undue discomfort should be performed. This is accomplished by placing the patient in the position of anticipated adjustment and gently provoking the joint. If the patient is resistant or experiences undue discomfort during joint testing, other forms of manual or adjunctive care should be considered. Once the patient has progressed to a point at which full assessment is possible, a complete examination to determine the nature and extent of the underlying dysfunction must be performed.

OUTCOME MEASURES

Patient-oriented outcome measures (OMs) are procedures used to measure a patient's clinical status and response to treatment. In the management of NMS conditions, this commonly incorporates measures that assess the patient's pain symptoms, function (impairment), disability (activity intolerance), and general health status (Box 4-5).^{64,65}

BOX 4-5 Outcome Measures for Spine Pain

- Regional mobility measures
- Pain-reporting instruments
- Physical capacity questionnaires
- Physical performance measures
- General health status

In the absence of definitive physical measures for the identification of manipulable spinal lesions, patient-oriented OMs provide a valid tool for measuring patient response to chiropractic treatment. The NMS disorders commonly treated by chiropractors are symptomatic or have a significant effect on the patient's ability to function, establishing the patient as an excellent candidate for functional outcome assessment.^{55,64,66}

Instead of relying solely on procedures traditionally used to identify joint dysfunction/subluxation syndromes, practitioners should also apply procedures that measure the effect their treatment is having on the patient's symptoms and function. In this context, the name and nature of the disorder become less of a focus, and more attention is paid to how the patient is functioning and responding to treatment. The critical issues are to establish functional goals and monitor and document the patient's progress using reliable OMs.

OMs do not necessarily represent the pathophysiologic status of the condition being treated. Instead, they answer questions about the quality or the perception of the patient's life in comparison to the preillness state. OMs that evaluate functional status typically allow the assessment of multiple dimensions of patient functioning (e.g., physical and psychosocial). Many have well-demonstrated reliability and validity and stand as appropriate measures for monitoring the patient's response to treatment.⁶⁴ As such, they can be used to decide if a specific approach to dealing with patient complaints is effective and efficient compared with other approaches. It is the use of reliable and valid OMs in clinical studies and practices that will help quell the critical echoes of unscientific claims.

OMs incorporate self-reporting instruments and physical assessment procedures. Self-reporting instruments generally take the form of questionnaires that are used to quantify the degree of pain or the severity of disability as a result of impairment. Examples of tools that measure pain symptoms include the visual analog scale, which measures and rates a patient's pain intensity and response to treatment; pain drawings, which identify the location and quality of pain; and the McGill pain questionnaire, which measures sensory, cognitive, and motivational elements of pain. Pain intensity can also be evaluated through palpation or with algometry. Palpatory assessment and location of pain have consistently demonstrated excellent reliability (see Chapter 3).

The patient's perception of disability or activity intolerance is commonly measured by any of a number of self-reporting instruments. The Oswestry Disability Questionnaire⁶⁷ and the Roland-Morris Questionnaire⁶⁸ are common instruments applied in LBP disorders. The Neck Disability Index⁶⁹ has been developed and applied for assessing disability associated with neck pain. Other measures that may be incorporated include evaluation of general health and well-being (e.g., Sickness Impact Profile, SF 36, EuroQol, and COOP Charts) and patient satisfaction surveys.⁶⁵

The measurement of physical capacity for selected regional muscles and joints can be evaluated by a variety of physical tasks that measure ROM, muscle strength, and endurance. Normative values have been established for such procedures and can be effectively and economically used to monitor treatment progress.⁷⁰ Four low-tech tests have been studied and have shown good reliability and correlation with spinal pain and disability (Box 4-6).⁷¹

BOX 4-6 Spinal Physical Capacity Tests**REPETITIVE SQUATTING**

Patient stands with feet about 15 cm apart, squats until the thighs are horizontal, and then returns to the upright position. Patient repeats every 2 to 3 seconds, to a maximum of 50.

REPETITIVE SIT-UPS

The patient lies supine with the knees flexed to 90 degrees and ankles fixed. The patient then sits up, touching the thenar aspect of the hand to the patella, and then curls back down to the supine position. Patient repeats to a maximum of 50.

REPETITIVE ARCH-UPS

The patient lies prone with the inguinal region at the end of the table, arms at the sides, ankles fixed (by the examiner or a strap), holding his or her trunk off the table at a 45-degree flexion angle. The patient rises to a horizontal position and lowers back down, with a maximum of 50 repetitions.

STATIC BACK ENDURANCE TESTS

The patient lies prone on the table with the inguinal region at the edge of the table, arms at the sides, ankles fixed (by the examiner or a strap), holding his or her trunk off the table in a horizontal position. The patient maintains the horizontal position for long as possible, for a maximum of 240 seconds.

Broader functional capacity or whole-body movement testing can also be measured. Testing in this arena is more complicated and time consuming. Functional capacity testing is often designed to simulate specific workplace demands and includes such procedures as “lifting, carrying, and aerobic capacity, static positional tolerance, balancing, and hand function.”⁶⁴

CONTRAINDICATIONS TO AND COMPLICATIONS OF ADJUSTIVE THERAPY

As mentioned previously, the clinical corroboration of subluxation/dysfunction syndromes is not, in and of itself, an indication for adjustive therapy. Dysfunction may be associated with, or concomitant with, conditions that contraindicate various forms of manual therapy. A *complication* is defined as a problem that occurs after the application of a procedure. A *contraindication* is a problem identified before a procedure is applied that makes application of the treatment inadvisable because of its potential to cause harm or delay appropriate treatment.

Manual therapy is contraindicated when the procedure may produce an injury, worsen an associated disorder, or delay appropriate curative or life-saving treatment. Although certain conditions may contraindicate thrusting forms of manual therapy, they may not prohibit other forms of manual therapy or adjustments to other areas.^{72,73}

When manual therapy is not the sole method of care, it may still be appropriate and valuable in the patient's overall health management and quality of life. For example, manual therapy, if not contraindicated, may help a cancer patient gain some significant pain relief and an improved sense of well-being. “Such

palliative care should be rendered concomitantly and in consultation with the physician in charge of treating the malignancy.”⁷²

All disorders listed as potential contraindications to adjustive therapy are not necessarily *absolute contraindications* to thrust manipulation. Certainly, some disorders contraindicate any form of thrust manipulation, but many potentially risky conditions depend on the stage of the disorder and its pathologic process. Many of the disorders or defects identified as potential contraindications to manipulation are therefore *relative contraindications*. A *relative complication* implies that caution should be used in applying adjustive therapy and consideration given for possible modifications in the adjustive treatments provided. The decision to treat depends on the individual circumstances of the presenting case. For example, what is the patient's age and state of health? What is the nature of the potentially complicating pathologic condition? Is the disorder in a state of remission or exacerbation, or is it in its early or late stages of development?

Serious injuries resulting from adjustive therapy are very uncommon.⁷⁴⁻⁸⁶ Suitable adjustive therapy is less frequently associated with iatrogenic complications than many other common health care procedures.⁸³ The majority of spinal manipulation complications arise from misdiagnosis or improper technique. In the majority of situations, it is likely that injury can be avoided by sound diagnostic assessment and awareness of the complications and contraindications to manipulative therapy. Conditions that contraindicate or require modification to spinal manipulation are listed in Table 4-1.

Although the incidence of injury from manipulation is extremely low, mild associated transitory discomfort is not unusual. Adverse reactions and reported complications to spinal thrust manipulation run the gamut from mild increased local discomfort to very rare but serious permanent neurologic complications or death.^{87,88} The best available evidence indicates that chiropractic care is an effective option for patients with mechanical spine pain³⁷ and is associated with a very low risk of associated serious adverse events.⁸⁹⁻⁹¹

Senstad, Leboueuf-Yde, and Borchgrevink,²⁵ using a prospective clinic-based survey, studied the frequency and characteristics of side effects to spinal manipulative therapy (SMT). Information regarding any unpleasant reactions after SMT was collected on 580 patients and 4712 spinal manipulative treatments by Norwegian chiropractors. The researchers report that at least one reaction was reported by 55% of the patients some time during the course of a maximum of six treatments. Treatments were not limited to manipulation (36% of visits were soft tissue manipulation and 25% had both soft tissue and thrust manipulation). It is unknown to what degree soft tissue manipulation may have affected the rate of reported side effects. Therefore the findings of this study outline the rate of side effects for common chiropractic treatments, but do not provide a precise rate for thrust manipulation alone.

The most common reactions were increased musculoskeletal pain. Increased local discomfort accounted for 55%, headache 12%, tiredness 11%, or radiating discomfort 10%. The reactions to treatment usually did not interfere with activities of daily living and were rated as mild or moderate in 85% of the cases; 64% of reactions appeared within 4 hours and 74% disappeared within 24 hours. A prospective multicenter cohort study (2007) evaluating cervical manipulation and adverse events found very similar results.⁸⁹

TABLE 4-1 Conditions That Contraindicate or Require Modification to High Velocity–Low Amplitude Spinal Manipulative Therapy

Condition	Potential Complication from Manipulation	Method of Detection	Management Modifications
Atherosclerosis of major blood vessels	Blood vessel rupture (hemorrhage) Dislodged thrombi	Palpation Auscultation X-ray examination Visualization Doppler ultrasound	Soft tissue and mobilizing techniques with light or distractive adjustments Referral to vascular surgeon
Vertebrobasilar insufficiency	Wallenberg syndrome Brainstem stroke	History Doppler ultrasound Angiography MRA	No cervical thrusting techniques Referral to anticoagulant therapy
Aneurysm	Rupture Hemorrhage	Irregular pulse Abdominal palpation Auscultation X-ray examination	Referral to vascular surgeon
Tumors	Metastasis to spine Pathologic fracture Disease progression	Palpation X-ray examination Laboratory findings MRI CT	Referral*
Fractures	Increased instability Delayed healing	Radiograph CT	Referral*
Severe sprains	Increased instability	Stress x-ray examination Motion palpation	If severe, referral* If not, manipulation of areas of fixation
Osteoarthritis (late stage)	Neurologic compromise Increased pain	Radiograph	Mobilization Gentle manipulation Distractive adjustments
Uncarthrosis	Vertebral artery compromise or dissection	Radiograph	Gentle traction Mobilizing and soft tissue techniques
Clotting disorders	Spinal hematoma	History of anticoagulant therapy Pulse Bruises	Forceful manipulation contraindicated
Osteopenia (osteoporosis)	Pathologic fracture	History of long-standing steroid therapy Postmenopausal females Malabsorption syndrome Nutritional deficiencies Anticonvulsive medication X-ray examinations	Forceful manipulation contraindicated Mobilizing technique with light distractive adjustments
Space-occupying lesions	Permanent neurologic deficits	MRI CT (myelography)	Referral*
Diabetes (neuropathy)	Unresponsiveness to pain	Laboratory findings Examination of lower extremities Skin (trophic changes) Pulse	Referral*
Malingering Hysteria Hypochondriasis	Prolonged treatment Treatment dependency	Symptom amplification Waddell scale Libman test	Referral* for psychologic evaluation Active care
Alzheimer disease	Inappropriate response or unresponsiveness to pain or treatment	Mental status evaluation	Gentle manipulation Mobilizing and soft tissue techniques

MRA, Magnetic resonance angiography; MRI, magnetic resonance imaging; CT, computed tomography.

*NOTE: Although referral for medical treatment of the specific pathologic process is deemed appropriate and necessary, it does not preclude the patient from receiving manipulative therapy to unaffected areas or, in some cases, to the areas of pathology for symptomatic relief or quality-of-life enhancement.

The study involved 79 chiropractors and 529 subjects over 12 months. The most common adverse events were with a mild to moderate transitory increase in musculoskeletal pain (70% to 75%). No serious adverse events were reported during the study period.

The overwhelming majority of reported side effects fall within the category of acceptable reactions. Their occurrence is likely a normal product of manual therapy and the mobilization or stimulation of periarticular soft tissues. There were no reports of any serious complication in this study, and 5% of patients or less reported uncommon and transitory reactions of dizziness, nausea, or hot skin.

Kleynhans⁷⁷ has suggested labeling reactions as *normal* and *adverse* to distinguish those postadjustive reactions that are expected from those that are unwanted. Normal reactions reflect the minor increased discomfort that is anticipated to occur in a significant percentage of patients who have been successfully treated. Adverse reactions reflect the more uncommon reactions that lead to more significant discomfort and temporary or permanent impairment.

Dvorak and colleagues⁸⁴ have proposed a more detailed division of postadjustment effects, including two major categories (reactions and complications) and four subcategories (Box 4-7).

BOX 4-7 Adjustive Side Effects

REACTIONS

Adequate Reaction

- Onset 6 to 12 hours
- Mild subjective symptoms
- Local soreness
- Tiredness
- Headache
- No decreased work capacity
- Less than 2 days' duration
- Spontaneous remission

Exceeding Reaction

- Onset 6 to 12 hours
- Objective worsening of signs and symptoms
- Interferes with work
- More than 2 days in duration
- Spontaneous remission

COMPLICATIONS

Reversible Complication

- Onset within 2 days
- Requires diagnostic or therapeutic interventions
- Tissue damage
- Patient can return to preoccurrence status

Irreversible Complication

- Onset within 2 days
- Requires diagnostic or therapeutic interventions
- Permanent tissue damage and impairment result

Reactions are transient episodes of increased symptoms that resolve spontaneously. They are not associated with any organic worsening of the underlying condition or new iatrogenic injury. Complications are associated with new tissue damage and require a change in therapeutic approach.

Reactions are further subdivided into *adequate (acceptable)* and *exceeding*. Adequate (acceptable) reactions are transient episodes of increased discomfort or mild associated symptoms that resolve spontaneously. Adequate (acceptable) reactions are subjective complaints that do not last longer than 2 days and do not interfere with the patient's work capacity. Exceeding reactions are associated with more pronounced discomfort, objective worsening of the signs and symptoms, decreased work capacity, and a duration longer than 2 days.

Complications are divided into reversible and irreversible categories. With *reversible complications*, the pathologic condition associated with the incident is reversible, and the patient eventually returns to a preoccurrence state. *Irreversible complications* result in some degree of permanent disability.

The low documented risk of serious injury resulting from spinal adjustive therapy does not release the doctor from the responsibility of informing the patient about the procedures to be performed and of the potential for any significant associated negative consequences.⁹¹ The patient must understand the nature of the procedure and give written, verbal, or implied consent before therapy is applied. The patient's consent to treatment must be documented in his or her health record. Any unauthorized diagnostic evaluation or treatment is unacceptable and exposes the doctor to the potential charge of malpractice as well as assault and battery.

Patients have the right to know about significant risks and treatment options before consenting to examination and care.^{82,91} Despite the concern that detailed discussion of rare complications would unduly alarm patients and lead many to reject beneficial treatment,⁹² patients should be informed in circumstances in which "there is risk of significant harm."⁹³

What constitutes a material and significant risk is debatable but typically interpreted widely by the courts. In a Canadian case (*Mason v. Forgie*) involving cervical manipulation and subsequent cerebrovascular accident (CVA), the rare but serious potential complication was deemed material. In Canada, this has led to professional guidelines requiring informed written consent before applying a patient's first cervical thrust manipulation.⁹²

In the United States, guidelines and formal policies have not yet been developed along the explicit lines that they have in Canada. However, lack of documented informed consent is felt by the profession's largest malpractice insurer, National Chiropractic Mutual Insurance Company (NCMIC), to be a significant cause of action for filing malpractice suits. This company recommends that all practitioners contact an attorney in their area who specializes in health care law for advice on the standards for obtaining informed consent.

CERVICAL SPINE

Critics of manipulative therapy in general, and chiropractic specifically, emphasize the possibility of serious injury from cervical

manipulation while downplaying the benefits of cervical manipulative therapy.⁸⁸ Although case reports of serious complications associated with cervical manipulation are rare events,^{86,87,89} it has required only the rare occurrence to “malign a therapeutic procedure that in experienced hands gives beneficial results with few side effects.”⁹³

Case reports of serious complications from cervical spine manipulation include a range of neurovascular complications including cerebrovascular strokes from injuries to the vertebral or carotid arteries, cervical myelopathy or radiculopathy secondary to meningeal hemorrhage or herniated discs, Horner syndrome, and diaphragmatic paralysis.^{88,94} Other non-neurovascular injuries such as pathologic fracture, dislocations of cervical vertebrae, disc herniation, dislocations of atlas on axis as a result of agenesis of the transverse ligament (found in Down syndrome), and rupture of the transverse ligament (found in inflammatory arthropathies) have also been reported.^{87,88} The case reports of postmanipulative complications represent a very small percentage of patients receiving spinal manipulation. They inform us that rare postmanipulative complications may develop and continued clinical research is indicated. However, they are primarily retrospective and cannot be used to establish a predicative cause-and-effect relationship between any specific form of manual therapy and the development of serious complications.⁹⁵

Cervical Artery Injury and Cerebrovascular Events

The proposed serious side effect of cervical manipulation that receives the most attention is damage to the vertebral artery and subsequent vertebrobasilar artery (VBA) stroke. Although a biologically plausible mechanism has been proposed, a causal relationship between cervical manipulative therapy and VBA strokes has not been established.^{86,96-98} The initial injury is speculated to result from manipulation-induced disruption and dissection of the vessel wall. Damage to the vessel wall is speculated to induce an occlusive vertebrobasilar infarct secondary to thrombosis or embolism formation. The literature also contains reports of postmanipulative internal carotid artery dissection (ICAD) and neurovascular complications. However, a literature review conducted in 2003 identified only 13 cases. The authors concluded that the “medical literature does not support a clear causal relationship between chiropractic cervical manipulation and ICAD.”⁹⁹

Vertebral Artery Anatomic Considerations. Any discussion concerning the biologic plausibility and potential causal relationship between cervical manipulation and vertebral artery injury should begin with a review of the relevant anatomic relationships. The vertebral artery, the first branch from the subclavian trunk, becomes closely related to the spine by entering the transverse foramen at the sixth cervical vertebral level. It then passes through the transverse foramen from C6 to C1, lying directly in front of the cervical nerves and medial to the intertransverse muscles (Figure 4-3).

Accompanying the artery is the vertebral plexus of veins and the vertebral nerve, composed of sympathetic fibers arising from the inferior (stellate) ganglion. After leaving C2, they pass with the artery through the transverse foramen of the atlas, necessitating a sharp deflection outward, a tortuous course around the poste-

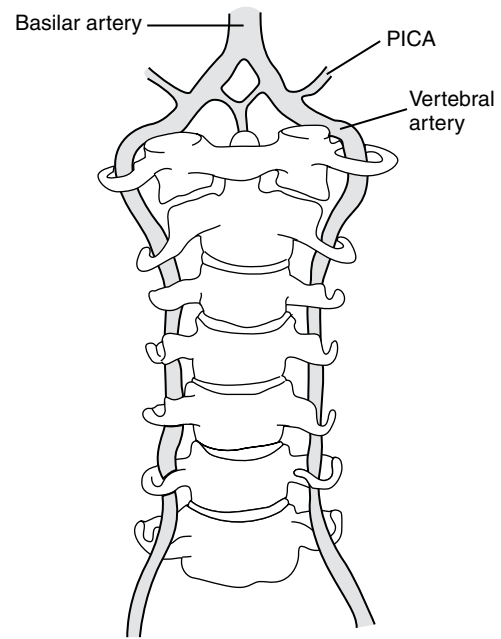


Figure 4-3 Relationship of the vertebral artery to the cervical spine. PICA, posteroinferior cerebellar artery.

rolateral aspect of the superior articular process of the atlas. As the artery heads posterior, it passes by the atlanto-occipital joint capsule and through the arcuate foramen, which is formed by the posterior atlanto-occipital membrane. As the artery travels over the atlas, it lies in a groove in the posterior arch of the atlas, which it shares with the first cervical nerve. This groove can range in depth from a shallow indentation to a complete bony ring. It then turns upward and runs through the foramen magnum into the cranial cavity and passes to the lower border of the pons, where it joins the opposite vertebral artery to become the basilar artery. The basilar artery runs a relatively short course and then splits to form the circle of Willis, which is joined anteriorly by the internal carotid arteries.

At the foramen magnum, a branch comes off of each vertebral artery to unite with the anterior spinal artery that descends on the anterior surface of the cord. These branches give off further branches, forming the posterior spinal arteries that supply the cord down to the level of T4. Another branch of the vertebral artery, the posteroinferior cerebellar artery (PICA), leaves the vertebral artery just before their conjunction. The PICAs are the largest branches of the vertebral artery and run a tortuous course along the lateral aspect of the medulla, to which they are the main blood supply.¹⁰⁰ The vertebrobasilar system also supplies the inner ear, the cerebellum, most of the pons and brainstem, and the posterior portion of the cerebral hemispheres, especially the visual cortex.

Branches from the vertebral artery also supply blood to the facet joint structures, the NRs, and the dorsal root ganglia. These branches then form free anastomoses with the anterior and posterior spinal arteries, both of which are derivatives of the vertebral artery.^{101,102} Most vertebral arteries are markedly unequal in diameter. The diameter of one, usually the left, may be three times larger than that of the right. One vessel may be congenitally absent.¹⁰³

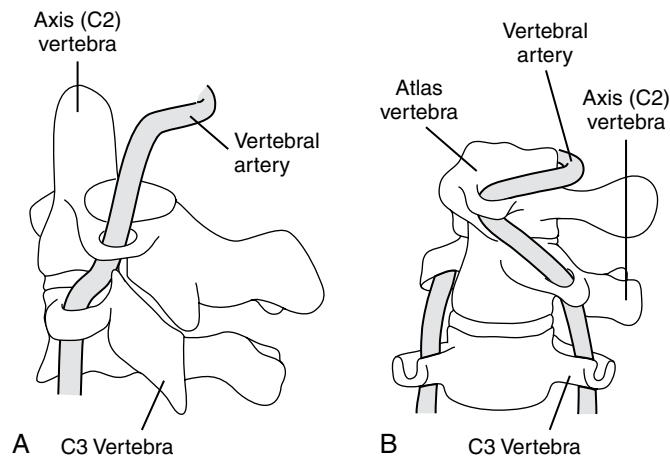


Figure 4-4 Diagram illustrating the relationship of the vertebral artery to the upper cervical spine. **A**, In the neutral position, the vertebral artery passes through the transverse foramen without any traction or compression. **B**, During right rotation, the left vertebral artery is tractioned as the atlas rotates forward on the left.

Theoretic Mechanical Model of Vertebral Artery Injury. The anatomy of the cervical spine and the relationship of the vertebral arteries to neighboring structures make the arteries potentially vulnerable to mechanical compression and trauma. Variation in the diameter of the arteries is thought to contribute to obstruction and thrombosis, and attention has been drawn to the potential susceptibility of vertebral arteries at the atlantooccipital articulation. Specific head and neck movements have been proposed as the source of potential mechanical injury to the vertebral artery and provide the potential link to cervical spine manipulative therapy. End-range neck movements are speculated to affect vessel wall integrity by inducing injurious compression or stretching of the arterial wall.¹⁰⁴ Rotation with extension has been proposed as the most risky movement. The contralateral vertebral artery is postulated as the vessel most at risk because of vessel stretching or compression that occurs with rotation of the atlas (Figure 4-4).

The postulated sites and mechanisms for extraluminal vertebral artery obstruction associated with head movement include the following:

1. Skeletal muscle and fascial bands at the junction of the first and second vertebral segments
2. Adjacent osteophyte, particularly at C4–5 and C5–6
3. Between the C1–2 transverse processes, where the relatively immobile vertebral arteries may be stretched or compressed with rotary movements
4. By the C3 superior articular facet on the ipsilateral side of head rotation

Traumatic compression or stretching of the artery wall may lead to a subintimal hematoma or intimal tear (Figure 4-5). A subintimal hematoma may lead to partial or complete occlusion of the lumen. Tearing of the intimal layer can lead to pooling of blood that serves as a space-occupying lesion. Blood rushing past an intimal tear can also potentially dissect away the vessel wall, creating a subintimal hemorrhage or dissecting aneurysm (see Figure 4-5). A tearing of the intima results in exposure of the subendothelial tissue and clot formation. With repair, no further problems may be encountered, or a biochemical cascade and repair process may

be triggered, resulting in thrombus formation. The propagating thrombus may impair blood flow, increase turbulence, and lead to further clotting and thrombus growth (see Figure 4-5). Blood flow may break off a portion of the thrombus, resulting in a floating embolus and infarct where it lodges in a distal arterial branch. In the case of the vertebral artery, this may result in occlusion of the PICA. An infarct in the PICA results in a brainstem stroke referred to as *Wallenberg syndrome*. It is characterized by clinical findings associated with structures innervated by the cranial nerves. A less common occurrence is occlusion of the basilar artery and more serious neurologic complications (locked-in syndrome) with conservation of only vertical ocular mobility and blinking.

Attempts to determine the relationship between neck movements and their effects on vertebral artery blood flow have led to a number of Doppler ultrasound studies conducted on both cadaveric and human volunteers. Cadaveric studies have implicated rotation as the single most likely movement to cause reduction in blood flow. Lateral flexion and extension movements individually were found to have little effect in altering blood flow. With pure rotation, the contralateral artery was compromised more often. Reduction in blood flow occurred toward the end of rotation but still within the normal range of head motion. However, when rotation was combined with extension, the ipsilateral artery was involved as frequently as the contralateral artery.^{102,105,106}

Studies conducted on healthy volunteers and subjects who have a history of dizziness or positive positional tests have demonstrated mixed results. All studies conducted through 1996 have used Doppler ultrasound evaluation of vertebral blood flow velocity. Results have ranged from complete reduction of blood flow to no measurable change.¹⁰⁷

Beginning in 1998, Licht and associates¹⁰⁷ published the results of a series of studies conducted with the aid of more advanced digitized color-flow duplex Doppler ultrasound techniques. The researchers found modest reductions in vertebral artery blood flow in full contralateral rotation and a mild increase in vertebral artery blood flow in ipsilateral rotation.¹⁰⁷ Licht and associates¹⁰⁸ believed that many of the previous studies, which had demonstrated significant variations in the effects of neck positions on vertebral artery blood flow, may have had less-than-accurate recordings as a result of less sophisticated technology. Potential errors were speculated to have resulted from inadvertently investigating the wrong vessel, establishing an inappropriate angle of insonation, or missing the vertebral artery as the patient's head was rotated.

In 1999, Licht, Christensen, and Houlund-Carlson¹⁰⁹ expanded the investigation and reported for the first time on the effects of cervical rotation on blood volume flow through the vertebral arteries. Measures of blood volume were more representative of vertebral artery perfusion and clinically more relevant. In the evaluations of the same 20 asymptomatic volunteers, no significant changes in vertebral artery blood volume was noted, despite reductions in contralateral blood flow velocity. Blood flow volume was also unchanged 3 minutes after manipulation in subjects deemed to have a cervical dysfunction.

In 1999, Yi-Kai and co-workers¹¹⁰ using transcranial Doppler, found vertebral artery flow to be decreased with extension and rotation in both cadaveric and human subjects. The most marked reductions were noted when extension was coupled with rotation.

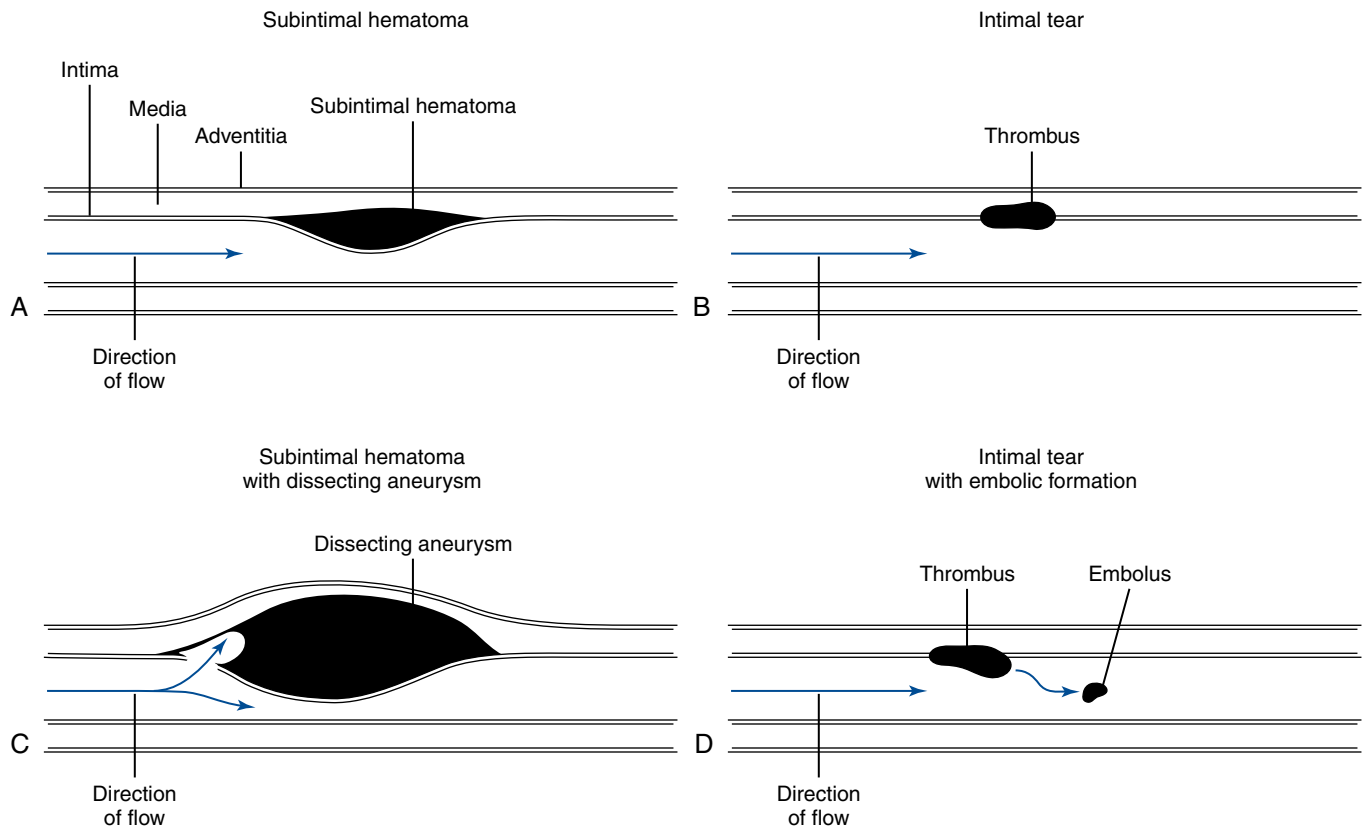


Figure 4-5 Diagram illustrating vessel injury and the pathologic sequence of events that can lead to vessel occlusion. **A**, Subintimal hematoma. **B**, Thrombus formation. **C**, Dissecting aneurysm. **D**, Embolus formation.

Extension coupled with rotation reduced blood flow in both vertebral arteries, but the reduction was most pronounced in the contralateral vertebral artery. In 2002 Haynes conducted Doppler velocimetric and magnetic resonance angiography (MRA) blood flow assessments on eight healthy middle-aged adults.¹⁰⁴ He concluded that end-range rotation did not demonstrate significant stretching, narrowing, or major blood flow change in the contralateral vertebral artery. However, vessel stenosis and potential stress from localized compression of the vertebral artery at the level of the C2 transverse foramen was noted.

The cadaveric, human subject Doppler and MRA vertebral artery studies do suggest a relationship between cervical movements and vertebral artery blood flow, but they do not answer the question of whether cervical manipulative therapy has any negative effects. To investigate the potential for vessel injury, Symons and colleagues applied manipulative-level forces to freshly dissected vertebral arteries.¹¹¹ They dissected six vertebral arteries from five fresh, unembalmed, postmortem cadavers and strained the arteries on a materials testing machine. They concluded that the strains associated with SMT “were almost an order of magnitude lower than the strains required to mechanically disrupt the artery and were similar to or lower than the strains recorded during range of motion testing.”¹¹¹ They concluded that under normal circumstances, a single thrust to the cervical spine would be very unlikely to mechanically disrupt the vertebral artery.

Although this study does provide some biologic evidence that healthy vertebral arteries are unlikely to be injured during cervical

manipulation, it cannot be generalized to clinical practice and it does not address the issue of whether underlying arteriopathy may make the vertebral arteries more susceptible to dissection. Potential pathophysiologic models of vertebral artery dissection (VAD), not associated with major trauma, have been presented. They are based on the hypothesis that VAD is unlikely to occur unless there is some environmental trigger or risk factor (e.g., infection, oral contraceptives, smoking, atherosclerosis, trivial trauma associated with neck movements such as sporting events or manipulative therapy) superimposed on an underlying genetic predisposition (e.g., connective tissue disease, hyperhomocysteinemia, vessel abnormality).¹¹² Further research is needed to evaluate the validity of this hypothesis and determine whether VAD risk factors can be identified.

Based on reviews of case reports, Terrett¹¹³ concluded that 94.5% of the reported cases of apparent post-manipulation-induced stroke involved neck rotation. Evaluation of the literature also indicated that adjustments delivered to the upper cervical spine as compared with the lower cervical spine were more frequently associated with complications. Based on this analysis, Terrett¹¹⁴ and Terrett and Kleynhans¹¹⁵ reasoned that rate of injury could be reduced by avoiding rotational tension or rotational manipulation in the upper cervical spine. They subsequently recommended that rotational manipulation of the upper cervical spine be abandoned in favor of lateral flexion adjustments.

However, rotational-type adjustments are the most commonly applied thrusting procedures to the neck, and the higher incidence

of injury may simply be a product of their common application. A 1999 assessment⁹⁷ of the literature supports this hypothesis. Their literature review failed “to show a consistent position or movement of the neck that could be considered particularly dangerous.”⁹⁷ Almost all forms of manipulation have been implicated and, if the relationship is temporal and not causal, or occurring in patients with genetic predilections to trivial trauma, then almost all potential minor activities of daily living could precede a VAD. Adjustive procedures reported to minimize rotation and vertebral artery injury, such as Gonstead and activator instrument adjustments, have also been associated with reported adverse reactions. Moreover, primarily nonmanipulating practitioners (e.g., neurologists, vascular surgeons, and pathologists) have written the reports of vascular accidents associated with manipulation. Their interpretations of accounts, events, and procedures reported by patients, relatives, or witnesses might be lacking in understanding and accuracy.¹¹³ A review of the English literature before 1996 revealed that 60.87% of the cases reported had no description of the manipulation used, and only two of the reports had accurate detailed information about the manipulative methods applied.⁹⁷ This brings into question much of the data and conclusions that have been drawn about who is delivering the manipulation and the relationship between specific types of manipulation and vertebral artery injury.

If a practitioner wishes to minimize rotational tension during the application of cervical adjustments, he or she need not abandon rotational adjustive procedures. A more prudent approach would be to apply only rotational manipulation when indicated and apply it in a manner that minimizes full rotational tension with extension. It is important to distinguish between an adjustive procedure that induces segmental rotation using maximal rotational prestress from other adjustive procedures designed to minimize full rotational stress and tension on the vertebral artery. Inducing coupled lateral flexion in the direction opposite the induced rotation can prevent full rotational tension (Figure 4-6). Both influence segmental rotation and mechanically may be similar. However, the end-range procedure may place unnecessary stress on other

structures, including the vertebral arteries. It is recommended that a “premanipulation” position be held for a short while and that it be explained to the patient that this is the position that will be used for treatment, thereby giving the patient an opportunity to say whether the position causes any symptoms or discomfort.

Incidence of Manipulation-Associated Vertebral Artery Injury and Stroke

VAD and VBA strokes are exceedingly rare events. “It is estimated that VBA dissections regardless of the etiology comprise only 1.3 in 1000 cases of stroke per year. The dissection rate in the general population is estimated to be 0.97 to 1.2 per 100,000 individuals,^{86,116} with major medical centers encountering only 0.5 to 3 cases of this disorder per year.”⁹⁷ Because of the rarity of this condition, estimates of the potential incidence of manipulation-linked VAD and stroke have relied on analysis of case reports, series, surveys, and observational studies.^{80,81,83,88,113,117-124} Based on a number of citations, the estimated incidence of VBA stroke following or occurring during cervical manipulation is reported to range from less than 1 in 2 million to 1 in 3.8 to 5.8 million cervical manipulations.^{98,125}

In 1983, Dvorak and Orelli¹²¹ conducted one of the first comprehensive surveys on incidence of complications after cervical manipulation. They surveyed 203 practitioners of manual medicine in Switzerland and found a rate of one serious complication per 400,000 cervical manipulations, but reported no deaths among an estimated 1.5 million cervical manipulations. In 1995, Dabbs and Lauretti⁸³ reported an estimated rate of less than one stroke per 2 million cervical manipulations, based on a review of the literature and CVA claims settled in a 3-year period by the National Chiropractic Mutual Insurance Company. An extensive survey conducted by Klougart, Leboueuf-Yde, and Rasmussen⁸⁰ evaluated the records of all the Danish Chiropractors’ Association members from 1978 to 1988 and found one case of VBA stroke for every 1.3 million cervical manipulations. In the 10-year review of Danish chiropractors’ records, they found only five cases, with one case resulting in death. Another extensive literature review, performed to formulate practice guidelines, concluded that “the risk of serious neurological complications from cervical manipulation is extremely low, and is approximately 1 or 2 per million manipulations.”¹²² A comprehensive study published by Haldeman, Kohlbeck, and McGregor⁹⁷ in 1999 reviewed the English literature for all reported cases of VBA dissection and occlusion and documented 367 primary case reports. Of this pool, 160 (44%) were described as spontaneous and 115 as postmanipulation (31%), and 58 were associated with minor trauma and 37 with major trauma.⁹⁷ Postmanipulation-linked cases represented a smaller percentage of cases than spontaneous VAD.

Dobbs and Lauretti⁸³ estimated that one VAD would occur per 100,000 chiropractic patients. This was based on the assumption of one VBA-associated stroke per million manipulations, and 10 to 15 treatments per mechanical neck pain syndrome. Their estimates approximate those of a recent best-evidence review by Miley and colleagues.¹¹⁸ They estimated that within 1 week of treatment, approximately 1.3 cases of VAD will occur for every 100,000 patients. No relationship was noted in patients older than 45 years of age. Of all CVAs, it is estimated that approximately one fourth will be fatal¹²⁶ and one third will resolve with mild or no residual effects.⁹⁶ This results in an estimated death rate of 1 per 400,000 patients



4-6

Figure 4-6 Right rotational adjustment in the upper cervical spine with coupled left lateral flexion and avoidance of coupled extension to minimize full rotational tension.

(0.0000025%) who seek chiropractic care.⁸³ For comparison, a geriatric population of patients treated with nonsteroidal anti-inflammatory drugs (NSAIDs) for osteoarthritis had an estimated rate of serious complication of 0.4% and an estimated death rate from gastric hemorrhage of 0.04%. This rate of complication results in an estimated annual mortality rate of 3200 deaths per year in the United States from NSAID-induced ulcers among geriatric patients treated for osteoarthritis.⁸³ These rates of serious complication and death are considered rare by medical standards and are many magnitudes the estimated incidence of reported serious complication associated with cervical manipulation.⁸³

Because the estimates of association between cervical manipulation and VBA stroke have been predominantly based on evaluation of case reports and surveys, some have suggested that the risk of manipulation-linked VBA strokes may be understated.⁸⁸ On the other hand, there is also evidence to suggest that the incidence of chiropractically attributed VBA strokes are overestimated.^{127,128} Terrett¹²⁷ concluded that many of the reported cases were attributed incorrectly to chiropractors. A significant number of the cases reviewed implicated chiropractic manipulation when the therapist performing the procedures was a medical doctor, physiotherapist, or person without formal health care training. In addition, the larger health care community, public press, and legal community have become increasingly aware of a possible relationship between manipulation and complications.¹²⁹ In this environment, it seems unlikely that serious complications of cervical manipulation would be significantly under-reported.¹²⁸

There have been three recent epidemiologic studies addressing the possible association of cervical SMT and VBA stroke. Two case controls and one very large population-based case control-case crossover study have been performed. The first by Rothwell, Bondy, and Williams,¹³⁰ published in 2001, compared 528 cases of VBA stroke to 2328 matched controls. They identified a five-fold increased risk of VBA stroke in individuals younger than age 45 who had visited a chiropractor within the previous week. The results were based on the identification of only six identifiable cases and an estimated incidence rate of 1.3 per million cases. Smith and co-workers,¹³¹ in 2003, compared 100 nondissection-related stroke patients to 51 individuals diagnosed with cervical artery dissection. No significant association between stroke or transient ischemic attack (TIA) and neck SMT was identified. However, a subgroup analysis did identify 25 cases of VAD in which a visit to a chiropractor was six times more likely to have occurred within the previous month than in the control group. The study was criticized for several methodologic shortcomings, including selection, information, and recall bias.¹³²

Although both studies identified a possible temporal relationship between SMT and VAD, it is not possible to attribute a definitive causal relationship between cervical manipulation and VAD and VBA stroke by retrospective case control studies. It is possible that all, or some percentage, of the postmanipulative-associated VBA strokes are spontaneous and temporally not causally associated with cervical manipulation. VAD and VBA stroke may be associated with chiropractic care because patients with VAD are seeking treatment based on symptoms associated with a dissection already in progress.¹³³ Spontaneous VAD may initially present as neck pain and headaches. Neck pain and headaches are a common

presentation for patients seeking chiropractic care. Furthermore, in a number of the reported postmanipulation cases, symptoms of vessel damage and stroke do not materialize until hours or days after treatment. In such circumstances, it is possible that the treating doctor was administering manipulation to a patient with a spontaneous artery dissection already in progress or to a patient who developed a spontaneous dissection after treatment.

To further investigate the question of whether chiropractic SMT is temporally or causally connected to VAD, Cassidy, Boyle, and Cote⁸⁶ compared the incidence of VBA stroke with chiropractic visits and primary care provider (PCP) visits. The hypothesis was that if chiropractic care increases the risk of VBA stroke, then the incidence of VBA stroke should be higher with chiropractic visits than PCP visits. The study concluded that VBA stroke was a very rare event in both patient populations, with no evidence of an increased risk of occurrence with chiropractic care as compared with PCP care. The study population included all residents older than 9 in Ontario, Canada. It evaluated all hospital-admitted VBA strokes (818) between 1993 and 2002. In individuals younger than 45 years, visits to chiropractors and PCP providers were associated with a threefold increased rate of VBA stroke. There was no increased association between chiropractic visits and VBA stroke in individuals older than age 45.

Because it is unlikely that PCP care is associated with any management procedures that are likely to cause stroke, the results of this study support the authors' conclusions that the increased association between chiropractic visits and PCP visits is likely the product of patients seeking care for symptoms associated with a VBA dissection before a stroke has occurred (VBA prodrome).⁸⁶

Screening and Prevention of Vertebral Artery Dissection.

Chiropractors have the potential to affect the development or outcome of a VAD by either identifying patients with signs of a dissection in progress or by avoiding diagnostic or therapeutic procedures that could induce a VAD. Recent evidence indicates that chiropractic cervical SMT is most likely temporally and not causally associated with VAD and VBA stroke in that patients seek care for symptoms associated with an undiagnosed VAD in progress.⁸⁶ In this situation, clinicians need to be trained to identify and immediately refer any patient with signs of an evolving VAD.¹³³

Other theoretic models have been presented suggesting that VAD may also be associated with patients who may have a pre existing genetic predisposition to arteriopathy. This model suggests that cervical artery dissection "is a product of an underlying predisposition triggered specifically by risk factors associated with environmental exposure, with or without trivial trauma."¹¹² In this situation the identification of potential risk factors is paramount. Genetic risk factors capable of compromising vessel wall integrity have been proposed and include connective tissue disease (e.g., Ehlers-Danlos syndrome, Marfan syndrome), hyperhomocysteinemia, migraine, and vessel abnormalities. Potential triggers include "(1) environmental exposure (e.g., infection, oral contraceptives), (2) trivial trauma (common neck movements, sporting activities, manipulative therapy), and (3) atherosclerotic-related disease (e.g., hypertension, diabetes mellitus, smoking)."¹¹² Although numerous risk factors have been postulated for VAD, none have been clearly established.

At this time no clinical diagnostic tests have been developed to identify patients at risk for a VAD. However, it is essential that chiropractors stay abreast of the evolving literature and, when possible, develop the ability to identify potential risk factors for VAD.

The common risk factors associated with atherosclerosis (hypertension, diabetes, smoking, oral contraceptive use, and high cholesterol levels) are less likely to be associated with VAD than non-VAD ischemic strokes.¹³⁴ With the exception of cigarette smoking, the other typical vascular risk factors demonstrated a negative association with VAD. This correlates with the findings that most patients who have developed postmanipulative VBA strokes are young to middle-aged individuals who are apparently healthy; suffer from musculoskeletal complaints such as head, neck, or shoulder pain; and have no significant history of hypertension or hypotension. The most common age range for VBA stroke is younger than 45, contradicting the impression that this is a problem in the older adult population.¹¹³ Furthermore, a history of successful cervical manipulation without complications does not appear to reduce the risk of future complications with manipulation.^{93,96,113,135}

A 2005 systematic review of risk factors associated with cervical artery disease (CAD) identified associations between aortic diameter, diameter change of the carotid artery during the cardiac cycle, alpha-1 antitrypsin genetic protease inhibitor deficiency, migraine, trivial trauma, and age younger than 45.¹³⁴ A weak association was found with high levels of homocysteine and recent infection. Most of the reviewed studies had several sources of potential bias or inadequate data analysis, leading the authors to conclude that the relationship between arteriopathy and CAD has been insufficiently studied.¹³⁴

The potential for trivial trauma (including manipulation-induced trauma) to induce VAD has been widely discussed in the health care literature and the popular media. A number of physical activities and specific movements temporally associated with VAD and VBA stroke has been reported. The majority of these activities are not associated with significant trauma and are likely the result of a noncausal temporal relationship or the product of trivial trauma in a patient with pre existing arteriopathy (Box 4-8).¹³⁶ A 2005 systematic review did not find any studies that suggested “common neck movements pose an independent risk factor for VAD.”¹³⁴

Examination. Cervical manipulation should be preceded by an appropriate problem-based history and physical examination. The assessment should include a systems review and family health history.⁹⁸ Any identified cerebrovascular risk factors or concerns should stimulate a “close observation of neurologic status.”⁹⁸ Currently there is no established history or physical examination findings that predict whether a patient will develop a VAD. However, there are clinical findings that appear to be more associated with the development of VAD. The identification of these findings should raise the clinician’s index of suspicion and concern for the possibility of developing a VAD.⁹⁸ The most extensive monograph covering cervical spinal manipulation and cervical artery incidents recommends the factors listed in Box 4-9 as the most important elements to consider in the clinical assessment of a patient being considered for neck manipulation.⁹⁸ Those listed in Box 4-10 are important features warning of possible CAD.

BOX 4-8

Activities Associated with Vertebrobasilar Artery Stroke

Childbirth
Head movements by surgeon or anesthetist during surgery
Calisthenics
Yoga
Overhead work
Neck extension during radiography
Neck extension for a bleeding nose
Turning the head while driving a vehicle
Archery
Wrestling
Emergency resuscitation
Stargazing
Sleeping position
Swimming
Dancing
Fitness exercise
Beauty salon activity
Tai Chi

From Terrett AGJ: *Vertebrobasilar stroke following manipulation*, West Des Moines, Iowa, 1996, National Chiropractic Mutual Insurance Company.

BOX 4-9

Potential Warning Signs or Risk Factors for Cervical Artery Dissection

1. Sudden severe pain in the side of the head or neck, which is different from any pain the patient has had before
2. Dizziness, unsteadiness, giddiness, and vertigo
3. Age <45
4. Migraine
5. Connective tissue disease
 - Autosomal dominant polycystic kidney disease
 - Ehlers-Danlos type IV
 - Marfan syndrome
 - Fibromuscular dystrophy
6. Recent infection, particularly upper respiratory

From Triano J, Kawchuk G: *Current concepts in spinal manipulation and cervical arterial incidents*, Clive, Iowa, 2006, NCMIC Chiropractic Solutions.

Signs and symptoms indicative of vertebral artery insufficiency (the five “Ds” and three “Ns”) and carotid artery insufficiency are listed in Box 4-10.

The most important risk factors for developing a CVA appear to be signs of vertebrobasilar ischemia (VBI) (e.g., dizziness, drop attacks, dysarthria, and nystagmus) and a sudden onset of severe pain in the side of the head or neck, which is different from any pain the patient has had before.⁹⁸ These may be signs of a VAD in process and warrant further evaluation and possible need for immediate referral.

Unfortunately, dizziness, vertigo, and disequilibrium are symptoms that are not unique to patients suffering from VBI.

BOX 4-10 Signs and Symptoms of Vertebrobasilar Ischemia

New and sudden onset of head, neck, or face pain unfamiliar to the patient from prior experience

Five “Ds” and three “Ns”:

- Dizziness, vertigo, giddiness, light-headedness
- Drop attacks, loss of consciousness
- Diplopia, other visual disturbances
- Dysarthria
- Dysphagia
- Ataxia of gait, walking difficulties, incoordination of extremities
- Nausea, vomiting
- Numbness on one side of the face or body
- Nystagmus

Signs and symptoms of carotid artery ischemia:

- Confusion
- Dysphasia
- Headache, anterior neck or fascial pain
- Hemianesthesia
- Hemiparesis or Monoparesis
- Visual field disturbances

From Triano J, Kawchuk G: *Current concepts in spinal manipulation and cervical arterial incidents*, Clive, Iowa, 2006, NCMIC Chiropractic Solutions.

Disequilibrium secondary to cervical dysfunction is a common presentation, especially in patients who have had cervical trauma.^{137,138} The dilemma faced by the doctor is how to differentiate vascular from nonvascular disequilibrium. A patient who presents with VBI-like symptoms or has these symptoms triggered with positional testing may be suffering from cervical dysfunction, which could respond positively to manual therapy.¹³⁹ Unfortunately, reliable clinical tools are not presently available to differentiate vascular from nonvascular disequilibrium. Therefore if the clinician has serious suspicion of VBI, he or she should refer the patient for a cerebrovascular evaluation before administering manual therapy. In the majority of cases in which cervical dysfunction and disequilibrium are suspected, the doctor can proceed with a cautious trial of therapy. Indicated manual therapy includes soft tissue manipulation, mobilization, and gentle adjustments. Any gently preformed adjustments should not be applied in any prethrust positions that aggravate the patient's symptoms. If one or two treatments of initial therapeutic trial substantially decrease the patient's pain, it is safe to assume that the pain is of musculoskeletal origin and proceed with additional procedures.⁹⁸

If a patient develops any postmanipulation symptoms that could indicate VBI, it is prudent to assume a vascular causal condition. Although VBI is an unlikely cause of the symptoms, therapy should be changed accordingly because of the disastrous consequences that could develop if manipulative treatment is continued and appropriate referral not made.¹¹⁵ If mild postmanipulation symptoms (e.g., dizziness and disequilibrium) dissipate, it is possible that the symptoms are cervicogenic in nature. At subsequent visits, it may be suitable to proceed with the manual therapies outlined previously.

Evaluation procedures intended to identify patients at risk of manipulation-associated vascular compromise have been proposed. Specific “functional” procedures have also been advocated and applied in clinical practice.^{136,140-143} There are a number of different procedures designed to functionally test the vertebral arteries (de Kleyn, George, Hautant, Houles, Wallenberg tests, etc.), but they all attempt to provoke signs of VBI by inducing extension and extreme rotation of the neck. Unfortunately, all of the applied functional testing procedures alone or in combination do not increase the chance of identifying the patient at risk of having a manipulation-linked VBA stroke. The applications of functional vascular tests do not have any diagnostic value and are no longer considered to be standard of care screening diagnostic procedures.⁹⁸ Terrett¹¹³ made the following concluding remarks concerning functional vertebral artery vascular tests: “It makes no sense to subject the patient to a screening procedure that is invalid and only gives the practitioner a false sense of security regarding the degree of risk for SMT.” This can only lead to the conclusion that the tests should be abandoned for clinical and medicolegal purposes, and should not be used for nonclinical risk management reasons.

Bruits and carotid arterial bruits specifically have been proposed as contraindications and are possible indications of vascular pathologic conditions, but are not by themselves contraindications to SMT.¹¹³ Furthermore, the reliability of auscultation has been questioned. Ziegler and colleagues¹⁴⁴ concluded that the presence of bruits over the carotid artery is a very unreliable indicator of CAD. CAD is even less frequently affiliated with SMT⁹⁹ than VAD, and the vertebral artery cannot be auscultated. If a bruit is heard and is associated with other symptoms (such as headache, neck pain, or whooshing sounds) or other pathologic conditions (such as hypertension), further evaluation and referral are indicated before applying cervical manipulation.

Conclusions. Postmanipulation VAD and VBI are extremely rare events. The majority of chiropractors go through their entire careers without ever encountering this event.

An association between VAD dissection and chiropractic cervical manipulation has not been established and the relationship may be temporal and not causal.⁸⁶ Definitive risk factors for developing a VAD have not been established¹¹² and there are no clearly identified neck positions or manipulative procedures associated with an increased risk of inducing this injury.⁹⁷

At the present time (2010), no specific adjustment can definitively be said to have more risk than another. The current limited understanding of the mechanism VAD and its relationship to rotational manipulation does not support recommendations to avoid all rotational manipulation of the upper cervical spine. This position is not supported by the clinical literature⁹⁷ and is an over-reaction to a procedure that when performed by a skilled practitioner, is quite safe and therapeutically beneficial^{37,38,145-156}

Despite the adherence to sound practice standards, the rare postmanipulation ischemic stroke will likely continue to occur because of our inability to identify those patients at risk of developing a spontaneous or postmanipulative-associated VBA stroke. It is therefore absolutely imperative that the clinician be able to recognize the signs of a VBI and take the appropriate steps to minimize the pathologic effects.

Although it is uncommon to experience VBI-like symptoms after manipulation,²⁴ if they do occur, specific steps must be followed. The most important first step is to not administer another cervical adjustment.¹⁵⁷ If the patient is experiencing a VBA stroke, further manipulation will only delay appropriate referral and management.

Symptoms indicative of a TIA or stroke represent a potential medical emergency. Health care practitioners are responsible for recognizing the symptoms of these events and taking appropriate action. If a patient demonstrates or develops any pre- or postmanipulative symptoms indicative of VBI, it is prudent to assume a potential vascular cause. Even if symptoms abate, there is a set of signs or symptoms that should trigger consideration for an immediate referral. Every clinical situation is potentially different, but the treating clinician must have good clinical justification to not refer a patient for evaluation if any of the symptoms or findings outlined in Box 4-11 are encountered. If an immediate referral is indicated, the practitioner should call 911 and have the patient transported to the nearest emergency room. It is important to communicate the patient's status, and the practitioner's concern that the patient may be suffering from a stroke. It is important to communicate any precipitating events, including whether the symptoms developed after the delivery of cervical manual therapy. If indicated, advanced imaging can confirm the presence of an ischemic stroke, and immediate administration of anticoagulant therapy is in order. This therapy must be delivered within 3 hours to effectively dissolve an offending clot. A quick referral and effective communication will expedite necessary care and have dramatic effects on decreasing morbidity and improving outcomes.

If more moderate, nonurgent but suspicious (e.g., dizziness, disequilibrium) postmanipulative symptoms develop, the patient should be closely monitored. Allow the patient to rest quietly and if the condition does not improve within a reasonable time, the patient should be transported to the nearest emergency room for evaluation. Appropriate evaluative and procedural steps are outlined in Box 4-12.

Postmanipulation VBI-like symptoms are not necessarily indicative of vertebral artery injury or a developing VAD. Maigne¹⁰ postulated that VBI-like symptoms can be the product of stimulation of the vertebral nerve and the accompanying sympathetic plexus. This stimulation in turn is hypothesized to induce spasm of the vertebrobasilar arteries and a transient cascade of symptoms, including vertigo, temporary loss of balance, nausea, and head-

BOX 4-11**Postmanipulation Symptoms
Indicating Need for Immediate Referral**

Impaired or loss of consciousness
Slurred speech
Drop attacks (sudden loss of strength in lower extremities)
Visual field disturbances
Difficulties with speech or swallowing
Paresis or paralysis of any limb
Paresthesia in one or both sides of the face
Ataxia or clumsiness of upper or lower extremities

BOX 4-12**Steps to Follow with Possible
Postmanipulative Stroke Patient**

1. Do not administer another cervical adjustment.
2. Do not allow patients to ambulate; keep them comfortable.
3. Note all physical and vital signs (pallor, sweating, vomiting, heart and respiratory rate, blood pressure, and body temperature).
4. Check the pupils for size, shape, and equality.
5. Check the eyes for light and accommodation reflexes.
6. Test the lower cranial nerves (facial numbness or paresis, swallowing, gag reflex, slurred speech, and palatal elevation).
7. Test cerebellar function (dysmetria of extremities, nystagmus, and tremor).
8. Test the strength and tone of the somatic musculature.
9. Test for somatic sensation to pinprick.
10. Test for muscle stretch and pathologic reflexes.
11. If condition does not abate and referral is deemed necessary, communicate with the provider as to findings, probable diagnosis, recommendation for an MRA, and consideration of anticoagulant therapy.

From Ferezy JS: *Neural ischemia and cervical spinal manipulation: The chiropractic neurological examination*, Rockville, Md, 1992, Aspen.
MRA, Magnetic resonance angiography.

aches. Terrett and Kleynhans cite Maigne, who has labeled this pattern of postmanipulation symptoms as *sympathetic storms*.¹¹⁵ Although this is an engaging hypothesis, further investigation has demonstrated limited neural control of vertebral blood flow,¹⁵⁸ casting doubt on this theory. Another more plausible postulated mechanism for postmanipulation, nonvascular VBI-like symptoms is manipulation-induced transient altered sensory and proprioceptive input from cervical joints.¹¹⁴

THORACIC SPINE

Adjustive complications in the thoracic spine are rare. Reviews of the literature reveal very limited information on types and rates of postmanipulation injuries in the thoracic spine. Studies designed to measure the incidence of adverse reactions typically do not report incidence rates by spinal region.

The apparent low rate of serious injury in the thoracic spine is probably a consequence of the region's comparative stability and the limited potential of manipulative treatments to damage associated neurologic or vascular structures. Although the rate of serious injury is lower, it appears that the rate of mild (acceptable) reactions to manipulation is similar or higher than other regions of the spine. The only study to report comparative rates of adverse reactions found the largest number of reported mild (acceptable) reactions to manipulation to be in the thoracic spine.¹⁵⁹

As mentioned earlier, adverse reactions that exceed a mild to moderate increase in discomfort are rare. They include sprains to the costovertebral and costotransverse articulations, strains of the

intercostal muscles, rib fractures, and rare reports of transverse process fracture and hematomyelia.^{77,160}

Excessive thoracolumbar torque in the side-posture position, as well as inappropriately applied posterior-to-anterior (P-A) techniques, may cause thoracic cage injuries, particularly in older adults. These problems are usually a result of excessive force in relation to the patient's size and physical condition. They can be avoided by appropriate technique selection, application, and evaluation.

LUMBAR SPINE

The incidence of serious complication from lumbar manipulation is extremely low. A review of the “obtainable literature indicates that, on average, less than one case occurs per year.”⁸² Reported complications have been classified by Terrett and Kleynhans⁸² and are listed in Box 4-13. Loads measured during the application of lumbar and pelvic SP manipulation were comparable to those encountered by airline baggage handlers and deemed to be below an injury threshold.¹⁶¹

The most frequently described serious complications from SMT in the lumbar spine is compression of the cauda equina by a midline disc herniation at the level of the third, fourth, or fifth intervertebral disc (IVD).^{77,85,162,163} The resultant cauda equina syndrome (CES) is characterized by paralysis, weakness, pain, reflex change, and bowel and bladder disturbances. Any patient who has bilateral radiculopathies with distal paralysis of the lower limbs, sensory loss in the sacral distribution, and sphincter paralysis may have CES and should be considered a nonmanipulable case and a surgical emergency.¹⁶²

Estimating the rate of serious lumbar manipulation complications is difficult because of the lack of prospective documentation of complications and the uncertainty as to the number of manipulations delivered. In a review of 80 years of literature, Haldeman and Rubinstein¹⁶² reported on 13 cases of CES that were apparently the result of manipulative therapy. Their literature review identified 29 cases, but 16 of the cases were patients manipulated under anesthesia. Manipulation under anesthesia is an uncommonly performed procedure, and including those cases does not accurately reflect the risk of lumbar manipulation. In many of the reported cases, both the chiropractic doctor and the emergency room physician failed to comprehend the nature of the problem and take appropriate action. This lack of prompt, appropriate

treatment likely increased the incidence of serious complication and residual impairment.

Shekelle and co-workers⁷⁸ estimated the rate of post-lumbar manipulation CES to be approximately 1 per 100 million manipulations. The rate was calculated by dividing the number of estimated lumbar manipulations delivered in the United States from 1967 to 1992 by the reported number (4) of postmanipulation cases of CES in the United States. A 2004 review on the safety of lumbar manipulation estimated that the risk of lumbar disc herniation (LDH) and CES at 1 event per 3.72 million manipulations.⁸⁵

SIDE-POSTURE MANIPULATION AND INTERVERTEBRAL DISC

Despite the extremely low rate of complication, controversy continues to surround the question as to whether SP rotary adjustments can injure the lumbar IVDs. The debate is primarily a theoretic one, based on two opposing anatomic and biomechanical models. One position postulates that SP lumbar manipulation produces a torsional shear force that is damaging to the discs. The other postulates that the lumbar facets limit lumbar rotation and protect the discs by preventing undue torsional stress. The following discussion looks at the underlying information central to positions presented by these opposing models.

Those advocating a position that lumbar SP rotary manipulation can potentially injure the disc often cite the biomechanical work and theories of Farfan. Farfan and associates were the first to advance the theory that repetitive rotational torsion and stress could be damaging to the lumbar IVDs.¹⁶⁴ He estimated that approximately 90% of the torsional strength of a lumbar motion segment is provided by the disc and facet joints, with the annulus providing the majority of the torsional resistance. His model postulates that repetitive end-range torsional loading can lead to tears in the annulus and disc degeneration. The injury process is hypothesized to begin with circumferential separation of the outer annular fibers, followed by the development of radial fissures, internal disruption of the disc, and possible production of disc protrusions and herniations.

A number of more recent studies bring into question the pure rotational model of disc failure and its relationship to SP lumbar manipulation. These experiments support the position that the posterior elements of the spine, including the facet joints and ligaments, rather than the disc, are the key structures resisting torsion in the lumbar spine.¹⁶⁵⁻¹⁶⁷ The physiologic range of rotational motion of the whole lumbar spine is approximately 10 to 15 degrees or approximately 2.5 degrees for each joint.¹³⁹ The lumbar joint space is small, and the articular cartilage must compress significantly (up to 60%) to allow up to 3 degrees of segmental movement. The primarily sagittally oriented lumbar facets provide an interlocking mechanism that minimizes rotational mobility and stress to the IVD. Movement must exceed 3 degrees of axial rotation (4% strain) before the annular fibers begin to demonstrate microscopic failure. Full macroscopic failure does not occur until 12 degrees.¹⁶⁸ Therefore, impaction of the zygapophyseal joints provides protection for the IVD by limiting tension to the annulus fibrosus to less than 4% strain.

Using a cadaveric model, Adams and Hutton¹⁶⁵ demonstrated that the torsion of the lumbar spine is resisted primarily by the

BOX 4-13 Reported Complications of Lumbar Manipulation

Disc-related complications
Diagnostic error
Vascular complications from thrombosis
Fracture in presence of osteoporosis
Manipulation in patient on anticoagulant therapy
Rib fracture
Inguinal and abdominal hernia
Unknown

facets and that the compressed facet is the first structure to yield at the limit of torsion. Significant injury to the articular cartilage and soft tissues was demonstrated before significant mechanical stress was transferred to the IVD.^{165,169} The capsular ligaments of the tension facet (facet being distracted during rotation) and the supraspinous and interspinous ligaments were found to be uninvolved or unimportant. This suggests that pure rotational damage to the IVD could occur only after significant disruption of the posterior joints. The same studies established that the disc was more vulnerable to flexion injuries.¹⁷⁰ Flexion is not inhibited by the articular facets, and distortion and disruption of the posterior annulus may occur with excessive flexion, especially when coupled with positions of lateral bending, loading, and rotation.

Bogduk,¹⁶⁸ aware of the protective effects of the posterior joints, has postulated a biomechanical model of injury to the disc that does not necessitate a preceding disruption of zygapophyseal joints. His model incorporates excessive rotation coupled with flexion. Flexion is presumed to tense the annular fibers, leaving less available stretch before their rotational limits are exceeded. Moreover, with the spine in a flexed position, the inferior and superior articular processes are less engaged, allowing for more segmental rotation. With excessive rotation, the normal axis of rotation is envisioned to shift from its central location in the posterior one third of the disc to the impacted facet (compression facet). The compression facet becomes the new axis of rotation, allowing for excessive pivoting of the superior vertebra, resulting in shear and torsion on contralateral facet and annular circumferential discal tears (Figure 4-7).

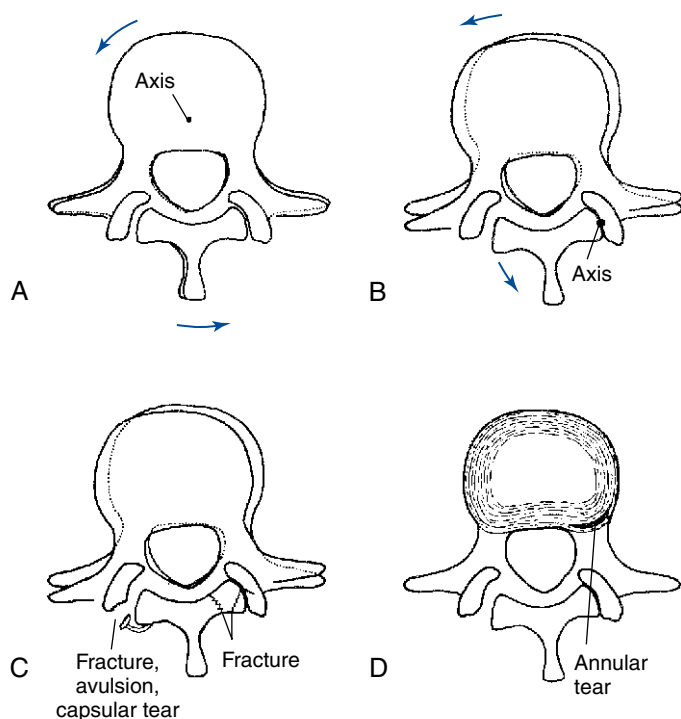


Figure 4-7 Forced rotation beyond the normal range may shift the axis of rotation from its central location (A) to the impacted facet (B). Continued rotation may lead to fractures of the impacted facets or capsular tears or avulsion fractures of the contralateral joint (C) and circumferential tears of the annular fibers (D).

Broberg¹⁷¹ studied the response to compression, shear, bending, and axial rotation of an IVD using a theoretic disc model. He reported that the stiffness of the IVD increases considerably with axial load. This finding implies that most experimental data obtained at zero axial load may reflect poorly on real situations involving weight bearing and axial loading. Within normal physiologic limits, bending, shear, or axial rotation does not seem to constitute a risk of fiber rupture, except in combination with very high axial loads. Moreover, with pure compression, the likelihood of fiber rupture is not very great because end-plate failure occurs earlier, before the rupture is manifest.¹⁷²

It must be remembered that all of the previously described theories and speculations are based on studies conducted primarily on cadavers. Most of the studies examining the effects of torsion have been focused on the lumbar spine because of the high prevalence of LBP in society, and many of the studies were performed on cadaver spine sections with the posterior elements removed. The effects of torsional forces on the cervical and thoracic segments have not been adequately examined or studied. In the cervical region, the facets do not interlock as in the lumbar spine, and greater axial rotation and torsion are available. The cervical spine is the most mobile region of the spine, yet the incidence of disc herniation here is much less frequently reported than in the lumbar spine. Conclusions reached with these studies, especially any inference in their application to living human beings, must therefore be viewed with caution. The IVD may respond very differently to loads under normal physiologic loads or in circumstances in which there is associated disc degeneration or motion segment instability.

The clinical literature evaluating the potential risk of disc injury from lumbar manipulation reveals a very low incidence of reported manipulation-induced disc herniation. A review of the literature through 1993 by Assendelft, Bouter, and Kripschild revealed only 56 case reports of lumbar manipulation complications attributed to disc herniation.⁸¹ Nearly half (49%) of the cases occurred during manipulation under anesthesia, and the majority of the cases (82%) progressed to CES. In addition, a number of the reported cases cannot be clearly cited as evidence of manipulation-induced disc herniation.¹⁶³ In a number of the cases, the symptoms either developed over time or at a point after treatment at which the patient was involved in other activities that may have triggered a worsening in his or her condition.¹⁶²

Despite the very low level of documented postmanipulation disc herniations in the literature, disc problems account for the greatest percentage of malpractice claims filed against chiropractors. NCMIC insures the overwhelming majority of chiropractors, and the company paid claims on 1403 malpractice cases from 1991 through 1995.¹⁷³ This results in an annual average of 280 paid claims per year for this company. During this time, the percentage of filed claims for disc problems decreased slightly from 29% in 1991 to 26.8% in 1995. In 1995, the incidence was slightly higher in the lumbar spine (13.8%) than the cervical spine (12.2%).¹⁷⁴ If the percentage of disc complications for filed claims and claims that were settled are the same, the number of claims paid by NCMIC for disc-related problems in 1995 is approximately 75. Of this number, approximately 37 resulted from lumbar manipulation.

Although the total number of yearly claims paid for post-lumbar manipulation disc-related problems is a very low percentage of patients receiving treatment, it is potentially an artificially elevated number. It is likely that the natural history of disc herniation has led to a mistaken connection of causation between manipulation and disc herniation. Patients with disc herniation often present initially with back pain that over time may progress to include leg pain. This often develops as associated NR inflammation and compression persist. If the initial evaluation of a patient is equivocal and the patient is not informed that he or she may be suffering from a disc herniation, subsequent progression of symptoms and the subsequent diagnosis of a disc herniation may lead the patient to erroneously assume that the manipulative treatment he or she received caused the disc herniation.

Although the debate on the risk of disc injury with lumbar manipulation has not been definitely resolved, the following tentative conclusions can be suggested:

1. The lumbar IVDs are protected from rotational stress and injury by the lumbar posterior joints.
2. Marked force would have to be applied to injure the disc with a rotational force.
3. Movement beyond normal range must be applied to injure the disc and likely would occur only after significant injury had been subjected to the posterior joints.
4. The disc is most vulnerable to flexion injuries. Loaded positions combining flexion and rotation are probably the most risky.
5. The forces involved in skillfully delivered SP rotational manipulation are not sufficient to injure a healthy disc.
6. In patients with disc herniation, manipulative positions that incorporate excessive flexion and rotation should be avoided.
7. Before applying adjustments in patients with disc herniations, an evaluation of lumbar movements should be conducted.
8. Adjustments should not be delivered in positions and directions that implicate increased NR compromise (i.e., directions that increase the intensity or distal distribution of the patient's leg pain).^{174,175}

EFFECTS OF ADJUSTIVE THERAPY

MUSCULOSKELETAL

Treatment of NMS dysfunction and disease has historically been the major reason for which chiropractors are consulted,^{3-8,176} and NMS disorders are the conditions most commonly covered for chiropractic care by insurance companies and government health care programs.^{3,8} Chiropractic patients have repeatedly expressed satisfaction with the quality and effectiveness of chiropractic care. In comparative studies for the treatment of back pain, patients consistently rate chiropractic care as superior to medical care.¹⁷⁷⁻¹⁸⁶

Furthermore, authors who have reviewed the literature on spinal manipulation have concluded that sufficient evidence exists to support the use of spinal manipulation in the treatment of a number of painful NMS conditions. This is most notable in the case of mechanical back and neck pain and headache, in which a large body of controlled clinical trials and systematic reviews has consistently

shown “spinal manipulation to be superior to sham/placebo or as effective or more effective than an array of other comparison treatments.”^{37,38,74,180,187-198} There is presently more evidence supporting manipulation as a therapy for LBP than for any other alternative.³⁷

Guidelines on the management of LBP have also concluded that SMT is a safe and appropriate treatment choice. The first major U.S. government-directed guideline on the management of LBP was published in 1994 by the Agency for Health Care Policy and Research (now the Agency for Healthcare Research and Quality AHRQ).¹⁹² This document represented a synthesis of the best evidence regarding the assessment and management of acute LBP in the adult population of the United States. It consulted a panel of experts drawn from the professions involved in treating LBP. There were a number of principal conclusions. Most notably for the chiropractic profession were the recommendations that relief of discomfort can be accomplished most safely with nonprescription medication or spinal manipulation. Bed rest in excess of 4 days was deemed to be nonhelpful in most circumstances, and patients were to be encouraged to stay active and return to work as soon as possible. Numerous subsequent professional, national, and international guidelines on the treatment of LBP have reached similar conclusions.^{46,199-202} Most recent is the 2007 joint clinical practice guideline from the American College of Physicians and American Pain Society, which recommends that “patients who do not improve with self-care options consider the addition of nonpharmacologic therapy with proven benefits.”⁴⁶ This recommendation was made on moderate-level evidence and recommends the use of spinal manipulation for acute LBP and the following nonpharmacologic options for chronic or subacute LBP: intensive interdisciplinary rehabilitation, exercise therapy, acupuncture, massage therapy, spinal manipulation, yoga, cognitive-behavioral therapy, or progressive relaxation.⁴⁶

The chiropractic profession has also consistently demonstrated cost-effective treatment for back pain. Since 1980, the majority of studies investigating the comparative cost-effectiveness of chiropractic care have shown chiropractic treatment for LBP to be more cost-effective than medical care.^{180,184,188,191,203-207} An extensive review conducted in 1993 for the provincial government of Ontario, Canada, concluded that chiropractic care was more cost-effective and would generate considerable cost savings if chiropractic services for treatment of LBP were increased.¹⁸⁰ Incorporation of chiropractic services within a managed care organization decreased the use of radiographs, low-back surgery, hospitalizations, and average back pain episode costs.²⁰⁸ A large multicenter, community-based trial conducted in the United Kingdom found that the addition of manipulation to “best [medical] care” improved back function in both the short- and long-term. The authors concluded that spinal manipulation is a cost-effective addition to “best care” for LBP in general practice.^{209,210}

There are several exceptions in which the cost of chiropractic care per episode of acute LBP was higher than care provided by medical primary care providers.^{181,211} Chiropractic per-visit costs were significantly lower, but total costs were higher because of the higher number of visits per episode. The number of visits per episode varied significantly among providers, indicating that total costs were significantly elevated by a small percentage of providers who delivered service well above the mean. Furthermore, medical costs may have been artificially decreased in one study because of the exclusion of associated hospital costs.

NON-MUSCULOSKELETAL

In addition to their successes treating musculoskeletal disorders and dysfunction, most chiropractors have also noted positive health effects from adjustive and manual therapy in areas outside the musculoskeletal system. From the time of chiropractic's origins, chiropractors have viewed their healing art as having wide-ranging health benefits.²¹² Philosophically, this is symbolized by the chiropractic holistic health care viewpoint, which stresses the important relationship between the structure and function of the NMS system and its effects on homeostatic regulation and health maintenance.²¹³

Unfortunately, clinical research in the area of manual therapy and somatovisceral disease is minimal. The functional visceral conditions that may respond to chiropractic care, the circumstances under which they may respond, and the degree to which they may respond have yet to be systematically studied and clearly identified. It is still unknown and under debate if the removal of mechanical malfunction of the spine may be helpful in treating functional disorders. At present, there have been no appropriately controlled studies that establish that spinal manipulation or any other somatic therapy represents a valid curative strategy for the treatment of any internal organ disease.^{214–235}

Consequently, the profession should be cautious in implying or guaranteeing a positive outcome for the manipulative treatment of visceral disease. Further research involving large patient populations will be required before the somatovisceral effects of adjustive therapy can be substantiated.²³⁶ At the same time, the profession should not discount the potential positive health effects noted in clinical practice. Patients without contraindications to manual therapy who have a possible somatovisceral disorder should not be refused treatment, but they should not be solicited with the implied guarantee of a positive result.

Although the clinical effectiveness of chiropractic SMT for mechanical spine pain has been demonstrated, very little is known about how manipulation is producing a therapeutic effect. Several hypotheses exist as to the mechanism by which chiropractic therapy affects the underlying NMS causes of joint dysfunction and somatovisceral disorders. They include concepts that may be broadly divided into mechanical and physiologic. The following discussion touches on some of the proposed mechanisms, but is by no means comprehensive.

MECHANICAL HYPOTHESES

In the mechanical arena, manual therapy is directed toward reversing or mitigating the soft tissue pathologic condition and mechanical dysfunction associated with disorders or injuries of the NMS system. The soft tissue derangements responsible for mechanical dysfunction may be initiated by trauma, repetitive motion injuries, postural decompensation, developmental anomaly, immobilization, reflex changes, psychosocial factors, or aging and degenerative disease. These injuries and disorders often result in soft tissue fibrosis, adaptational shortening, loss of flexibility, joint instability, and altered joint mechanics.^{30,237–241} The scope of manual therapies available to treat mechanical joint dysfunction is extensive. The selection and application of each should be based

on an understanding of the pathophysiology of the disorder being treated and knowledge of the procedure's potential therapeutic effects and treatment outcomes. The major objective of adjustive therapy is improved health and function through the alleviation of musculoskeletal pain, and aberrant function.

In the early stages of soft tissue injury and repair, manual therapy is directed toward decreasing pain and inflammation, preventing further injury, and promoting flexible healing. Early appropriate manual therapy and mobilization may minimize the formation of extensive fibrosis and the resulting loss of extensibility.^{30,237,239–246} Excessive immobilization can retard and impair the healing process and can promote further atrophy and degeneration in articular soft tissue and cartilage.^{240–254} By promoting an early return to activity, the detrimental effects of immobilization may be minimized. Early activation promotes strong, flexible repair and remodeling and breaks the pattern of deconditioning and illness behavior, which can be detrimental to recovery.^{66,255,256} Gentle distractive adjustments, passive joint mobilization, friction massage, and effleurage are commonly applied manual therapies in this stage.

If the initial injury to the connective tissue is minor, repair may proceed quickly without significant structural change or resulting impairment. If the tissue damage is marked, however, the ensuing fibrous repair may result in "a scar, visible or hidden, which has matured to fill the injured area, but lacks the resilience, strength, and durability of the original tissue. Such an asymmetric scar, produced either by injury, degeneration, or surgical trauma, may produce disturbances of biomechanical performance."²⁵⁵ Therefore, when injury or degenerative disease results in contracture, stiffness, joint hypomobility, and chronic pain or impairment, manual therapies shift toward a more vigorous approach and are directed toward the restoration of mobility and function. They include adjustments, mobilization, therapeutic muscle stretching, connective tissue massage, trigger-point therapy, myofascial release techniques, and the like.²¹⁶ In this stage, manual therapies are most effective when coupled with activities and exercises that promote soft tissue remodeling and muscle strength. However, applying spinal exercises without first incorporating an assessment and treatment of joint dysfunction may be less effective. If joint hypomobility persists, active exercise may stimulate movement at the compensatory hypermobile joint instead of the hypomobile joints. This may lead to the further breakdown and attenuation of the joint stabilizing structures, which further complicate joint stability.

Forces Generated During Adjustive Therapy

As mentioned previously, the clinical value of SMT for mechanical spine pain has been demonstrated. However, the specific mechanism by which adjustments effect a reduction in symptoms has not been determined.²⁵⁷ Adjustive therapy is assumed to have its effect through the application of an external force. It is taken for granted that this force will deform the spine, move its articulations, and stretch and stimulate associated soft tissues. The last decade has seen significant evaluation and measurement of the forces produced in the application of HVLA adjustments and research is expanding on how those forces may be transferred to the body. However, information regarding the effects of manipulative forces on biologic tissue is limited.^{161,257}

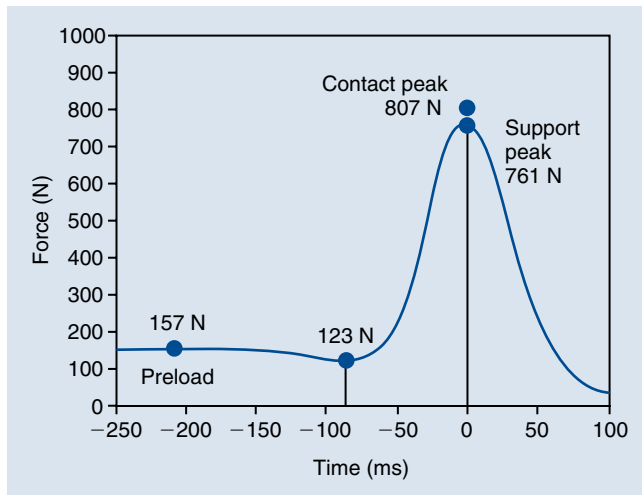


Figure 4-8 Comparison of force versus time for a typical adjustive thrust.

External forces associated with adjustments have been calculated by recording loads transmitted through flexible transducers placed on the surface of patients, through the forces transmitted to a load cell placed in the table below the patient²⁵⁸ or through computer modeling.¹⁶¹ The typical manual HVLA adjustment is characterized by a prethrust (preload) period and a thrust period. The force magnitudes and durations of these periods have been calculated and are illustrated in Figure 4-8.^{257,258}

Herzog²⁵⁷ and Herzog, Kawchuk, and Conway²⁵⁹ measured forces during the application of supine cervical, prone thoracic, and side-lying sacroiliac adjustments. The peak thrust forces averaged 400 N for the thoracic spine and ranged between 220 and 550 N of peak force during the application of sacroiliac adjustments. The peak forces, when converted from newtons to pounds, range from 50 to 125 lbs of force. These forces corresponded to approximately one third to two thirds of the treating doctor's body weight. Thrust duration times measured in the thoracic spine ranged from 100 to 150 ms and never exceeded 200 ms.²³⁶ Force measurements in the cervical spine were markedly less than other regions with preload and peak forces and averaged 100 N of peak force. Thrust duration times were also significantly less than in the thoracic and sacroiliac regions, ranging from 80 to 100 ms. Patient loads measured during the application of lumbar and pelvic side posture manipulation were comparable to those encountered by airline baggage handlers. The loads were deemed to be below an injury threshold. The transmitted loads were complex and varied based on patient position (PP) and method selected.¹⁶¹

Kirstukas and Backman²⁵⁸ revisited the characteristics of prone thoracic adjustments. They measured prone unilateral thrusts in the thoracic spine using contact pressure measurements and table force measuring equipment. Two separate chiropractors applied six unilateral adjustive thrusts, divided equally over two sessions, to the apex of the subjects' thoracic spines. The results demonstrated significantly greater thrust forces than Herzog, Kawchuk, and Conway,²⁵⁹ with one chiropractor averaging 630 N and the other 960 N of peak thrust force. Thrust duration times averaged 96 ms and were consistent between doctors.

Kirstukas and Backman²⁵⁸ and Herzog, Kats, and Symons²⁶⁰ have reported on the distribution of thoracic prone manipulative forces and the differences between applied forces, area of maximal contact pressure, and peak "effective" applied force. In the thoracic spine Kirstukas and Backman estimated mean peak contact pressures at 680 kPa (100 psi) for one doctor and 1486 kPa (215 psi) for the other.²⁵⁸ Peak contact pressure was focused under the doctor's proximal hypothenar to an area only a small fraction of the total area covered by the doctor's contact hand. They labeled this region the *intense contact area* and have defined it as the area over which two thirds of peak contact pressure readings are recorded. Herzog, Kats, and Symons²⁶⁰ also determined that prone thoracic adjustments had an "effective" peak force and contact area that was much more focused than the full area of anatomic contact. Based on these experiments, it appears that short-lever, prone thoracic adjustments will have a significantly more focused area of effective applied force than the overall applied force.²⁶¹

Although adjustive pretension and peak forces may vary between doctors, certain consistent characteristics of HVLA adjustments stand out. They all produce a high-velocity force with a consistent preload phase (preadjustive tension) and a rapid acceleration phase. There is a consistent small drop in preload force before the impulse is delivered.²⁶² The adjustive thrust has a very short duration and short-lever adjustments have a focused area of contact pressure and force. It also appears that trained chiropractors have the ability to modify prethrust tension, peak velocity, and duration of adjustive thrust. These features are modified according to the area that is being treated and the amount of prethrust tissue resistance that is encountered.

Movements Generated During Adjustive Therapy

Our knowledge concerning the specific movements induced by adjustive thrusts is limited but growing. The expanding body of information on this topic does confirm that spinal movements are produced with adjustive thrusts, but also indicates that the location and directions of movement may not fully match our clinical assumptions.

The first significant study evaluating HVLA manual procedures was limited to evaluating the movements generated by unilateral P-A thrusts in the lower thoracic spine of fresh-frozen cadavers.²⁶³ Segmental translational and angular movements were measured. The movements were recorded by using bone pins embedded in the spinous process of three adjacent vertebra and high-speed cinematography. P-A and lateral translational movements averaged 0.5 mm and ranged up to 1 mm. Axial rotations averaged approximately 0.5 degree and were noted up to nearly 1 degree. Sagittal rotations were greater, averaging approximately 1 degree, and were recorded up to approximately 2 degrees.

Significant movement was localized to the contacted segment and motion segments immediately inferior and superior to the point of contact (Figure 4-9). None of the vertebral motion segments had pre existing fixations, and all had returned to their resting state within 10 minutes after the application of the adjustive thrust.

Although this study cannot be generalized to living subjects, it is the first study to demonstrate that high-velocity thrusts can

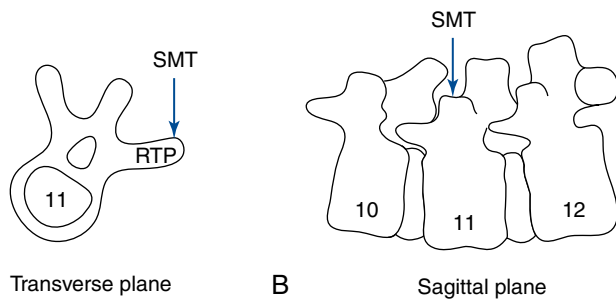


Figure 4-9 Diagram of the movements produced when a posterior-to-anterior adjustment is applied to the right T11 transverse process (RTP). **A**, Transverse view, demonstrates the transverse plane rotation that is induced. **B**, Sagittal view, illustrates the anterior translation and sagittal plane rotation (extension) that occurs at T10–11 and T11–12.

generate measurable spinal joint movement focused at the level of adjustive contact and adjacent joints. This study also supports the clinical assumption that unilateral prone thoracic P-A paraspinal contacts are likely to generate forces that induce localized sectional rotation and extension.

To evaluate lumbar manipulative movements, Ianuzzi and Khalsa measured strains in the lumbar facet joint capsules of cadaveric specimens during physiologic ROM movements and simulated manipulation.²⁶⁴ They found that simulated manipulative forces induced movements primarily in the direction of applied loads, were dispersed over fairly large areas, and induced strains across facet joint capsules that were similar whether they were delivered at the targeted joint or a nearby joint. They concluded that “despite the fact that vertebral rotations varied significantly in magnitude or direction with manipulation site, facet joint capsule strain magnitudes were similar regardless of where the manipulation was applied.”²⁶⁴ Although generalizability to clinical practice and manual therapy is difficult because this study was conducted on cadaveric specimens with manipulations simulated and produce by linear mechanical actuators, it does imply “that segmental specificity may not be as important as previously postulated in the efficacy of SM.”²⁶⁴

In two separate studies, Cramer and co-workers^{265,266} used MRI to study the effect of side posture positioning and manipulation on zygapophyseal (Z) joint movement and gapping in the lumbar spine. Both studies were conducted on healthy student volunteers (N 16, N 64) ages 22 through 30. Patients were scanned in the supine neutral position, in left rotated side-lying posture with left side up and then post-rotation resisted mamillary push adjustment (see Figure 5-230a). The positioning and adjustments were applied to induce left lumbar rotation and gapping of the left lumbar Z joint. Evidence of preadjustive positional gapping and postadjustive gapping was evaluated by three radiologists. Lumbar side posture spinal positioning demonstrated increased separation (gapping) of the Z joints over the neutral control position (mean 1.18 mm). Lumbar adjustments induced mean separation of 1.89 mm an increase of 0.71 mm over the nonadjusted side posture positioning controls. The increased postadjustive separation was noted only in the group that was scanned in side posture position versus the neutral position. It should be noted that the down-side facet joints demonstrated an average -0.74 mm compression during

side posture positioning and -0.89 mm compression postadjustive side posture position. The average postadjustive side posture distractive gapping of the lumbar Z joints over the neutral control was 2.24 mm—a significant amount of movement for a lumbar Z joint. These studies establish that side posture lumbar-resisted mamillary adjustments induce increased rotational distraction and gapping in the up-side superior Z joints (side of adjustive contact) during side posture-resisted mamillary adjustments.

A number of additional studies have been conducted to evaluate whether adjustments induce joint cavitation and whether the cavitation can be localized to a targeted joint. These studies did not attempt to measure the specific movements that might be associated with joint cavitation. The studies employed skin-mounted microphones or accelerometers capable of detecting and localizing sound or vibrations associated with joint cavitation. The studies included assessment of supine cervical thumb pillar rotation adjustment,²⁶⁷ side posture lumbar spinous pull, spinous push and mamillary push adjustments,^{268,269} side posture lower sacroiliac SI push adjustments,²⁶⁸ and prone thoracic crossed bilateral transverse and bilateral thenar transverse adjustments.²⁶⁹ Based on the information provided by these studies, the following generalities can be noted for the specific adjustments performed and joint cavitation:

1. Thrust adjustments commonly produce cavitations.
2. Single-level joint cavitation is uncommon in side posture pelvic, lumbar, and supine cervical adjustments.
3. Supine cervical thumb pillar rotation adjustments overwhelmingly produce cavitation in the side opposite the contact (94%).
4. Side posture pelvic adjustments commonly generate cavitation in the lumbosacral spine.
5. Prone thoracic adjustments and cavitation are more localized to the level of contact than side posture and pelvic adjustments.
6. The targeted joint is more likely to cavitate when multiple cavitations are produced.
7. Generalized cavitation accuracy is achievable with side posture lumbar adjustments.

If level of cavitation is representative of level of focused adjustive force, it seems likely that adjustments are not as focused and specific as clinically assumed. This raises the question of whether adjustments need to be joint-specific to have maximal clinical effect and the need to advance clinical research to address this question. The majority of procedures evaluated to date have been based on the premise that a precise level of spinal dysfunction needs to be ascertained before effective treatment can be rendered. Considerable effort is expended during the evaluation of patients to ascertain whether a specific joint malposition or restriction exists. Adjustments are then selected and applied with the presumption that the correct method and vector must be selected to induce the appropriate movement and therapeutic effect. However, if generalized adjustive clinical effects are equivalent to single-level clinical effects, then adjustive therapy decision-making might change significantly.

Presently, many of the joint assessment tools, especially segmental motion palpation, have poor interexaminer reliability for identification of a specific level of joint restriction. If identification of regional dysfunction were sufficient to establish effective

treatment, then spinal joint motion palpation may have more clinical utility if applied within this context. This also has the potential to dramatically change the clinician's perspective and alleviate many of the clinical frustrations that occur when trying to establish a specific level of dysfunction.

Clinical research addressing the assumption that clinical outcome is better with specific identification of level of dysfunction and application of specific adjustment is limited and addressed by only one study at this point.²⁷⁰ The study evaluated patients with neck pain who were randomized to receive cervical spine manipulation at restricted levels identified by motion palpation versus manipulation at levels randomly generated by a computer. The results show that both groups had similar, and in some cases dramatic, improvements in symptoms directly after receiving one HVLA cervical adjustment. The results indicate that cervical end-play (EP) assessment-directed manipulation did not improve same-day outcomes in pain or stiffness. The outcome lends support to the hypothesis that spinal manipulation may have a more generalized, nonspecific mechanism of action in relieving symptoms. It implies that the mechanical effects associated with manipulation may lack spatial specificity and the adjustive vector may not be as important as generally thought.

Although the evidence from this study indicates that using EP to identify level of dysfunction does not improve the measured outcome, it is still premature to abandon the specificity model. It is the only study to clinically investigate this topic and it has a number of limitations that significantly affect its clinical implications. Firstly, it measured the effects of only one adjustment on immediate and same-day pain and stiffness reduction. It is likely that manipulation has a dose-dependent therapeutic effect,²⁷¹ and this trial did not approximate the typical course of adjustive treatments. Adjustive treatments for a cervical mechanical pain syndrome average in the 6 to 12 range and occur over weeks. EP assessment may also not be a valid indicator for same-day postmanipulative pain and yet valid in directing therapy that has an effect on other clinical outcomes and pain and function over time. The immediate pain and stiffness relief noted by both groups may also be attributable to placebo or nonspecific effects associated with assessment and treatment concealing differences between groups that might develop over time.

Cavitation

As discussed earlier, adjustive thrusts are frequently associated with a cracking sound. Typically, this occurs at the end range of passive joint motion when a quick thrust overcomes the remaining joint fluid tension. However, any procedure that produces joint separation has the potential to cause the cracking sound. The separation of the joint is theorized to produce a cavity within the joint, the induction of joint cavitation, and an associated cracking sound.

Cavitation is the "formation of vapor and gas bubbles within fluid through the local reduction of pressure" and is a well-established physical phenomenon. Evidence strongly suggests that it also occurs during the application of spinal adjustive therapy, although this premise has not been proven conclusively.^{23,272,273}

It has long been known that a liquid confined in a container with rigid walls can be stretched. If stretched sufficiently, cavitation occurs. The pressure inside the liquid drops below the vapor pressure,

bubble formation and collapse occur, and a cracking sound is heard.²⁷⁴ The case for synovial joint cavitation and cracking is supported by experimental evidence conducted on metacarpophalangeal (MP) joints, the cervical spine, and the thoracic spine.^{23,275-282} Experiments conducted on MP joints indicate that there is a linear relationship between an applied load and joint separation up to the point of joint cavitation.^{276,278} At the point of joint cavitation, there is a sudden increase in joint separation without a proportional increase in the applied load (Figure 4-10). When the joint is reloaded after cavitation, there is no second cavitation, and the joint separates to the same degree with a much more linear relationship between the applied load and the degree of joint separation (Figure 4-11). The inability of the joint to undergo a second cavitation persists for approximately 20 minutes, and has been labeled the *refractory period*. The bubbles formed within the MP joint cavitation consist of water vapor and blood gases and have been measured at 80% carbon dioxide. The bubbles persist for approximately 30 minutes before the gas is absorbed back into solution.²⁷⁶⁻²⁸⁰

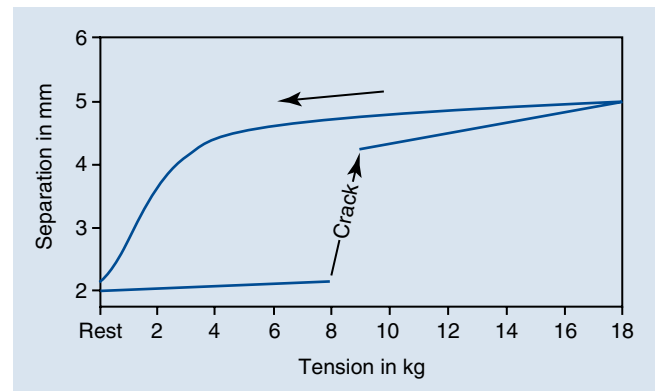


Figure 4-10 Force displacement curve representing the effects of joint separation and cavitation: As the joint tension increases with joint surface separation, a quick and dramatic separation occurs, and a cracking noise is produced.

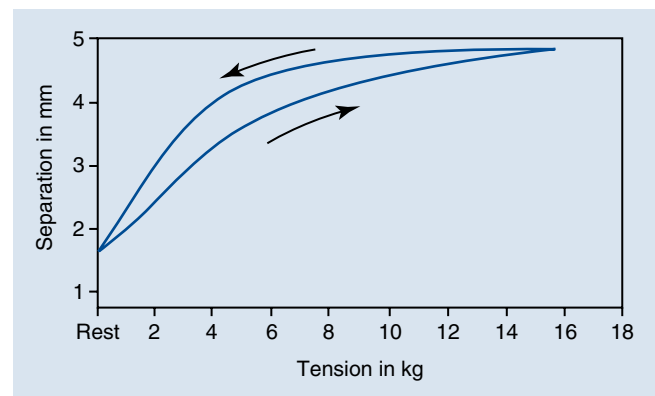


Figure 4-11 Force displacement curve illustrating that immediate reloading of the joint after cavitation is not associated with a second cavitation, and the joint separates to the same degree with a much more linear relationship between the applied load and the degree of joint separation.

In manipulative experiments conducted on the thoracic spine, joint cavitation typically occurred just before the peak adjustive force. In a few cases, the cavitation occurred just after the delivery of the peak force.²⁸¹ In the experiment conducted on the MP joints, a small percentage of the manipulated joints did not produce an audible crack. It is postulated that the joint capsule in these individuals was very tight, not allowing for sufficient joint separation to produce cavitation²² (Figure 4-12). This observation might offer an explanation for the clinical occurrence in which some individuals need several adjustive treatments before joint cavitation is produced. Over time, treatments might produce enough flexibility in the joint capsule to permit joint cavitation.

Several mechanisms have been proposed for how joint cavitation produces the audible crack. Speculation centers on the formation and collapse of gas bubbles or a rapid stretch of the capsular ligament. Unsworth, Dowson, and Wright²⁷⁸ suggested that cracking is not the result of bubble formation but the result of the rapid collapse of bubbles caused by fluid flow. The crack is viewed as a postcavitation phenomenon generated by the collapse of bubbles as the newly formed bubbles rush from the higher-pressure periphery to the relative low-pressure pocket generated in the center of the distracted joint. Meal and Scott²⁷⁹ have more recently shown that the crack produced in the MP joint and in the cervical spine are actually double cracks separated by several hundredths of a second. The significance of two separate recorded cracks is a matter of speculation. The two sounds may be a direct consequence of cavitation, the first crack being the product of gas bubble formation and the second crack associated with the rapid collapse of gas bubbles. Other possibilities include cavitation plus soft tissue vibrations, stretching, or artifacts to account for the second sound.

Brodeur²⁷² has presented a slightly different model of joint cavitation and cracking based on a mechanism described by Chen and Israelachvili.²⁸³ Within this model, the capsular ligament plays a primary role in the production of joint cavitation and cracking. During the first phase of joint manipulation, as the joint is being loaded and the joint surfaces are being distracted, the joint and the capsular ligament are seen as invaginating (drawing inward) to maintain a constant fluid volume within the joint space. As

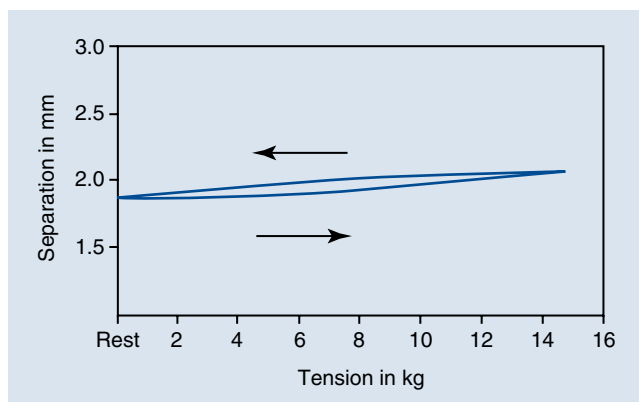


Figure 4-12 Force displacement curve in joints in which no audible release could be generated. In these individuals, it is postulated that the joint capsule is very tight, not allowing for sufficient joint separation to produce cavitation.

distractive pressure is increased, the capsular ligament reaches its elastic limits and snaps away from the synovial fluid, producing cavitation at the capsular-synovial interface. A rapid increase in joint volume follows, and the gas bubbles formed at the periphery rush to form a single coalesced bubble in the center of the joint space (Figure 4-13). Brodeur²⁷² speculates that the “snap-back” of the capsular ligament is the event responsible for the audible crack. He also proposes that this mechanism offers an explanation for why some individuals with very tight or loose joint capsules do not crack. “For loose joints, the volume of the articular capsule is larger and traction of the joint does not cause a sufficient tension across the ligament to initiate the snap-back of the joint capsule. Similarly, an overly tight joint reaches the limits of its anatomic integrity before the joint capsule can begin to invaginate.”²⁷²

Besides the cracking itself, cavitation is considered to be associated with several postadjustive phenomena: a transitory increase in

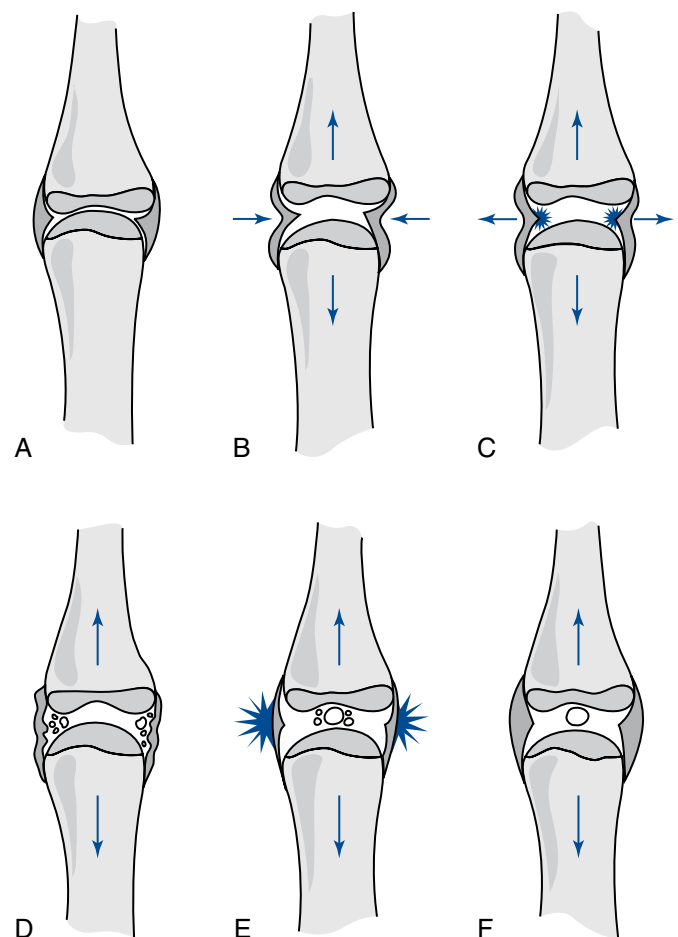


Figure 4-13 Model of the changes in the periarticular structures during a manipulation. **A**, The joint in its resting position. **B**, Long-axis distractive load applied to the joint. **C**, Once the tension exceeds a certain threshold, the energy stored in the capsular ligament initiates an elastic recoil that causes the capsule to snap back from the synovial fluid. Cavitation occurs at the capsular ligament-synovial fluid interface. **D**, The sudden increase in joint volume temporarily decreases tension on the capsular ligaments. **E**, The distractive forces continue to traction the joint, stimulating high-threshold receptors. **F**, The joint volume is increased, gases have coalesced into the central area, and the joint is significantly distracted relative to its resting position.

passive ROM, a temporarily increased joint space, an approximate 20-minute refractory period during which no further joint cracking can be produced, and increased joint separation. Sandoz²⁴ has labeled the postadjustment increase in joint range of movement *paraphysiologic movement* because it represents motion induced only after cavitation (see Figure 3-23).

The postcavitation refractory period, discussed previously, and associated phenomena may be explained by microscopic bubbles of carbon dioxide remaining in solution for approximately 30 minutes. During this period, the bubbles will expand with any subsequent joint separation, maintaining the pressure within the joint. The postcavitation expanded joint space appears as a radiolucency on a radiograph of the distracted joint. The postcavitation increase in joint space appears to be temporary and corresponds to the refractory period. Because the pressure within the joint cannot drop until the gas bubbles are reabsorbed, no further cavitation can occur during this time.²⁷⁸ Furthermore, the force contributed by the stretching of fluid will be absent, causing a decrease in force holding the joint surfaces together and thus resulting in the increased passive ROM noted by Sandoz^{23,277} and Mireau and colleagues.²⁸⁴

As noted earlier, the crack associated with joint cavitation may not be the product of the formation of gas bubbles, but rather a rapid collapse of gas bubbles. In this model, the temporary increased joint space cannot be explained by the persistence of gas bubbles. An alternate explanation postulates that the increased joint space persists from the excess synovial fluid that rushes to the decompressed center of the joint. The joint does not immediately return to its precavitation resting space because synovial fluid is viscoelastic and slow-moving. The flow of excess synovial fluid between the joint surfaces takes time to reestablish equilibrium and allow the joint to return to its precavitation resting position.²⁷⁸

A study conducted by Mireau and colleagues²⁸⁴ brings into question whether the temporary increase in joint space after manipulation is a product of gas bubble formation. They compared the resting joint spaces of subjects who did and did not have an audible crack with manipulation of the MP joints. Only 68% of the 62 subjects manipulated experienced an audible crack, yet the resting joint space increased for both groups, with no statistical difference noted between the groups. If the inaudible-crack group was able to achieve a post-treatment increase in joint space, it suggests that joint cavitation may have occurred, but without the intensity necessary to record an audible release, or that some other unknown phenomenon is at work for both groups.

Mireau and colleagues²⁸⁴ also studied the postmanipulation joint mobility of the subjects who recorded an audible release and those who did not. Both groups had 6 lbs of long-axis distraction applied after treatment. In the audible-crack cohort, an increase in joint space of 0.88 mm was noted, and an increase in joint space of 0.45 mm was recorded for the group without an audible crack. These findings suggest that there is some different physical effect between those who experience an audible release and those who do not. Perhaps a more profound separation of joint surfaces and stretching of periarticular tissues is associated with joint cracking. This supposition is further reinforced by the noted difference the researchers reported between those individuals receiving a third MP mobilization versus manipulation. The groups receiving joint manipulation had a significantly larger post-treatment ROM.

Although articular cracking (cavitation) is commonly used by chiropractors as evidence of a successfully delivered adjustment,²⁷² the process of cavitation is not assumed to be therapeutic in and of itself. Rather, it represents a physical event that signifies joint separation, stretching of periarticular tissue, and stimulation of joint mechanoreceptors and nociceptors. These events, in turn, are theoretically responsible for alleviating or reducing pain, muscle spasm, joint hypomobility, and articular soft tissue inflexibility.^{236,272} Whether cavitation represents movement that is necessary to produce a better outcome as compared with patients who do not cavitate is largely unanswered. One study has compared the outcome of patients who did and did not cavitate with manipulation. The population was a cohort of 71 LBP patients who received a single sacroiliac manipulation. Subjects were reassessed 48 hours after the manipulation for changes in ROM, numeric pain rating scale, and modified Oswestry Disability Questionnaire. Both groups improved (21 noncavitators) and there were no clinically significant differences between groups. This study was limited to one area of the spine, evaluated only one adjustive method, and the application of only one manipulation. These factors limit the study's generalizability and clinical implications.

The presence or absence of cavitation (an audible crack) is also commonly presented as a means for distinguishing mobilization and thrust manipulation (adjustment).²⁷² Manipulation purportedly produces a cavitation, and mobilization does not. Thrust manipulation is much more frequently associated with joint cracking than mobilization. However, deep mobilization may also be associated with cavitation. The original studies conducted on cavitation in the MP joints were the product of joint mobilizations.²⁷⁶ If manipulation and mobilization were differentiated by the presence or absence of cavitation, a thrust manipulation, not associated with an audible release, would have to be reclassified as a mobilization. Any therapy that induces enough joint separation to overcome the fluid tension between synovial joint surfaces can produce joint cavitation. Therefore, manipulation and mobilization should be distinguished by the velocity of their application, not by the presence or absence of an associated joint cavitation.

Whether repetitive joint cavitation is associated with any negative side effects is a matter of debate. Brodeur²⁷² reviewed the literature and concluded that the investigations were very limited and inconclusive. It appears that habitual joint cracking is not associated with an increase in cartilage damage or osteoarthritic changes, although one study did note an increase in joint swelling and loss of grip strength in habitual joint crackers.

There are other potential causes of noises associated with various forms of manual therapy that are not a product of cavitation. With the development of cross-linkages in traumatized soft tissues, a manual procedure can break them apart, theoretically producing an audible tearing sound. With some mobilizing or manipulating procedures, the necessary movements of the parts can cause muscle tendons to move over bony protuberances, producing an audible snapping sound. Bony outgrowths can produce impingement that, with movements of the involved parts, can produce an audible clunking sound. Degenerative joint disease can produce crepitus on joint movement, producing an audible crackling sound.

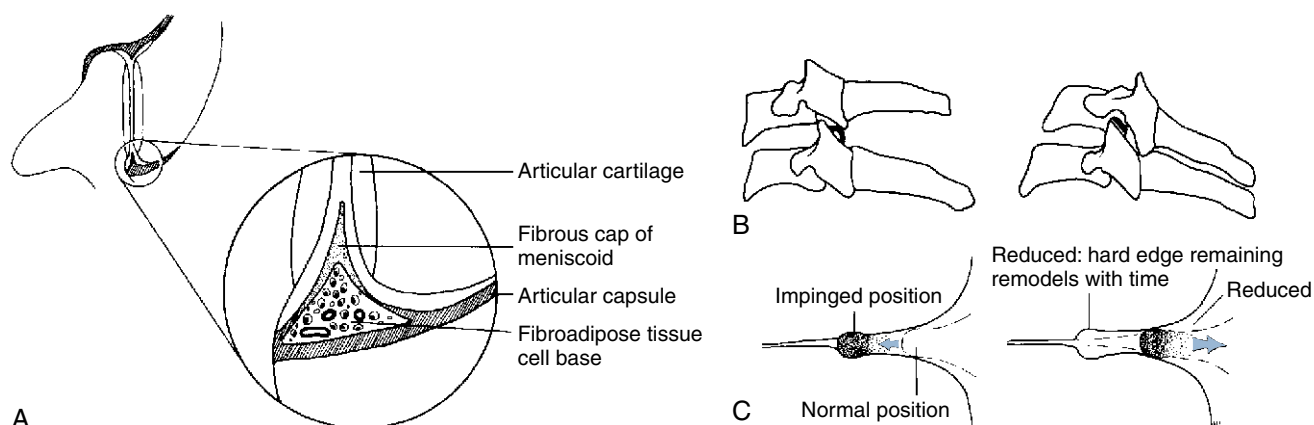


Figure 4-14 Position and postulated incarceration of synovial joint meniscoids. **A**, Diagram of the structural components of a meniscoid in a lumbar facet joint. **B**, Meniscoid entrapment in cervical facet joints restricting extension and flexion movements. **C**, Entrapment of meniscoids is postulated to produce deformation of the articular cartilage surface; after reduction and over time the articular cartilage will remodel. (A modified from Dupuis,¹⁸⁶. C modified from Lewit⁹⁸.)

JOINT FIXATION

Joint fixation implies a partial or complete restriction in joint movement. The restriction may be in one or more directions, and when used in chiropractic circles, it typically refers to a partial loss of joint movement (hypomobility), not a complete loss of movement. Several theories concerning the cause of joint fixation have been advanced. Derangements of the posterior joints, intercapsular adhesions, and intradiscal derangement have been proposed as interarticular sources; segmental muscle spasm and periarticular soft tissue fibrosis and shortening have been proposed as extra-articular sources.

Interarticular Adhesions

Interarticular adhesions refer to the hypothesis that joint fixation or hypomobility may be a product of adhesions that have developed between the articular surfaces of the Z joints.²⁸⁵ This process is speculated to result from joint injury, inflammation, or immobilization.^{241,247,249,254,286-289} Joint injury or irritation leading to chronic inflammation and joint effusion may induce synovial tissue hyperplasia, invasion of fibrous connective tissue, and consequent interarticular adhesions.^{56,57,247} In addition, Gillet²⁸⁹ has suggested that prolonged joint immobilization secondary to periarticular ligamentous shortening may eventually lead to fibrous adhesion formation between joint surfaces. Adjustive therapy is postulated to induce gaping of the involved joints breaking the adhesions between joint surfaces and improving or restoring joint mobility.

Interarticular Block

The term *interarticular block* refers to a reduction (blockage) of joint movement that is a product of some derangement within the synovial joint, internal to the joint capsule. Entrapment of the interapophyseal meniscus within the posterior spinal joints has been hypothesized as a cause of episodic acute back pain and joint locking.^{10,290-295} The menisci are purportedly drawn into a position between the joint margins during poorly coordinated spinal movements or by sustained stressful postures (Figure 4-14, A). With resumption of normal postures, pain results from impaction

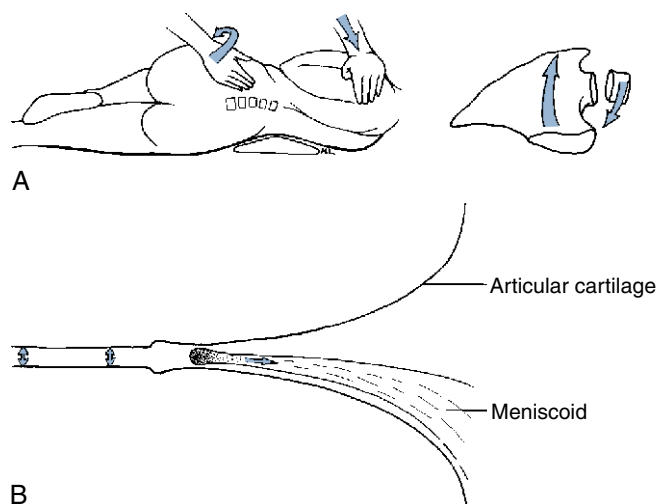


Figure 4-15 Techniques producing joint distraction have the potential to produce cavitation and reduce entrapment or extrapment of meniscoids. **A**, Technique applied to induce flexion, lateral flexion, and rotation in the left lumbar facets. **B**, Separation and expulsion of entrapped meniscoid.

of the menisci or traction of the articular capsule, inducing reactive muscle spasm and joint locking. The development of a painful myofascial cycle is initiated as prolonged muscle contraction leads to muscle fatigue, ischemia, and more pain. If spasm and locking persist, the articular cartilage may mold around the capsular meniscus, causing it to become more rigidly incarcerated within the joint (see Figure 4-14, B and C).²⁹⁴⁻²⁹⁶

To interrupt the cycle of pain, muscle cramping, and joint locking, distractive adjustments have been presented as a viable therapy capable of inducing joint separation, cavitation, and liberation of the entrapped meniscoid (Figure 4-15).

Bogduk and Engel²⁹⁷ question the plausibility of meniscus entrapment as a source of acute joint locking and make a compelling case for meniscoid extrapment. They contend that meniscoid entrapment would require the meniscus to have a firm apex strongly bound to the capsule by connective tissue. Their

morphologic studies did not confirm such an anatomic entity. They did imply, however, that a piece of meniscus torn and dislodged from its base could form a loose body in the joint, capable of acting as a source of back pain amenable to manipulation.

Bogduk and Jull²⁹⁸ favor instead the theory that the meniscoids become extrapped rather than entrapped. In their model of dysfunction, as the joint goes into flexion, the meniscoid is drawn out of the joint, and on return into extension, the meniscoid fails to properly reenter the joint cavity. Instead it lodges against the edge of the articular cartilage, where it buckles, serving as a space-occupying lesion that causes pain by distending the joint capsule (Figure 4-16).²⁹⁷ Manipulation that produces passive flexion should reduce the impaction, and rotation should gap the joint, encouraging the meniscoid to reenter the joint cavity.²⁹⁸

Other theories of interarticular soft tissue entrapment suggest that impingement of synovial folds or hyperplastic synovial tissue are additional sources of acute back pain and locking.^{299–302}

Bony locking of the posterior joints at the end-range of spinal motion have also been proposed. It is suggested that the developmental incongruencies and ridges in joint surface anatomy, combined with the complex coupled movements of the spine, may lead to excessive joint gapping at the extremes of movement, which may in turn lead to bony locking as the surfaces reapproximate.³⁰⁰ In both circumstances, distractive adjustive therapy has the potential to reduce the locking.

Interdiscal Block

Interdiscal block refers to internal derangement of the disc that leads to alterations or reductions in normal motion of the spinal motion segments. The mechanical derangements of the IVD that may lead to joint dysfunction are postulated to result from pathophysiologic changes associated with aging, degenerative disc disease, and trauma. Farfan³⁰³ has proposed a model of progressive disc derangement based on repetitive rotational stress to the motion segment. He postulates that repetitive torsional loads of sufficient number and duration may, over time, lead to a fatigue injury in the outer annular fibers. The process begins with circumferential distortion and separation in the outer annular fibers, followed by progression to radial fissuring and outward migration of nuclear material. The rate of fatigue and injury depends on the duration and magnitude of the force applied. In the individual with disrupted segmental biomechanics, the process is potentially

accelerated as an altered axis of movement leads to increased rotational strain on the IVD.

As presented earlier, the significance of torsional stress on the IVD, especially without coupled flexion, has been questioned. The sagittal orientation of the lumbar facets and the protective rotational barrier they provide bring into question the susceptibility of the lumbar discs to rotational torsion.^{165,169,170,303,304} Regardless of the mechanism or process, there is little doubt that internal disc derangement can lead to episodic or prolonged painful alterations or reductions in spinal movement.

Further complicating discal injury and internal disc disruption are the likely inflammatory and potential autoimmune reactions triggered by cellular disruption. Naylor³⁰⁵ has suggested that a discal injury with its associated connective tissue repair and vascularization is sufficient to create an antibody-antigen inflammatory reaction by exposing proteins of the nuclear matrix. The net effect is diminished protein polysaccharide content of the nucleus pulposus, loss of fluid content, and progression and acceleration of nuclear degeneration. As the nucleus atrophies, the disc becomes more susceptible to loading, and additional tractional forces may be transferred to the annulus, inducing mechanically based pain as the intact outer fibers are excessively stretched.¹⁴⁰

Interwoven into the natural history of degenerative disc disease may be episodes of acute mechanical back pain and joint locking. Others^{24,29,12,305–309} have postulated that incidents of blockage may occur during movements of trunk flexion as nuclear fragments become displaced and lodged along incomplete radial fissures in the outer fibers of the posterior annulus (interdiscal block) (Figure 4-17). Consequently, when extension is attempted, the displaced fragment cannot return to its central position and becomes compressed. The compressed fragment produces radial tension on the posterior annulus, causing pain and potential local muscle guarding and joint locking. Cyriax³⁰⁸ proposes that these lesions may induce tension on the dura mater, inducing low back pain (LBP) and muscle splinting. Once local pain and muscle spasm are initiated, a self-perpetuating cycle of pain, cramping, and joint locking may result. Adjustive therapy has been proposed as a viable treatment for interrupting this cycle of acute back pain and joint locking. In addition to the distractive effect on the posterior joints, adjustive therapy is thought to have a potential direct effect on the IVD, either by directing the fragmented nuclear material back toward a more central position or by forcing the nuclear fragment toward a less mechanically and neurologically insulting position between the lamellae of the annulus.³⁰⁹

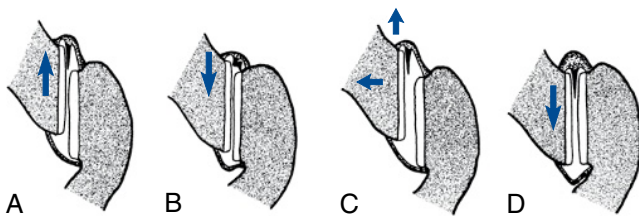


Figure 4-16 Theory of meniscoid extrapment. **A**, On flexion, the inferior articular process of a zygapophyseal joint moves upward, taking a meniscoid with it. **B**, On attempted extension, the inferior articular process returns toward its neutral position, but the meniscoid, instead of reentering the joint cavity, buckles against the edge of the articular cartilage, forming a space-occupying lesion under the capsule. **C**, Manipulation gaps the joint, allowing the meniscoid to return to its neutral resting position (**D**).

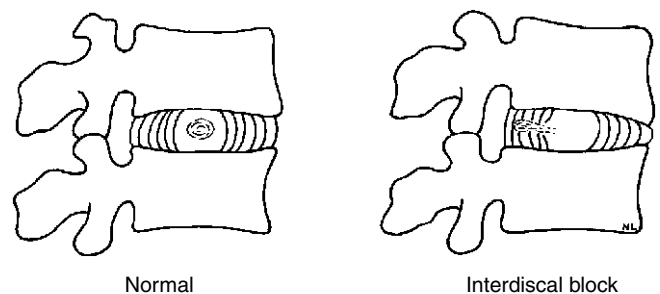


Figure 4-17 Fragments of nuclear material migrate in annular defects, creating an interdiscal block.

Two separate mechanical concepts have been proposed as models for how this might occur. The Gonstead adjustive technique has presented a model using adjustments to close down the side of nuclear migration (slippage) and force the material back toward the center (Figure 4-18).³¹⁰ The second concept, presented by Sandoz,²⁴ proposes a model in which distractive side posture adjustments combine disc distraction with rotation to induce helicoid traction and draw the herniated nuclear material back toward the center (Figure 4-19).

Internal derangement of the disc without associated NR dysfunction is difficult to conclusively differentiate from other mechanical disorders of the motion segment. Repetitive end-range loading and centralization of the patient's symptoms, especially in the presence of leg pain, has demonstrated value in helping

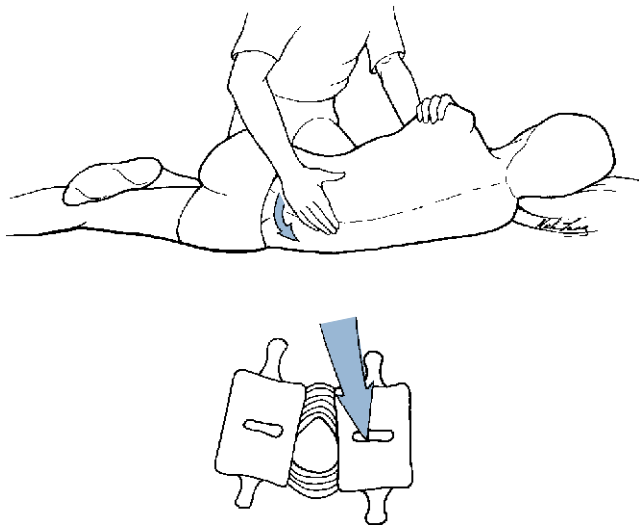


Figure 4-18 Techniques designed to close the side of nuclear migration (open wedge) are performed to force nuclear material toward the center of the disc.

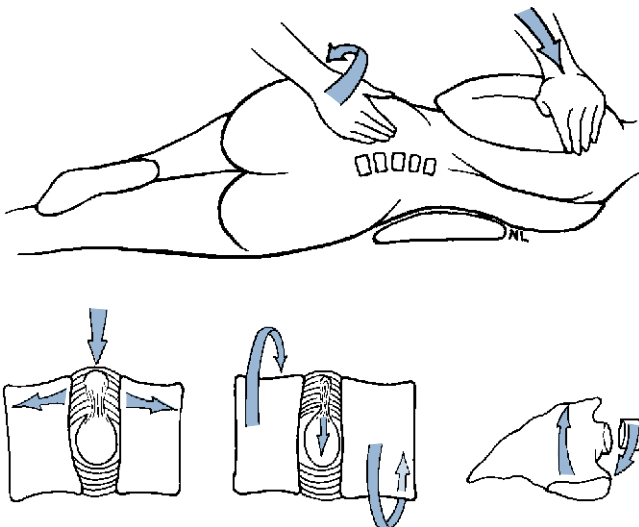


Figure 4-19 Techniques using distraction combined with rotation induce a helicoid traction that is intended to draw nuclear material toward the center of the disc.

clinically diagnoses this condition,³¹¹ but consensus has not been reached on the clinical criteria and standard of care for definitively establishing this disorder. Consequently, clinical research evaluating the effects of chiropractic HVLA adjustive treatment on IVD syndromes has focused primarily on biomechanical studies investigating chiropractic management of disc protrusion or herniation confirmed by imaging.

Levernieux³¹² noted reduction in disc herniation with axial traction, and Matthews and Yates³¹³ reported epidurographic reductions in disc herniations with manipulation. In contrast, Christman, Mittnacht, and Snook³¹⁴ reported a notable improvement in 51% of their patients treated with manipulation, but they reported no change in disc hernia as measured with myelography. Sandoz²⁴ concluded that the contradictory findings between these two studies can be accounted for by the fact that epidurography may measure smaller derangements of the disc, whereas myelography reveals only larger protrusions that are less amenable to manipulative care. It is doubtful that manipulation can reduce an external protrusion, but Sandoz²⁴ has suggested that manipulation may have a role to play in shifting the herniation away from the NR, minimizing the mechanical conflict and associated inflammation. In such circumstances, treatment is expected to be more protracted.²⁴

Well-designed and well-conducted clinical trials on HVLA adjustive therapy for disc herniation and associated radiculopathy (sciatica) are very limited. Clinical trials, uncontrolled descriptive studies, and case reports on the manipulative treatment of lumbar disc herniations are few, but they do indicate that this patient population may benefit from chiropractic manual therapy.^{24,304,314-324}

The incidence of complications arising from the manipulative treatment of disc herniation patients is extremely low. However, this procedure may carry some very minimal risk. Accordingly, modifications of side posture manipulative techniques have been suggested in the treatment of patients with marked disc herniations. To minimize the risk of further annular injury, side posture adjusting or mobilization postures, which minimize excessive lumbar flexion and compression, have been proposed.³⁰⁴ Procedures and positions that increase the patient's leg pain are assumed to be more stressful to the annular fibers and are to be avoided. Those that reduce or centralize back pain while decreasing the patient's leg pain are presented as potentially the safest and most effective. Disc herniation patients suffering progressive neurologic deficits or midline herniations with an associated CES should not be considered for manipulation.³²⁵⁻³²⁸

Periarticular Fibrosis and Adhesions

As mentioned previously, acute or repetitive trauma may lead to articular soft tissue injury. In the process of fibrotic repair, adhesions and contractures may develop, resulting in joint hypomobility. Distractive adjustments are advanced as procedures capable of effectively treating these derangements by stretching the affected tissue, breaking adhesions, restoring mobility, and normalizing mechanoreceptive and proprioceptive input.^{30,237-239}

It is further postulated that manipulation may sever the adhesive bonds, stretch tissue, and promote mobility without triggering an inflammatory reaction and recurrence of fibrosis. However, when articular or nonarticular soft tissue contractures are encountered,

incorporation of procedures that minimize inflammation and maintain mobility should be considered. Viscoelastic structures are more amenable to elongation and deformation if they are first warmed and then stretched for sustained periods.³²⁹ Therefore, the application of moist heat, ultrasound, and other warming therapies might be considered before applying sustained manual traction or home-care stretching exercises.

Joint Instability

Although emphasis has been placed on the adjustive treatment of mechanical disorders resulting from joint hypomobility, manipulative therapy also may have a role in the treatment of clinical joint instability. *Clinical joint instability* can be defined as a painful disorder of the spine resulting from poor segmental motor control or a loss of stiffness in the controlling soft tissues that leads to a loss of motion segment equilibrium and an increase in abnormal translational or angular movements.^{330,331} Common proposed causes of joint instability include acute trauma, repetitive-use injuries, compensation for adjacent motion segment hypomobility,³³² ineffective neural control, degenerative disc disease, and muscle weakness or poor endurance.³³³ Clinical joint instability is not to be confused with gross orthopedic instability resulting from marked degeneration, traumatic fracture, or dislocation.

Joint instability may predispose the patient to recurring episodes of acute joint locking and may be seen more frequently in individuals who have some degree of hypermobility that is a result of advanced training in athletics such as gymnastics or ballet dancing.³³² Adjustive therapy applied in this condition is not intended to restore lost movement but rather to reduce the episodic pain, temporary joint locking, joint subluxation, and muscle spasm that are commonly encountered in patients with unstable spinal joints. Adjustive therapy delivered in these circumstances is considered to be palliative. It should not be applied during an extended period, and it should be incorporated with stabilization therapy, appropriate exercise, and lifestyle modification.^{332,333}

NEUROBIOLOGIC HYPOTHESIS

Analgesic Hypothesis

The reduction of pain and disability from spinal manipulation is well recognized and clinically documented.^{147,194,199,334-340} “Numerous studies suggest that SM alters central processing of noxious stimuli because pain tolerance or pain threshold levels can increase after manipulation.”²⁶¹ The mechanisms by which manipulation inhibits pain, however, are matters of speculation and still under investigation. Proposed hypotheses have suggested that manipulation has the potential to remove the source of mechanical pain and inflammation or induce stimulus-produced analgesia.

The case for decreasing pain by removing its mechanical source is empiric and deductive. The pain associated with mechanical disorders of the musculoskeletal system is a product of physical deformation, inflammation, or both.³⁴¹ It is reasoned that manual therapy effective at reversing or mitigating underlying structural and functional derangements will remove the source of pain and the associated pain-producing agents as structures are returned to normal function.

The argument for stimulus-produced analgesia is bolstered by experimental evidence that suggests that chiropractic adjustments induce sufficient force to simultaneously activate both superficial and deep somatic mechanoreceptors, proprioceptors, and nociceptors. The effect of this stimulation is a strong afferent segmental barrage of spinal cord sensory neurons, capable of altering the pattern of afferent input to the central nervous system and inhibiting the central transmission of pain (Figure 4-20).³⁴¹⁻³⁴⁵

Gillette³⁴² suggests that spinal adjustments may initiate both a short-lived phasic response triggered by stimulation of superficial and deep mechanoreceptors, and a longer-lived tonic response triggered by noxious-level stimulation of nociceptive receptors. The phasic response is hypothesized to initiate a local gating effect, but pain inhibition terminates with cessation of therapy. The tonic response initiated by noxious levels of mechanical stimulation is more powerful and capable of outlasting the duration of applied therapy.³⁴⁴

Adjustments that induce joint cavitation and capsular distraction may be a source of nociceptive stimulation capable of initiating relatively long-lasting pain inhibition. This concept supports the premise that the slight discomfort that may be associated with adjustments is causally associated with a positive therapeutic effect.³³⁴

The potential for spinal adjustments to act directly on the pain system opens up the possibility that manipulation may have the ability to diminish persistent pain that is neuropathic in origin.³⁴⁵ Chronic neuropathic pain may result from plastic changes and central sensitization of the nervous system. Central sensitization refers to plastic changes in the nervous system that result from persistent amplification of nociceptive synaptic transmission. This can result in the persistence of pain states even after the offending periphenal pathologic injury and inflammation have resolved.³⁴⁴

The short-term bursts of proprioceptive and nociceptive input associated with adjustments, much like transcutaneous electrical nerve stimulation and acupuncture, have also been theorized to increase the levels of neurochemical pain inhibitors.³³⁷ Both a local release of enkephalins, initiated by stimulation of the neurons of substantia gelatinosa, and a systemic increase in plasma and cerebrospinal fluid endorphin levels, initiated by stimulation of the hypothalamic pituitary axis, have been proposed. Both substances act as endogenous opioid pain inhibitors and may play a role in the analgesic effects of adjustments.

Doctor reassurance and the laying-on of hands may also impart a direct analgesic effect, which must be factored into the equation when calculating the effects of adjustments and manual therapy. The contact established during a skilled evaluation of the soft tissues indicates the doctor's sense of concern and skill. Paris³³¹ states that with the addition of a skilled evaluation involving palpation for soft tissue changes and altered joint mechanics, the patient becomes convinced of the clinician's interest, concern, and manual skills. If the examination is followed by treatment and an adjustive cavitation (crack), further positive placebo effects may be registered. The astute clinician accepts and reinforces this phenomenon if it influences the patient's recovery. This does not excuse misrepresentation or irresponsible exaggeration of the therapeutic effect.

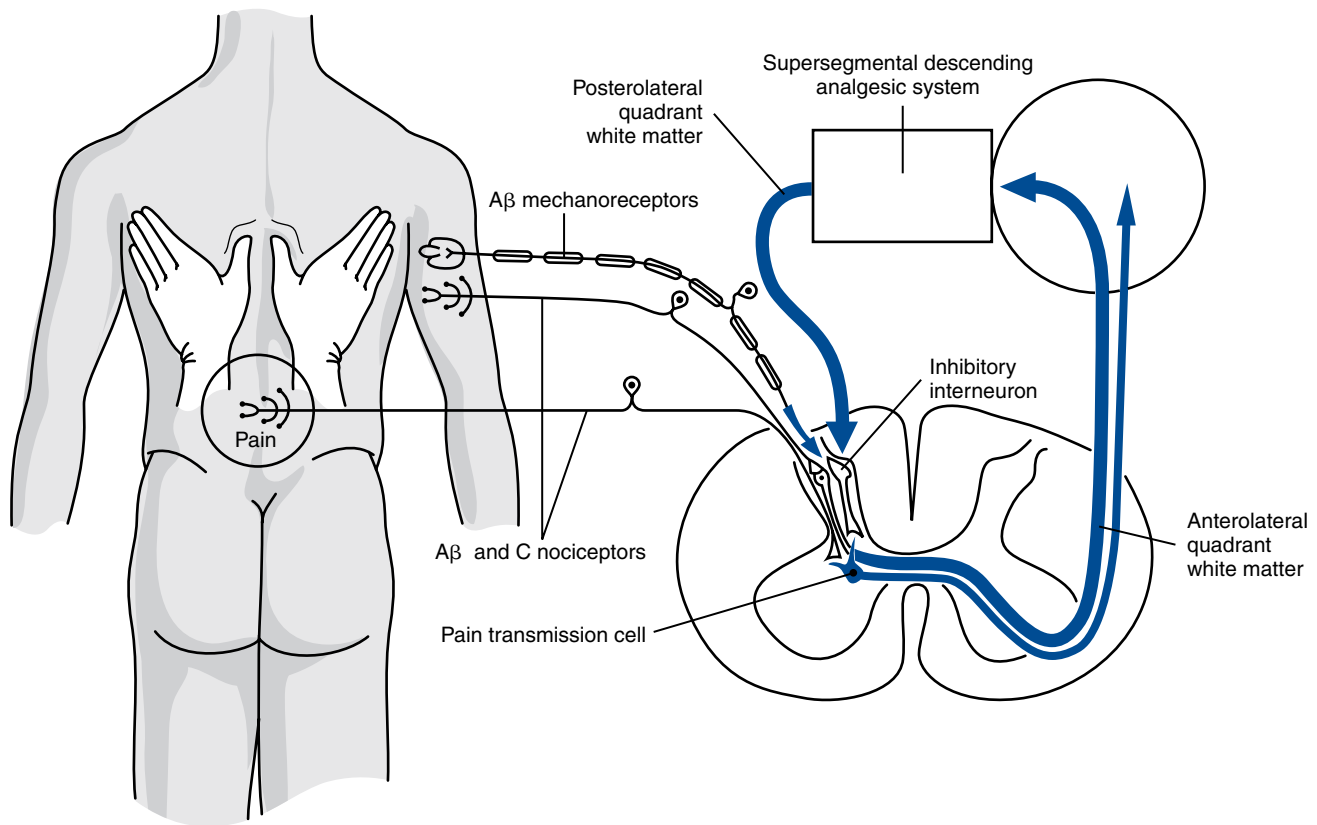


Figure 4-20 Diagram suggesting the mechanism by which a high-velocity chiropractic adjustment inhibits the central transmission of pain through activation of mechanoreceptors and nociceptors. (Modified from Gillette, Cassidy JD, Lopes AA, Yong-Hing K: The immediate effect of manipulation versus mobilization on pain and range of motion in the cervical spine: A randomized controlled trial, *J Manipulative Physiol Ther* 15:570, 1992.)

Muscle Spasm (Hypertonicity)

Numerous authors have presented the potential causative role of hypertonic muscles in the development of joint dysfunction and spinal pain.^{24,238,289,292,346-349} The concept that restricted joint movement may result in increased segmental muscle tone or spasm is supported by the knowledge that muscles not only impart movement but also impede movement. Joint movement depends on a balance between its agonist and antagonists. If this balance is lost and antagonistic muscles are unable to elongate because of involuntary hypertonicity, the joint may be restricted in its range or quality of movement.

Increased resting muscle tone or spasm may be initiated by direct provocation or injury to myofascial structures or indirectly by stimulation or injury to associated articular structures. Direct overstretching and tearing of muscle lead to stimulation of myofascial nociceptors and protective muscle splinting. The intersegmental muscles of the spine may be especially vulnerable to incidents of minor mechanical stress and overstretching. They are not under voluntary control. They act primarily to stabilize and integrate segmental movements in response to global movements of the trunk. As a result, they may be especially vulnerable to unguarded movements and the induction of reactive splinting.

Korr³⁴⁶ suggests that unguarded and uncoordinated movements may approximate the short segmental muscles of the back and reduce annulospiral receptor activity in the muscle spindle complex and produce muscle spasm. Maigne²⁹¹ envisions a similar lesion (articular strain), but speculates that it results from abnormal

sustained postures or poorly judged movements that induce minor intersegmental muscle overstretching and cramping. Both speculate that segmental muscle spasm, once initiated in the back, may be hard to arrest. Contracted segmental muscles of the back, unlike the voluntary appendicular muscles, are not easily stretched by the contraction of antagonistic muscle groups. As a result, this condition may not be inhibited by active stretching and therefore may be less likely to be self-limiting.²⁹¹ Research published in 2000 demonstrated that muscle spasm reduced the ability of paraspinal muscle stimulation to evoke cerebral potentials.³⁵⁰ “Spinal manipulation reversed these effects, reducing muscle spasm and restoring the magnitude of the evoked cerebral potentials.”²⁶¹

Myofascial Cycle

A central complicating feature of many of the internal and external derangements of the motion segment is the induction of a self-perpetuating myofascial cycle of pain and muscle spasm. The articular soft tissues are richly innervated with mechanoreceptors and nociceptors, and traction or injury to these structures may lead to the initiation of local muscle splinting. With time, the continued muscle contraction may lead to further muscle fatigue, ischemia, pain, and maintenance of muscle spasm and joint locking (Figure 4-21).

High-velocity adjustments are suggested as treatments that may be effective in interrupting this cycle. Several theories exist as to the mechanism by which adjustments relieve muscle spasm. Both are speculated to induce a reflex response in muscle—one

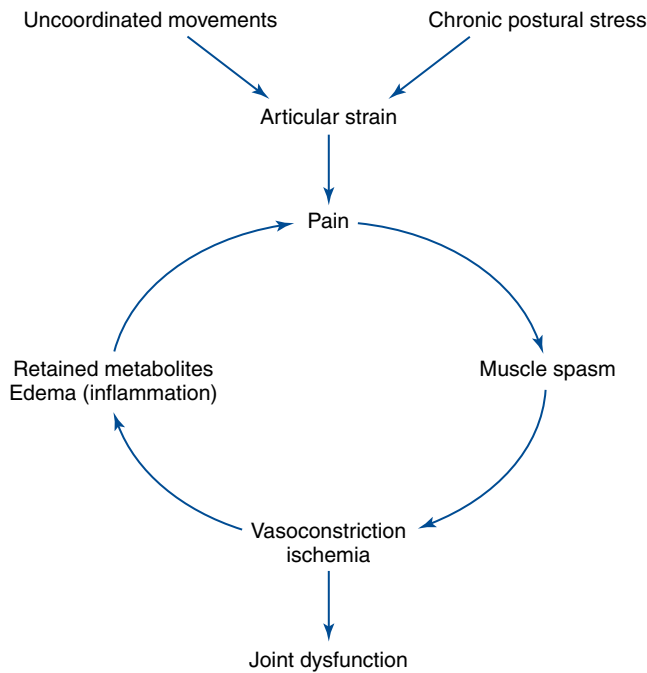


Figure 4-21 The self-perpetuating cycle of myofascial pain and muscle spasm.

through direct action on muscle and the other reflexly through joint distraction (cavitation). The direct muscle model³⁴⁶ speculates that quick traction and excitation of the Golgi tendon organ (GTO), located in the muscle tendon junction, act as brakes to limit excessive joint movement and possible injury by inhibiting motor activity. The concept is that adjustments induce a strong stretch on the muscle tendon complex, activate the GTO, and induce reflex muscle relaxation (autogenic inhibition). Although this model seems reasonable, evidence suggests that the GTO has a less profound effect than initially envisioned. Watts and associates³⁴⁹ found that stimulation of the GTO produces a very meager inhibitory effect on motor neuron activity. This information implies that the GTO plays a more minor role in the inhibition of muscle spasm than initially proposed and brings into question its relationship to postadjustment muscle relaxation.

In contrast, stimulation of articular low- and high-threshold mechanoreceptors and nociceptors has demonstrated a notable inhibitory effect on segmental motor activity.³⁴² Mechanoreceptors and nociceptors are also widely embedded in articular soft tissues, muscle, and skin. High-velocity adjustments induce enough force to stimulate these structures and induce a burst of somatic afferent receptor activity.^{281,342} Based on this information, it seems reasonable to assume that joint and soft tissue mechanoreceptors and nociceptors have the potential to play a material role in the inhibition of muscle spasm and the interruption of painful myofascial cycles and joint locking.

Clinical investigations on the effects of spinal manipulation on muscle activity are very limited. Investigations have centered on the effects of manipulation during and after the application of manipulation. Using surface electromyograph (EMG), Herzog and others^{351–353} investigated the immediate effects of thoracic SMT on paraspinal muscle activity. They applied prone unilateral quick (HVLA) and slow (3- to 4-second) “manipulations” to the

thoracic transverse processes. Both procedures consistently induced momentary increased muscle activity during their application. The high-velocity manipulations were associated with a fast, burst-like EMG signal, and the slow manipulation with a gradual increase in EMG activity. Cavitations induced during the application of the slowly applied manipulation were not associated with increased EMG activity, leading the researchers to speculate that cavitation alone is not sufficient to induce a reflex muscular response. The myoelectric response recorded during thoracic adjustments did conflict with the application of the adjustive forces. In contrast, Triano and Schultz¹⁶¹ were unable to record any significant myoelectric activity or muscular responses with the application of HVLA SP lumbar-adjusting procedures.

Investigations into more prolonged effects on resisting muscle activity, although very limited, have shown reductions in paraspinal muscle activity and imbalance with full-spine adjusting procedures.^{352,353}

Nerve Root Compression

Chiropractic, osteopathy, and manual medicine has envisioned manual therapy affecting not only somatic disorders, but also visceral disorders through neurologic means.¹⁷ The early paradigm presented in chiropractic stressed a model of altered NR function as the basis for secondary somatic or visceral dysfunction. It was theorized that subluxations induce structural alteration of the intervertebral foramina, leading to compression of the contained neurovascular structures and altered function of the NR as electrical transmission or axoplasmic flow is impaired. The postulated net result of this process (nerve interference) was dysfunction or disease in the somatic and visceral structures supplied by the affected NR.^{17,354–359} The subluxation-induced narrowed intervertebral foramen (IVF) was hypothesized to induce NR dysfunction through direct bony compression (pinched-hose model) or indirectly by increasing pressure around the NR and its vascular structures.

In 1973, Crelin³⁶⁰ challenged the anatomic plausibility of subluxated motion segments producing NR compression. His anatomic dissections and measurements, made at the lateral borders of the IVF, demonstrated a minimum of 4 mm of space around the NR. He concluded that the space was more than adequate and that the NR was not anatomically vulnerable to compression. More recently in 1994, Giles³⁶¹ revisited the issue of NR vulnerability but at a different anatomic site. His measurements were taken at the interpedicular zone and demonstrated an average of only 0.4 to 0.8 mm of space around the NR and the NR ganglion. He concluded that the NR was anatomically vulnerable, but at the interpedicular zone, not at the lateral borders of the IVF. Furthermore, “dorsal roots and dorsal root ganglia [DRG] are more susceptible to the effects of mechanical compression than are axons of peripheral nerves because impaired or altered function is produced at substantially lower pressures.”³⁶¹

A potential site of anatomic vulnerability does not, by any means, validate chiropractic models of subluxation-induced NR dysfunction. The plausibility of uncomplicated subluxations commonly inducing NR compression still seems unlikely.^{355–360} It does, however, raise an interesting issue about the potential for spinal motion segment dysfunction to contribute to NR compression when it is associated with other compromising joint patholo-

gies.^{300,361,362} Disc herniation and exposure of the NR to discal material increase spontaneous nerve activity and the mechanical sensitivity of the NR and possible mechanical hyperalgesia. Spinal NRs already compromised by disc herniation, degenerative joint and disc disease, or central or lateral stenosis and the associated inflammation may become more serious when associated with dysfunction that fixes the joint in a more compressive and compromising position. In such circumstances, adjustive therapy that reduces a position of fixed subluxation and root irritation may have an effect on reducing NR traction, compression, or inflammation.

Reflex Dysfunction

Beginning with the work of Homewood,³⁵⁸ the profession has gradually moved away from reliance on NR compression and toward a more dynamic model of subluxation-induced neurodysfunction. As presented in Chapter 3, the reflex paradigm presents a model in which somatic dysfunction or joint dysfunction induces persistent nociceptive and altered proprioceptive input. This persistent afferent input triggers a segmental cord response, which in turn induces the development of pathologic somatosomatic or somatovisceral disease reflexes^{357-359,363-368} (Figure 4-22). If these reflexes persist, they are hypothesized to induce altered function in segmentally supplied somatic or visceral structures.

Chiropractic adjustive therapy has the potential for arresting both the local and the distant somatic and visceral effects by normalizing joint mechanics and terminating the altered neurogenic reflexes associated with joint dysfunction. For example, a patient with a strained posterior joint capsule accompanied by reflex muscle spasm may have nociceptive bombardment of the spinal cord. If the nociceptive bombardment is of sufficient strength and duration, it may cause segmental facilitation. The spinal adjustment may reduce the strain on the joint capsule and reduce muscle spasm that stops nociception from these tissues into the spinal cord. At the same time, adjustments stimulate many different types of mechanoreceptors. The result is a reduction of a harmful somatosomatic and potential somatoautonomic reflex. This model has become the focus of more attention and investigation as

chiropractors search for an explanation to the physiologic effects that they have clinically observed to be associated with spinal adjustive therapy. This relationship is not consistent, and the frequency of response is undetermined, but the anecdotal and empiric experiences of the profession are significant enough to warrant serious further investigation.

An additional model of subluxation-induced neurodysfunction focuses attention on the potential direct mechanical irritation of the autonomic nervous system. The paradigm for irritation of sympathetic structures is based on the anatomic proximity and vulnerability of the posterior chain ganglion, between T1 and L2, to the soma of the posterior chest wall and costovertebral joints. Altered spinal and costovertebral mechanics are hypothesized to mechanically irritate the sympathetic ganglia and to induce segmental sympathetic hypertonia.³⁶⁸ The target organs within the segmental distribution then theoretically become susceptible to altered autonomic regulation and function as a result of altered sympathetic function.

In contrast to the sympathetic chain, the parasympathetic system, with its origins in the brain, brainstem, and sacral segments of the spinal cord, does not have anatomic proximity to the spinal joints. Models of mechanically induced dysfunction of the parasympathetic system propose dysfunction in cranial, cervical, and pelvic mechanics as potential sources of entrapment or tethering of the parasympathetic fibers. Altered cervical, cranial, or craniosacral mechanics are theorized to induce traction of dural attachments and the cranial nerves as they exit through the dura and skull foramina. The treatment goal in mechanically induced autonomic dysfunction is to identify the sites of joint dysfunction and implement appropriate manual therapy to balance membranous tension.³⁶⁹

From the discussion of spinal dysfunction and its potential neurobiologic effects on health, it must be remembered that spinal dysfunction and pain may be the product of, not the cause of, somatic or visceral dysfunction or disease.³⁷⁰ Spinal pain and dysfunction may be secondary to a disorder that needs direct treatment. Manual therapy may be a fitting component of appropriate care, but would be inadequate as the singular treatment. The patient with caffeine-induced gastritis who develops secondary midback pain and dysfunction (viscerosomatic) should not receive manual therapy without also being counseled to discontinue ingestion of caffeinated beverages. The spine is a common site of referred pain, and when a patient with a suspected mechanical or traumatic disorder does not respond as anticipated, the possibility of other somatic or visceral disease should be considered.

Neuroimmunology

An interaction exists between the function of the central nervous system and the body's immunity that lends support to the chiropractic hypothesis that neural dysfunction is stressful to the body locally and globally. Moreover, with the resultant lowered tissue resistance, modifications to the nonspecific and specific immune responses occur, as well as altered trophic function of the involved nerves. This relationship has been termed the *neurodystrophic hypothesis*.

Selye³⁷¹⁻³⁷³ demonstrated neuroendocrine-immune connections in animal experiments and clinical investigations. Physiologic, psychologic, psychosomatic, and sociologic components compose the stress response. From studies of overstressed animals, Selye

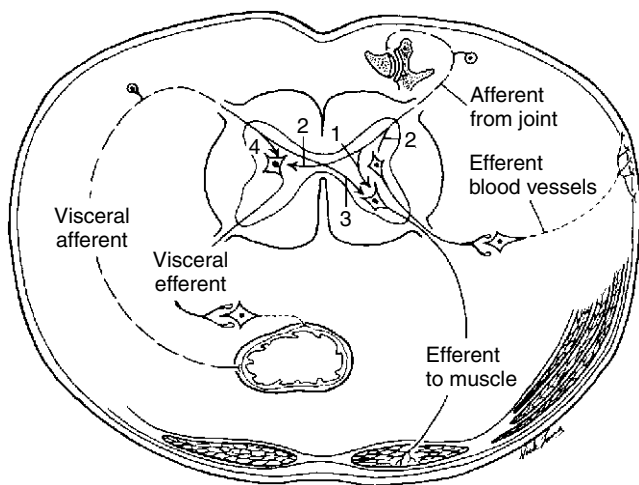


Figure 4-22 Afferent and efferent pathways from and to the viscera and somatic structures that can produce (1) somatosomatic, (2) somatovisceral, (3) viscerosomatic, and (4) viscerovisceral reflex phenomena. (Modified from Schmidt,¹⁸⁸)

observed nonspecific changes that he labeled the *general adaptive syndrome*. He also observed very specific responses that depended on the stressor and on the part of the animal involved, which he termed *local adaptive syndrome*. Furthermore, he established a stress index comprising major pathologic results of overstress, including enlargement of the adrenal cortex, atrophy of lymphatic tissues, and bleeding ulcers. Selye also felt that long-term stress would lead to diseases of adaptation, including cardiovascular disease, high blood pressure, connective tissue disease, stomach ulcers, and headaches.

Stressors can produce profound health consequences.³⁷⁴ Theorists propose that stressful events trigger cognitive and affective responses that, in turn, induce sympathetic nervous system and endocrine changes, and these ultimately impair immune function.³⁷⁵⁻³⁷⁹ Stressful events cannot influence immune function directly. Instead, stress is thought to affect immune function through central nervous system control of the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic-adrenal-medullary axis.^{377,380-383} Stressors produce reliable immune changes.³⁷⁴ Segerstrom and Miller³⁸⁴ analyzed different types of stressors separately and found that the immunologic effect of stressors depends on their duration.

However, because all individuals do not develop the same syndrome with the same stressor, Mason³⁸⁵ suggested that emotional stimuli under the influence of internal (genetics, past experiences, age, and sex) or external (drugs, diet, and hormone use) conditioning are reflected in the responses of the endocrine, autonomic, and musculoskeletal systems³⁸⁵ (Figure 4-23).

Stein, Schiavi, and Camerino³⁸⁶ convincingly demonstrated psychosocial and neural influences on the immune system. They showed that the hypothalamus has a direct effect on the humoral immune response, explaining how psychosocial factors can modify host resistance to infection. Moreover, Hess³⁸⁷ produced sympathetic and parasympathetic responses by stimulating different parts of the hypothalamus. The sympathetic response (ergotropic response) is characteristic of the fight-or-flight mechanism, whereas the parasympathetic response (trophotropic response) produces relaxation that promotes a restorative process. Table 4-2 lists the characteristics and physiologic responses of the ergotropic and trophotropic states.

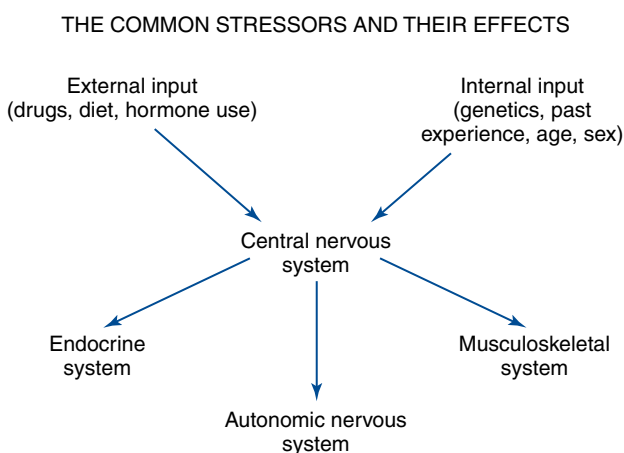


Figure 4-23 Internal and external conditioning can affect emotional stimuli, resulting in autonomic, endocrine, or musculoskeletal changes.

TABLE 4-2 Characteristics of the Ergotropic and Trophotropic Responses

Ergotropic Responses	Trophotropic Responses
Primarily sympathetic	Primarily parasympathetic
Excitement, arousal, action	Relaxation
Movement of body or parts	Energy conservation
Increased heart rate, blood pressure, respiratory rate	Decreased heart rate, blood pressure, respiratory rate
Increased blood sugar	Increased gastrointestinal function
Increased muscle tension	Decreased muscle tension
Increased dioxide consumption	Decreased dioxide consumption
Increased carbon dioxide elimination	Decreased carbon dioxide elimination
Pupil dilation	Pupil constriction

The three classically separated areas of neuroscience, endocrinology, and immunology, with their various organs—the brain; the glands; and the spleen, bone marrow, and lymph nodes, respectively—are actually joined to one other in a multidirectional network of communication, linked by information carriers known as *neuropeptides*. The field of study is called *psychoneuroimmunology (PNI)*. PNI is a scientifically solid field of study, grounded in well-designed experiments and in the resolute tenets of behaviorism.³⁸⁸ The first components of the process of linking the systems of the body together, and ultimately the body and mind, are the receptors found on the surface of the cells in the body and brain. Almost every peptide receptor, not just opiate receptors, could be found in this spinal cord site that filters all incoming bodily sensations. It has also been found that in virtually all locations at which information from any of the five senses enters the nervous system there is a high concentration of neuropeptide receptors. These regions are called *nodal points*.³⁸⁸

Today's health care provider should recognize the interconnectedness of all aspects of human emotion and physiology. The skin, the spinal cord, and the organs are all nodal points of entry into the psychosomatic network. Health care providers that incorporate touching and movement in their treatment of patients affect them all.

Leach³⁸⁹ points out that there is a paucity of studies that directly link vertebral lesions with immunologic competence, although his review of the literature suggests that such a connection is possible. Fidelibus,³⁹⁰ after conducting a recent review of the literature, concluded that the concepts of neuroimmunomodulation, somato-sympathetic reflex, and spinal fixation provide a theoretic basis for using spinal manipulation in the management of certain disorders involving the immune system, including asthma, allergic rhinitis, and the common cold. He further postulates that musculoskeletal dysfunction can result in immune dysfunction and that, by removing the musculoskeletal dysfunction, spinal manipulation

can affect the immune dysfunction. As mentioned previously, chiropractic manipulation did not have a positive outcome in treating childhood asthma in a population of children who were less than optimally responsive to medication.²¹⁶

Two studies on infantile colic^{233,391} indicate that chiropractic treatment results in a reduction of the daily length and number of colic periods. Klougart and associates²³⁴ found that 94% of the infants studied were helped by chiropractic treatment within 14 days from the start of treatment. Wiberg, Nordsteen, and Nilsson³⁹¹ compared spinal manipulation with dimethicone medication. The infants in the chiropractic group exhibited a 67% reduction of daily hours of colic, whereas the dimethicone group had a 38% reduction. However, a 2001 randomized placebo controlled study that blinded the parents from the therapy found no difference between placebo and spinal manipulation in the treatment of infantile colic.²³⁵

Vernon and colleagues³³⁷ reported a slight, but statistically significant, increase in B-endorphin levels in asymptomatic males after cervical manipulation, whereas Sanders et al³⁹² and Christian, Stanton, and Sissons³⁹³ found no change in B-endorphin levels in either symptomatic or asymptomatic male study participants after chiropractic manipulation.

Whelan and associates³⁹⁴ examined 30 asymptomatic male chiropractic students in a randomized clinical trial to determine the effect of HVLA cervical manipulation on salivary cortisol secretion. They found no effect of chiropractic manipulation on salivary cortisol and concluded that in asymptomatic subjects familiar with chiropractic manipulation, neither the sham nor cervical manipulation induces a state of anxiety sufficient to disrupt the homeostatic mechanisms and activate the HPA axis.

Teodorczyk-Injeyan, Injeyan, and Ruegg³⁹⁵ report that SMT in asymptomatic subjects down-regulates production of the inflammatory cytokines tumor necrosis factor- α and interleukin 1 β (IL-1 β). They also determined that this change in cytokine production was unrelated to serum substance P levels.

The work of Brennan and others³⁹⁶⁻⁴⁰³ remains the only extended line of investigation into the effect of chiropractic SM and immune function. They reported that a single manipulation in the thoracic or lumbar spine produced a short-term priming of the polymorphonuclear cell response to an *in vitro* particulate challenge. They observed an enhanced chemiluminescent respiratory burst in both asymptomatic and symptomatic study participants.^{396,398,403} This enhanced polymorphonuclear cell activity was associated with slight, but statistically significant, rise in plasma substance P. Further investigation suggested that this systemic effect depends on both the applied force and vertebral level.^{398,403} In follow-up, Kokjohn and co-workers⁴⁰⁴ hypothesized that the force applied to the thoracic spine by manipulation is sufficient to result in increased plasma levels of substance P, which may prime circulating phagocytic cells for enhanced respiratory burst. However, whether the effect is significant in fighting infection has not been determined, and the exact mechanism whereby manipulation affects phagocytic cells remains speculative, because significant levels of plasma substance P were not determined.

The available studies suggest mechanisms by which spinal influences may mediate a clinically significant effect on immune function, but few studies have directly examined those mechanisms,

and the evidence to date is conflicting. Consequently, there are clearly both plausible mechanisms to explore and clinical practice-driven justification for additional basic science studies in this area.²⁶¹

CIRCULATORY HYPOTHESIS

Beneficial vascular responses to adjustive therapy are theorized to result as a product of stimulation of the autonomic nervous system or through improved function of the musculoskeletal system. Experimental and clinical evidence suggests the importance of an adequate blood supply for optimal function.⁴⁰⁵ It was observed long ago that vasoconstriction resulting from sympathetic hyperactivity reduces blood volume substantially, posing a threat of relative ischemia in the area involved.⁴⁰⁶ Disturbances ranging from ischemia to hypoxia can generate influences that adversely affect the musculoskeletal system.

As discussed previously, joint subluxation/dysfunction has been submitted as a source of altered segmental sympathetic tone. If joint dysfunction can induce a sympathetic response robust enough to induce local or segmental vasoconstriction, spinal subluxation/dysfunction may be associated with decreased circulation to segmentally supplied tissues. Cutaneous signs are found in altered texture, moisture, and temperature. Chiropractic adjustments would then have the potential to improve circulation by restoring joint function and removing the source of sympathetic irritation.

Musculoskeletal integrity and function are additional factors directly affecting the circulatory system. The venous and lymph systems are driven by skeletal muscle movements and changing intrathoracic and intra-abdominal pressures. A healthy respiratory pump depends on a functioning diaphragm and flexible spine and rib cage. Conditions or injuries that lead to the loss of musculoskeletal mobility and strength result in a potential net loss of functional capacity of the musculoskeletal system and its ability to move blood and lymph. Muscle injury or disuse leads to an accompanying loss of vascularization in the affected tissues, and additional blood and lymph flow impedance may occur. Blood vessels pass through muscle, and it is reasonable to assume that marked contraction of the muscle will impede circulatory flow, especially on the venous side, where pressures are low. Therapy directed at improving mobility and skeletal muscle strength has the potential to improve the functional capacity of the musculoskeletal system and improve circulation.⁴⁰⁷

It has not been established, however, whether manipulation specifically acts through the nervous system to affect the blood supply or by altering the adverse musculoskeletal influences that are interfering with the controls and regulations of vasomotor function. It is likely that both concepts are possible.

APPLICATION OF ADJUSTIVE THERAPY

Once a working diagnosis is established and a decision is reached to use adjustive therapy, the chiropractor and patient need to establish the therapeutic goals of treatment and decide what specific adjustive methods to apply (Figure 4-24). The decision is influenced by factors such as the presence or absence

ADJUSTIVE THERAPY DECISION-MAKING

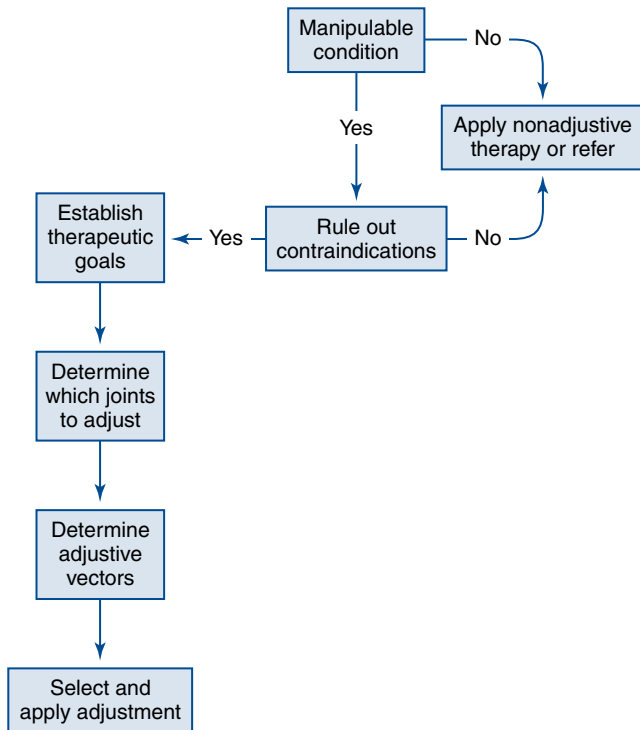


Figure 4-24 Factors to consider before selecting and applying an adjustment.

of complicating disorders and the patient's age, size, flexibility, physical condition, and personal preferences. The ability to make a correct assessment and decision is affected by the doctor's knowledge of anatomy, biomechanics, contraindications to adjustments, and adjustive mechanics.

Before adjustments can be applied, the doctor must determine which joints or spinal regions to adjust and what adjustive movements and vectors to generate (see Figure 4-24). The decision is a clinical one based on the presenting condition and physical findings (Box 4-14). The final decision must be placed within the context of the local anatomy and the geometric plane of the articulations, the nature of the patient's health status, and any underlying disease processes. These factors and the mechanical characteristics of the adjustment to be applied will influence positioning of the patient, the specific contacts, the

degree of appropriate preadjustive tension, the magnitude of the applied force, and the direction of the adjustive thrust (Box 4-15). The ultimate goal is to select and apply a safe, comfortable, and effective adjustment that allows the doctor to localize and focus the adjustive forces to a specific region or motion segment.

JOINT ANATOMY, ARTHROKINEMATICS, AND ADJUSTIVE MOVEMENTS

Knowledge of spinal and extremity joint architecture, facet plane orientations, and arthrokinematics is necessary for sound application of adjustments. Most adjustive techniques are directed at producing joint distraction. Spinal adjustments are more likely to induce effective movements when the clinician has a fundamental understanding of how joints are configured and what adjustive vectors and forces are likely to efficiently generate joint movement without producing joint injury.

The application of prone adjustive technique can be used to illustrate this point. In the thoracic spine, the articular surfaces are relatively flat. The superior articular processes underlie (are anterior to) the inferior articular processes and on average form an angle of approximately 60 degrees to the horizontal. During segmental flexion in the thoracic spine, the posterior joint surfaces glide apart along their joint surfaces. During extension, the posterior joint surfaces glide together. With maximal extension, there is the potential for the articular surfaces to tip apart at their superior margins (Figure 4-25).

When thoracic dysfunction is treated with prone thoracic adjustments, it is common for the adjustive vectors to be delivered in a direction that approximates either the disc plane or the facet planes. The thrusts that parallel the disc plane are perpendicular to the spine and will likely induce forward translation of the contacted segments (Figure 4-26). This thrust is also likely to induce angular movements of extension at the contacted level as the superior and inferior segments move toward the shallow depression that is created by the forward translation of the contacted area (see Figure 4-26).⁴⁰⁸ There is also a possibility that gapping will occur in the facet joints superior to the point of contact resulting from forward translation of the contacted area and its superior facet.

In contrast, a thrust delivered P-A and inferior-to-superior (I-S) along the facet planes is commonly applied to induce more gliding distraction in the facet joint inferior to the point of contact (Figure 4-27). This approach is applied to induce the gliding movements that occur during segmental flexion. Therefore, the traditional approach is to direct the adjusting vector perpendicular to the thoracic spine (P-A), when treating a joint with decreased extension (flexion malposition) (see Figure 4-26) and more superior along the facet planes (P-A and I-S) when treating a joint with decreased flexion (extension malposition) (see Figure 4-27).

However, recent findings on the biomechanical properties of prone thoracic adjusting bring into question whether altering the P-A direction of adjustive vectors on prone stationary patients can effectively change the movement induced in the spine.³⁵ For example, can changing a prone adjusting vector from a perpendicular P-A orientation to a more P-A and I-S vector induce segmental flexion? Bereznick, Ross, and McGill³⁵ make a compelling

BOX 4-14 Questions and Factors in the Determination of Which Joints to Adjust

- Is the condition affecting one or multiple levels?
- Is the condition affecting one or both sides of the spine?
- Will one or multiple adjustments be needed?
- Site and side of subjective and palpable pain
- Side of reactive soft tissue changes (e.g., altered muscle tone)
- Site and direction of restricted or painful motion
- Site and directions of restricted end play or joint play

BOX 4-15 Factors Governing the Selection of and Specific Application of Adjustive Methods**ANATOMIC LOCATION OF JOINT DISORDER OR DYSFUNCTION**

Morphology of tissues: size, strength and mobility of structures
Some areas necessitate more power (mass and leverage).

PATIENT'S AGE AND PHYSICAL CONDITION

Ability to assume specific positions; degree of pretension (force, mass, leverage, and depth of thrust) the patient can withstand; stress to adjacent spinal or extremity joints and soft tissues

PATIENT'S SIZE AND FLEXIBILITY

Large or inflexible patient: need increased mechanical advantage in the development of pretension and thrust
Table selection: height, articulating vs. nonarticulating, release or drop pieces, mechanized
Method: leverage and type of thrust (e.g., push vs. pull)
Flexible patient
Focus force preloading of joint: removal of articular slack, use of non-neutral patient positions
Selection of method: shorter lever methods

PRESENCE OF MITIGATING DISORDERS OR DEFECTS

Pre existing congenital or developmental defects
Pre existing degenerative defects
Coexisting disease states
Adjacent motion segment instability (focus force minimize stress to adjacent joints)

DOCTOR'S TECHNICAL ABILITIES AND PREFERENCES

Patient treatment preferences
Cannot compromise safety and effectiveness

SPECIFIC MECHANICAL AND PHYSICAL ATTRIBUTES OF ADJUSTIVE METHODS**Adjustive Localization and Pretension**

Patient position
Doctor position
Contact points
Leverage

Adjustive Thrust

Leverage
Velocity
Amplitude (depth)
Mass
Point of delivery
Pause-nonpause

Short Lever Preferred to Long Lever

Issue of specificity
Patient of manageable size
Flexible patient
Patients with clinical motion segment instability

Long Lever Preferred to Short Lever

Spinal regions where additional leverage is desired
Patient size and flexibility demand additional leverage and power

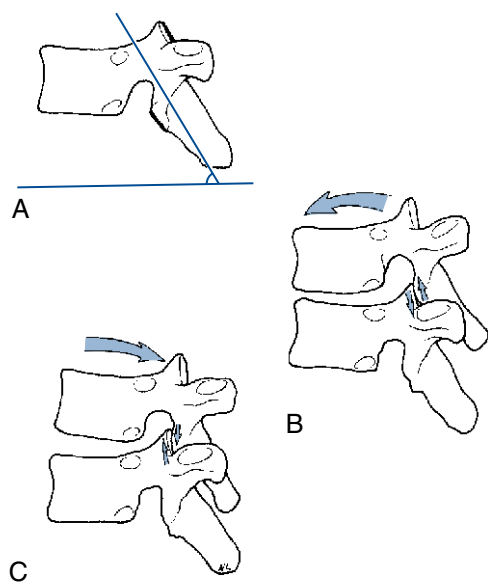


Figure 4-25 **A**, The thoracic facets lie at a 60-degree angle to the transverse plane. **B**, The facets separate and glide apart on flexion. **C**, The facets approximate and glide together with extension. With maximal extension, the articular surfaces may gap at their superior margins

case that challenges this assumption by demonstrating that surface adjustive contacts cannot establish fixed contacts on underlying bone, fascia, or muscle. They demonstrated that the interface between the superficial structures (skin and subcutaneous tissue) and the underlying bone, muscle, and fascia is essentially frictionless.³⁵ Therefore, any forces directed at the spine, other than perpendicular P-A forces, end up deforming and stretching overlying structures without adding any directional forces (i.e., flexion) to the spine. In this model, the more an adjustive force is directed away from a perpendicular (P-A) orientation to the spine, the less likely is a deformation and cavitation of the spine.

This emerging biomechanical research should lead the profession to question and further investigate some of its adjustive mechanics assumptions and clinical applications. If changes in prone thoracic vectors do not always induce the precise movements we anticipate, but are associated with a good clinical outcome, then perhaps it is not necessary to be precise with adjustive vectors in all circumstances. Maybe a P-A thoracic thrust that induces extension deformation of the spine and distraction in the facet joints is effective at mobilizing the spine in a number of directions. If this is the case, the profession can move beyond the frustrations of trying to demonstrate clinically reliable and valid

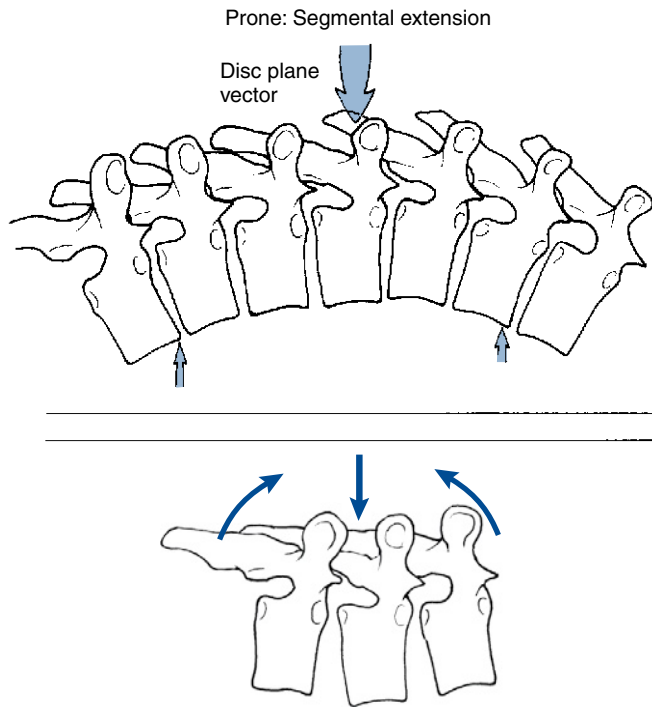


Figure 4-26 Effects of an adjustive force applied in a P-A direction along the disc plane.

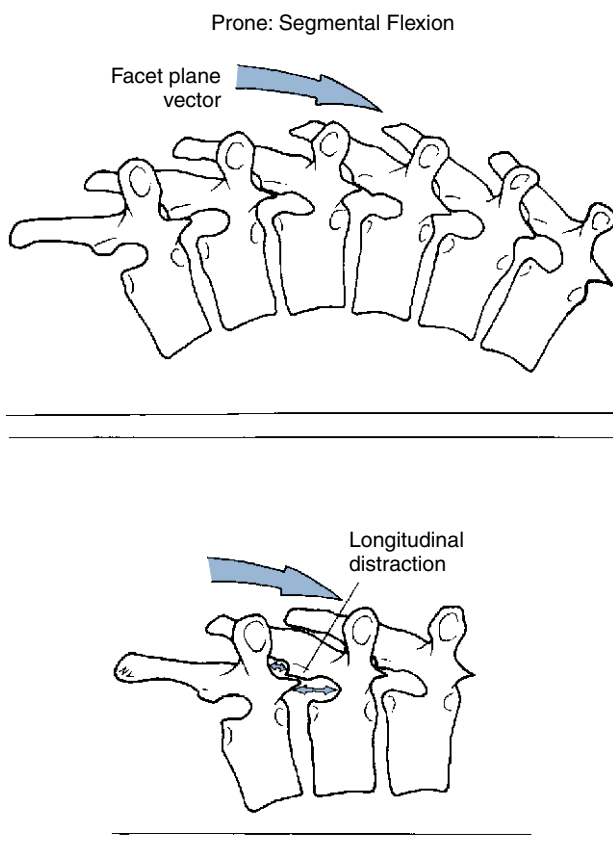


Figure 4-27 Hypothetical effects of an adjustive thrust applied in a P-A and I-S vector along the facet planes.

methods for determining precise levels and directions of spinal malpositions and restrictions. If the central clinically effective component of adjustive therapy is the production of spinal movement it also frees the clinician to deliver a potentially more effective prone thoracic adjustment. For example, the more forces are directed perpendicular (P-A) to the spine, the more likely they are to induce spinal movement. The more the vectors are directed I-S away from the spine, the more they are absorbed and dissipated into the superficial soft tissues.

On the other hand, spinal adjustive therapy may be less effective than it could be because we have not developed the understanding and adjustive tools to the level necessary to be precise and specific. Perhaps outcomes could be improved by furthering our understanding of adjusting biomechanics and the application of methods that could be counted on to produce specific movements and effects. If simply changing our vector in a prone neutral position is unlikely to induce any movement other than extension or rotation, are there other options available that will produce different effects? For example, can changing patient position produce different effects? If we maintain segmental flexion or lateral flexion at the spinal level of desired effect, will a prone adjustment be more effective at inducing the desired movement? Can a supine adjustment, with the patient maintained in a flexed position, produce more flexion? In the context of our present understanding, it seems reasonable to apply modifications in PP to try to effect different spinal movements and possibly improve outcomes. Whether these approaches generate different spinal movements and improved patient outcomes awaits further biomechanical and clinical research.

ADJUSTIVE LOCALIZATION

Adjustive localization refers to the preadjustive procedures designed to localize adjustive forces and joint distraction. They involve the application of physiologic and unphysiologic positions, the reduction of articular “slack,” and the development of appropriate patient positions, contact points (CPs), and adjustive vectors. These factors are fundamental to the development of appropriate preadjustive articular tension and adjustive efficiency. Attention to these components is intended to improve adjustive specificity and to further minimize the distractive tension on adjacent joints. The proper application of these principles should maximize the doctor’s ability to focus his or her adjustive forces to a specific spinal region and joint.

Physiologic And Unphysiologic Movement

Knowledge of the physiologic movements (normal coupled movements) of the spine and extremities is important in the process of determining how to localize and apply adjustive therapy. Localization of adjustive forces depends on an understanding of the normal ranges of joint movement and how combinations of movement affect ease and range of joint movement. Each spinal region and extremity joint has its own unique range and patterns of movement. Knowing the ranges and patterns of movement allows the doctor to know what combination of movements is necessary to produce the greatest range of movement and what combination is necessary to limit movement.

The spine can flex, extend, laterally flex, and rotate, but in combination, these movements can act to either limit or increase movement. Performance of movement in one plane limits movement in another plane; flexion of the spine limits the amount of lumbar rotation, and lumbar rotation limits the amount of flexion. An additional coupling of motion in a third plane can combine to further restrict or enhance the ROM. For example, the greatest range of combined lumbar rotation and lateral flexion is achieved if rotation and lateral flexion are executed in opposite directions and coupled with extension instead of flexion.

Combined movements that allow for the greatest total combined range are referred to as *physiologic movements*, and combined movements that lead to limited movement are referred to as *unphysiologic movements*. Right lateral flexion combined with left rotation and extension is an example of physiologic movement in the lumbar spine. Right lateral flexion combined with right rotation or flexion is an example of unphysiologic movement.

Unphysiologic movements bring the joints to positions of tension earlier in their ROM, limiting their overall ROM. Positioning sections of the spine in unphysiologic postures during the application of adjustive therapy is a strategy referred to as *joint locking*.⁴⁰⁹ Application of this procedure helps focus the adjustive forces to the affected region or joint and minimizes mobility at adjacent joints. When adjacent spinal regions are placed in unphysiologic positions, a block of resistance may be created superior or inferior to the joint to be adjusted, establishing earlier preadjustive tension. Joints placed in their unphysiologic positions have greater impact between joint surfaces, which may decrease the likelihood of parapsysiologic joint movement and gapping at those joints.

The region and motion segment to be adjusted is placed in the transition area between unphysiologic motion and physiologic motion or between sections placed in unphysiologic locking (Figure 4-28). The joints to be adjusted must have sufficient slack remaining so that the adjustive thrust may induce gapping or gliding within the joint's physiologic range. If an adjustive thrust is delivered against a joint placed in its close-packed position, there is a greater risk of inducing joint injury. Placing joints in unphysiologic positions may be especially valuable in circum-

stances in which clinical joint instability is suspect at adjacent levels.

Reduction of Articular Slack

Articular slack refers to the joint play (JP) present in all synovial joints and their periarticular soft tissues. Although it is a normal component of joint function, available slack should be reduced during or before delivery of an adjustive thrust to improve the likelihood of inducing joint cavitation. Reducing articular slack helps isolate tension to the specific periarticular soft tissues that may be limiting JP and impeding joint motion. The removal of articular slack and the development of preadjustive tension also help focus the adjustive thrust to the desired spinal level or extremity joint. The energy and force generated by adjustive thrusts may be dissipated into superficial soft tissue and adjacent articular soft tissue if preadjustive tension is not first established.^{410,411}

The doctor may reduce articular slack by passively distracting the involved spinal region or joint or by altering patient positions to move the joints from their neutral position toward their elastic barrier. Joint distraction induced by the doctor may be developed by the gradual transfer of body weight through the adjustive contacts or by directing tractional forces through the adjustive contacts. The degree of preadjustive tension is gauged by the doctor's sense of joint tension and by the patient's response to pressure. Excessive traction or compression of joints during the application of adjustive procedures can lead to jamming of joints, uncomfortable contacts, and patient splinting. It is common for chiropractic students to overdo articular slack reduction and preadjustive tension when first learning adjustive techniques.

Lighter contacts and less preadjustive tension are necessary when patient discomfort and splinting are encountered. Joints with limited mobility need less movement to reduce articular slack and are often adjusted closer to their neutral positions. Joints with greater flexibility usually necessitate patient positions that move the joint from neutral positions toward the elastic barrier.

Patient Positioning. Preadjustive joint tension and localization are significantly affected by patient placement and leverage. Localization of adjustive forces may be enhanced by using patient placement to position a joint at a point of distractive vulnerability. Locking adjacent joints and positioning the joint to be adjusted at the apex of curves established during PP enhance this process (Figure 4-29). Joint localization and joint distraction may be further enhanced if forces are used to either help (assist) or oppose (resist) the adjustive thrust. Assisted and resisted patient positions refer to principles involved during the adjustive setup and development of preadjustive tension.

Assisted and Resisted Positioning. The notion of applying assisted and opposing forces during the performance of manipulation was first described relative to thoracic manipulation by the French orthopedist Robert Maigne.⁴¹² In the chiropractic profession, Sandoz²⁴ was the first to describe similar terms. Sandoz proposed using the terms *assisted* and *resisted* to describe patient positions that either assist or resist side posture (SP) lumbar adjustive thrust.²⁴ Both methods are used to improve the localization of preadjustive tension. Their application is based on the mechanical

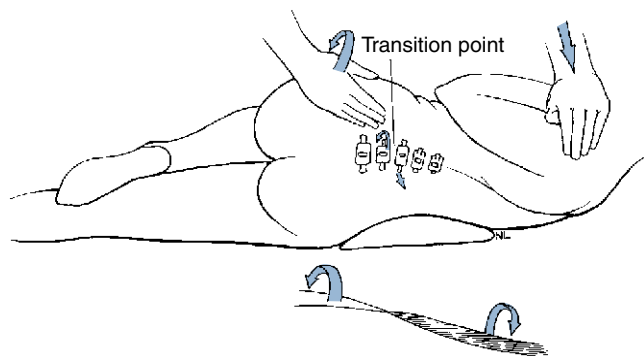


Figure 4-28 The joints above the level to be adjusted (L3 and L4) are placed in unphysiologic position (flexion, left rotation, and right lateral flexion) to develop locking of the joints above the level to be adjusted.

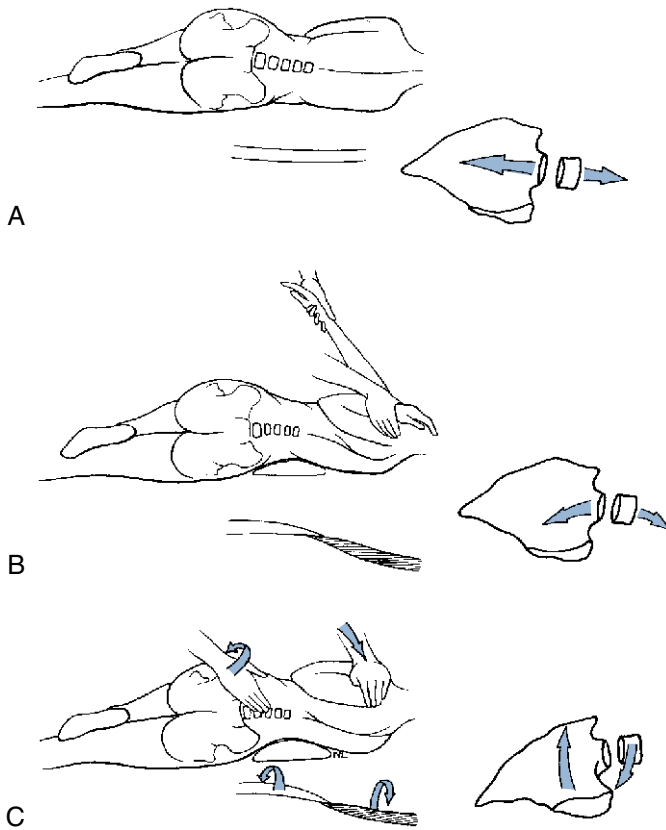


Figure 4-29 Proper patient positioning is necessary to develop appropriate preadjustive joint tension. **A**, Sagittal plane movement (flexion) and separation of the posterior element of the joint. **B**, Coronal plane movement (lateral flexion) and separation of the joint away from the table (left facet joints and disc). **C**, Transverse plane movement and development of counter-rotational tension and gapping of the left facet joints.

principle that the region of maximal tension will be developed at the point of opposing counter-rotation.⁴¹³

Assisted and resisted patient positions are distinguished from each other by the positioning of vertebral segments relative to the adjustive thrust. In both circumstances, the trunk and vertebral segments superior to the adjustive contacts are prestressed in the direction of desired joint movement. In the assisted method, the contacts are established on the superior vertebral segments, and movement of the trunk and the thrust are in the same direction (Figure 4-30, *B*). Resisted procedures use patient positions in which the segments superior to the adjustive contact are prestressed in a direction opposing the adjustive thrust. In the resisted method, the contacts are established on the lower vertebral segments, and the direction of adjustive thrust is applied opposite the direction of trunk movement (see Figure 4-30, *A*).

Sandoz²⁴ has suggested that resisted positions bring maximal tension to the articulations superior to the established contact (e.g., contact at the L3 mammillary inducing tension at the L2-3 motion segment and above) and assisted positions bring maximal tension to the articulation inferior to the established contact (e.g., L2 spinous contact inducing tension at the L2-3 motion segment and below). In the assisted method, the area of countertension is inferior to the point of contact because the inferior segments are

stabilized or rotated in a direction opposite the adjustive thrust (see Figure 4-30). In the resisted approach, the site of counter-tension is superior to the point of contact because the segments above the point of contact are rotated in a direction opposite the adjustive thrust (see Figure 4-30). Research by Cramer and co-workers²⁶⁵ has demonstrated that side posture-resisted lumbar mammillary push adjustments induce positional and postadjustment gapping in the articulations superior to the level of contact. In principle, either method can be used to induce the same joint motion within the same articulations. With assisted patient positions, the thrust is oriented in the direction of joint restriction; with resisted patient positions, the thrust is directed against the direction of joint restriction.

Assisted and resisted patient positions have been most frequently discussed relative to the development of rotational tension of the spine. In theory, the same methods and principles may be applied to treat dysfunction in lateral flexion or flexion and extension. To treat a loss of right lateral bending in the lumbar spine using the assisted method, the patient is placed on the right side with a roll placed under the lumbar spine to induce right lateral flexion. A contact is then established over the left mammillary of the superior vertebra, with an adjustive vector directed anteriorly and superiorly (Figure 4-31). To treat the same restriction with a resisted method, the same patient positioning should be maintained, but the left mammillary process of the inferior vertebra is contacted with a thrust delivered anteriorly and inferiorly (see Figure 4-31). Although both techniques are directed at distracting the left facet joints, one is assisting and the other is resisting the direction of bending.

To treat a loss of lumbar flexion with a side posture-assisted method, the patient should be placed on either side and segmental flexion induced, the superior vertebrae of the involved motion segment should be contacted, and the thrust should be anterior and superior. Conversely, without changing PP, the same restriction could be treated with a resisted method by simply contacting the lower vertebrae and thrusting anteriorly and inferiorly (Figure 4-32). The same principles described for flexion can easily be applied to treat an extension restriction, the only difference being the prestressing of the patient into segmental extension.

When applying side posture adjustive thrusts in the treatment of lateral flexion, flexion, or extension, it is typically less stressful to the doctor's wrist and shoulder to couple P-A thrusts with an I-S vector, as opposed to a coupled superior-to-inferior vector. The superior-to-inferior vector induces a posture of wrist extension and internal shoulder rotation that is uncomfortable and possibly injurious. Therefore, lateral flexion and flexion adjustments may be more safely and comfortably delivered with assisted patient positions and extension adjustments delivered with resisted patient positions.

The principles presented for assisted and resisted lateral flexion and flexion-extension side posture adjustments are potentially limited by the same biomechanical issues discussed previously relative to prone thoracic adjustments. Biomechanical research indicates that it is very unlikely adjustive contacts can establish effective tension with underlying bone, fascia, or muscle.²⁶⁹ In this context it seems unlikely that adjustive vectors directed superiorly or inferiorly will generate forces helpful in assisting in the production of lateral flexion or flexion-extension movements. It seems more plausible

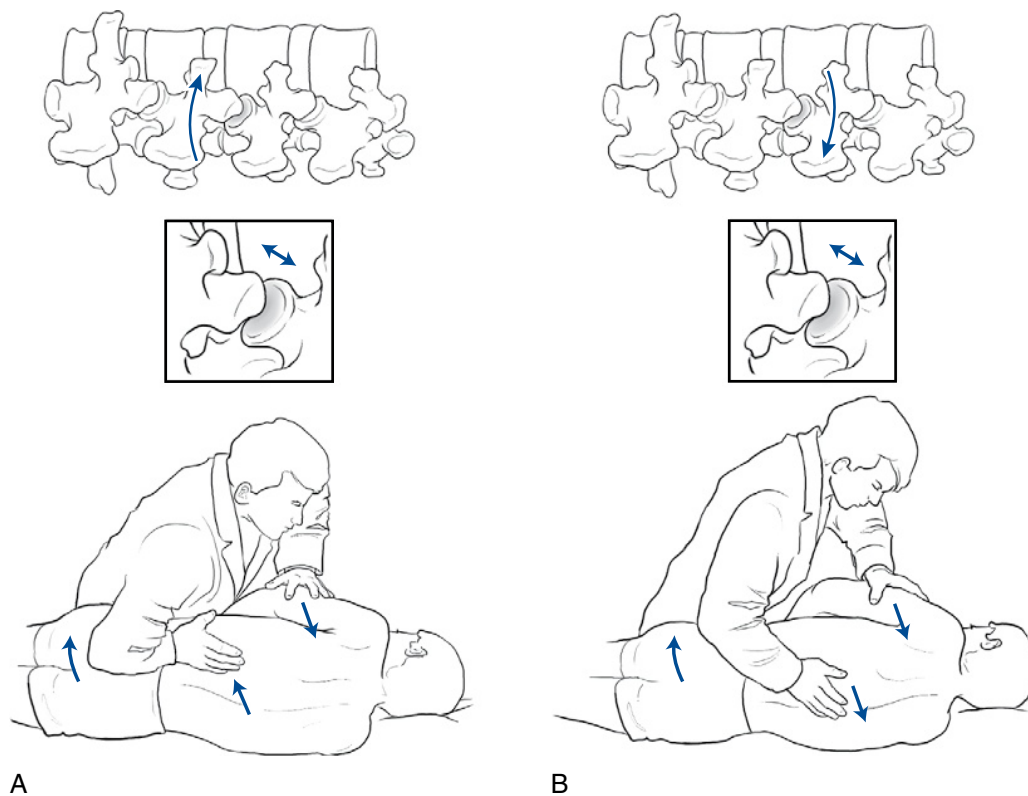


Figure 4-30 A, Resisted patient positioning with mammillary contact established on the inferior vertebra. B, Assisted patient positioning with spinous contact established on the superior vertebra. Both procedures are applied to produce left rotation.

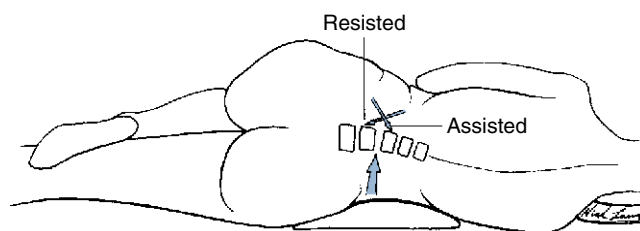


Figure 4-31 Adjustment for loss of right lateral flexion. With a resisted method, the contact is established on the left mammillary process of the inferior vertebra. The assisted method incorporates a contact established on the left mammillary process of the superior vertebra.

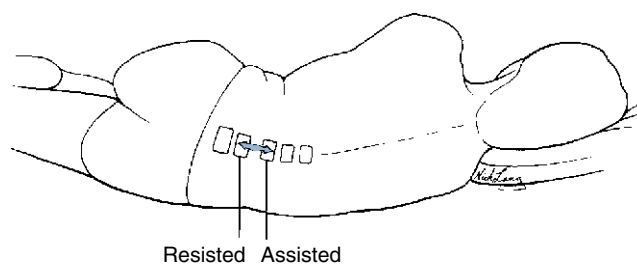


Figure 4-32 Adjustment for loss of flexion using resisted (inferior vertebra) or assisted (superior vertebra) methods.

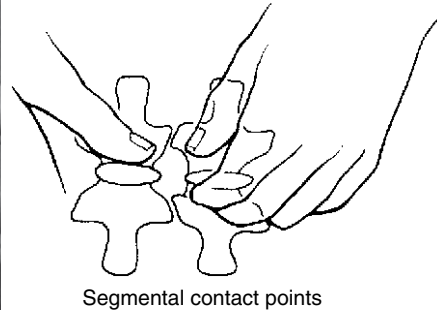
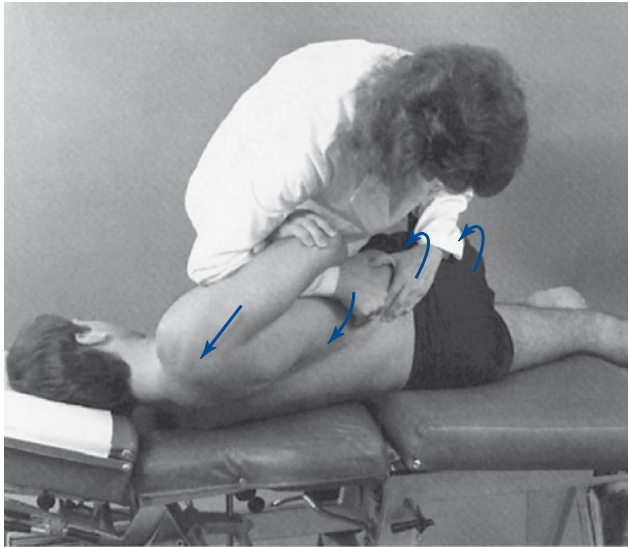
that attention to PP and factors that assist in deforming the spine in lateral flexion or flexion-extension would be potentially most effective.

Although the classification scheme of assisted and resisted patient positions is useful for contrasting different methods, it does create a possible void for those procedures in which both hands establish adjustive contacts and both deliver opposing adjustive thrusts. Counterthrust procedures, commonly applied during rotational spinal adjustments, do not conform to the strict definitions of assisted or resisted PP because these terms are defined relative to the delivery of one thrust, not two. In methods applying counterthrust techniques, both arms thrust; one arm establishes an assisted position and thrust as the other develops a resisted position and thrust. Based on the previous guidelines, they do not fit either category. To distinguish them

from single-thrust patient positions, we suggest referring to them as *counterthrust procedures* (Figure 4-33).

Neutral Positioning. *Neutral patient positions* refer to circumstances in which the patient and articulations are left in a relatively neutral position during the delivery of an adjustive thrust. Any preadjustive reduction of articular slack is established through the doctor's contacts without significant alterations in PP (Figure 4-34). Neutral positioning may be practical in some procedures such as prone spinal adjustive positions but impractical in others such as side posture rotational adjustments in which rotational leverage works to the doctor's advantage.

Principles of Patient Positioning. To take advantage of the potential increased specificity and efficiency that modifications in PP offer, the doctor must be aware of the various options available and the principles that underlie them. Although one approach is



Segmental contact points

**Figure 4-33** A counterthrust procedure applied to treat a lumbar right rotation restriction.**Figure 4-34** Prone thoracic bilateral thenar transverse adjustment applied to induce segmental extension with neutral patient positioning.

not necessarily superior to the other, each method has unique attributes that may make it more appropriate in certain circumstances. To make the appropriate distinction and effectively deliver adjustments, the doctor needs a clear understanding of each method's unique mechanical characteristics and differences. For example, a thrust delivered against the left L3 mammillary of a patient lying on the right side with shoulders in neutral may not have the same mechanical effect as in the patient whose shoulders are rotated toward the table into left rotation.

With the patient in the neutral position, the thrust against the left L3 mammillary is typically and traditionally applied to induce right rotation of L3 relative to L4 and the segments below (Figure 4-35). If the same thrust is delivered with the patient's shoulders rotated into left rotation (resisted position), maximal tension and cavitation may be induced in left rotation at the ipsilateral articulations above (L2-3 and superior). If the doctor wishes to induce right rotation at the L3-4 motion segment with an adjustive technique that involves shoulder counter-rotation and a mammillary contact, the patient should be placed on the opposite side (left) with a mammillary contact established at L4 instead of L3 (Figure 4-36).

Adjustive Specificity

Adjustive specificity describes the degree to which an adjustment is localized to a specific spinal region or joint. Historically the chiropractic profession has emphasized the value and application of methods believed to focus maximal effect in one joint. Application of the principles of PP and joint localization maximizes the potential for specific effects, but does not ensure that adjustive setups and thrusts will produce movement only at the desired level. The spine is a closed kinetic chain, making it highly unlikely that spinal movements can be induced at one joint at a time.^{264,269} Any adjustive thrust will have some effect on the other components of the three-joint complex and the joints superior to and inferior to the contacted vertebrae.²⁶⁴

The adjustive objective is not to eliminate all adjacent movement but to stress the skills that increase the probability of producing regional and focused joint cavitation while minimizing movement and tension at unwanted spinal regions and adjacent joints.

Ross, Bereznick, and McGill²⁶⁹ conducted some groundbreaking work evaluating the level of applied adjustment and the level of induced joint cavitation. They were able to localize the level of thoracic and lumbar joint cavitations by fixing accelerometers to the skin over the spinal column and measuring the relative time it took for cavitation-induced vibrations to reach each accelerometer. Lumbar adjustments produced multiple levels of cavitation in most cases (2 to 6). The average cavitation site was 5.29 cm off the target level (at least one vertebra away) with a range of 0 to 14 cm. In the thoracic spine, the average cavitation site was 3.5 cm off the target site, with a range of 0 to 0.95. Their research indicates that the tested procedures did not produce the frequency of targeted joint-level joint cavitations desired. Lumbar SMT was accurate approximately half the time. However, because lumbar adjustments were associated with multiple cavitations, at least one cavitation also typically emanated from the targeted joint. In the thoracic spine, SMT appears to be more accurate. Other studies evaluating cervical rotational adjustments and side posture lumbar

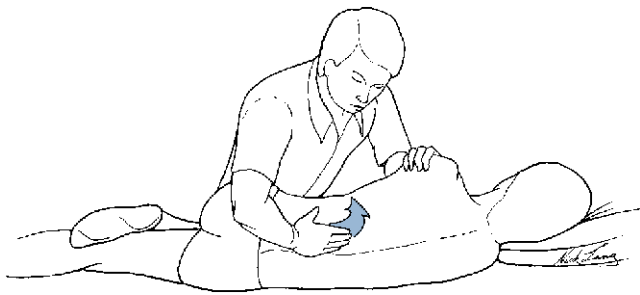


Figure 4-35 Assisted adjustment applied to induce right rotation of the L3-4 joint using an L3 mammillary contact and neutral patient position.

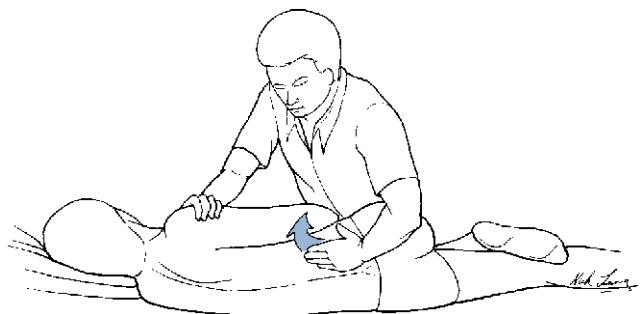


Figure 4-36 Resisted method applied to induce right rotation of the L3-4 joint. In this method, a contact is established on the right mammillary of L4, with the patient lying on the left side and the patient's shoulders rotated posteriorly to induce right rotation.

and SI adjustments also indicate less precision in producing cavitations to side or level to targeted joint.^{267,268}

Research evaluating HVLA adjustive specificity depends on the chiropractor's initial judgment of which joint is being targeted. This depends on the accuracy of the segmental contacts, the methods the chiropractor applies, and the biomechanical assumptions he or she has about the applied adjustment. The chiropractic profession has a history of assuming that adjustive contacts can be focused to one vertebra. It is also common to assume that the joint below the level of vertebral contact is the joint being targeted for treatment (adjusted). There are some presumptions in this model that seem improbable. First, it is unlikely that surface adjustive contacts can be precise enough to contact just one vertebra. In addition, surface contacts do not appear capable of hooking or binding to underline vertebra and individual vertebra.³⁵ As mentioned previously, individual vertebra are part of a closed kinetic chain, making it highly improbable that chiropractic adjustments can induce movement of a single vertebra.^{264,269}

It is also unlikely that all adjustive methods are uniform in their biomechanical effects and equal in their ability to focus adjustive forces. It is possible that some methods are more likely to affect joints above the level of contact or equally affect joints on either side of the contact level. For example, side posture-resisted mammillary lumbar and SI adjustments have demonstrated cavitations commonly occurring at levels above the contacted level.^{268,269} If chiropractors were to apply selected side posture lumbar methods with the intent of targeting joints above the level of adjustive contact, the specificity outcomes might be different. As our understanding of adjustive biomechanics deepens, our assumptions may

evolve and our concepts regarding adjustive specificity may change. It seems likely that HVLA adjustments will be viewed more in the context of being region-specific (several joints) rather than single-level-specific.

The emerging biomechanical information concerning the characteristics of HVLA adjustments raises the important clinical question of whether single-level localization of adjustive forces or cavitation is materially associated with clinical outcomes. What we say we do and what we really do may be two very different things. Although this information should initiate reevaluation of our clinical assumptions and possibly change our clinical approach, it must also be emphasized that it is biomechanical research and not clinical research. Clinical research is necessary to answer questions of clinical effectiveness. Basic science research cannot answer the question of which adjustive procedures are the most effective. They can guide the research, but answers to the questions of clinical effectiveness must be addressed with patient-centered clinical research. The likelihood that adjustments have a regional rather than a precise single-level effect does not diminish the demonstrated clinical effectiveness of chiropractic adjustive therapy. It is possible that the principles we apply to achieve joint specificity have a clinical effect and advantage not related to level of precise joint cavitation. Furthermore, much of the research on adjusting specificity is based on measuring the sites of joint cavitation, and it is possible that the sites of cavitation do not always correlate to the site of the focused adjustive force. It is possible that adjustive forces are relatively focused and yet induce cavitation at multiple sites or adjacent sites because the targeted joint is more fixed than adjacent joints. Adjustive therapy rarely reaches maximal clinical effect with several adjustments and, over time, the applied adjustments may start to induce cavitation at the targeted joint as it becomes more mobile and capable of cavitating. Clinical research comparing different adjustive approaches is necessary to determine if there are clinical differences or advantages to one approach versus another.

Research evaluating the premise that "specific" HVLA adjustments produce better outcomes has not been conducted. This research question cannot be clinically addressed until biomechanical evidence exists demonstrating that there are adjustments capable of producing a specific targeted effect. Up to this point, the overwhelming majority of clinical outcomes research on chiropractic adjustive treatment of mechanical spine pain has been conducted using standard approaches and methods that assume specificity does matter. The adjustments and vectors selected were applied with this principle in mind. Although the elements associated with different adjustive methods and vectors may produce better results, it is uncertain if this is a product of a localized specific effect. The outcome may have nothing to do with how a joint is moved or the precision of the level of effect. There are a number of possible clinical effects, and some may be sensitive to the direction adjustive forces generated and not germane to how the spine deforms and moves.

ADJUSTIVE PSYCHOMOTOR SKILLS

There is a wide range of adjustive procedures within the chiropractic profession; some are unique to the profession, and some are practiced by a wide variety of manual therapists. Each grouping of adjustments has its own mechanical characteristics that depend

on adjustive contacts, PP, doctor positioning (DP), and adjustive vectors. Efficient and effective selections cannot be made without an understanding of each adjustment's unique physical attributes. Several of the technique approaches are practiced as a package or system (see Appendix 1). They are often the product of clinical practice and usually include analytic procedures of assessment. It is not uncommon for chiropractors to limit their practice to primarily one of these many systems or approaches.

We believe that the adherence to one methodologic approach may be a disadvantage. A therapy or technique that works for one patient or problem may not work on a different problem or patient. An integrated approach that incorporates alternative technique approaches may provide effective options. Adjustive technique is a psychomotor skill that requires personal development and modification. Limiting alternatives to one approach may exclude techniques that fit the physical characteristics of the doctor or the patient.

Although some techniques differ dramatically, most thrust techniques share common basic mechanical characteristics and psychomotor skills. To effectively perform adjustive techniques, the chiropractor must have a foundation in these common principles and psychomotor skills. Each individual joint complex has specific anatomic and biomechanical considerations that affect adjustive therapy. As each spinal region and extremity joint is presented, the unique relationship between regional anatomy, biomechanics, and adjustive mechanics is discussed (see Chapters 5 and 6).

Patient Positioning

PP denotes the placement of the patient before and during the delivery of an adjustment. It is an essential component of effective adjustive treatment. It is a learned skill, which is often overlooked during the instruction and learning of adjustive technique. Proper attention to PP is critical to patient comfort and protection. Patients placed in awkward positions are apprehensive and unlikely to relax. Improper selection can leave the doctor at a mechanical disadvantage and in a position of increased risk of injury. The doctor is also vulnerable to injury as he or she assists patients in their positioning.

Whenever possible, the doctor should allow the patient to position himself or herself. The patient should be instructed on how to comfortably assume or modify his or her position on an adjustive bench. If it is necessary to assist a patient, the doctor should ensure that his or her back is in a stable position and that the patient is close to his or her center of gravity. Whenever possible, the doctor should use the power available in his or her legs to assist with lifting, pushing, or pulling movements.

As previously described, PP is critical to the development of joint preadjustive tension, adjustive localization, and efficiency. Adjustive localization and efficiency are products of adjustive leverage, preadjustive tissue resistance, and joint locking. All these factors in turn depend on PP. Increased tissue resistance and locking of adjacent joints are developed by inducing opposing forces through non-neutral PP. By positioning the joint to be distracted at the apex of secondarily established curves, joint distraction is increased, and the dysfunctional joint and spinal section is established as the area to receive the most distractive forces (see Figure 4-29).

There is a variety of postures available, each offering its own advantages and disadvantages. The selection of a specific position is governed by the specific mechanical features of each patient position, the clinical condition being treated, and the specific preferences of the doctor and patient. The standard PP options include prone, supine, standing, sitting, knee-chest, and side posture position. Within each adjustive description presented in Chapters 5 and 6, the PP section describes and illustrates the mechanics of PP, the type of adjusting table used, the position of the table's sectional pieces, and the appropriate use of any additional pillows or rolls. When indicated, the positioning of the extremities is described to ensure proper segmental tension.

Equipment Varieties and Management

The development of the equipment used by chiropractors and other practitioners of manipulation has taken place over time. Almost all procedures make use of a table or bench of some sort. The first chiropractic table had a flat, wooden surface atop ornate turned legs. It had no padding and no face opening, providing little comfort to the patient. It was not until 1943 that the first pad was designed for the adjusting table surface.⁴¹⁴ As new tables were developed, attention was paid to PP and the location of the clinician, providing for increased leverage and an advantageous adjacent stance.⁴¹⁵

A wide range of specialized adjusting tables and equipment is now available to enhance patient comfort and adjustive efficiency (Figure 4-37). Table options include flat benches, articulated tables, elevation tables, high-low (tilting) tables, knee-chest tables, manual and automatic distraction tables, and drop-piece tables. Some equipment is designed for the application of specific techniques, but most tables may be used with any of the common adjustive methods.

Regardless of the equipment used, some general habits should be developed. A doctor should select a table height advantageous to his or her physical attributes, use clean face paper on the headpiece of the adjusting table, and regularly apply a disinfectant to the table. The appropriate table height varies depending on the patient's size, the doctor's specific physical attributes, and the body area being adjusted. The average table height for pelvic, lumbar, and thoracic adjusting is the distance from the floor to the middle or superior

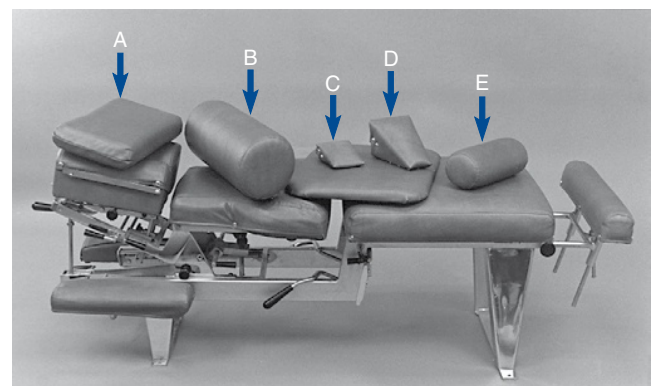


Figure 4-37 Specialized tables and equipment are used to enhance patient comfort and adjusting efficiency. **A**, Headrest pillow. **B**, Pelvic or Dutchman's roll. **C**, Dorsal or pediatric block. **D**, Pelvic block. **E**, Sternal roll.



Figure 4-38 A, Typical adjusting bench with brachial cut out. B, Pelvic bench. (Courtesy Lloyd Table Company, Lisbon, Iowa.)

aspect of the doctor's knee. For supine cervical adjusting, a higher table may be selected to minimize stress on the doctor's back.

Adjusting Bench. An adjusting bench (Figure 4-38) is a padded, nonarticulated, flat table with a face slot. It typically has a brachial cut out to allow comfortable placement of the patient's shoulders in prone positions. A pelvic bench is very similar to the standard adjusting bench. It is usually wider than the articulated adjusting tables and lacks the brachial cut out commonly featured on other adjusting benches (see Figure 4-38). The pelvic bench is useful for side posture or supine adjustive methods, but is uncomfortable on patients' shoulders in the prone position. The lack of articulated sections limits the ability of adjusting benches to modify patient positions and spinal postures. However, the use of wedges or cylindrical cushions are effective ways to achieve similar modifications in side posture or prone PP (Figure 4-39).

Articulated and Hydraulic Tables. An articulated table has movable head, thoracic, pelvic, and foot pieces to properly accommodate the patient in both the prone, side posture, and supine positions (Figure 4-40, A). High-low tables tilt from a vertical to a horizontal position, making it easier for a patient to get on and off the table (see Figure 4-40, B). Elevation tables have the ability to adjust to variable heights for different procedures as well as for different-sized doctors (see Figure 4-40, C).

When the patient is in the supine position on an articulating table, the headrest should be closed and elevated, and all other sections should be lowered to a level position. When performing cervical or upper thoracic adjusting, the headpiece may be slightly lowered. For prone positioning, to achieve a relaxed neutral posture, the footrest, pelvic, and thoracic sections should be elevated slightly, and the headrest should be lowered slightly.

Knee-Chest Table. The knee-chest table (Figure 4-41) gets its name from the position the patient assumes when on the table. The patient's chest and face are supported by a head and chest piece and the patient's knees rest on the padded base of the table. The chest piece should be situated so that the patient's spine remains parallel to the floor.⁴¹⁶ The lower thoracic and lumbar

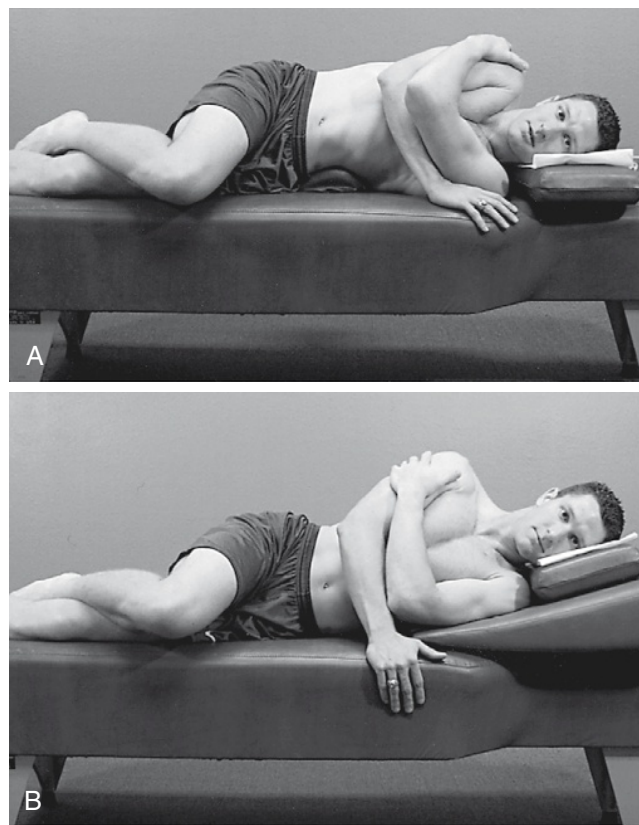


Figure 4-39 Use of rolls and wedges to modify preadjustive patient positioning. A, Use of a cylindrical roll to induce lateral flexion toward the table (right lateral flexion). B, Use of a wedge to induce lateral flexion away from the table (left lateral flexion).

spine are left in an unsupported and unrestricted position. It is this feature that provides the table's most unique and potentially effective attribute. In this position the doctor has the mechanical advantage to easily develop full adjustive pretension, especially into extension. Consequently, this table may be most effective when applied in the treatment of lower thoracic and lumbar extension restrictions. It has also been suggested for those patients with large abdomens for whom the prone position is uncomfortable. Patients beyond the first trimester of pregnancy may be more comfortable and have less anxiety in the knee-chest position than prone when having P-A thrusts applied to the lower back.

The attributes of the knee-chest table are also the features that contribute to its greatest inherent risk for hyperextension injuries. The risk of injury can be minimized by gently developing pretension and delivering shallow and nonrecoiling adjustive thrusts.

Although cervical, thoracic, and lumbar techniques can be performed in the knee-chest position, lower thoracic and lumbar dysfunctions are the areas more commonly adjusted in this position.

In a predicament, the knee-chest position can be approximated by having the patient kneel on a pillow at the head end of the traditional table with the face on the headrest and forearms on the armrests. The kneeling modification cannot duplicate the comfort and modifications available in a knee-chest table and should be used only in unusual circumstances.



Figure 4-40 Articulated and hydraulic tables. **A**, Stationary: (1) footrest, (2) pelvic section, (3) thoracic section, and (4) headrest. **B**, High-low with vertical to horizontal tilt. **C**, Elevation table to variable heights. (Courtesy Lloyd Table Company, Lisbon, Iowa.)

Some doctors and patients are quite apprehensive about knee-chest positioning. In such circumstances, an articulated table may be used to achieve a similar position. This may be accomplished by slightly raising the pelvic piece and allowing the thoracic piece to drop away.

Drop Tables. Mechanical drop pieces are available on any or all of the sections of an articulated table (Figure 4-42). Drop mechanisms allow for the elevation of sectional pieces and the subsequent free fall of those sections when sufficient adjustive force is applied against the patient. The drop sections elevate a fixed amount (approximately $\frac{1}{2}$ inch), but the degree of resistive tension



Figure 4-41 Knee-chest table.

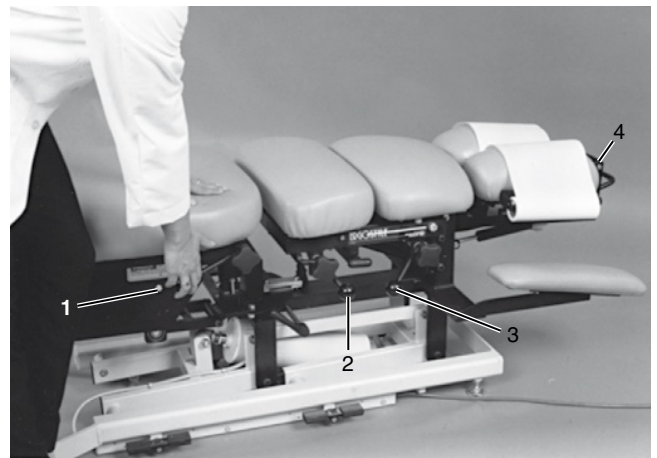


Figure 4-42 Mechanical drop pieces on stationary articulated table: (1) pelvic section cocking lever, (2) lumbar section cocking lever, (3) thoracic section cocking lever, (4) cervical section cocking lever. (Courtesy Lloyd Table Company, Lisbon, Iowa.)

varies. The amount of tension varies depending on the size of the patient, the extent of established preadjustive tension, and the force of the adjustive thrust. The degree of tension established in the drop mechanism should not be ascertained by thrusting against the patient. Tension should be determined by placing the patient on the table and thrusting against the table, not the patient.

Although no supporting clinical data exist, drop-piece mechanisms have been promoted as a technology for increasing adjustive efficiency. One position suggests that the degree of adjustive effort and force may be reduced because the drop of the table decreases the counter-resistance of the table and the patient. The other assertion is that the force of the adjustive thrust is enhanced by the counter-reactive force generated across the joint when adjustive thrusts are maintained through the impact of the drop piece.

Proponents of the first approach set low resistive tension on the drop mechanism and apply multiple light shallow recoil thrusts. The thrust is typically terminated before the drop mechanism has completely terminated its drop. In the second approach, resistive tension of the drop mechanism is increased to the point at which it can withstand the patient's weight and additional loading applied from the doctor as he or she establishes pretension.

The thrust is nonrecoil and maintained until the drop mechanism has terminated its drop. One of the potential disadvantages of the drop mechanism is the noise generated during the dropping action, which makes it difficult to perceive specific joint movement with the thrust.

Distraction Tables. The distraction table (Figure 4-43) offers a form of mechanical assistance for the application of manual therapy by having a fully movable pelvic section. The mobile pelvic piece provides a long-lever action that allows the lumbar spine to be positioned in or mobilized in flexion, extension, lateral flexion, or rotation, as well as the combined movement of circumduction.

Technique procedures applied to mechanical distraction tables commonly use a manual vertebral contact and either a manual or motorized mobile pelvic section to create distraction. Distraction tables can be used to evaluate spinal mobility, mobilize spinal articulations, or assist the doctor in the application of thrust techniques. Most chiropractic table manufacturers (e.g., Leader, Lloyd, Zenith Cox, Chattanooga and Hill) make a table that provides continuous passive spinal distraction. This motion is produced as the motorized pelvic section of the table rhythmically depresses toward the floor and back to a neutral position. Additional tension in rotation and lateral flexion can be added by prepositioning the table into the desired direction of rotation or lateral flexion. Some tables also provide the added feature of linear axial distraction, focusing on the long axis of the body (Figure 4-44).

When applying motion-assisted procedures for spinal joint dysfunction, the patient is typically positioned on the table so that the pelvis is on the pelvic section. All recumbent positions (prone, supine, and side posture) can be used. Because the use of linear distraction is considered an enhancement to the clinician's physical application, virtually all recumbent techniques can be performed. There are, of course, specific considerations for each joint to be adjusted, such as the segmental contact point (SCP), vector of thrust, and clinician position. Doctors should take caution not to use excessive flexion with segmental distraction; excessive flexion has the potential to overstretch the posterior joints and posterior portion of the IVD.

Cervical Chair. The cervical chair (Figure 4-45) is a padded chair with a movable backrest. The backrest is adjusted so that

the patient's spine remains straight and the area to be adjusted lies just below the doctor's forearm when the elbow is flexed to 90 degrees. The patient should sit with legs comfortably straightened and hands relaxed on the thighs. The cervical chair is used exclusively for adjustments applied to the cervical spine and upper thoracic spine.

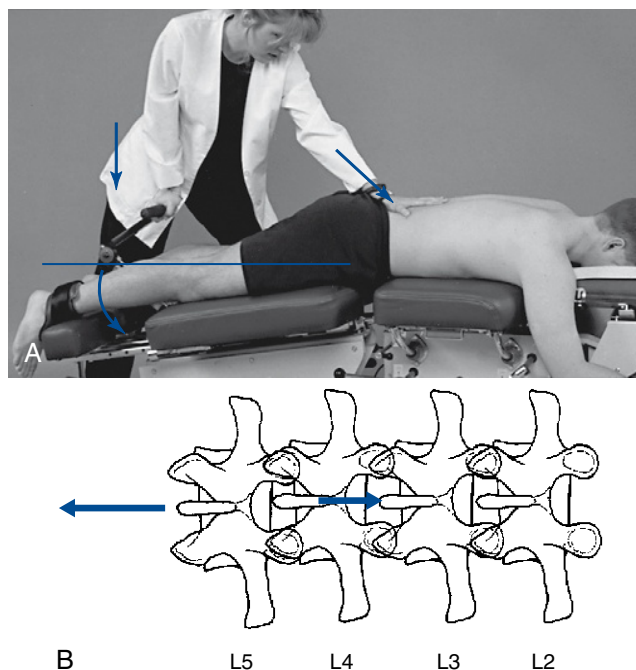


Figure 4-44 A, Flexion-distraction of L3-L4 motion segment. Patient is prone, with ankles strapped (optional). Clinician stands adjacent, in a lunge position (fencer stance), with the treating hand over the L3 spinous process and other hand on the handle of the pelvic section. Clinician depresses the pelvic section in a pumping action four to five times while maintaining cephalic pressure on the spine, then repeats the four to five pumps for one to two additional cycles, with a 30-second rest between. B, Diagrammatic representation of contact over the L3 spinous process, with the distractive vector shown.



Figure 4-43 Flexion distraction table. (Courtesy Lloyd Table Company, Lisbon, Iowa.)



Figure 4-45 Cervical chair.

Doctor Positioning

Chiropractic is a physically demanding profession associated with significant risk of occupational injury. Providing adjustive treatments subjects the doctor's spine and upper extremities to numerous stressful postures and repetitive movements involving pushing, pulling, twisting, bending, and lifting. A study was undertaken to determine the prevalence and types of work-related injuries among a random sample of chiropractors and to identify factors associated with these injuries.⁴¹⁷ Many chiropractors (40.1%) reported experiencing injuries while working. Most of those injuries were classified as soft tissue injuries and occurred while either performing (66.7%) or positioning (11.1%) a patient for manipulation. The clinician's body parts most commonly injured were the wrist, hand, and fingers (42.9%); shoulder (25.8%); and low back (24.6%). These injuries were most often related to side posture manipulation to the lumbar spine.⁴¹⁷ To avoid fatigue and injury, it is critical that DP involve sound body mechanics.

Good body mechanics start by selecting an appropriate table height to maintain a balanced and relaxed stance. If the table is too high, the doctor is at a mechanical disadvantage, unable to use the strength and leverage of his or her lower torso and legs. Instead, the doctor must rely on the strength of his or her upper body. Excessive dependence on the upper body can lead to underpowered adjustments and repetitive stress injuries to the upper extremities. If the table is too low, unnecessary stress may be applied to the doctor's back as he or she attempts to accommodate the height of the table. Accommodations to lower tables should be made by bending at the knees and hips and abducting the thighs, not by slouching with the trunk (Figure 4-46).

Whenever possible, the doctor should establish postures that maintain symmetric and neutral joint positioning. Delivering

thrusts through articulations positioned at end range or in close-packed positions places additional tension on the joint capsule and surrounding soft tissues. To perform safe and effective adjustments, the doctor needs to establish a stable kinetic chain through the spine and extremities. Core spinal stability and muscular bracing of the involved extremity joints are essential to the application of manual therapy and adjustments in particular. Common hazardous postures include excessive flexion and twisting of the trunk, excessive internal rotation and abduction of the shoulder, and unsupported extension of wrists.

Proper attention to DP applies equally to the cervical spine. Unfortunately, this region is frequently overlooked during discussion and presentations of adjustive technique. The doctor should maintain a stable neck position and avoid excessive cervical flexion to observe segmental contacts. Flexion of the neck encourages slouching of the upper back and excessive stress on the posterior soft tissues, and it weakens the stability of the neck and upper back (Figures 4-46 and 4-47).

Another critical element in the efficient and effective use of DP is the orientation of the doctor's center of gravity relative to the level of his or her adjustive contacts. The doctor's center of gravity should be placed as close as possible to the SCP and positioned so that his or her body weight can effectively be used to establish preadjustive joint tension (see Figure 4-47). The effective use of body weight (mass) can minimize the effort expended in developing preadjustive tension and in delivering an adjustive thrust. If the mass of the adjustive thrust is increased, force can be increased during the adjustment without increasing the velocity.^{410,411,418,419} Placing his or her center of gravity behind the line of drive (LOD) allows the doctor to transfer appropriate body weight into the adjustive set-up and thrust. Using body weight and leg strength saves energy for the adjustive thrust and minimizes the workload on the upper extremities. This helps minimize muscular effort and fatigue. As much as possible, the doctor's legs should bear the workload, thereby protecting his or her own back.

There are a number of named doctor stances used to describe the doctor's position during the delivery of adjustments. They commonly denote the position of the doctor's lower extremities and trunk in relation to the adjusting table and patient. Figure 4-48 illustrates two of the common stances; other modifications are discussed and illustrated in the regional sections on adjusting.

Contact Point

The CP designates which hand is the thrusting hand and the specific area of the hand that develops the focus of the adjusting contact. Attention to localizing a portion of the hand as the CP helps focus the adjustive force.^{258,260} However, it is also possible for adjustive contacts to be established too firmly on or near a bony prominence (e.g., pisiform). Excessively bony or penetrating contacts can prevent an adjustment from succeeding by generating unnecessary splinting and resistance from the patient. Uncomfortable contacts in the thoracic and lumbar spine may be associated with postures involving excessive extension of the wrist or arching of the hand. Uncomfortable contacts in the neck are often encountered when the lateral and bony edge of the index finger, rather

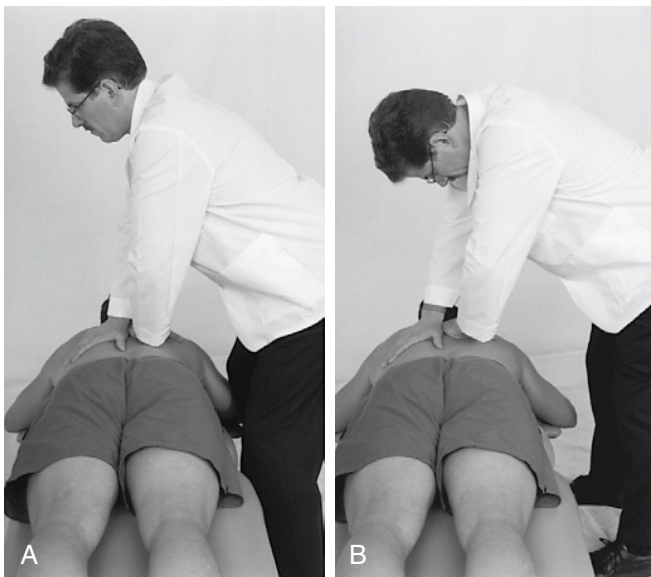


Figure 4-46 A, Illustration of sound body mechanics and doctor accommodating the table by bending hips and widening his stance and maintaining neutral spinal posture. B, Example of poor body mechanics illustrating the doctor excessively flexing his spine and slouching over the patient.



Figure 4-47 A, Illustration of poor side-posture doctor positioning. The doctor has dropped his head and upper back into excessive flexion and has positioned his torso and center of gravity to superior and anterior to his contact point. This results in ineffective use of body weight and a stressful position on the doctor's shoulder. B, Illustration of sound side-posture doctor positioning. The doctor's center of gravity and body weight are effectively positioned to reinforce the thrusting vector and establish a neutral and stable position for his shoulder.

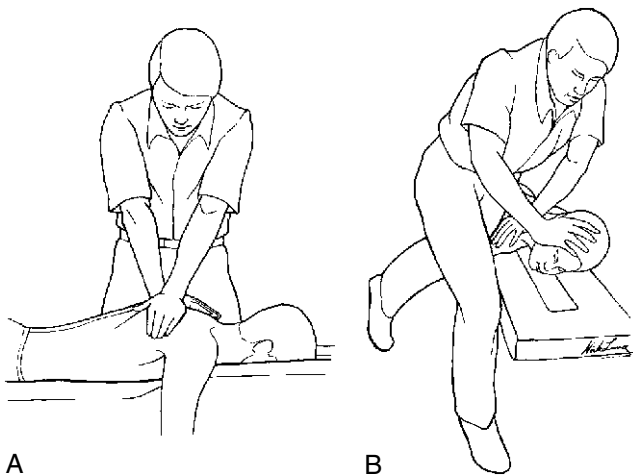


Figure 4-48 Two common doctor positions. A, Square stance: feet are parallel and aligned in the coronal plan. When accommodating a lower table, the doctor attempts to maintain a neutral spinal posture by widening the stance and bending at the knees and hips. B, Fencer's stance (lunge position): legs are separated at shoulder width or greater and angled to the torso. The knees are bent, and the doctor's back heel is off the floor. This position allows the doctor to efficiently transfer weight forward and inferior toward his front foot.

than the more padded palmar lateral surface of the finger, is used as the contact. The CP may be described anatomically or by a numbering convention developed to represent the common CPs (Figure 4-49). This text describes the contacts anatomically.

Indifferent Hand

The indifferent hand (IH) specifies which hand is used to stabilize the patient, fixate adjacent joints, or reinforce the contact hand. The points of patient contact and forces necessary to maintain positioning and stabilization are also presented within this category. The IH is not always passive during the delivery of an adjustment. There are circumstances in which the IH moves from the realm of stabilization into either an assisting or counter-resisting thrust. In such circumstances, both extremities deliver an adjustive thrust. In the illustrations throughout the text, when thrusting forces are delineated from stabilization forces, an arrow is used to demonstrate adjustive vectors, and a triangle is used to demonstrate stabilization points (Figure 4-50).

Segmental Contact Point

The SCP specifies anatomically where the adjustive contact or contacts are to be established on the patient. The SCPs are listed and described specifically in this chapter and in Chapters 5 and 6. When possible, they are illustrated in photographs or drawings. The SCPs are typically referenced as *bony landmarks*. This is intended to be illustrative and clarify the underlying focal point of the adjustive force (Figure 4-51).

Segmental contacts focused at specific bony landmarks cannot be established without contacting overlying or adjacent soft tissues. Adjustive contacts established at or near the level of the dysfunctional joint are referred to as *short-lever (direct) adjustments*. Adjustive contacts established at some distance from the level of the dysfunctional joint are referred to as *long-lever (indirect)*

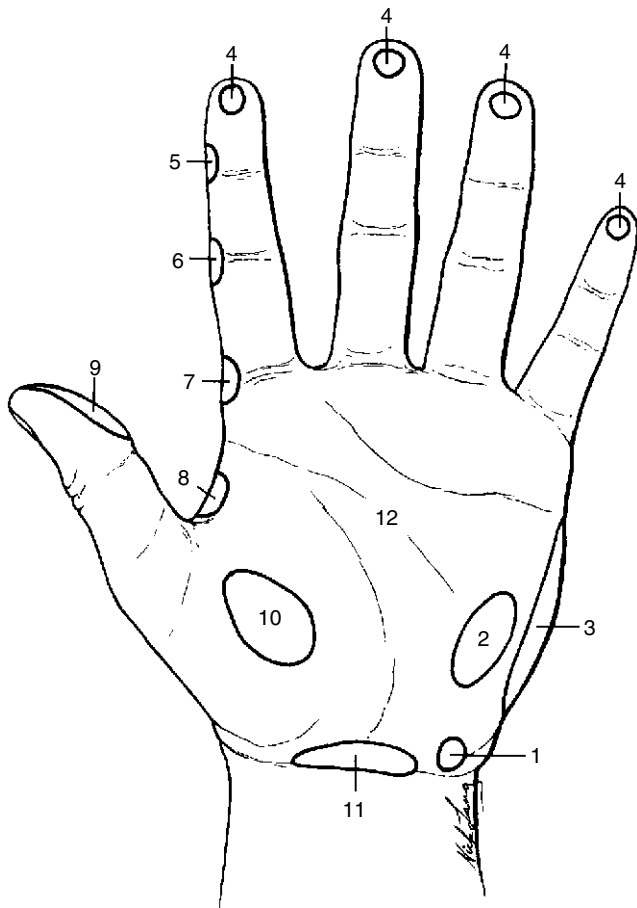


Figure 4-49 Contact points on the hand: (1) pisiform; (2) hypothenar; (3) metacarpal or knife-edge; (4) digital, used typically with the index and middle fingers; (5) distal interphalangeal; (6) proximal interphalangeal; (7) metacarpophalangeal or index; (8) web; (9) thumb; (10) thenar; (11) calcaneal; and (12) palmar.

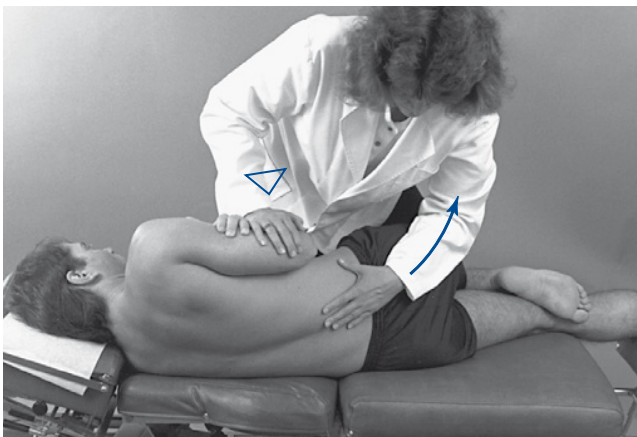
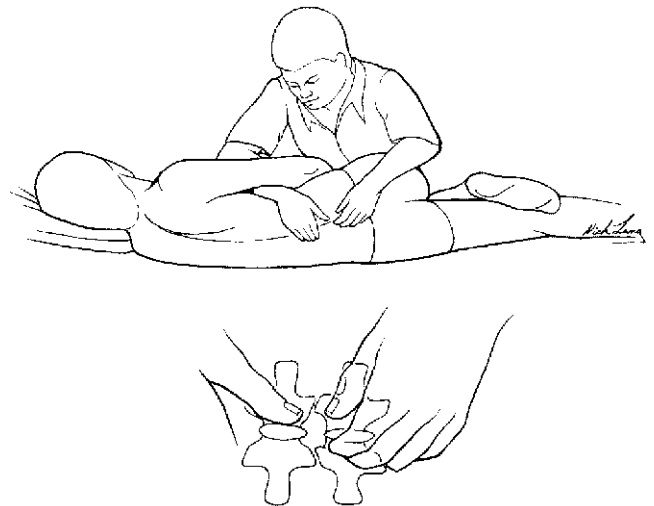


Figure 4-50 Arrows indicate adjustive vectors; triangle indicates stabilization.

adjustments, and adjustments that combine short- and long-lever contacts are referred to as *semidirect adjustments*.

In spinal adjusting, a single thrusting contact is conventionally taken on the superior vertebrae of the dysfunctional motion segment. Methods that incorporate thrusting contacts on the lower



Segmental contact points

Figure 4-51 Segmental contact points are bony landmarks located close to the joint(s) to be adjusted. In this illustration, segmental contacts are illustrated for a spinous push-pull adjustment. Bony landmarks are illustrative and clarify the focal area of contact. They are not meant to imply that contact points are limited to bony structures. Overlying and adjacent soft tissue structures are obviously also contacted

vertebra or both vertebrae of the involved motion segment are also effective and in common use. Contacts established on the lower vertebra of the dysfunctional motion segment establish a resisted method; contacts on the superior vertebra establish an assisted method; and contacts established on adjacent vertebrae establish a counter-resisted method (see Figure 4-51). Assisted and resisted methods are summarized in Table 4-3.

Hand contacts established near the level of desired adjustment are assumed to improve the specificity of the adjustment, and research indicates chiropractors are capable of developing an area of focused force within the broader area of their contact.^{260,261} Whether specific short-lever contacts are universally associated with more specific successful joint cavitations is in doubt. It appears that short-lever prone thoracic adjustments do induce relatively specific effects as compared with side posture lumbar adjustments.²⁶⁹ However, research indicates that employing a short-lever contact and thrust in side posture lumbar adjustments works against the doctor's ability to induce joint cavitation.⁴²⁰ The authors conclude that "successful generation of cavitation during side posture lumbar manipulation requires emphasizing forces to areas on a patient remote from the spine such as the pelvis and/or lateral thigh."⁴²⁰ Although focused short-lever contacts may produce a more local force in the spine, it is apparent that additional added points of leverage are necessary to induce sufficient lumbar axial rotation and cavitation.

If a local focused force is desired, then errors in the placement of adjustive contacts may lead to the localization of adjustive forces at undesired segmental levels. However, this does not imply that it is always desirable or possible to establish a segmental contact over a single vertebra. What is important is the ability to locate contacts in a manner that focuses the adjustive forces and desired movements in the joints or region to be adjusted.

TABLE 4-3 Comparison of Assisted, Resisted, and Counter-Resisted Spinal Adjustive Methods*

	Assisted	Resisted	Counter-Resisted
Segmental contact point	Superior vertebra	Inferior vertebra	Adjacent vertebra
Adjustive vector	Direction of joint restriction	Opposite direction of joint restriction	Superior contact: direction of restriction Inferior contact: opposite direction of restriction

*To be biomechanically efficient with adjustive methods, the patient positioning used should prestress the patient dysfunction joint(s) in the direction of the restriction of movement and the indicated level of contact taken. The patient can remain in the neutral position and still have these principles apply.

A number of the adjusting methods used by chiropractors involve close physical contact between the patient and the doctor. The nature of this contact, if not properly explained, can lead to misunderstandings and complaints of inappropriate touching. It is paramount that doctors explain the procedures they are going to use and receive permission to proceed before applying treatment. Explanation of procedures is essential, followed by the questions “Do you understand?” and “Is it okay?” These give the patient an opportunity to question or refuse treatment.

The chiropractic educational process demands the development of highly perfected manual palpation and therapy skills. Students learn these skills by voluntarily practicing on each other. In the process there tends to be a desensitization to touch, disrobing, examination, and treatment procedures through familiarity. However, naïve patients will not feel that familiarity. Therefore, it is important to be attentive to procedures that chiropractors may take for granted but that patients may look on in an entirely different manner. Casual and unconscious contact with sensitive body parts may go unnoticed by the practitioner but not by the patient. Doctors must be mindful and aware of the potential to inadvertently touch sensitive areas during the application of adjustive procedures.

Methods to be especially conscious of include supine thoracic adjustments and side posture lumbar or pelvic adjustments. During supine adjustments, unwanted contact between the doctor and the breasts of the female patient or between the breasts

of the female doctor and the patient can become an issue. This can be minimized by placing a small pillow or roll between the patient’s breasts and arms or between the doctor and the patient’s arms (Figure 4-52). In side posture adjustments, inadvertent contact between the doctor’s genitals and the patient’s thigh can occur. This can easily be avoided if the doctor is simply aware of this potential and positions himself or herself accordingly.

Any examination or treatment procedure performed on a member of the opposite sex that involves exposure or contact with the genitals or rectal region should be performed only when an assistant is in the room. The internal mobilization or manipulation of the coccyx is an example of a procedure for which this is warranted.

Tissue Pull

Superficial tissue traction (pull) is typically applied during the establishment of an adjustive contact. Proper tissue pulls are necessary to ensure that a firm contact is established before a thrust is delivered. If this is not taken into consideration, the CP may slip during the thrust and dissipate the adjustive force into superficial soft tissues and decrease the doctor’s ability to impart a force to the spine. The IH may be used to draw the tissue slack as the CP is established. Tissue pulls are commonly initiated in the direction of the adjustive thrust and, as such, will not be listed separately. Prone patient P-A thrusts are a common exception. In this circumstance the direction of tissue pull is often irrelevant. Tissue

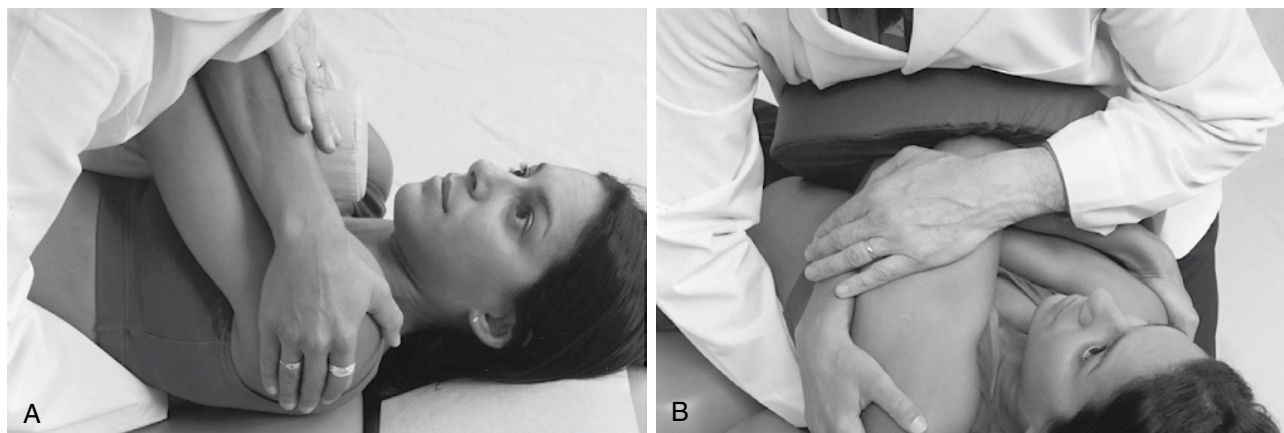


Figure 4-52 A, Supine thoracic adjustment in the midthoracic spine illustrating the use of a small roll to pad the patient’s anterior chest. B, Use of a rectangular pillow to minimize contact between the doctor’s anterior chest and patient.

pulls up or down the spine are appropriate and are applied to prevent the doctor's contacts from sliding. The direction of tissue pull is based on the region of the spine and the doctor's preference.

Vector (Line of Drive)

The vector, or LOD, indicates the direction of the adjustive force (thrust). Historically, the profession has described the direction of adjustive thrusts in anatomic terms. For example, an adjustive vector delivered with a patient in the prone position with a ventral and cephalic orientation is described as a P-A vector and an I-S vector. This text adheres to this standard and illustrates the direction of adjustive vectors in drawings and pictures with the aid of solid arrows (see Figure 4-50).

Attention to alignment is necessary to ensure anatomically sound, specific, and efficient adjustments. To produce joint distraction and movement without producing injury, the doctor must have knowledge of the functional anatomy and kinematics and match the adjustive vector accordingly. Misguided Vs may lead to unwanted joint compression, joint tension, ineffective dissipation of forces, or joint cavitation at undesired levels. A single adjustive thrust and cavitation may not free multiple directions of joint restriction.⁴²¹ Therefore, at times, a single articulation may be adjusted in multiple directions, with different adjustive Vs applied for each adjustive thrust.

Thrust

The *adjustive thrust* can be defined as the application of a controlled directional force, the delivery of which effects an adjustment. The *adjustive vector* describes the direction of applied force; the *adjustive thrust* refers to the production and implementation of that force.

The adjustive force is typically generated through a combination of the practitioner's muscular effort and body weight transfer. The chiropractic adjustive thrust is a ballistic HVLA force designed to induce joint distraction and cavitation without exceeding the limits of anatomic joint motion.

The thrust is the adjustive component, which, if delivered incorrectly, carries the greatest risk of patient injury. Adjustive thrusts performed with too much force, depth, or pretension carry the risk of exceeding the limits of physiologic joint movement. It takes extensive training and time to perfect adjustive skills and the ability to sense and control the appropriate depth and force of an adjustive thrust. This skill cannot be effectively learned over the course of a few months or by attending weekend courses. Chiropractors have devoted years of training to refine their manipulative skills, and in the hands of skilled practitioners, manipulation carries a very low rate of complication.

There is a critical adjustive force that must be supplied by the doctor to bring a synovial joint to cavitation and influence its structural and functional relationships. The development of this force depends on a multitude of factors, including stiffness and elasticity of the joint and patient, the proportion of impacting energy entering the joint and patient, and the amount of joint distraction at which cavitation takes place. These parameters are governed by numerous properties of the patient, the doctor, the joint, and the adjustive process.³³⁵

The average adjustive force produced by spinal manipulation can be expressed in terms of the impact kinetic energy (mass and velocity) of the clinician and the combined mechanical resistance to deformation (stiffness and elasticity) of both clinician and patient.³⁴³ This necessitates acquiring reflex contractile speed and stabilizing contractions of specific muscles (frequently the triceps and pectorals), as well as having enough applied leverage and body mass. It is thought that mechanical assistance can be used to augment these physical attributes.

The advantage of leverage and use of the doctor's body mass to induce lumbar joint cavitation is illustrated by recent research that demonstrates that dropping the doctor's body weight through CPs established on the patient's posterior pelvis or lateral thigh is necessary to induce lumbar cavitation.⁴²⁰ The authors concluded that "successful generation of cavitation during side posture lumbar manipulation requires emphasizing forces to areas on a patient remote from the spine such as the pelvis and/or lateral thigh."⁴²⁰

The use of preadjustive tension can limit the dissipation of thrust energy that occurs because of damping forces. Preloading the joint limits further motion during the thrust so that force and energy are not lost to other areas.⁴¹⁰ Use of preliminary distraction means that the thrust has to supply only the remainder of the force necessary for joint cavitation, diminishing the physical requirements of the clinician. Therefore, the resulting enhanced efficiency facilitates a more gentle adjustment⁴¹⁹ with less exertion by the clinician.

If preadjustive tension or countertension can be produced through a mechanical device (adjusting table), theoretically even less force, speed, and energy will be required from the clinician. There are manual and motorized mechanical assistance components to adjusting tables. One such modification is the drop-section mechanism, representing a form of manual mechanical assistance. Another modification is a moving table section, representing a form of motorized mechanical assistance.

Adjustive thrusts may be delivered in a variety of ways. Some of the common distinguishing attributes include the physical means the doctor uses to deliver the thrust (e.g., arm-centered thrust vs. body-centered thrust) (Figure 4-53), the positioning of the joint when the thrust is delivered (e.g., in a neutral position compared with a point near the joint's end ROM), and whether the adjustment is delivered with or without an active recoil⁴²² or whether the thrust is delivered with a postpretension pause or nonpause.

Adjustive thrusts are not always manually delivered. A number of mechanical thrust devices have been developed. Some are designed for hand-held application (Figure 4-54), and others are simply positioned by the doctor and do not require the doctor to hold the instrument during the application of the thrust. Whether these devices produce the same physical and therapeutic effects as manual thrust techniques remains untested.

Recoil Thrust. The recoil thrust involves the application of an HVLA ballistic force, characterized by the delivery of an active thrust coupled with a passive recoil. The recoil thrust is produced by inducing rapid elbow extension and shoulder adduction, followed by passive elbow flexion. The active thrust is induced by simultaneously contracting the pectoral muscles and extensor muscles of the elbows. The recoil is induced by rapid cessation of

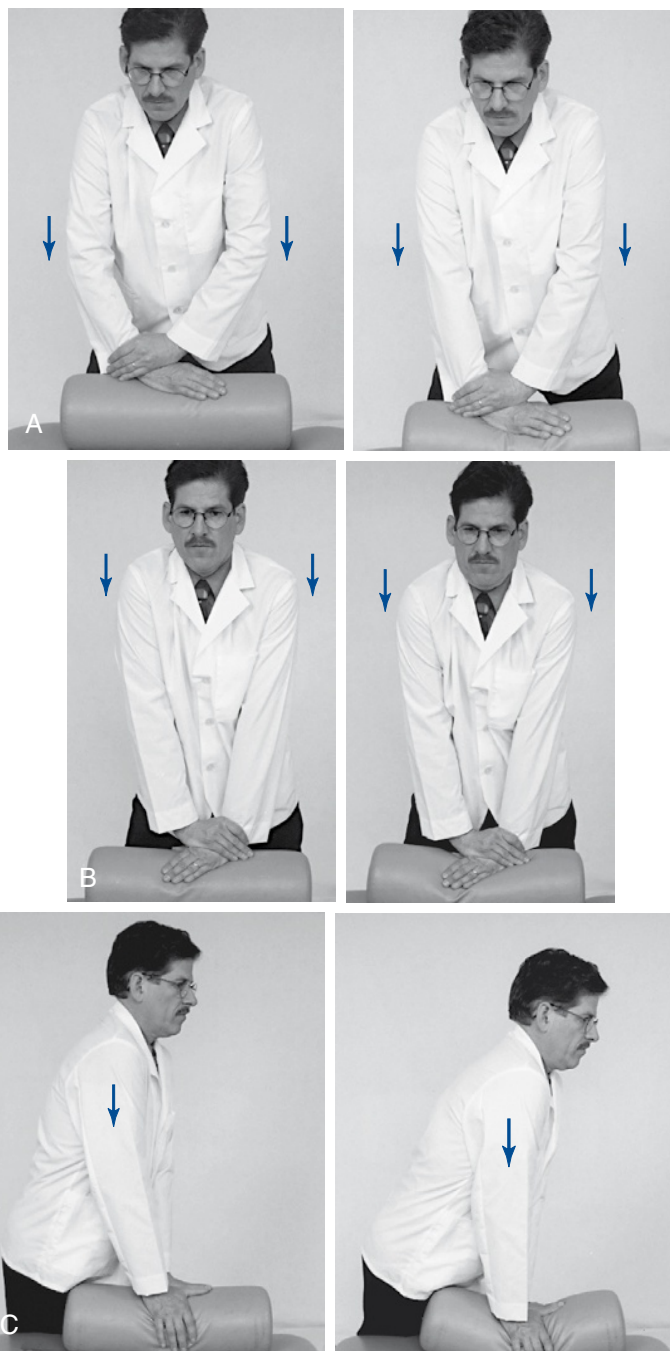


Figure 4-53 A, Illustration of recoil thrust. The body is held in stationary position, and thrust is generated by rapid acceleration at the elbows. The thrust is very shallow, with a quick termination, followed by an elastic recoil as full elbow extension is reached. B, Illustration of shoulder-drop thrust. The body is held in a stationary position, and the thrust is generated through a quick depression (elongation) at the doctor's shoulder. The doctor maintains a light contact after terminating the thrust to dampen reverberations generated by the shoulder thrust. C, Illustration of body-drop thrust. The thrust is generated by acceleration of the doctor's body weight through the adjustive contact. Transfer of body weight is generated by transferring weight from the doctor's heels toward the front of his feet. This is usually accomplished by inducing slight ankle dorsiflexion, knee flexion, and flexion of the trunk. Body-drop thrusts are often combined with shoulder-drop thrusts to produce a more rapid and rigid thrust and kinetic chain.



Figure 4-54 Prone manually assisted instrument (activator) adjustment illustrating sacroiliac (SI) joint application.

the thrust and the elastic rebound that results from impact with the patient and the stretch of the doctor's arms.¹⁸

This method typically involves establishing a segmental contact with one hand while the other hand reinforces the contact hand. Both arms thrust equally during the delivery of the adjustment; the vector is determined by the orientation of the doctor's episternal notch relative to the CP. This thrust is most commonly delivered with the patient in a relaxed position, with neutral joint positioning and little or no joint prestressing (see Figure 4-53).

Impulse Thrust (Dynamic Thrust). Impulse thrusts also use an HVLA force but are performed in a manner to minimize the normal elastic recoil that occurs after the quick cessation of an adjustive thrust. This is accomplished by maintaining mild pressure and contact with the surface for a short time after the termination of the adjustive thrust. The adjustive velocity may be varied, with either a slow or fast termination.

Impulse thrusts are most commonly delivered with the affected joint prestressed to reduce articular slack, but they should not be delivered with the joint stressed beyond its elastic limits. Impulse thrusts may be primarily arm centered or body centered, or their forces may be combined through the doctor's arms and body.

All adjustive thrusts involve relatively high-velocity forces, but vary in the degree of associated body weight coupled with the adjustment. When less mass and total force are desired, the thrust is typically delivered only through the upper extremities. This is commonly the case in the adjustive treatment of the cervical spine and small extremity joints and in the treatment of pediatric, geriatric, or frail patients.

During the delivery of arm-centered thrusts, the doctor's torso is stationary. The adjustive force is produced by the initiation of pushing, pulling, or rotation forces generated through the doctor's

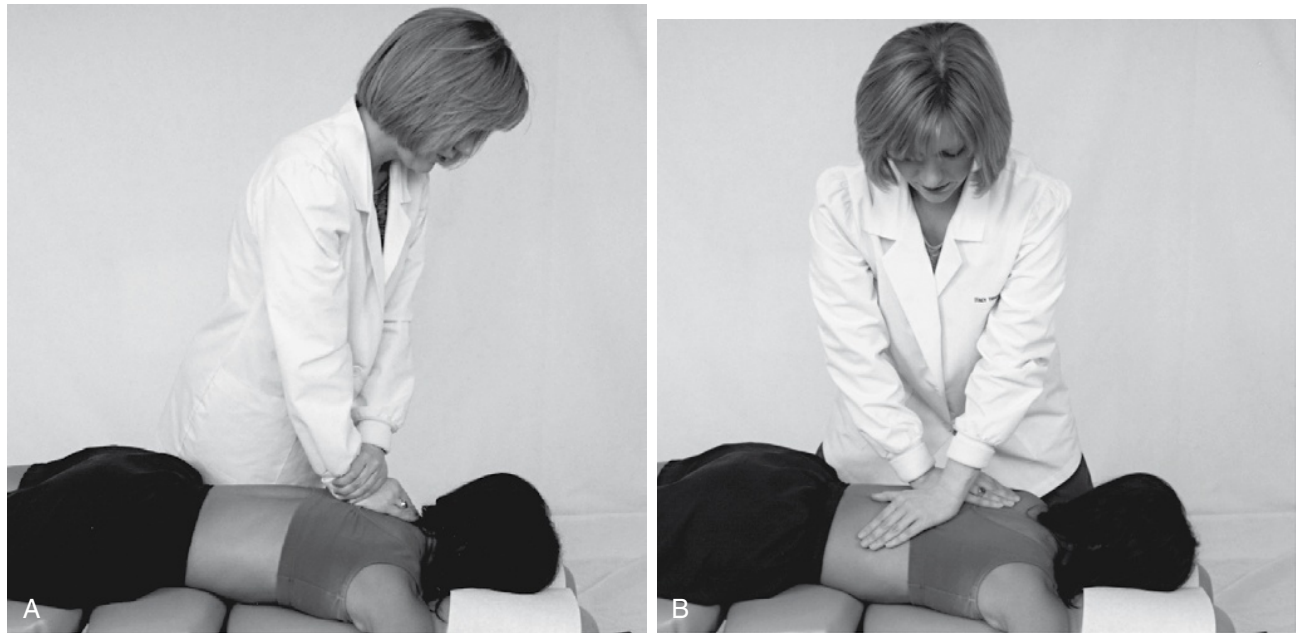


Figure 4-55 A, Prone unilateral hypothenar transverse push adjustment delivered to induce right rotation. B, Prone crossed bilateral hypothenar transverse counterthrust adjustment delivered to induce right rotation.

forearms, elbows, and shoulders (see Figure 4-53). Arm-centered thrusts may be delivered through one arm or both arms. When one arm is the focus of the adjustive force, the other arm (the IH) either reinforces the contact or stabilizes the patient at another site. When used for stabilization, the IH maintains the patient in a neutral position or induces positions or forces that assist or resist the adjustive force (Figure 4-55).

When more total force is desired, the doctor transfers additional weight from the trunk or pelvis into the adjustive thrust. In body-centered (body-drop) thrusts, the majority of the adjustive force is generated by propelling the weight of the doctor's trunk through the adjustive contacts (see Figure 4-53). This is accomplished with a quick and shallow flexion of the doctor's trunk and lower extremities, along with a simultaneous contraction of the abdominal muscles and diaphragm. Schafer and Faye⁴²¹ have described the abdominal and diaphragmatic contractions as a process similar to the event that occurs during sneezing.

During the delivery of body-drop thrust, it is critical that the upper extremities remain rigid. If the joints of the upper extremity give way during the delivery of an adjustive thrust, the adjustive force is dissipated. Rigidity is ensured by locking the upper extremity joints and by combining the trunk acceleration with a simultaneous shallow thrust through the upper extremities.

Adjustments delivered with prone patient position may be delivered as pure body-drop procedures, pure arm-centered thrusts, or combined body drop–arm thrusts. Lumbar and pelvic side posture adjustments, which commonly demand more total force, invariably involve the transfer of trunk and pelvic weight along with a simultaneous arm thrust. To transfer the additional body mass to the patient, the doctor typically establishes additional contacts along the lateral hip or pelvis of the patient (Figure 4-56, A).

A common technique variation in side posture lumbar adjusting couples a segmental contact with a reinforcing thrust through the doctor's leg. Instead of the doctor's weight resting against the patient's upper thigh and hip, a contact on the patient's knee is established. The impulse is then delivered by combining a pulling impulse through the arm with a quick extension of the doctor's knee (see Figure 4-56, B). In this method, the leg provides the additional leverage and force instead of the doctor's body weight.

Nonpause Thrust. After the removal of articular slack, a thrust may be delivered with or without a pause. When the thrust is performed without a pause, the slack is removed, and the thrust is delivered by accelerating and thrusting at the point of appropriate tension. An illustrated example is a wave crashing on the beach; removal of slack relates to the wave rolling toward the beach, and the thrust corresponds to the wave breaking against the shoreline. This approach is effective in maintaining adjustive momentum and avoiding patient guarding.

Pause Thrust. When thrusts are performed with a pause, the doctor takes a moment to assess the degree of established joint tension and tissue resistance before thrusting. This allows testing of the set-up and evaluation of the patient's responses to tension and pressure. If sufficient articular slack has not been removed or if abnormal binding induces patient discomfort, the doctor may modify the degree of preadjustive tension or the adjustive vector before applying the thrust.

After the pause, the doctor typically raises his or her trunk off of the patient slightly to regain momentum and accelerate his or her body weight into the thrust. During this process it is critical to maintain the majority of established preadjustive tension through the hands and points of secondary contact. The slight reduction in joint tension may aid the doctor in ensuring that the thrust is directed at the area of restriction and not too deeply into the

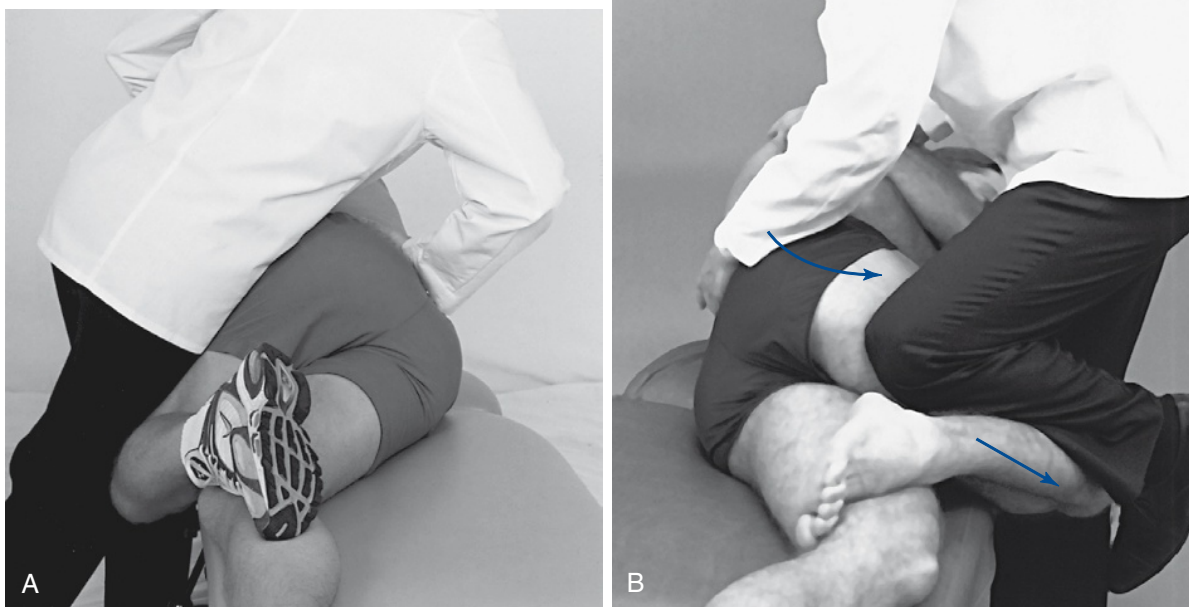


Figure 4-56 A, Side-posture resisted mammillary push adjustment with a thigh-to-thigh contact delivered to induce left rotation. B, Side-posture resisted spinous pull adjustment with a shin-to-knee contact delivered to induce right rotation.

joint's anatomic limits. However, if the doctor loses too much pre-tension, the adjustive force can dissipate and become nonfocused and uncomfortable.

Assisted, Resisted, and Counter-Resisted (Thrust) Methods.

Assisted methods incorporate contacts established on and above the superior vertebrae of the dysfunctional motion segment. They are applied to focus the adjustive force in the joints inferior to the level of segmental contacts. Assisted patient positions are incorporated if modifications in neutral PP are used. The adjustive Vs are directed to produce movement of the superior vertebra relative to the inferior vertebrae in the direction of joint restriction (direction opposite malposition).

The adjustive thrust may be focused through a single segmental contact or incorporate additional contacts and reinforcing thrusts applied at levels superior to the segmental contact. Figure 4-57 illustrates the application of a short-lever method incorporating a single level of focused thrust. Figure 4-58 illustrates a method incorporating a segmental contact coupled with a superior hand contact established on the patient's ipsilateral forearm. In this example additional leverage is provided through the superior contact, and both arms thrust to induce movement in the direction of joint restriction.

Resisted methods incorporate segmental contacts established on and below the inferior vertebrae of the dysfunctional motion segment. They are applied to focus the adjustive effect in the joints superior to the level of segmental contacts. Resisted patient positions are incorporated if modifications in neutral PP are used. The adjustive Vs are directed to produce movement of the joint in the direction of restriction (direction opposite malposition). This is accomplished by moving motion segments inferior to the dysfunctional joint in the direction opposite the joint restriction. Research by Cramer and colleagues²⁶⁶ has demonstrated that side

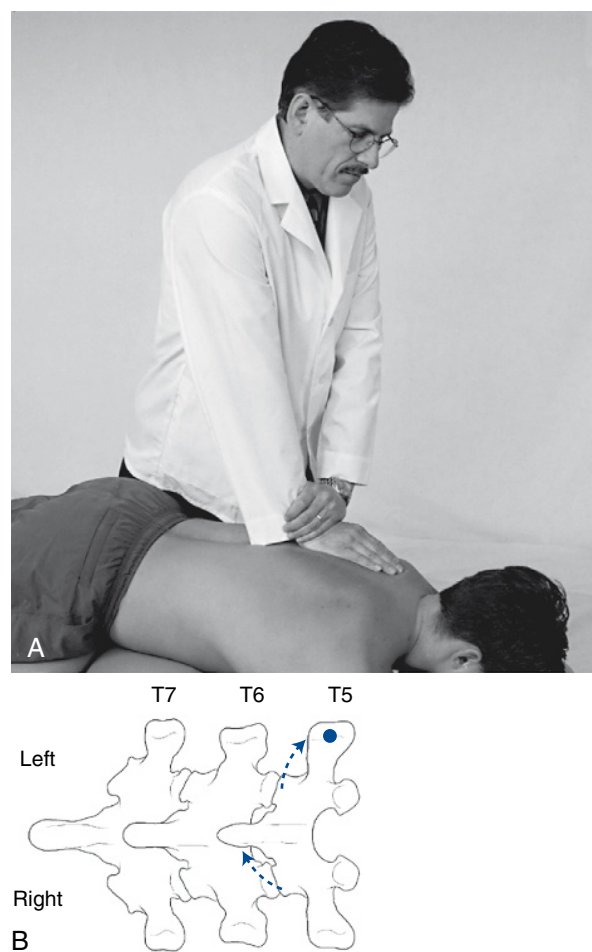


Figure 4-57 Prone unilateral hypothenar transverse push applied to treat a right rotation restriction at T5-6. Segmental contact is established over the left T5 transverse process.

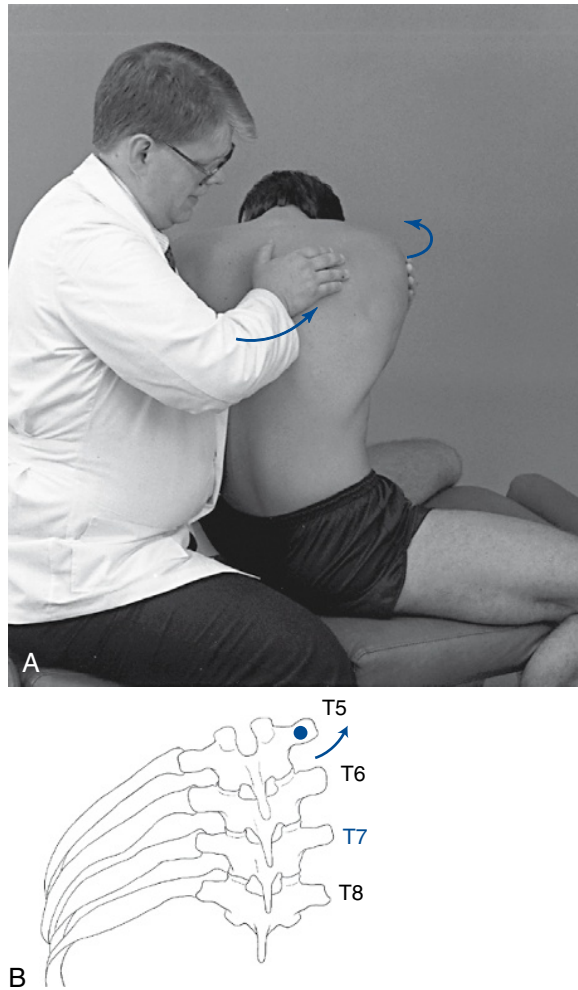


Figure 4-58 An example of an assisted sitting thoracic adjustment applied in the treatment of a left rotation restriction at T5-6. This technique incorporates a segmental contact on the transverse process of the superior vertebrae of the dysfunction motion segment with an assisting hand and thrusting contact established on the patient's ipsilateral forearm.

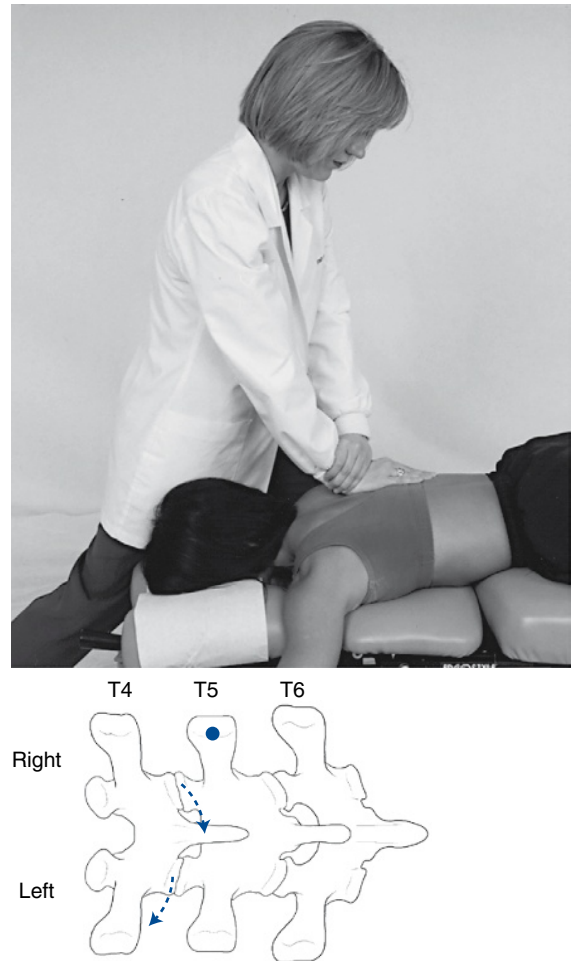


Figure 4-59 Prone thoracic resisted unilateral hypothenar transverse push adjustment applied to induce right rotation at the T4-5 joint. Adjustment is applied in the treatment of a right rotation restriction or a left rotation malposition at T4-5. Segmental contact is established over the right T5 transverse process.

posture-resisted lumbar mammillary push adjustments induce positional and postadjusting gapping in the articulations superior to the level of contact.

The adjustive thrust may be focused through a single segmental contact, but commonly incorporates additional contacts and reinforcing thrusts applied at levels inferior to the segmental contact. Figure 4-59 illustrates the application of a short-lever method incorporating a single level of focused thrust (very uncommon). Figure 4-60 illustrates a method incorporating a segmental contact coupled with a distal contact established inferior to the level of segmental contact. The vertebral segments superior to the contact are rotated in the direction of restriction, opposite the direction of the thrust, and preadjustive tension is localized to the articulations superior to the contact. Additional leverage is provided through the inferior contact established on the patient's leg. At tension, a thrust is delivered through both contacts to induce cavitation and movement in the direction of restriction.

Counter-resisted methods incorporate segmental contacts established on both sides of the joint or region to be adjusted.

Pretension and the adjustive thrusts are directed in opposing directions to maximize distraction across a given area and joint. The adjustive thrust may be focused through segmental contacts or may incorporate additional contacts and reinforcing thrusts applied at levels superior to and inferior to the segmental contacts. In the spine this procedure is most commonly applied in the treatment of rotational dysfunction. Figure 4-61 illustrates the application of a short-lever method incorporating a neutral patient position. Figure 4-62 illustrates a method incorporating a segmental contact established on adjacent spinous processes, coupled with additional points of leverage. The adjustive thrust is applied in opposing directions through the segmental contacts and contacts established on the patient's forearm and lateral pelvis.

Although for didactic purposes it is useful to separate the adjustive thrust into its component parts, it can be misleading and distracting to the student who is trying to perfect the art of adjusting. Instead of focusing on the act as a singular event, the novice often tries to develop a thrust by mentally producing each

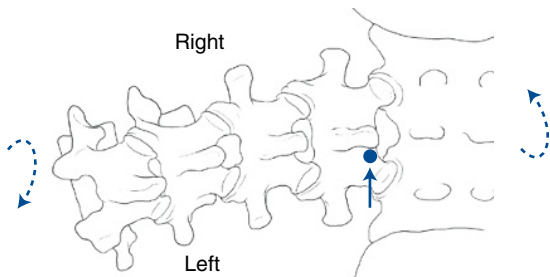
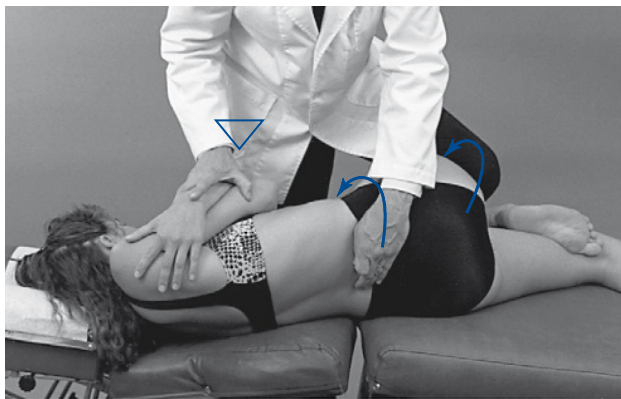


Figure 4-60 An example of a resisted side-posture adjustment applied in the treatment of a right rotation restriction at L4-5. This technique incorporates a segmental contact on the spinous process of the inferior vertebrae of the dysfunction motion segment with a resisted leg contact on the patient's leg.

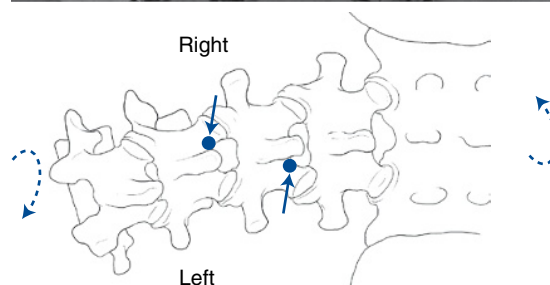
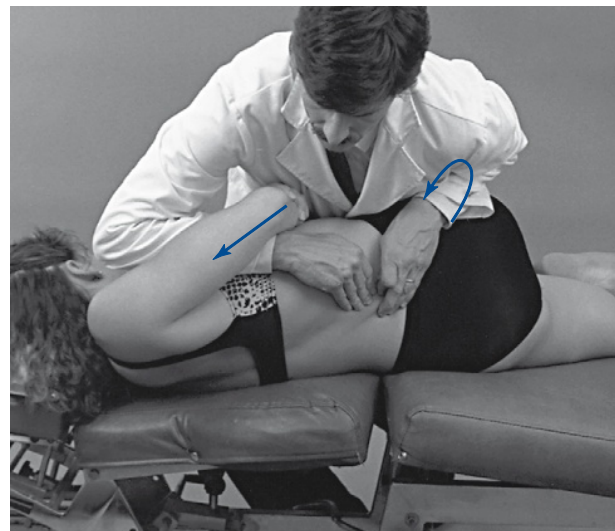


Figure 4-62 Side-posture lumbar spinous push-pull applied to induce right rotation at the L3-4 articulation. Adjustment is applied in the treatment of a right rotation restriction or a left rotation malposition at L3-L4.

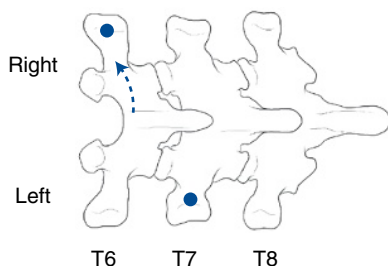


Figure 4-61 Prone thoracic crossed bilateral hypothernar transverse counterthrust technique applied to induce left rotation at T6-7. Adjustment is applied in the treatment of a left rotation restriction or a right rotation malposition at T6-7. Segmental contacts are established over the right T6 transverse process and the left T7 transverse process.

event separately, resulting in an unfocused, uncoordinated thrust. The thrust, developed through repetitive practice, is a fluid, habitual procedure, not a series of segmented and separated steps. Box 4-16 lists some basic rules and principles for the effective and safe use of chiropractic adjustive technique.

MOTION-ASSISTED THRUST TECHNIQUES

Motion-assisted thrust techniques are those procedures that incorporate a component of mechanical assistance in the development of adjustive pretension or the delivery of an adjustive thrust. The assisting mechanical forces are typically provided by non-motorized or motorized articulating adjustive tables. Because the critical force necessary to preload a joint and deliver an HVLA thrust may be difficult to achieve, the use of some form of mechanical assistance may be desirable. In addition, some forms of mechanically assisted technique tend to produce a long-axis tractive force on the articulations being treated. Long-axis distractive movement is a potentially important JP component of all synovial joints. It is not a general focus of most manually applied techniques for the spine, although this movement is a major focus in extremity manipulation. Incorporating it into spinal technique may provide some additional therapeutic effect.

Mechanically assisted techniques have been postulated to augment a practitioner's physical attributes, allowing for the

BOX 4-16 Basic Rules for Effective Adjustive Technique

1. Select the most efficient and specific technique for the primary problem.
2. Position the patient in a balanced, relaxed, and mechanically efficient position.
3. The doctor should be relaxed and balanced with his or her center of gravity as close to the contact points as possible.
4. The contacts should be taken correctly and specifically.
5. Articular and soft tissue slack should be removed before thrusting.
6. Any minor alterations in position or tension should be made before thrusting.
7. Visualize the structures contacted and the direction of your adjustive vector.
8. Guard against the loss of established preadjustive joint tension. Do not noticeably back off before thrusting.
9. The thrust must be delivered with optimum velocity and appropriate depth.
10. Maintain stability and rigidity through the upper extremities during the delivery of the adjustive thrust.
11. During the thrust, use additional body weight if appropriate (body-drop). This is especially important in side-posture pelvic and lumbar adjusting, in which most of the adjustive force is derived from a body-drop thrust.
12. It is just as important to know when not to adjust as to know when and where to adjust.
13. *Primo est non nocere*—First, do no harm.

development of forces that would not be otherwise achievable. The use of drop-section table pieces or motorized moving table sections can theoretically provide additional support for producing the needed force. Motion and mechanically assisted procedures are relatively new concepts and must be clinically studied. Although they hold promise and are based on sound principles, no clinical data exist to support effectiveness or efficiency. Each of these approaches is discussed here, and specific applications are described where applicable in Chapters 5 and 6.

Drop-Section Mechanical Assistance

The first drop headpiece was introduced in chiropractic in 1952; B.J. Palmer stated that the principle behind the drop head piece constituted one of the greatest advancements in chiropractic.⁴²² Dr. J. Clay Thompson developed adjusting tables with cervical, thoracolumbar, and pelvic drop-piece sections in 1957, with the stated intention of providing a mechanical advantage for producing an HVLA adjustment with minimal discomfort for the patient.

Dr. Thompson believed drop-table procedures used Newton's laws of motion to develop a certain amount of kinetic energy not seen in other forms of chiropractic technique. He theorized that the mechanical drop mechanism reduced the muscular effort needed by the clinician to produce the adjustive thrust. Therefore, the muscular strength of the clinician is not a limitation in providing manipulative therapy. Moreover, it is thought that when the drop piece releases, the amount of force exerted on the joints is minimal and therefore more comfortable for the patient. Finally, because the patient cannot resist the effects of the drop sections, it is reasoned that joint movements are more easily achieved.

Another theory proposes that the mechanical advantage gained by drop pieces is the shear reactive force that is generated at the termination of the drop. In this model the doctor sets more resistance in the drop mechanism and maintains adjustive force through the termination of the drop. There are, however, no studies to support either of these contentions.⁴²³

The Thompson table, and all drop tables, feature mechanical drop sections that drop a small distance on the delivery of

BOX 4-17 Drop-Table Procedure

1. Position the body part over the drop section.
2. Cock the drop section, checking its tension.
3. Establish contacts over the part to receive the thrust.
4. Generate a thrusting action to make the section drop.
5. The thrust may be repeated to patient tolerance.

a chiropractic thrust. The amount of resistance to pressure can be independently adjusted in each drop section. The patient is positioned on the table with the segment to be adjusted on a drop section with the tension properly set so that the patient's body weight will not cause the section to drop (Box 4-17). When additional force is applied and the resistance of the drop section is overcome, the section drops and terminates its fall at a preset short distance.

Tables equipped with drop-section mechanisms have levers used to set each drop section in a "cocked-up" position. Some tables use a pneumatic cocking mechanism that is operated by a foot pedal, freeing the clinician's hands from having to locate the levers. There are, of course, specific considerations for each joint to be adjusted, such as the SCP, vector of thrust, and clinician position. Specific procedures for each joint are described and demonstrated in detail in other works.⁴²⁴

Motorized Mechanical Assistance (Motion-Assisted Adjusting)

Motorized mechanical traction is provided by adjusting tables that provide continuous, rhythmic mechanical movement and distraction of the articulations to be mobilized or adjusted. Assisted mechanical distraction of spinal joints began with manually operated tables (McManis table)⁴²⁵ and progressed to include tables that provide motorized movement and distraction (Cox, Leander, and Hill). The fundamental principle and potential advantage of motion-assisted adjusting is the delivery of an adjustive thrust

across a joint that has been mechanically distracted. The preadjustive tension established at the involved joints is established through the movements of the motorized table, freeing the doctor to conserve energy and focus on his or her adjustive contacts, sense of joint tension, and adjustive thrust.

In addition, traction tables are assumed to induce some additional long-axis distraction in the joint to which it is applied. The movement of long-axis distraction (y-axis translation) in spinal segments is not specifically addressed with many manipulative approaches. In the extremity joints, considerable attention and significance are placed on the evaluation and manual treatment for loss of long-axis distraction and its role in producing joint dysfunction.^{42,426}

Using a motorized distraction table may increase the element of long-axis distraction during manipulative treatments. Because motion-assisted palpation and treatment may also be performed with the patient recumbent, many different patient presentations (e.g., acute, chronic, aged, and obese) may be accommodated by this technique.

Most mobilization and adjustive techniques are applicable to motorized distraction tables. Mechanized distraction tables simply provide additional preadjustive tension and joint distraction. This has the potential to decrease the amount of muscular effort and force the doctor must generate to preload a joint before delivering an adjustive thrust. When adjustive procedures are applied, segmental contacts and tissue pulls are established in the same fashion as they would be for an adjustment delivered on any adjusting table. The adjustive thrust is typically delivered at full excursion of the mechanized table as the doctor senses maximal distraction of the joint.

The fundamental components of motion-assisted adjusting can be illustrated with prone thoracic or lumbar adjustments. In the prone positioning, the adjustive thrust is delivered as the caudal

section stretches. The intermittent distraction opens the involved motion segment, facilitating the adjustment and reducing the required force needed for the thrust. In this tractive state the thrust can also be delivered repeatedly with less force being produced by the clinician. The table is in motion, creating distraction of the patient with the force of the treating hands directed primarily headward. A pull-push effect is thus created along the long axis (y-axis) of the body, facilitating the mobilization of the joint and the restoration of long-axis distraction movement (Figure 4-63). In addition, lateral flexion can be induced using a roll for producing prestress and a pulling vector while the table creates long axis distraction (y-axis) movement (Figure 4-63).

The science of chiropractic has made significant strides in the investigation of the art of chiropractic. The profession now has a body of credible research to document some of what it claims. Advocates of manipulative therapy in the healing arts of chiropractic, medicine, osteopathy, and physical therapy have independently concluded that the HVLA thrust is an important clinical intervention for the treatment of dysfunctional conditions associated with the NMS system. The acceptance of spinal manipulation by other health care professions, industries, and the general population continues to grow despite controversies that still exist in clinical practice. The controlled delivery of the adjustive thrust demands much discipline and skill. An adjustive thrust delivered incorrectly carries the risk of patient injury. It takes extensive training and time to perfect adjustive skills and the ability to sense and control the appropriate depth and force of an adjustive thrust. This skill cannot be effectively learned over the course of a few months or by attending weekend courses. The authors hope that this chapter helps advance the development and perfection of adjustive psychomotor knowledge and skills necessary for the delivery of safe and effective chiropractic adjustments.

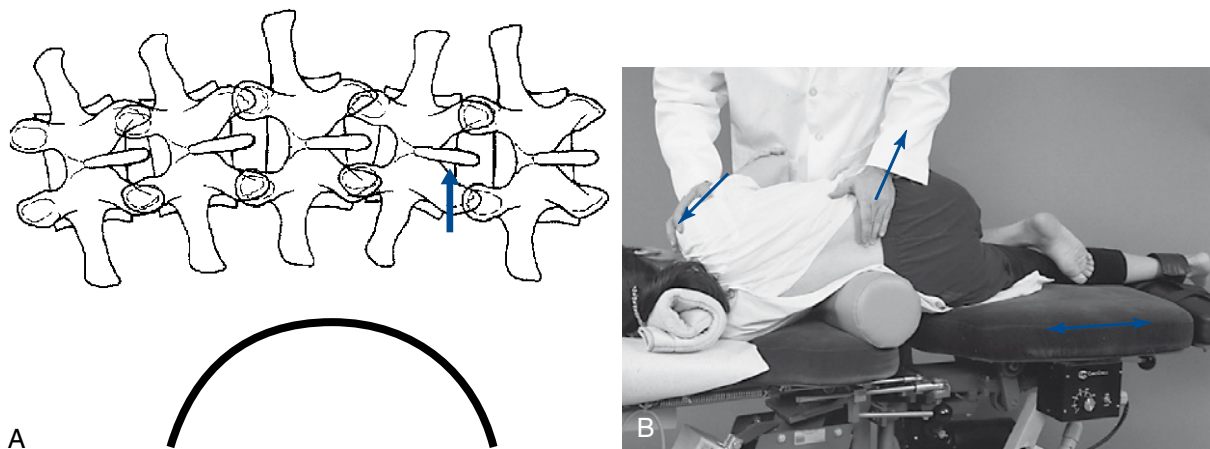


Figure 4-63 A, Diagrammatic representation of the contact point for a left lateral flexion restriction, right lateral flexion malposition, L4-L5. B, Motion-assisted thrust technique for intersegmental lateral flexion dysfunction (left lateral flexion restriction, right lateral flexion malposition, L4-L5).

THE SPINE: ANATOMY, BIOMECHANICS, ASSESSMENT, AND ADJUSTIVE TECHNIQUES

OUTLINE			
STRUCTURE AND FUNCTION OF THE SPINE	145	THORACIC SPINE	188
EVALUATION OF SPINAL JOINT FUNCTION	146	Functional Anatomy	188
Spinal Joint Scan	147	Thoracic Curve	189
IDENTIFICATION OF JOINT SUBLUXATION/DYSFUNCTION SYNDROME	151	Range and Patterns of Motion	189
CERVICAL SPINE	152	Kinetics of the Thoracic Spine	191
Functional Anatomy of the		Functional Anatomy and	
Upper Cervical Spine	152	Biomechanics of the	
Functional Anatomy of the		Rib Cage	191
Lower Cervical Spine (C3–C7)	157	Functional Anatomy and	
Evaluation of the Cervical Spine	162	Characteristics of the	
Overview of Cervical Spine		Transitional Areas	193
Adjustments	170	Evaluation of the Thoracic	
Upper Cervical Spine		Spine	195
Adjustments	174	Overview of Thoracic Spine	
Lower Cervical Spine		Adjustments	200
Adjustments	180	Overview of Rib Adjustments	211
		THORACIC ADJUSTMENTS	211
		Thoracocervical Adjustments	211
		Thoracic Adjustments	215
		Rib Adjustments	226
		Costosternal Adjustments	232
		LUMBAR SPINE	233
		Functional Anatomy	233
		Lumbar Curve	234
		Range and Patterns of Motion	235
		Kinetics of the Lumbar Spine	237
		Evaluation of the Lumbar Spine	238
		Adjustments of the Lumbar	
		Spine	245
		Lumbar Adjustments	253
		PELVIC JOINTS	262
		Functional Anatomy of the	
		Sacroiliac Joints	262
		Sacroiliac Motions	265
		Evaluation of the Pelvic	
		Complex	266
		Overview of Pelvic	
		Adjustments	274
		Pelvic Adjustments	274
		Pubic Symphysis Adjustments	280
		Coccyx Adjustments	281

STRUCTURE AND FUNCTION OF THE SPINE

The spine is, among its many other roles, the mechanism for maintaining erect posture and for permitting movements of the head, neck, and trunk. The pelvis helps to form the foundation for posture, and the cervical spine–occipital complex is essentially the postural accommodation unit. The spinal column simultaneously provides stability to a collapsible cylinder while permitting movements in all directions. It supports structures of considerable weight, provides attachments for muscles and ligaments, transmits weight onto the pelvis, and encases and protects the spinal cord while allowing transmission of neural information to and from the periphery.

The functional unit of the spine, the motion segment, is the smallest component capable of performing the characteristic roles of the spine. The motion segment consists of two adjacent vertebrae and their associated structures. It is classically viewed as a three-joint complex, divided into anterior and posterior elements. The disc and vertebral bodies form the anterior joint and the two zygapophyseal joints form the posterior joints (Figure 5-1). The intervertebral joint is therefore a three-joint complex throughout the spine, except for the atlanto-occipital articulation. Changes affecting the posterior joints also affect the disc and vice versa.

The articulations of the vertebral bodies are synchondroses, or cartilaginous joints, connected by the fibrocartilaginous intervertebral discs (IVDs). In the cervical and lumbar spines, a disc is approximately one third of the thickness of the corresponding vertebral body. In the thoracic spine, this ratio decreases to approximately one sixth of the thickness. This articulation forms

the anterior portion of the vertebral motion unit; its chief function is weight-bearing and shock absorption.

Two important ligaments help support the vertebral bodies. These are the anterior longitudinal ligament (ALL) and posterior longitudinal ligament (PLL) (see Figure 5-1). The ALL extends from the inner surface of the occiput to the sacrum. It starts as a narrow band that widens as it descends. It is thickest in the thoracic spine and thinnest in the cervical spine. The PLL runs from the occiput down the posterior portion of the vertebral bodies. It is a somewhat narrow structure that has lateral extensions and covers part of the IVD. It is also thickest in the thoracic spine and equally thin in the cervical and lumbar regions. In the lumbar spine, the PLL tapers, leaving the posterolateral borders of the disc uncovered and unprotected, with important clinical ramifications. Fibers from the PLL attach to the disc itself.

The articulations between the neural arches of vertebrae are diarthrodial joints (referred to as zygapophyseal joints, facet joints, or posterior joints). Each has a joint cavity enclosed within a joint capsule and lined with a synovial membrane (see Figure 5-1). The zygapophyseal joints are true synovial joints and form the posterior portion of the vertebral motion unit. They allow a guiding, gliding action, and the orientation of their joint surfaces is largely responsible for determining the amount and direction of regional spinal motions (Figure 5-2). Furthermore, the facet joints play a significant role in load-bearing. This varies between the facets and the disc, depending on the position of the spine. The facet joints bear an increasing percentage of the load as the spine moves toward an extended position.

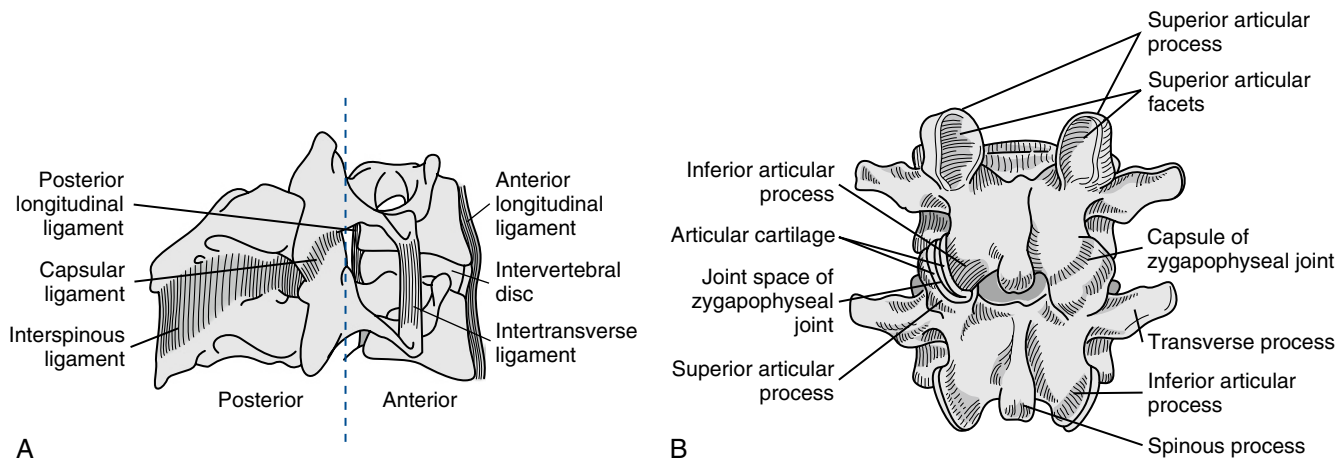


Figure 5-1 Spinal motion segment composed of two vertebrae and contiguous soft tissues: intrinsic ligaments (A) and the posterior joint and joint capsule (B). (B from White AA, Panjabi MM: *Clinical biomechanics of the spine*, ed 2, Philadelphia, 1990, JB Lippincott.)

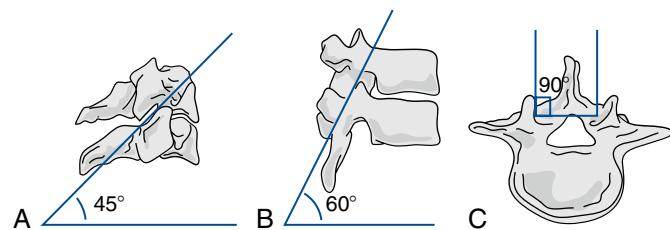


Figure 5-2 Facet planes in each spinal region viewed from the side and above. A, Cervical (C3–C7). B, Thoracic. C, Lumbar. (Modified from White AA, Panjabi MM: *Clinical biomechanics of the spine*, ed 2, Philadelphia, 1990, JB Lippincott.)

Support and stability for the posterior joints come from the small segmental ligaments and the joint capsule (see Figure 5-1). The ligamentum flavum, a strong and highly elastic structure, connects adjacent lamina. The interspinous and supraspinous ligaments attach from spinous process to spinous process. Occasionally a bursa forms between these two ligaments. The intertransverse ligaments are relatively thin and run from transverse process to transverse process.

Although each region of the spine has its own unique characteristics, typical vertebrae have common descriptive parts that include a vertebral body, two pedicles, two lamina, four articular processes, two transverse processes, and a spinous process (Figure 5-3). There are in each region, however, atypical vertebrae, which either lack one of these descriptive features or contain other special peculiarities. The atypical vertebrae are C1, C2, C7, T1, T9 to T12, L5, and the sacrum and coccyx. Specific anatomic descriptions and functional characteristics are covered under each specific spinal region.

EVALUATION OF SPINAL JOINT FUNCTION

The investigation for spinal function incorporates history-taking; physical examination; and, if appropriate, radiographic, laboratory, and special examinations. The interview and examination should be open-ended, efficient, and directed toward identifying the source and nature of the patient's complaint. This is not to imply that the

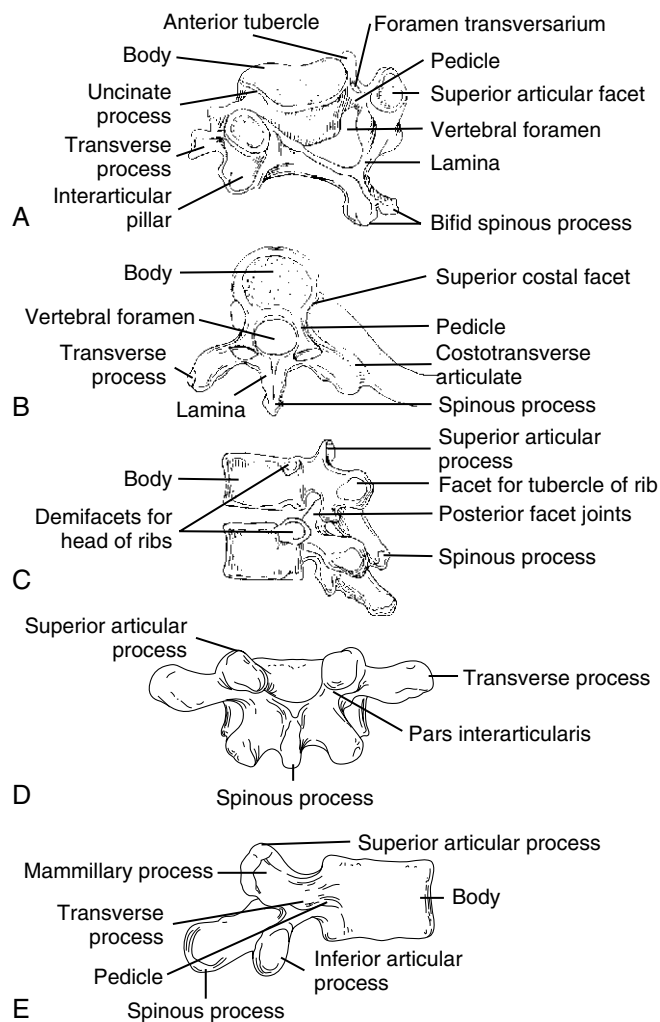


Figure 5-3 The structures that compose the typical cervical (A), thoracic (B and C), and lumbar vertebrae (D and E). (D and E from Dupuis PR, Kirkaldy-Willis WH. In Cruess RL, Rennie WRJ, eds: *Adult orthopaedics*, New York, 1984, Churchill Livingstone.)

examination should focus on just the site of complaint; the site of complaint does not necessarily correspond to the source of the dysfunction or pathologic condition. Complaints of pain or aberrant function may have visceral, not somatic, origin, and disorders within the neuromusculoskeletal (NMS) system may be secondary to somatic disease or dysfunction at distant sites. Consequently, the doctor must develop a method to efficiently scan regions of the spine and the locomotor system for possible sites of disease or dysfunction. Within this context, it is impractical to evaluate every joint of the musculoskeletal system during the initial evaluation. The spinal scanning examination should therefore be an abbreviated evaluation designed to quickly scrutinize key areas of spinal joint function. Sites of potential abnormality should then be examined in further detail to assist in the clinical localization of areas of potential joint dysfunction.

SPINAL JOINT SCAN

The scanning examination of the spine is designed to screen for alterations in structure or function indicative of possible joint subluxation/dysfunction syndromes. It incorporates the assessment of posture, global range of motion (ROM), mobility, and the location of any sites of palpatory pain (Box 5-1).

BOX 5-1 Physical Scanning Evaluation for Joint Dysfunction

GOAL

To locate possible areas of joint dysfunction in need of a further detailed examination.

COMPONENTS

Posture and gait

- Evaluate integration of activities of the musculoskeletal system.
- Evaluate asymmetries in sectional relationships of the spine and extremities.
- Perform rapidly during initial contact with patient.

GLOBAL RANGE OF MOTION

- Evaluate active movements of the cervicothoracic spine in flexion, extension, lateral flexion, and rotation.
- Evaluate active movements of the thoracolumbar spine in flexion, extension, lateral flexion, and rotation.
- Evaluate visually or quantifiably with instruments (inclinometer or goniometer).

MOTION SCAN: JOINT PLAY OR JOINT CHALLENGING

- Perform in the seated position (usually); posterior-to-anterior pressure is applied to the spinal segments while the patient's spine is passively extended, creating a resisted springing quality.
- A fluid, wavy, rocking motion should be produced; note any regions of restriction.
- Pain scan: palpable pain or skin sensitivity.
- Evaluate general areas, using light to moderate palpation or pinwheel.
- Note location, quality, and intensity of pain produced during the previous activities.

Posture Scan

The evaluation of static posture incorporates both lateral and posterior assessment. The patient stands with the heels separated approximately 3 inches and the forepart of each foot abducted about 8 to 10 degrees from the midline.

On the lateral analysis, visible surface landmarks that ideally coincide with a plumb line are the lobe of the ear, shoulder joint, greater trochanter, and a point slightly anterior to the middle of the knee joint and just anterior to the lateral malleolus (Figure 5-4).

On posterior postural examination, the plumb line should pass through the external occipital protuberance, the spinous processes, the gluteal crease, midway between the knees, and midway between the ankles (see Figure 5-4). Look for specific postural faults, including head tilt, head rotation, shoulder unleveling, lateral curves of the spine, pelvic unleveling, and pelvic rotation. Postural faults that are suspected of having a muscular basis should be followed up with evaluations of muscle length, strength, and volume. Although there is no single ideal posture for all individuals, the best posture for each person is the one in which the least expenditure of energy occurs because the body segments are balanced in the position of least strain and maximal support.

Global Range of Motion

Global ROM evaluation incorporates evaluation of all three cardinal planes of motion. Each range should be recorded and any reduced, aberrant, asymmetric, or painful movements noted. During a scanning examination of the spine, estimations of range are typically conducted without the aid of instrumentation; however, inclinometric measurements may be easily incorporated. Inclinometric measurements are more reliable than

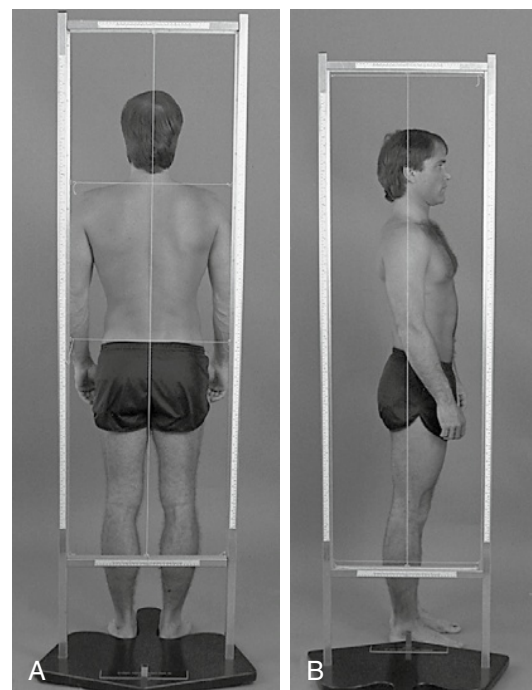


Figure 5-4 Posterior (A) and lateral (B) plumb line evaluation of posture.

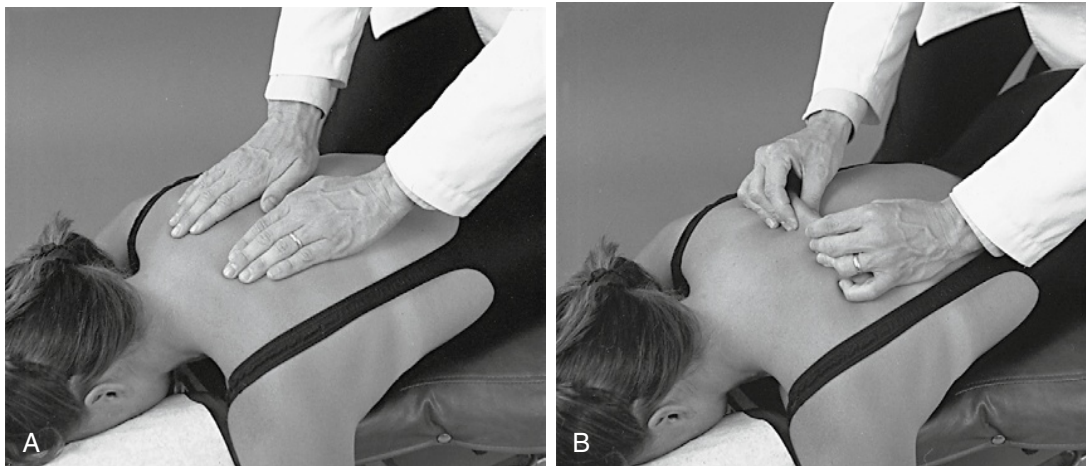


Figure 5-5 Evaluation of skin and superficial soft tissue sensitivity and texture with light palmar contacts (A) and skin-rolling technique (B).

visual estimates and are the standard of care in spinal impairment evaluations. The specific ranges and methods for evaluating regional mobility of the spine are discussed later under each separate spinal section.

Regional spinal movements that fall within normal ranges do not necessarily exclude segmental joint dysfunction. Segmental joint hypomobility may be masked by hypermobility at adjacent joints.

Pain Scan

The pain scan is designed to screen for sites of possible abnormal bony or soft tissue tenderness. The superficial soft tissues are assessed with light contacts through the palmar surfaces of the fingers (Figure 5-5, A) or by rolling the superficial layer between the fingers and thumbs (Figure 5-5, B). The deeper paraspinal tissues are evaluated with the same palmar contacts, but more pressure is applied to explore the deeper layer (Figure 5-6). Particular attention is directed to identifying any tenderness in the soft tissues over the posterior joints.

For evaluation of midline bony structures, the spinous processes and interspinous spaces may be scanned with the fingertips of one or both hands. When using a single-hand contact, the doctor rests the middle finger in the interspinous space and the index and ring finger on each side of the spinous process, spanning the interspinous space (Figure 5-7). The middle finger palpates for interspinous spacing and tenderness, and the index and ring fingers palpate for interspinous alignment and lateral spinous tenderness. When the fingers of both hands are applied, the fingertips meet at the midline to palpate interspinous alignment and tenderness (Figure 5-8).

The lumbar spine and thoracic spine are customarily examined in the prone position. Although the cervical spine may be evaluated in the prone or supine position, it is more commonly evaluated in the supine position with bilateral fingertip contacts.

Motion Scan

Evaluation of spinal mobility incorporates tests to scan regional joint play (JP), passive ROM, or end play. JP may be evaluated with the patient in a sitting or prone position, or in the supine

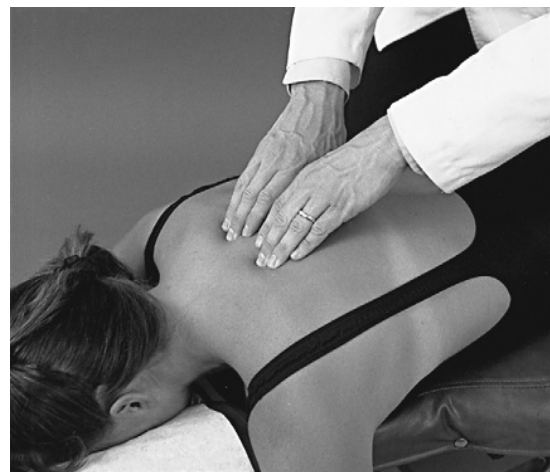


Figure 5-6 Evaluation of the sensitivity, tone, and texture of the deep paraspinal tissues, using the palmar surface of the fingertips.

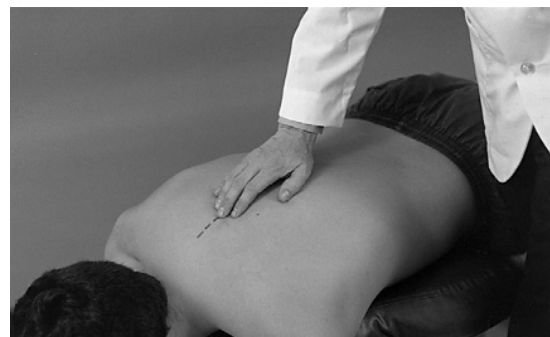


Figure 5-7 Single-hand palpation of spinous process alignment and tenderness.

position for the cervical spine. Passive ROM or EP is screened in the sitting position or supine position in the cervical spine. In both cases, the doctor establishes broad contacts against the spinous processes or broad bilateral contacts over the posterior joints. When JP is evaluated, the area evaluated should be positioned as close to the loose-packed neutral position as possible.



Figure 5-8 Two-hand palpation of spinous process alignment and tenderness.

When evaluating sitting JP, the doctor sits or stands behind the patient and places the nonpalpating arm across the patient's shoulders (Figure 5-9) or under the patient's flexed arms. The flexed-arm position is commonly used in the middle to upper thoracic spine and is developed by having the patient interlace his or her fingers behind the neck (Figure 5-10). In the cervical spine, the indifferent hand (IH) supports the crown of the patient's head (Figure 5-11).

With the patient prone, the doctor establishes bilateral contacts on each side of the spine or a reinforced contact over the spinous processes (Figure 5-12). To scan the spine, slide up or down, applying gentle posterior-to-anterior (P-A) springing movements. Regions of induced pain or inappropriate movement should be noted for further evaluation.

To screen sections of the spine for possible movement restriction, place the patient in the sitting position, with the arms crossed over the chest. The doctor may either sit behind the patient or stand at the patient's side (Figure 5-13). Trunk movement is controlled by establishing contacts across the patient's shoulder



Figure 5-10 Sitting joint play scan of the midthoracic region, using the flexed-arm position.

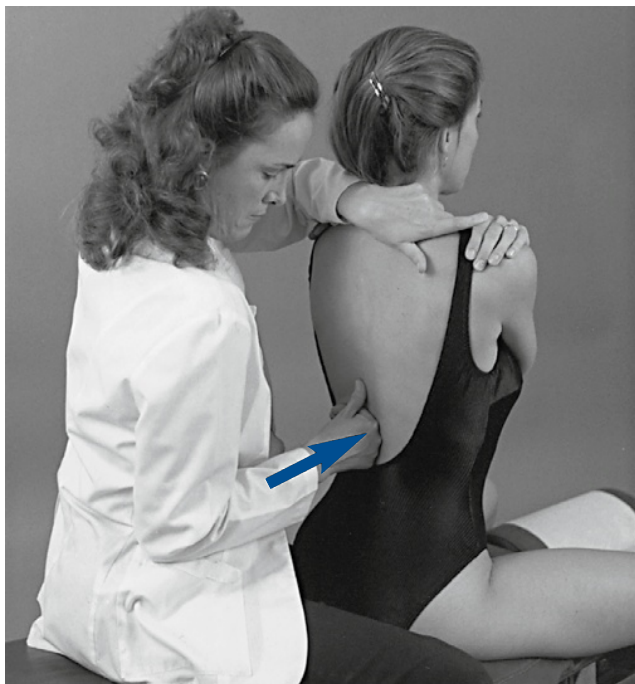


Figure 5-9 Sitting joint play scan of the thoracolumbar region.

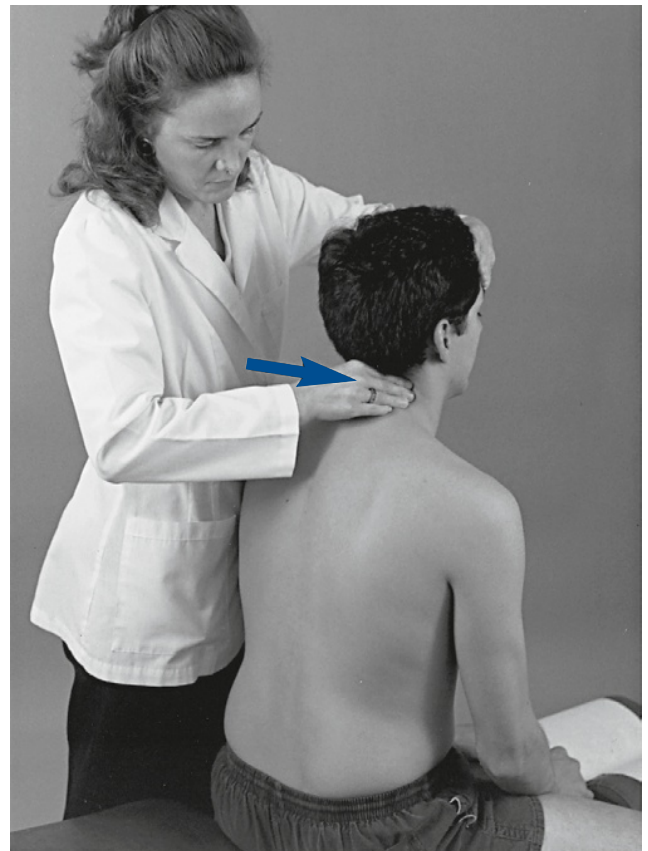


Figure 5-11 Sitting joint play scan of the midcervical region.

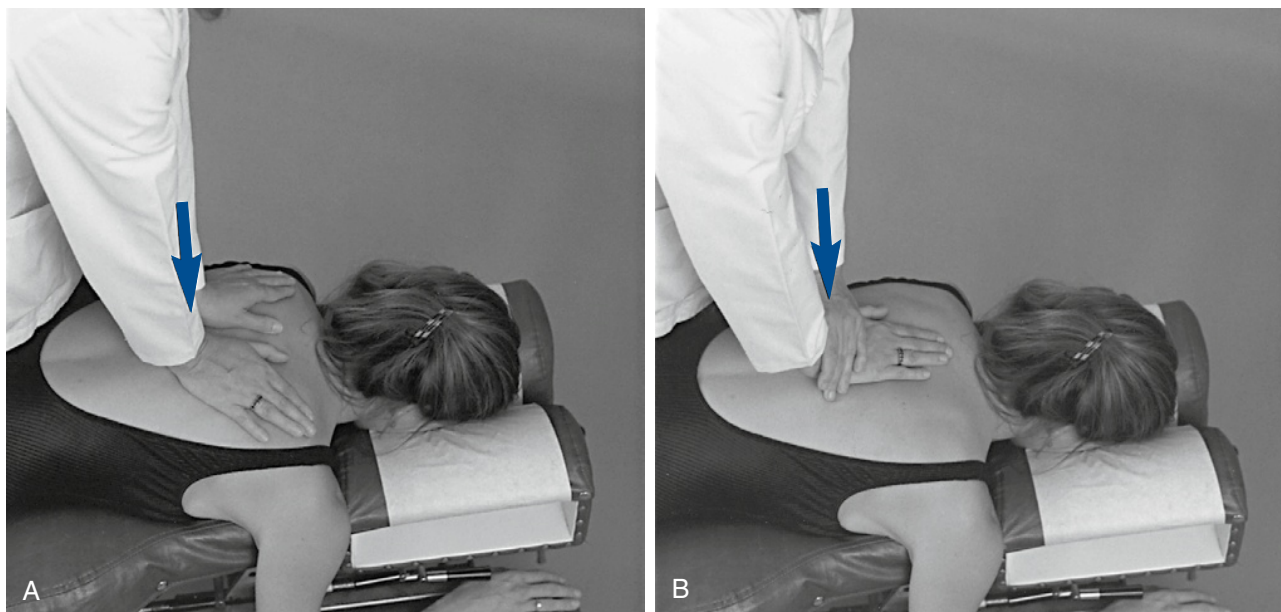


Figure 5-12 Prone joint play scan, using bilateral thenar contacts over the transverse processes (A) and reinforced hypothenar contact over the spinous process (B).

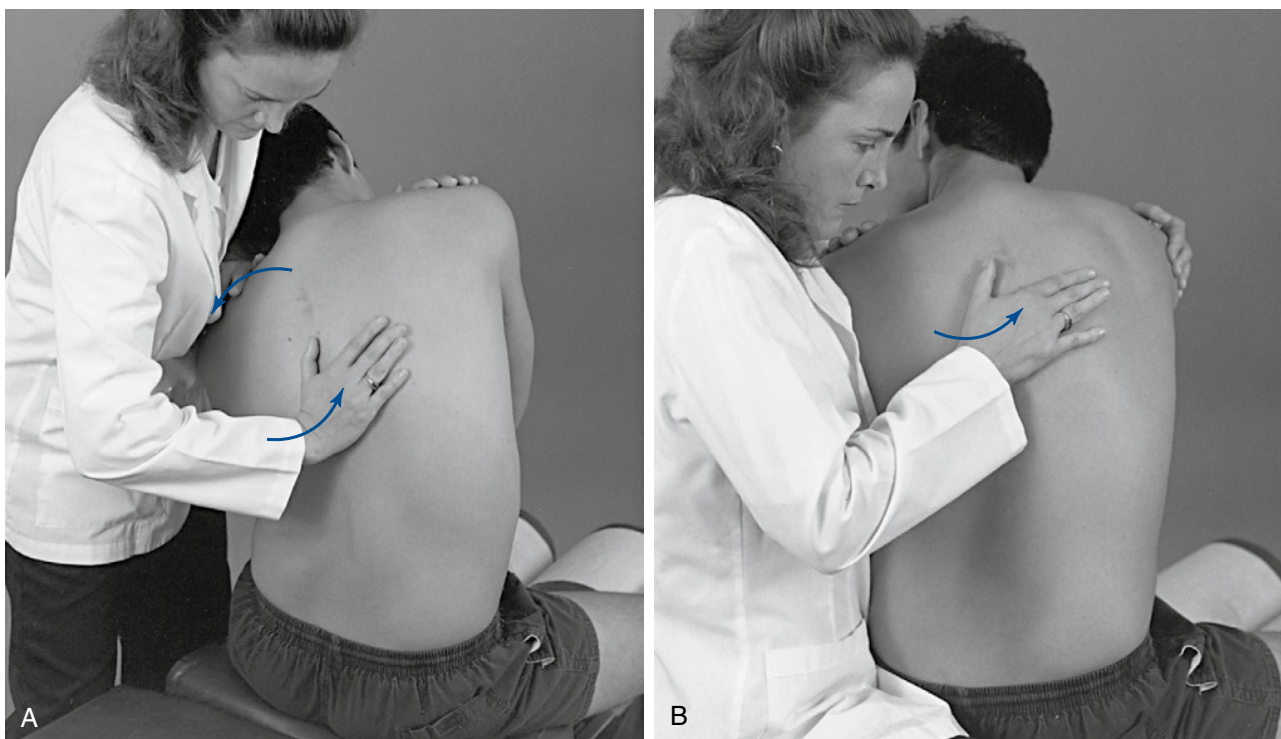


Figure 5-13 A, Evaluation of left lateral flexion movement, with the doctor standing. A broad thenar contact is established along the left side of the spinous processes. B, Evaluation of left rotation movement, with the doctor seated. A broad thenar contact is established along the left side of the spinous processes.

or by reaching around to grasp the patient's forearm. Cervical movement is directed by establishing a contact on the crown of the patient's head or forehead (Figure 5-14). These procedures are not designed to assess JP; rather, they are applied to evaluate full ROM with overpressure. Palpation contacts are established with the fingertips, palm, or thenar surface of the doctor's palpation hand.

The contacts should be broadly placed so that movement at two to three motion segments may be scanned together.

For lateral flexion and rotation assessment of the lumbar or thoracic region, contacts are established on the lateral surface of the spinous processes on the side of induced rotation or laterally bending (see Figure 5-13). In the cervical spine, the fingertips

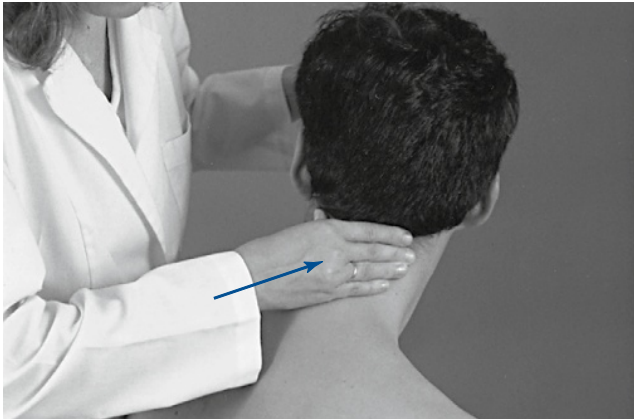


Figure 5-14 Evaluation of right cervical rotation, with the doctor's indifferent hand contacting the patient's forehead. The palmar surfaces of the doctor's right digits establish palpation contacts over the patient's right articular pillars.

establish the contacts over the articular pillars (see Figure 5-14). For spinal flexion and extension, the contacts are established with the dorsum of the hand or fingertip contacts over the interspinous spaces of several adjacent segments (Figure 5-15). To evaluate movement, guide the patient through the full ROM and induce gentle overpressure at end range. During the assessment, any regional sites of elicited pain or perceived increased or decreased resistance should be noted. JP and regional motion scanning of the cervical spine are commonly performed in a supine position.

Alignment Scan

Evaluation of joint alignment screens for asymmetric relationships on a sectional basis. Broad hand contacts are placed over the lateral transverse processes (paraspinal region), noting any posterior prominence indicative of rotational asymmetry. The index and middle fingers can also scan the interspinous spaces for widening or narrowing, indicative of flexion or extension asymmetries.

The lumbar spine and thoracic spine are customarily examined in the prone position. Although the cervical spine may be evaluated in the prone or supine position, it is more commonly evaluated in the supine position, using bilateral fingertip contacts.

IDENTIFICATION OF JOINT SUBLUXATION/DYSFUNCTION SYNDROME

As stated previously, the goal of manual joint assessment procedures is to identify possible sites of motion segment dysfunction. Many of the procedures used to scan the spine are also applied in the investigation and localization of dysfunction (Box 5-2). However, they are applied within a different context to identify more precisely the site and nature of the dysfunction/subluxation syndrome under question. They incorporate the detailed exploration of painful sites; the assessment of joint alignment and the texture, tone, and consistency of associated soft tissues; and the precise evaluation of intersegmental movements and end play.



Figure 5-15 Evaluation of upper lumbar flexion (A) and extension (B). The doctor's indifferent hand contact is placed across the posterior aspect of the patient's shoulders while the fingertips of the palpation hand contact the patient's interspinous spaces.

BOX 5-2 Isolation of Motion Segment Dysfunction (PARTS)

GOAL

To identify and define the specific dysfunction and specific tissues involved.

- P** Pain or tenderness (location, quality, and intensity) produced by palpation and pressure over specific structures and soft tissues
- A** Asymmetry of sectional or segmental components identified by static palpation of specific anatomic structures
- R** Range of motion decrease or loss of specific movements (active, passive, and accessory) distinguished through motion palpation techniques
- T** Tone, texture, and temperature changes in specific soft tissues identified through palpation
- S** Special tests or procedures linked to a technique system

The evaluation of painful tissues often incorporates the application of various directions of applied pressure to determine the directions of painful movement. These procedures are referred to as *joint challenging* or *joint provocation testing*.

Neither scanning (see Box 5-1) nor isolation evaluation, alone or in combination, constitutes a complete examination. The doctor of chiropractic must be competent in performing a complete physical evaluation to assess the nature of the patient's condition and to determine if the patient is suitable for chiropractic care.

Evaluation of segmental alignment involves comparing adjacent vertebral segments for symmetry and examining interspinous spaces, spinous processes, cervical articular pillars, thoracic transverse processes, rib angles, and lumbar mammillary processes. Sudden changes in interspinous spacing may identify flexion or extension malposition. Rotational malpositions may be identified by misalignment of adjacent spinous processes and unilateral prominence of the cervical articular pillars, thoracic transverse processes, or lumbar mammillary processes. The articular pillars, transverse processes, and mammillary processes are not as distinctly palpable as the spinous processes, but they are less susceptible to congenital or developmental anomaly. Unilateral contraction of segmental muscles produces a sense of fullness and may be mistaken for underlying rotational malposition of the articular pillars, transverse processes, or mammillary processes.

The localization of soft tissue changes also helps in specifying the nature and site of joint disease or derangement. Injured or inflamed joints may be associated with an overlying sense of increased warmth or puffiness. Joint disease or dysfunction is also commonly associated with local soft tissue reactive changes in the segmentally related tissue. This may lead to sites of asymmetric muscle tone and sites of abnormal tenderness (allodynia). Long-standing dysfunction may be associated with local areas of induration and contracture. These sites may palpate as areas of deep nodular or rope-like consistency.

Segmental motion palpation and end-play tests are applied to identify those segmental movements that are increased, restricted,

or painful. Pain elicited at one level and not adjacent levels helps localize the site of possible dysfunction. Increased resistance identifies a site of possible joint fixation, and increased movement identifies a site of possible clinical joint instability. The identification and location of soft tissue alterations, pain, and end-play restriction are fundamental to identifying the level and the direction of possible restriction. Furthermore, they are often essential to determining the type and directions of applied adjustive therapy.

Although all of the physical examination procedures discussed are an integral part of joint evaluation, it must not be forgotten that all have limitations. Many are based on the evaluation of symmetry in structure and function, and the degree of variation necessary to produce disease or dysfunction has not been determined. Asymmetry of structure and function is common, and minor abnormalities in alignment and motion may be within the range of normal variation. Furthermore, physical joint examination procedures depend on the skill of the examiner and are susceptible to errors in performance or interpretation. As discussed in Chapters 3 and 4 the present ability to precisely identify and adjust a single spinal segment may be limited and not directly related to clinical outcome. Based on this information, some have suggested that clinicians should focus on identifying regional sites (several segments) of dysfunction.¹⁻⁴ It must also be remembered that the identification of dysfunction does not necessarily identify the cause.

All of these concerns lead to the necessity of incorporating outcome measures in the evaluation of patient care and contrasting all of the history and examination findings before a diagnostic conclusion is reached and therapy is applied.

CERVICAL SPINE

The cervical spine has the precarious task of maintaining head posture while allowing for a great deal of mobility. The cervical spine must balance the weight of the head atop a relatively thin and long lever, making it quite vulnerable to traumatic forces. The cervical facets allow movement in all directions; the cervical spine is therefore the most movable portion of the vertebral column. The cervical spine has two anatomically and functionally distinct regions, which are considered individually.

FUNCTIONAL ANATOMY OF THE UPPER CERVICAL SPINE

The upper cervical spine is the most complex region of the axial skeleton. It is composed of the atlanto-occipital and atlantoaxial articulations, which serve as a transition from the skull to the rest of the spine. These two functional units are anatomically and kinematically unique. Neither has an IVD, and the atlantoaxial articulation incorporates three synovial joints.

The atlas has no vertebral body or spinous process (Figure 5-16). It consists of a bony oval, with the two lateral masses connected by the anterior and posterior arches. The lateral masses, formed from enlarged pedicles, have concave articular facets superiorly for articulation with the occipital condyles and circular inferior facets for articulation with the axis.

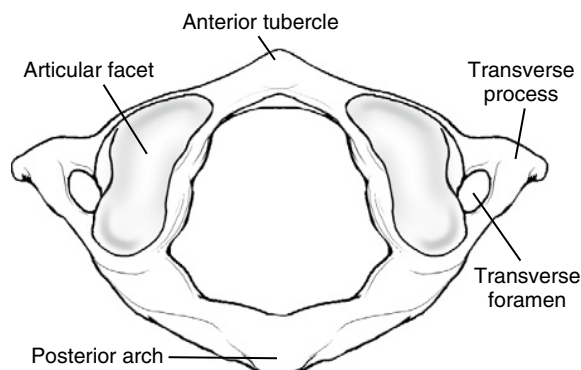


Figure 5-16 The structure of the atlas (C1).

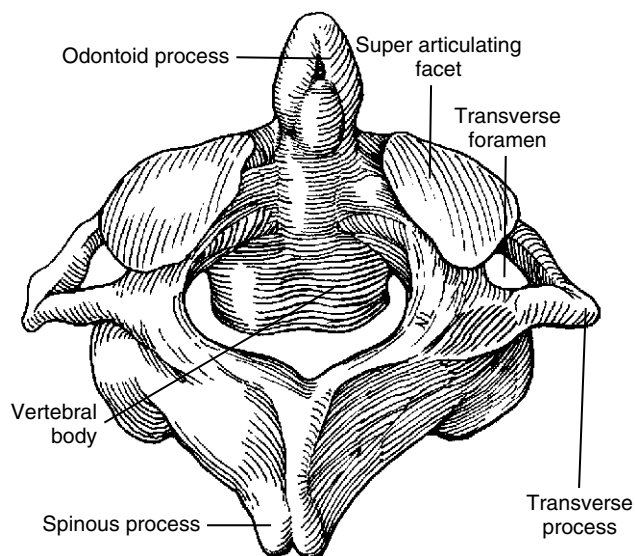


Figure 5-17 The structure of the axis (C2).

The outstanding feature of the axis (C2) is the presence of the odontoid process (dens) (Figure 5-17). The odontoid is formed by the fusion of the embryologic remnants of the vertebral body of the atlas to the superior aspect of the body of the axis. The spinous process of the axis is large and bifid, and it is the first palpable midline structure below the occiput. The superior articular surfaces project from the superior aspect of the pedicles to meet the inferior aspects of the atlas' lateral masses. Their surfaces are convex and lie in the transverse plane, with a slight downward lateral slant. The atlantoaxial articulation is formed by articular surfaces of the C1–2 lateral masses. Both articular surfaces are convex, allowing for considerable mobility in rotation. The atlanto-odontal articulation is formed by the anterior arch of the atlas and the odontoid process. The odontoid process is completely surrounded by the anterior arch of the atlas anteriorly, the lateral masses laterally, and the transverse ligament posteriorly (Figure 5-18). It is a trochoid joint, providing a pivot action.

The atlanto-occipital articulation is a freely movable synovial condyloid articulation (Figures 5-19 and 5-20). The articular surfaces of the condyles are convex and converge anteriorly, resembling curved wedges that fit into matching concave surfaces in the lateral masses of the atlas. Individual axes for each condyle exist,

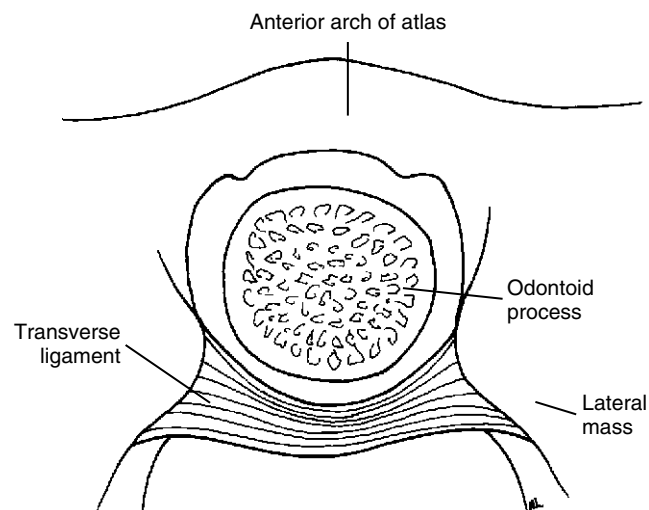


Figure 5-18 The atlanto-odontoid articulation viewed from above.

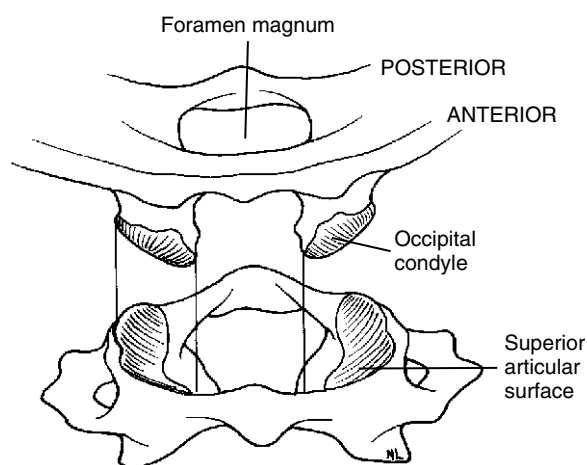


Figure 5-19 The atlanto-odontoid articulation has convex occipital condyles that fit into the concave lateral masses of the atlas.

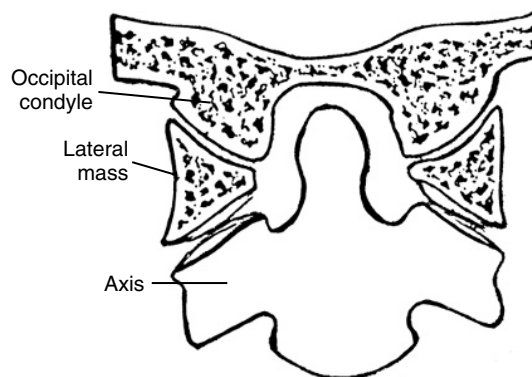


Figure 5-20 A coronal section through the atlanto-occipital and atlantoaxial articulations, showing the plane of the facets.

demonstrating that there is no single axis for axial rotation. The axis of movement for rotation occurs at two points (eccentrically located), thus resulting in very little active rotation. Each condyle can move a degree or two forward and backward without the other side moving much.

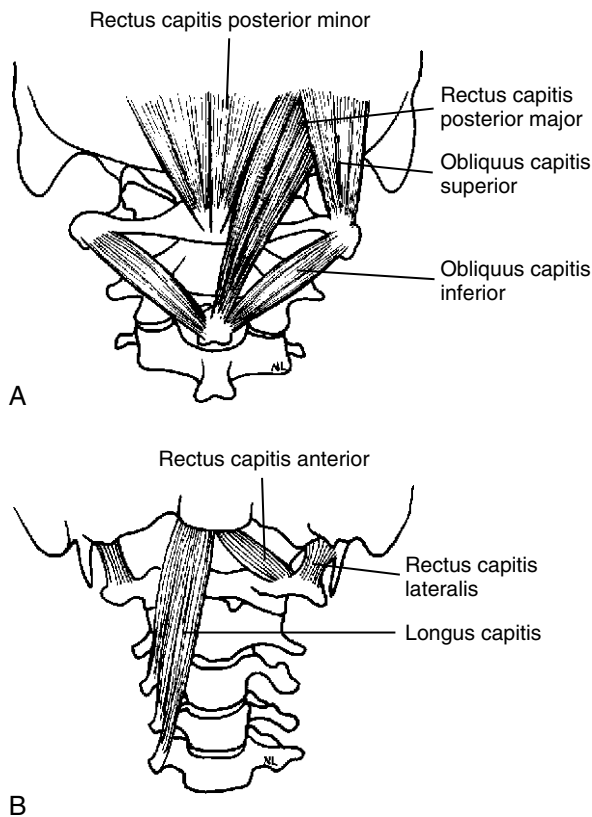


Figure 5-21 The suboccipital muscles. **A**, Posterior view. **B**, Anterior view.

The muscles that provide the forces necessary for movement, postural support, and primary stability of the upper cervical region include the rectus capitis posterior major, rectus capitis posterior minor, rectus capitis lateralis, rectus capitis anterior, superior oblique, and inferior oblique (Figures 5-21 and 5-22). All of these muscles are supplied with motor fibers from the first cervical nerve and proprioceptive and pain fibers via a communicating branch from the second cervical nerve.

The ligaments that provide added stability to the upper cervical spine include the transverse ligament of the atlas, alar ligaments, PLL, posterior atlanto-occipital membrane, anterior atlanto-

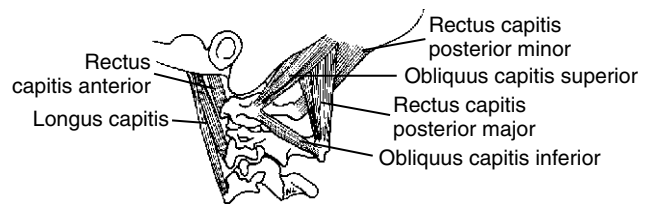


Figure 5-22 Lateral view of the suboccipital muscles.

occipital membrane, ligamentum nuchae, and the apical ligament (Figure 5-23). Because the ligaments of the upper cervical spine can be damaged by trauma, weakened by systemic inflammatory diseases, or congenitally absent or malformed, testing for their integrity should be done before manipulative therapy is begun. If instability is suspected, flexion-extension stress x-ray examinations should be performed.

Range and Pattern of Motion of C0–C1

The principle movement that occurs in the atlanto-occipital articulation is flexion and extension.⁵ The combined range is approximately 25 degrees (Table 5-1 and Figure 5-24). Flexion and extension movements at C0–1 are predominantly angular movements in the sagittal plane, without any significant associated coupled motions. During flexion the occipital condyles glide posterosuperiorly on the lateral masses of the atlas as the occipital bone separates from the posterior arch. During extension, the condyles slide anteriorly on the lateral masses of the atlas while the occipital bone approximates the posterior arch of atlas (Figure 5-25).

Axial rotation at the C0–1 articulation was previously thought to be very limited.⁶ However, recent studies have demonstrated a range of 4 to 8 degrees to each side.⁵ Rotational movement is limited by the articular anatomy and the connections of the alar ligaments. The movement that does occur is predominantly in the elastic range at the end of total cervical rotation, where it is usually coupled with some small degree of lateral flexion.⁷

Lateral flexion of the atlanto-occipital articulation approximates that of axial rotation. Although the articular design of the atlanto-occipital articulation should allow for greater flexibility in

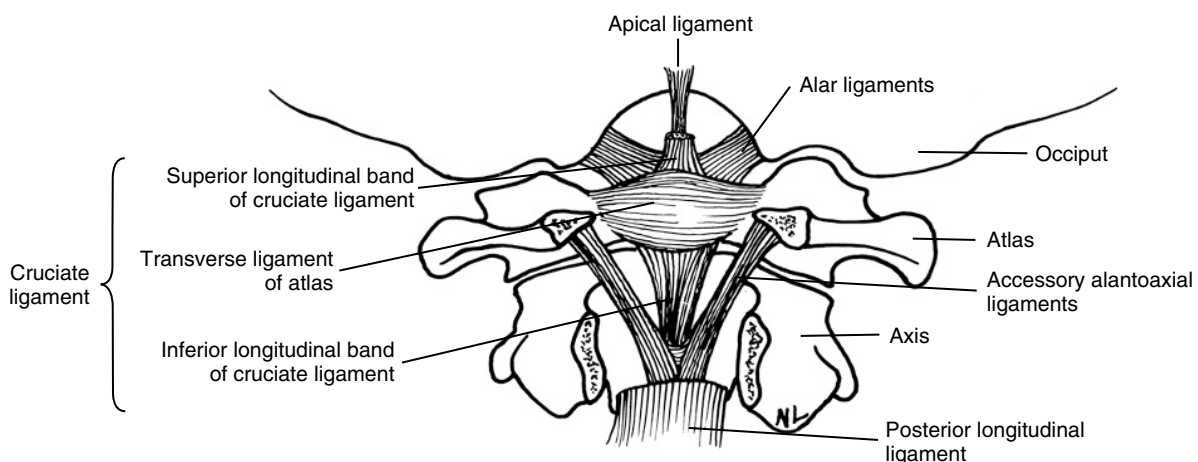


Figure 5-23 Upper cervical spinal ligaments shown with the posterior arch of the atlas and axis removed.

TABLE 5-1 Segmental Range of Motion for the Upper Cervical Spine

Vertebra	Combined Flexion and Extension	One-Side Lateral Flexion*	One-Side Axial Rotation
C0–1	25 degrees	5 degrees	5 degrees
C1–2	20 degrees	5 degrees	40 degrees

*Lateral glide or translation (laterolisthesis) occurs with lateral flexion movements of the neck.

Modified from White AA, Panjabi MM: *Clinical biomechanics of the spine*, ed 2, Philadelphia, 1990, JB Lippincott.

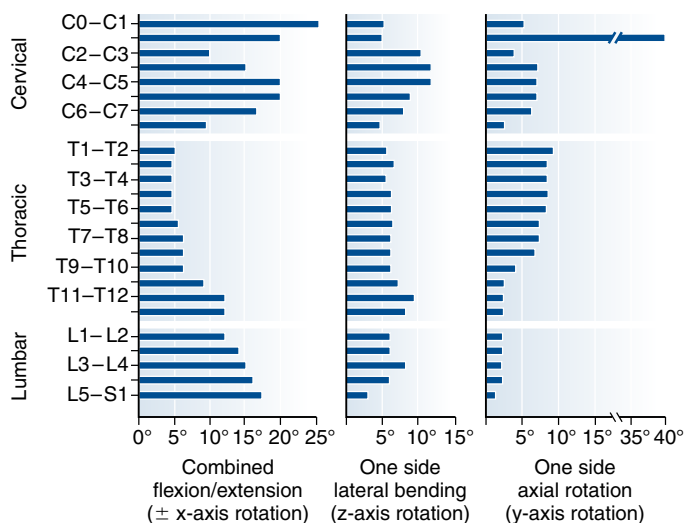


Figure 5-24 Representative values for rotatory range of motion at each level of the spine. (From White AA, Panjabi MM: *Clinical biomechanics of the spine*, ed 2, Philadelphia, 1990, JB Lippincott.)

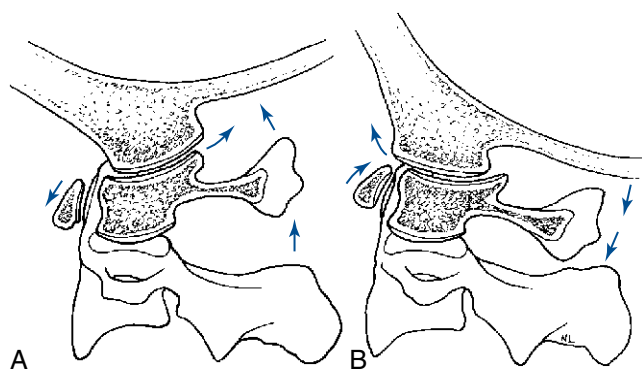


Figure 5-25 Flexion (A) and extension (B) of the occiput-atlas and atlas-axis.

lateral flexion, it appears that the attachments of the alar ligament function to limit this motion (Figure 5-26). Movement occurs primarily in the coronal plane, although it is typically associated with some small degree of coupled rotation in the opposite direction. This leads to rotation of the chin away from the side of lateral flexion. The predominant movements occurring at the articu-

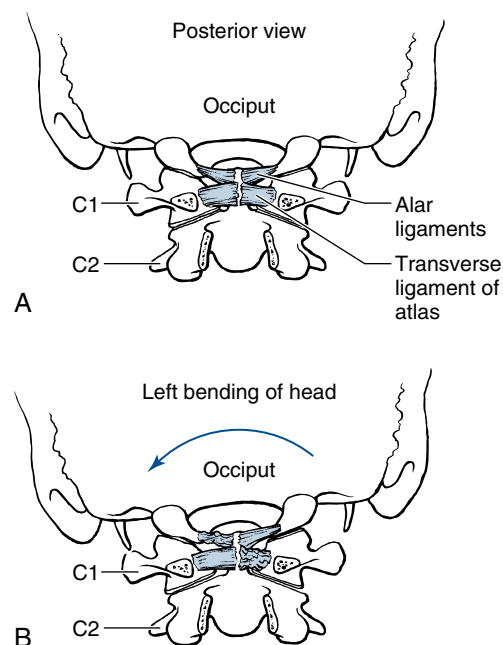


Figure 5-26 The role of the alar ligaments in lateral flexion of the atlanto-occipital articulation. **A**, Posterior view in the neutral position. **B**, Left lateral flexion. Motion is limited by the right upper portion and the left lower portion of the alar ligaments.

lar surface during lateral flexion are coronal plane rotation (roll) and translation (slide). Roll and slide occur in opposite directions because of the convex shape of the occipital condyles and the concave shape of the atlas articular surface. Rotation (roll) occurs in the direction of lateral flexion, and translation (slide) occurs in the direction opposite the lateral flexion (Figure 5-27).

The instantaneous axes of rotation (IAR) have not been experimentally determined for the atlanto-occipital articulation. The axes were estimated “by determining the centers of the arches formed by the outline of the joints in the sagittal and frontal planes”⁵ (Figure 5-28).

Range and Pattern of Motion of C1–2

The principal movement that occurs at the atlantoaxial joint is axial rotation. Segmental range averages 40 degrees to each side, contributing to more than half of the total cervical rotation. The first 25 degrees of cervical rotation occur primarily in the atlantoaxial joint.⁷ During rotation the lateral mass and articular surface slide posteriorly on the side of rotation and anteriorly

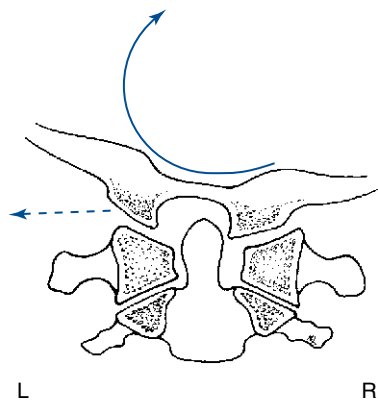


Figure 5-27 Right lateral flexion of the atlanto-occipital articulation, demonstrating rolling of the occiput to the right (*solid arrow*) and sliding to the left (*broken arrow*).

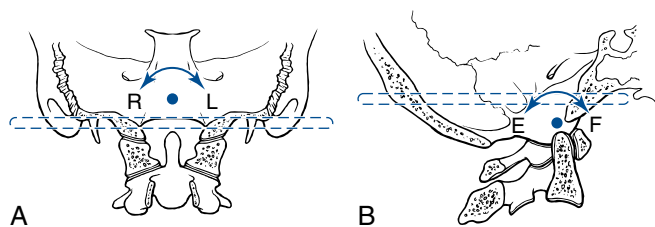


Figure 5-28 The theoretic location of the instantaneous axes of rotation (*dot*) in lateral flexion (*R and L*) (A) and flexion (*F*) and extension (*E*) (B). (From White AA, Panjabi MM: *Clinical biomechanics of the spine*, ed 2, Philadelphia, 1990, JB Lippincott.)

on the side opposite rotation. The motion occurs about a centrally located axis within the odontoid process (Figure 5-29). An additional subtle vertical displacement of the atlas takes place with rotation as a result of the biconvex structure of the articular surfaces (Figure 5-30).

Flexion and extension movements of the atlas on the axis occur as rocking movements as a result of the biconvex facet surfaces. The IAR is located in the middle third of the dens. In flexion, the posterior joint capsule and posterior arches separate, and the atlas articular surface glides forward. In extension, the posterior joint capsule and posterior arches approximate, and the atlas articular surface glides posteriorly (Figure 5-31). Also, the anterior arch of the atlas must ride up the odontoid process during extension and down during flexion. Flexion and extension movements of the atlantoaxial joint are also associated with small translational movements from 2 to 3 mm in the adult up to 4.5 mm in the child.⁵ Any movement greater than these ranges should trigger an evaluation to assess the stability of the C1–2 articulation and the integrity of the odontoid and transverse ligaments.

Compared with rotation, lateral flexion of the atlantoaxial articulation is limited, averaging approximately 5 degrees to each side.⁷ It has been suggested that lateral flexion is coupled with translation; however, this is a controversial subject.⁵ The associated translation is purported to occur toward the side of lateral flexion. In

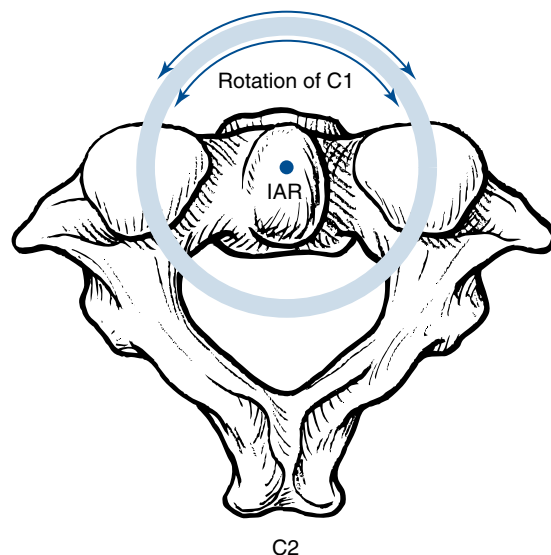


Figure 5-29 The theoretic location of the instantaneous axis of rotation for the atlantoaxial articulation in axial rotation.

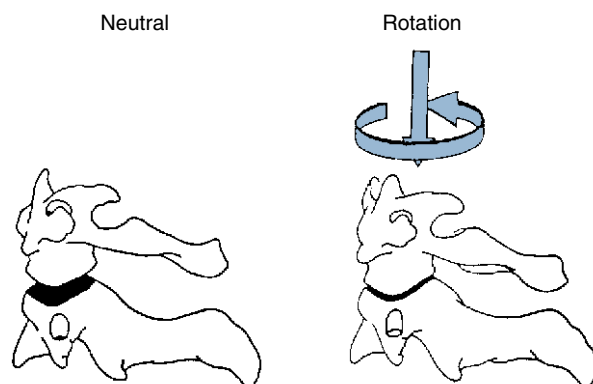


Figure 5-30 Because the articular surfaces are both convex, as the atlas rotates on the axis, a subtle vertical displacement occurs, causing the two segments to approximate one another.

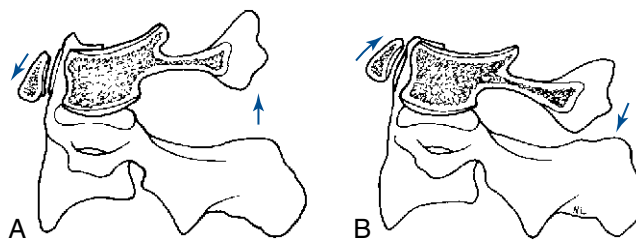


Figure 5-31 Flexion (A) and extension (B) of the atlantoaxial joint.

other words, right lateral flexion of the cervical spine would be associated with translation of C1 to the right (Figure 5-32).

Further clouding the issue is the apparent translation that may be visible on an anterior-to-posterior open-mouth (APOM) radiograph with rotational subluxation of the atlas. Rotational movement of the lateral masses about the odontoid process may induce an apparent lateral translation of the atlas on the APOM

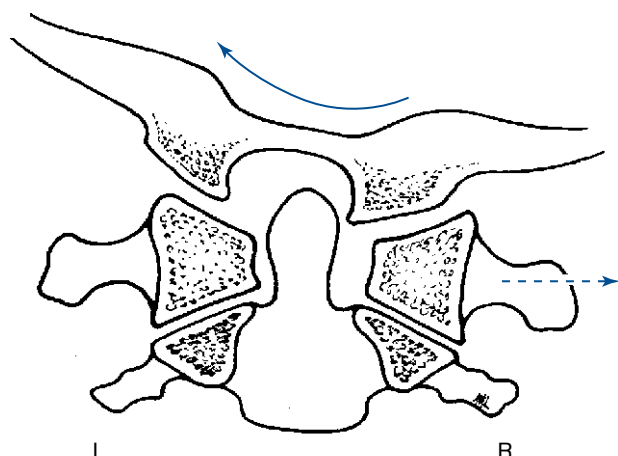


Figure 5-32 Right lateral flexion of the upper cervical spine (*solid arrow*) with translation of the atlas (*broken arrow*) toward the right.

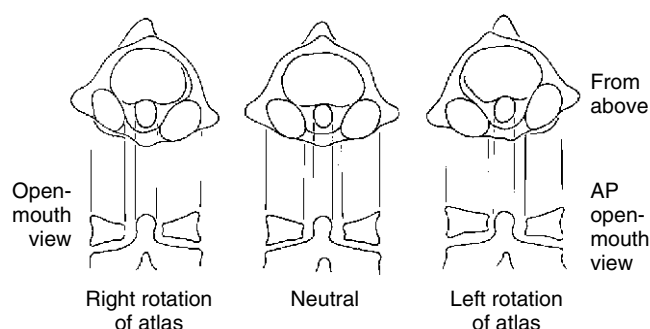


Figure 5-33 Atlas rotation produces a wider lateral mass and narrower appearance of the atlanto-odontal interspace on the side of posterior rotation. This may lead to a false impression of a lateral flexion or translational malposition of the atlas on the anterior-to-posterior open-mouth radiograph.

radiograph as a result of projectional widening and narrowing of the lateral masses (Figure 5-33).

FUNCTIONAL ANATOMY OF THE LOWER CERVICAL SPINE (C3–C7)

The typical cervical vertebrae (C3–C6) possess the same structural parts as all other true vertebrae, plus some unique and distinctive physical features (Figure 5-34). The spinous processes are bifid to allow for better ligamentous and muscular attachment. Each transverse process from C6 upward contains the transverse foramen, allowing for the passage of the vertebral artery. The body of the typical cervical vertebra has anterior and posterior surfaces that are small, oval, and wide transversely. The anterior and posterior surfaces are flat and of equal height. The posterior lateral aspect of the superior margin of the vertebral bodies is lipped, forming the uncinata processes, which serve to strengthen and stabilize the region. The uncovertebral articulations (joints of Von Luschka) are pseudojoints that have a synovial membrane with synovial fluid but no joint capsule (Figure 5-35). They serve as tracts that guide the motion of coupled rotation and lateral

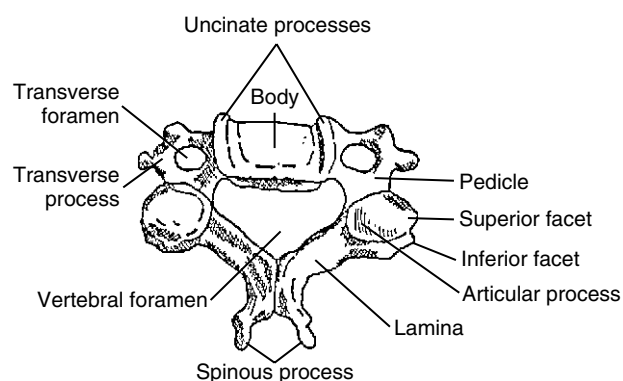


Figure 5-34 Structure of a typical cervical vertebra.

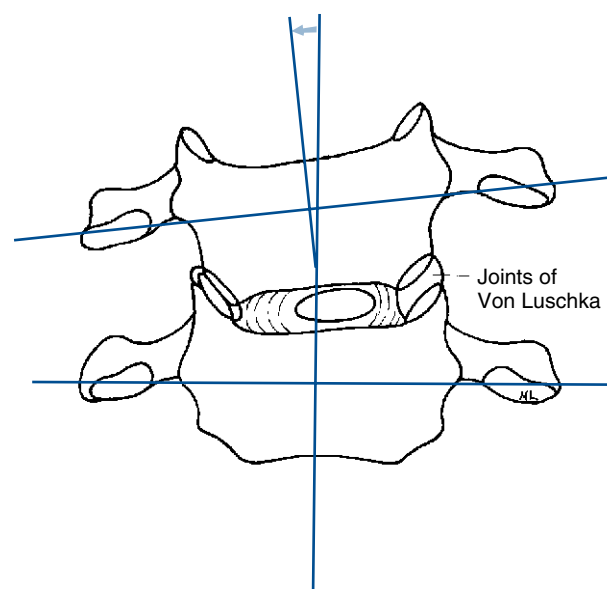


Figure 5-35 The uncinate processes limit pure lateral flexion to only a few degrees while serving as guides to couple lateral flexion with rotation.

flexion. They begin to develop at 6 years of age and are complete by 18 years of age.

The articular facets are teardrop-shaped, with the superior facet facing up and posteriorly and the inferior facet facing down and anteriorly, placing the joint space at a 45-degree angle midway between the coronal and transverse planes (Figure 5-36). The disc height-to-body height ratio is greatest (2:5) in the cervical spine, therefore allowing for the greatest possible ROM (Figure 5-37).

The short and rounded pedicles of cervical vertebrae are directed posterolaterally. The superior and inferior vertebral notches in each pedicle are the same depth. The laminae are long, narrow, slender, and sloping. The intervertebral foramina in this region are larger than in the lumbar or thoracic areas and are triangular in shape.

The C7 vertebra (vertebra prominens) is considered the atypical segment of the lower cervical spine. It demonstrates anatomic characteristics of both the cervical vertebra and the thoracic vertebra. It has a spinous process that is quite long and slender, with a tubercle on its end. The inferior articular processes are similar to those in the thoracic spine, and the superior processes match those of the typical cervical vertebra. C7 has no uncinate processes and no transverse foramen. The transverse processes are large,

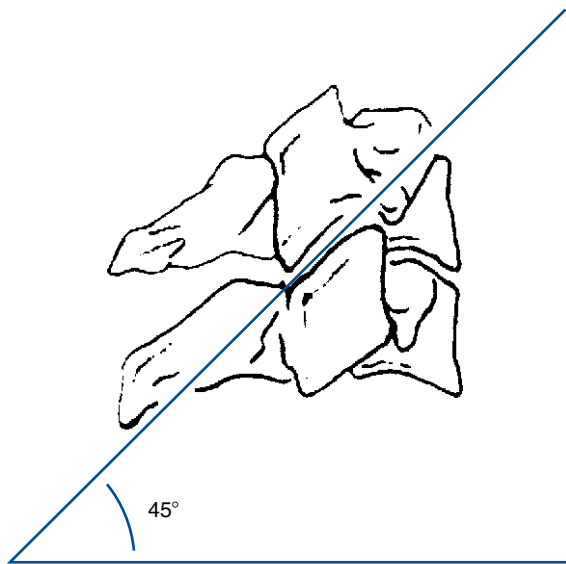


Figure 5-36 The cervical facet planes, demonstrating a 45-degree angle to the horizontal plane.

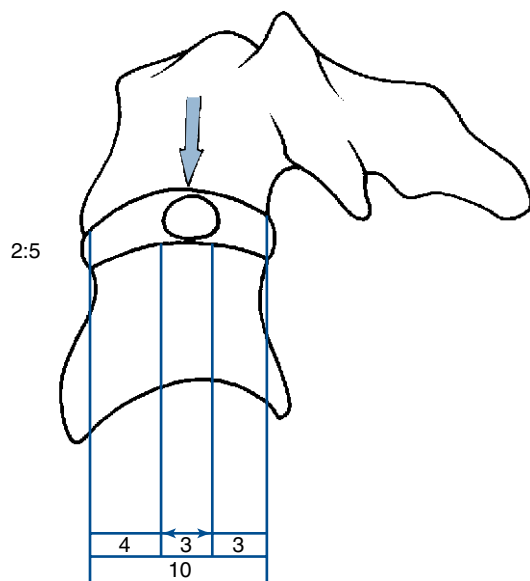


Figure 5-37 The location of the nucleus pulposus and the disc height-to-body height ratio in the cervical spine.

broad, and blunt. The transverse processes may become enlarged or develop cervical ribs, with the potential to create thoracic outlet compromise (Figure 5-38).

Cervical Curve

The cervical spine forms a lordotic curve that develops secondary to the response of upright posture. The functions of the cervical curve and the anterior-to-posterior (A-P) curves throughout the spine are to add resiliency to the spine in response to axial compression forces and to balance the center of gravity of the skull over the spine. The center of gravity for the skull lies anterior to the foramen magnum (Figure 5-39).

The facet and disc planes in large part determine the degree of potential lordosis. Congenital diversity in pillar height and facet

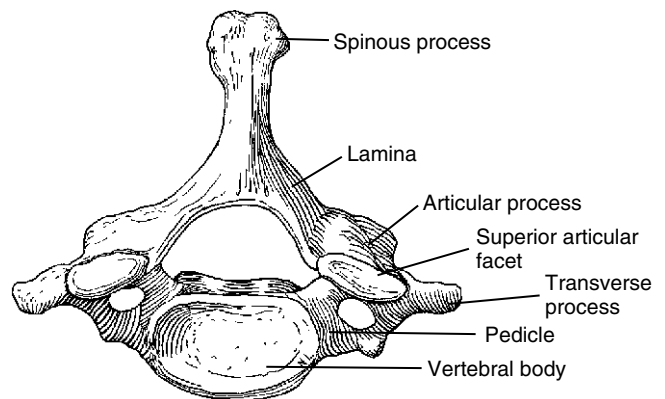


Figure 5-38 The structure of the C7 vertebra (vertebral prominence).

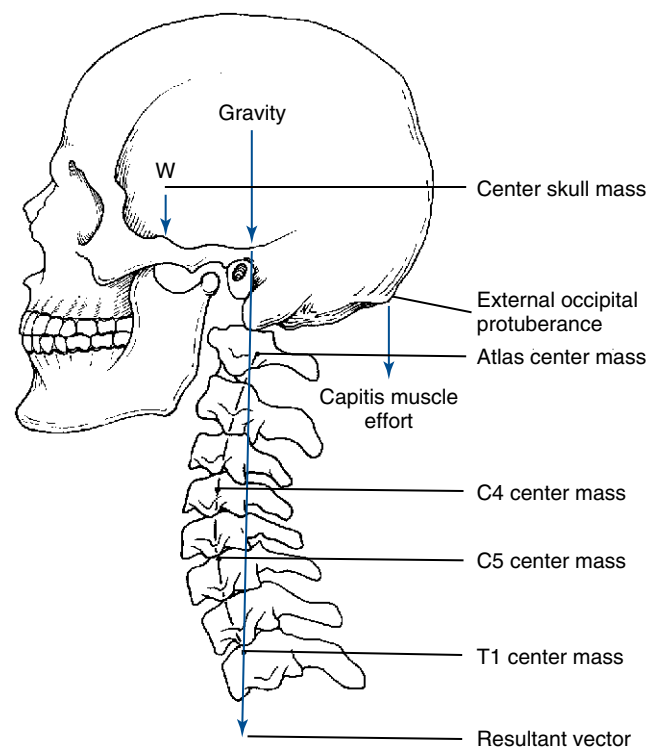


Figure 5-39 The center of gravity for the skull. If the cervical curve changes, the center of gravity shifts.

angulation therefore leads to significant variation in the degree of cervical lordosis present in the population. In addition, degenerative changes or stress responses in these structures may change the “normal” lordosis.

There are a number of opinions as to what the normal cervical curve should be and how it should be measured.^{6,8-14} There is also significant debate on what constitutes an abnormal curve and what biomechanical consequences, if any, will result from alteration in the cervical lordotic curve. A reduced cervical curve (hypolordosis) has the potential to shift more weight onto the vertebral bodies and discs and increase muscular effort as the posterior neck muscles work to maintain head position and spinal stability. An increased cervical curve (hyperlordosis) will potentially increase the compressive load on the facets and posterior elements (Figure 5-40).

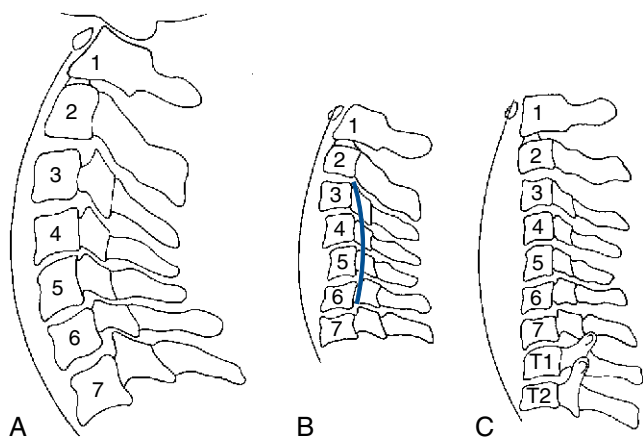


Figure 5-40 The cervical curve extending from C1 to T2. **A**, Normal. **B**, Hypolordosis with a kyphosis involving the middle segments. **C**, Alordotic.

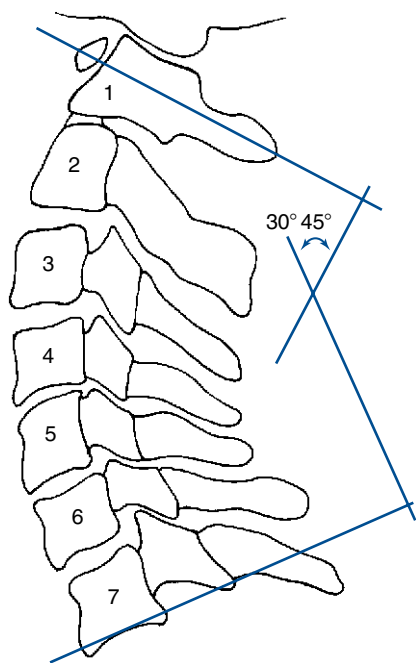


Figure 5-41 The angle of the cervical curve should be about 30 to 45 degrees when measured between lines drawn through C1 and C7.

Various methods for radiographically measuring lordosis have been suggested. The most common method involves direct measurement of the curve by forming an angle between a line extending through the center of C1, with a line drawn along the inferior endplate of C7 (Figure 5-41). Although the cervical lordosis apparently extends to the T1–2 motion segment, measurements commonly use the C7 level as the lowest point reliably viewed on a lateral cervical x-ray film. Another method presented by Jochumsen¹² proposes classifying the cervical curve by measuring the distance from the anterior body of C5 to a line running from the anterior arch of the atlas to the anterior superior aspect of the body of C7 (Figure 5-42). There is some agreement that the cervical curve midpoint is the C5 vertebra (C4–5 interspace).

The proposed optimal curve for the cervical spine can be extrapolated from the mechanical principle that states the strongest

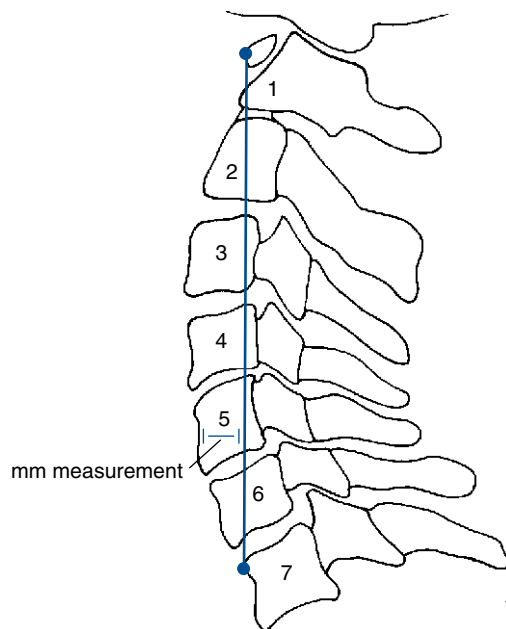


Figure 5-42 Jochumsen's measuring procedure for determining the adequacy of the cervical curve.

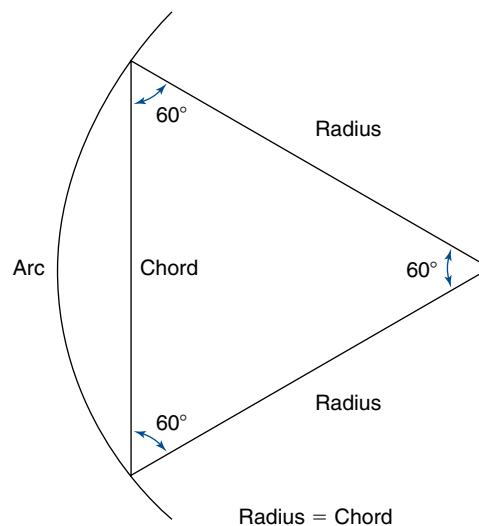


Figure 5-43 Diagram demonstrating the relationship formed when a chord equals the radius of an arc.

and most resilient curve is an arc that has a radius of curvature equal to the chord across the arc (Figure 5-43). The length of the radius, and hence the chord, should equal approximately 7 inches or 17 cm. As the radius increases, the curve increases (flattens, as in hypolordosis) and vice versa.

Range and Pattern of Motion of the Lower Cervical Spine

The lower cervical spine exhibits its greatest flexibility during flexion and extension movements (Table 5-2; see Figure 5-24). Lateral flexion exhibits slightly greater movement than rotation. Both rotation and lateral flexion decrease significantly at the thoracocervical junction.

Flexion and Extension. Movement averages approximately 15 degrees of combined flexion and extension per segment and is greatest at the C5–6 motion segment.¹⁵ Flexion and extension

TABLE 5-2 Segmental Range of Motion for the Lower Cervical Spine*

Vertebra	Combined Flexion and Extension	One-Side Lateral Flexion	One-Side Axial Rotation
C2–3	5 to 16 (10) degrees	11 to 20 (10) degrees	0 to 10 (3) degrees
C3–4	7 to 26 (15) degrees	9 to 15 (11) degrees	3 to 10 (7) degrees
C4–5	13 to 29 (20) degrees	0 to 16 (11) degrees	1 to 12 (7) degrees
C5–6	13 to 29 (20) degrees	0 to 16 (8) degrees	2 to 12 (7) degrees
C6–7	6 to 26 (17) degrees	0 to 17 (7) degrees	2 to 10 (6) degrees
C7–T1	4 to 7 (9) degrees	0 to 17 (4) degrees	0 to 7 (2) degrees

*Numbers in parentheses indicate averages.

Modified from White AA, Panjabi MM: *Clinical biomechanics of the spine*, ed 2, Philadelphia, 1990, JB Lippincott.

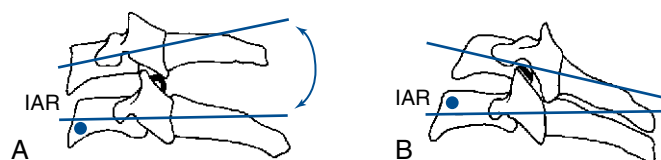


Figure 5-44 Sagittal plane movement of a cervical motion segment in flexion (A) and extension (B), locating the instantaneous axis of rotation and the stair-stepping appearance that occurs with combined tipping and gliding movements.

occur around an axis located in the subjacent vertebra and combine sagittal plane rotation with sagittal plane translation (Figure 5-44). This pattern of combined segmental angular tipping and gliding develops a stairstep effect, which is noted on flexion and extension radiographs.

With flexion, the articular joint surfaces slide apart, producing stretching of the facet joints and posterior disc and anterior disc approximation and compression. With extension, the opposite occurs. The disc is subjected to compression on the concave side and tension on the convex side. The side of the disc subjected to tension retracts and the side subjected to compression bulges.⁵ The net effect of these two opposing forces is to limit shifting of the nucleus pulposus during movements of flexion and extension and lateral flexion (Figure 5-45). Krag and colleagues¹⁶

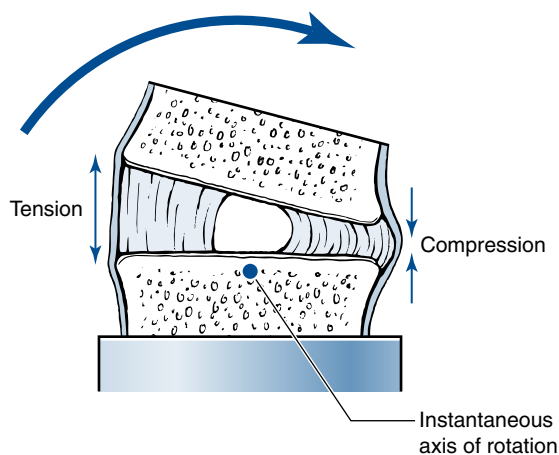


Figure 5-45 Representation of changes in the disc with flexion, as well as extension, or lateral flexion movements.

implanted small metal markers within the lumbar and thoracic IVDs and confirmed the bulging and retraction of the discs during lumbar segmental flexion movements. However, they did note some minor posterior migration of the nucleus that was not identified by previous mathematical models. This phenomenon has not been investigated for the cervical spine.

The coupled translation that occurs with flexion and extension has been measured at approximately 2 mm per segment, with an upper range of 2.7 mm.¹⁷ Translational movements do not occur evenly throughout the cervical spine.¹⁵ For every degree of sagittal plane rotation, more translation occurs in the upper cervical segments than in the lower cervical segments. This leads to a flatter arc of movement in the upper cervical spine (Figure 5-46). Accounting for radiographic magnification, White and Panjabi⁵

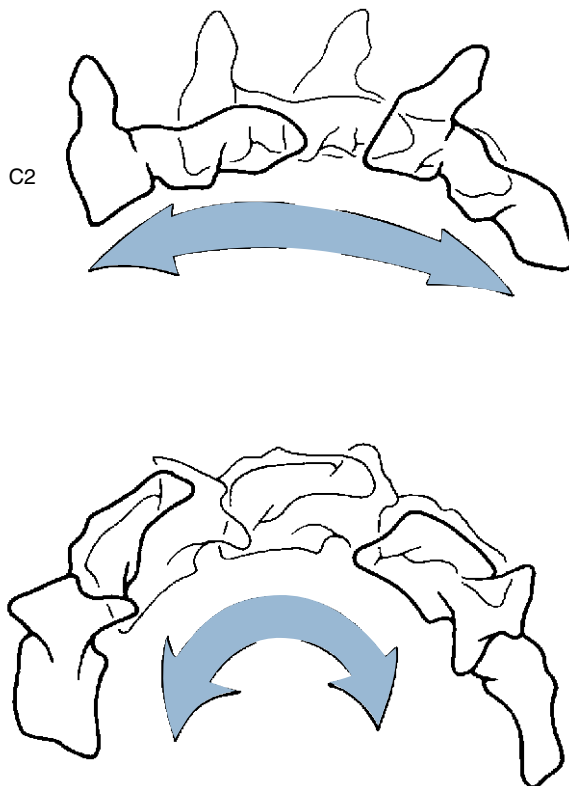


Figure 5-46 With active flexion and extension movements, more translation takes place in the upper segments than the lower segments, leading to a flatter arc of movement.

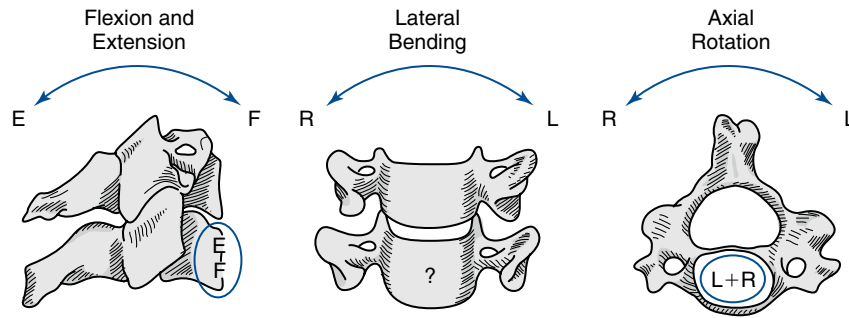


Figure 5-47 The theoretic locations for the instantaneous axis of rotation for each plane of movement in the lower cervical spine. (From White AA, Panjabi MM: *Clinical biomechanics of the spine*, ed 2, Philadelphia, 1990, JB Lippincott.)

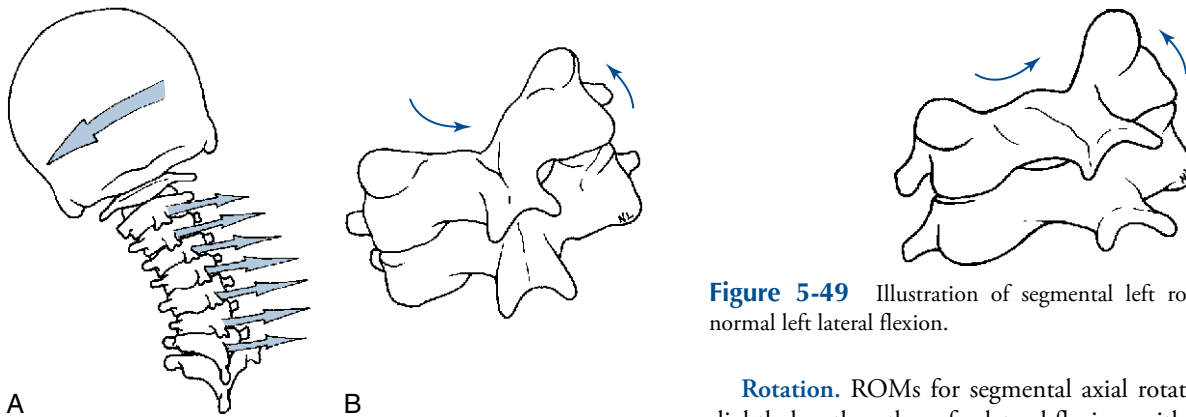


Figure 5-48 A, Left lateral flexion coupled with physiologic left rotation. B, Movement of the facet surfaces with left lateral flexion and coupled left rotation in the lower cervical spine.

have recommended 3.5 mm as the upper end of normal translational movement in the lower cervical segments. Translation beyond 3.5 mm suggests end range segmental instability.

Lateral Flexion. Lateral flexion averages approximately 10 degrees to each side in the midcervical segments, with decreasing flexibility in the caudal segments. The IAR for lateral flexion has not been determined. Speculation places the axis in the center of the subjacent vertebral body (Figure 5-47).

Lateral flexion in the lower cervical spine is coupled with rotation in the transverse plane. The coupling is such that lateral flexion and rotation occur to the same side. This leads to posterior vertebral body rotation on the side of lateral flexion, thereby causing the spinous processes to deviate to the convexity of the curve (Figure 5-48). The degree of coupled axial rotation decreases in a caudal direction.¹⁴ At the second cervical vertebra there are 2 degrees of coupled rotation for every 3 degrees of lateral bending, and at the seventh cervical vertebra there is only 1 degree of coupled rotation for every 7.5 degrees of lateral bending.

During lateral flexion the facets on the side of lateral flexion (concave side) slide together as the inferior facet slides inferomedially because of the coupled rotation. On the opposite side, the facets distract and the inferior facet slides superiorly. The IVD approximates on the side of lateral flexion and distracts on the opposite side.

Figure 5-49 Illustration of segmental left rotation coupled with normal left lateral flexion.

Rotation. ROMs for segmental axial rotation on average are slightly less than those for lateral flexion, with a similar tendency for decreased movement in the lower cervical segments, especially at the C7–T1 motion segments. The axis of rotation is also somewhat speculative and has been placed by Lysell¹⁴ in the anterior subjacent vertebral body (see Figure 5-47).

Rotational movements in the lower cervical spine demonstrate the same coupling as described for lateral flexion. In other words, left or right axial rotation is coupled with lateral flexion to the same side. This leads to a pattern of motion in which, on the side of cervical rotation (posterior body rotation), the inferior facet of the superior vertebra glides posteroinferiorly as the contralateral glides anterosuperiorly (Figure 5-49).

Cervical Kinetics

Nonsegmental muscles produce integrated global movement of the cervical spine as a result of the head's moving in relation to the trunk. Concentric and eccentric muscle activity is combined, with eccentric activity predominating during flexion, extension, and lateral flexion. *Concentric muscle activity* refers to the development of sufficient muscle tension to overcome a resistance, causing the muscle to visibly shorten and the body part to move. However, *eccentric muscle activity* occurs when a given resistance overcomes the muscle tension, causing the muscle to actually lengthen. Relaxation of a muscle against the force of gravity, creating a deceleration of the moving body part, is an example of eccentric muscle activity.¹⁵

The segmental (intrinsic) muscles function to coordinate and integrate segmental motion. The intrinsic muscles act as involuntary integrators of overall movement. Movements of the head initiate normal movements of the cervical spine, but with conscious

effort, movement may be initiated at lower segmental levels. They operate by the same concentric and eccentric principles as the larger nonsegmental muscles.

Flexion is initiated by anterior cervical muscles and controlled or limited by eccentric activity of the semispinalis, longissimus, and splenius muscle groups. Flexion is further limited by the elastic limits of myofascial tissue, nuchal ligament, joint capsule, PLL, ligamentum flavum, posterior IVD, anterior vertebral bodies, and the chin hitting the chest.

Posterior cervical muscles controlled or limited by the eccentric activity of the sternocleidomastoid (SCM), scaleni, and longus coli muscle groups initiate extension. Extension is further limited by the elastic limits of the myofascial tissue, anterior IVD, ALL, joint capsule, posterior vertebral bodies, and articular pillars.

Lateral flexion is initiated by ipsilateral contraction and controlled or limited by the contralateral eccentric activity of the splenius capitis, semispinalis cervicis, and longus coli muscle groups. Lateral flexion is further limited by the elastic limits of some myofascial tissue, contralateral joint capsule, periarticular ligaments, flaval ligament, IVD, ipsilateral joint capsule, and ipsilateral articular pillars.

Rotation is initiated by concentric contraction of the ipsilateral splenius capitis and cervicis, longissimus cervicis, and contralateral semispinalis muscles. Eccentric muscle contraction occurs simultaneously to guide and break movements and involves action of the contralateral splenius capitis, cervicis, longissimus cervicis, and ipsilateral semispinalis and scaleni muscles. Movement is further limited by capsular and periarticular ligaments and segmental muscles.

EVALUATION OF THE CERVICAL SPINE

Observation

Examination of the cervical spine begins with a visual examination of the alignment and ROM of the cervical spine in the sagittal, coronal, and transverse planes. Alignment in the coronal plane is evaluated by observing the orientation of the head relative to the trunk and shoulders, the leveling of the mastoid processes, and the symmetry of the cervical soft tissues. Observing the status of the cervical curve and orientation of the patient's chin assesses sagittal plane alignment. Tucking or elevation of the chin in the presence of a normal cervical curve may indicate upper cervical dysfunction. Observing the patient from the posterior and noting any turning of the head (Figure 5-50) may assess orientation of the head in the transverse plane.

Global ROM is most effectively evaluated in the sitting position. Take care to observe for recruitment of trunk movement and stabilize the shoulders if necessary. During flexion the patient should be able to touch the chin to the chest, and during extension look straight toward the ceiling. During rotation the patient should be able to approximate the chin to the shoulder, and during lateral flexion approximate the ear to within two to three fingers'-width of the shoulder (Figure 5-51). Variations with sex and age are quite common. If ROM is evaluated in circumstances other than screening evaluations, it should be conducted

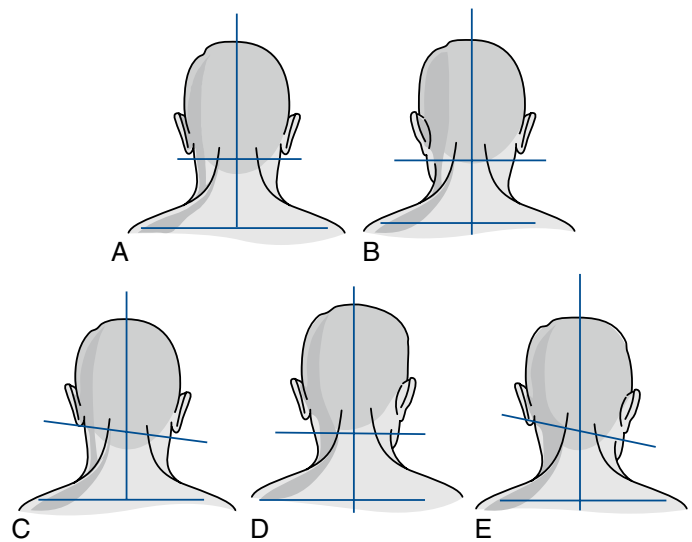


Figure 5-50 Common cervical postural presentations. **A**, Normal. **B**, Occiput in left posterior rotation. **C**, Occiput in right lateral flexion or atlas in left laterolisting. **D**, Atlas in right rotation. **E**, Axis in rotation and right lateral flexion. (Modified from Pratt NE: *Clinical musculoskeletal anatomy*, Philadelphia, 1991, JB Lippincott.)

with the use of inclinometry for more accurate recordings of the ranges (see Figure 3-11 and Table 5-3).

Static Palpation

Palpation for alignment, tone, texture, and tenderness of the bony and soft tissue structures of the neck is conducted with the patient in the supine or sitting position. During supine evaluation, stand or kneel at the head of the table, and during the seated evaluation, stand behind the patient.

Upper Cervical Spine. The suboccipital muscles are evaluated by using the palmar surfaces of the fingertips to make a bilateral comparison of tone, texture, and tenderness (Figure 5-52). Bony alignment of the atlanto-occipital joint is evaluated by placing the tip of the index finger in the space between the mandibular ramus and the anterior tip of the atlas transverse process and between the inferior tip of the mastoid process and the atlas transverse process (Figure 5-53).

Spacing between the atlas transverse process and the mandibular ramus and between the atlas transverse process and mastoid processes should be symmetric on both sides. Malpositions between C0 and C1 can affect the spacing between the mandibular ramus and the C1 transverse process. The space between the angle of the jaw and atlas transverse processes may be closed on the side of posterior occipital rotation and open on the contralateral side. With lateral flexion, the mastoids may be unlevel, and a decreased spacing may be noted in the interspace between the atlas transverse process and mastoid process.

Bony alignment of the atlantoaxial joint is evaluated by comparing the relative alignment of the atlas transverse processes and axis articular pillars. This is accomplished by establishing bilateral contacts with the doctor's index and middle fingers



Figure 5-51 Cervical global range of motion. **A**, Flexion. **B**, Extension. **C**, Right lateral flexion. **D**, Left rotation.

TABLE 5-3 Global Range of Motion for the Cervical Spine		
Motion	Normal Range	Range Without Impairment
Flexion	60–90 degrees	60 degrees
Extension	75–90 degrees	75 degrees
Lateral flexion	45–55 degrees	45 degrees
Rotation	80–90 degrees	80 degrees



Figure 5-52 Palpation of suboccipital muscle tone and texture.

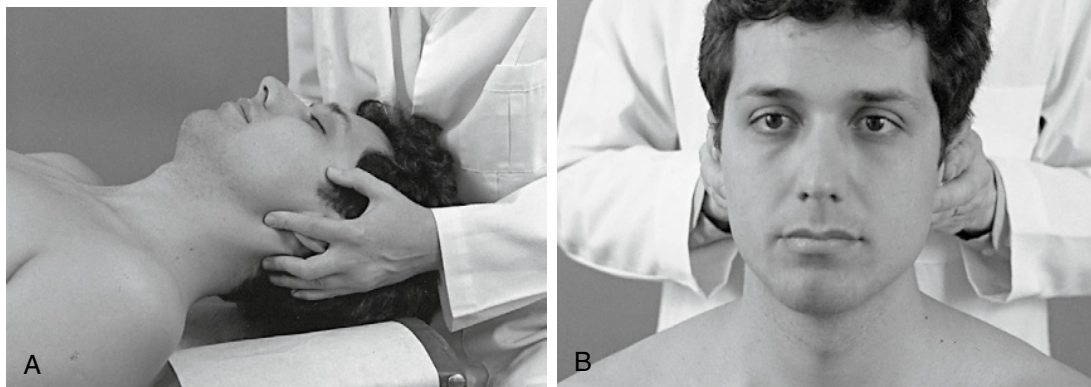


Figure 5-53 Palpation for flexion, extension, or rotation alignment (A) and lateral flexion alignment of the atlanto-occipital articulation (B).



Figure 5-54 Palpation for rotation and lateral flexion alignment of the atlanto-axial articulation.



Figure 5-55 Palpation for the alignment of the spinous processes in the lower cervical spine.

over each structure (Figure 5-54). Posterior prominence of the atlas or palpable stair stepping of the atlas and axis transverse processes indicates possible rotational malposition of the atlas. Lateral prominence of the atlas or narrowing of the lateral atlas-axis interspace indicates possible lateral flexion malposition of the atlas.

Asymmetry in suboccipital muscle tone and tender and taut suboccipital muscles are further indications of possible upper cervical joint dysfunction. However, the upper cervical spine is at the end of a kinetic chain, and asymmetries in tone and alignment are commonly encountered. They may be normal variations or sites of compensational adaptation instead of primary joint dysfunction.

Lower Cervical Spine (C2–C7). Palpating the spinous process, interspinous spaces, and posterior articular pillars assesses bony contour, tenderness, and alignment. In the sitting position, the interspinous spaces may be palpated with the middle finger while the index and ring fingers lay along the lateral margins to compare alignment of adjacent spinous processes (Figure 5-55). The spinous processes are bifid and difficult to palpate in the midcervical spine. They become more accessible if the neck is placed in a slight flexion. The articular pillars are not as accessible to direct palpation but are probably a more reliable landmark for detecting rotational malpositions.

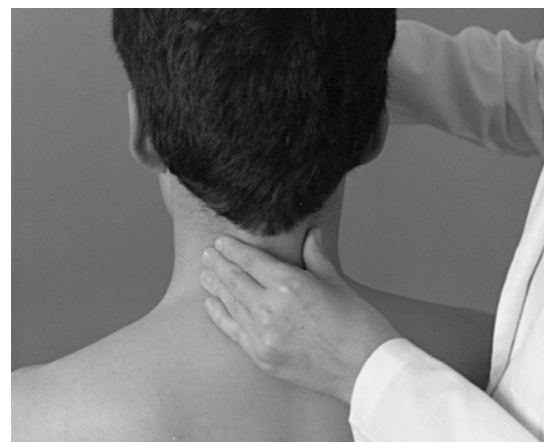


Figure 5-56 Palpation for the alignment of the articular pillars in the midcervical spine.

To evaluate the alignment of the articular pillars and the tone, texture, and tenderness of the paraspinal soft tissues, establish segmental contacts on each side of the spine. If the patient is in the sitting position, use the thumb and index fingers (Figure 5-56); if the patient is in the supine position, use the palmar surfaces of the fingers of both hands to make a bilateral comparison.

Motion Palpation

Cervical JP, segmental ROM, and end play may be evaluated with the patient in either a sitting or a supine position. Stand behind the seated patient or sit, kneel, or squat at the head of the table during a supine evaluation.

Joint Play. To evaluate JP and P-A glide with the patient in the seated position, position the patient's neck in a neutral position and establish segmental contacts bilaterally over the posterior joints, with the palmar surfaces of the index finger and thumb. With the patient's forehead supported by the IH, gently spring each individual motion segment in a fluid P-A gliding motion along the horizontal plane (Figure 5-57).

In the supine position, the doctor assesses P-A glide by contacting the posterior joints with the palmar surfaces of the fingertips. The patient's head rests on the table while the fingertips spring posteriorly to anteriorly against the articular pillars (Figure 5-58). Contact the posterolateral surface of adjacent vertebrae with the radial or palmar

surface of the doctor's index fingers to assess lateral-to-medial (L-M) glide. Testing is performed by springing toward the midline with one hand as the other hand counterstabilizes (Figure 5-59).

During P-A JP assessment, the doctor should feel a subtle gliding and recoil at each segment tested. The movement should be uniform on each side and pain free; unilateral resistance or a tendency for the spine to rotate out of the sagittal plane may indicate segmental dysfunction. L-M glide is less giving than A-P glide, and a perceptible decrease in movement should be noted when the adjacent vertebra is counterstabilized. Excessive sponginess and lack of elastic resistance with either procedure indicates possible hypermobility or instability.

Segmental Range of Motion and End Play (C0–1)

C0–1 Flexion and Extension. Atlanto-occipital flexion and extension may be evaluated by placing the tip of the index finger in the space between the mandibular ramus and the anterior tip of the atlas transverse process. The doctor's IH supports the top of the head in the sitting position and cups the patient's contralateral occiput and mastoid in the supine position. The patient's chin is elevated and tucked to instill extension and flexion in the upper cervical spine. The space between the mandibular ramus and atlas transverse process opens during extension and closes during flexion (Figure 5-60). Fixation in this plane leads to a loss of rolling of the occiput on the atlas and unchanged spacing between the angle of the jaw and the atlas transverse process.

To evaluate end play, apply additional springing overpressure at the end ROM. For flexion, the contacts are established under the inferior rim of the occiput, with pressure applied upward.



5-57

Figure 5-57 Sitting joint play evaluation for posterior-to-anterior glide in the midcervical spine.



Figure 5-58 Supine joint play evaluation for posterior-to-anterior glide in the midcervical spine.



Figure 5-59 Supine joint play evaluation for lateral-to-medial glide. The doctor contacts adjacent levels and applies medial-to-lateral pressure with the cephalad hand as the caudad hand counterstabilizes.

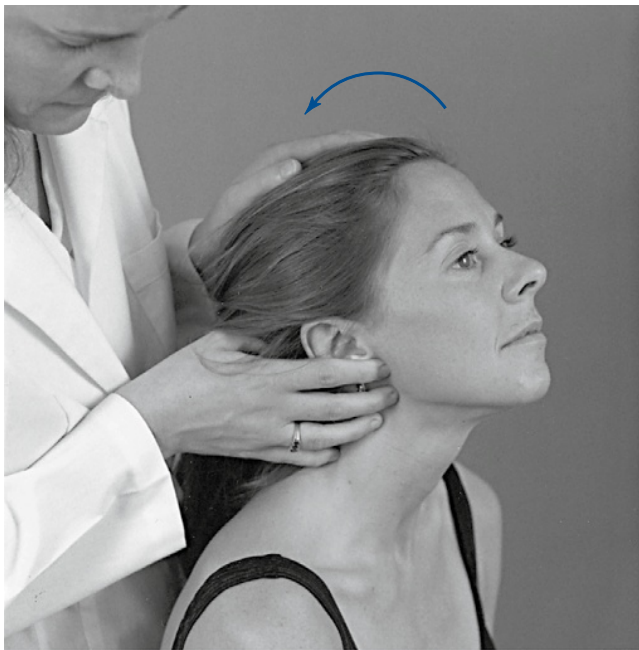
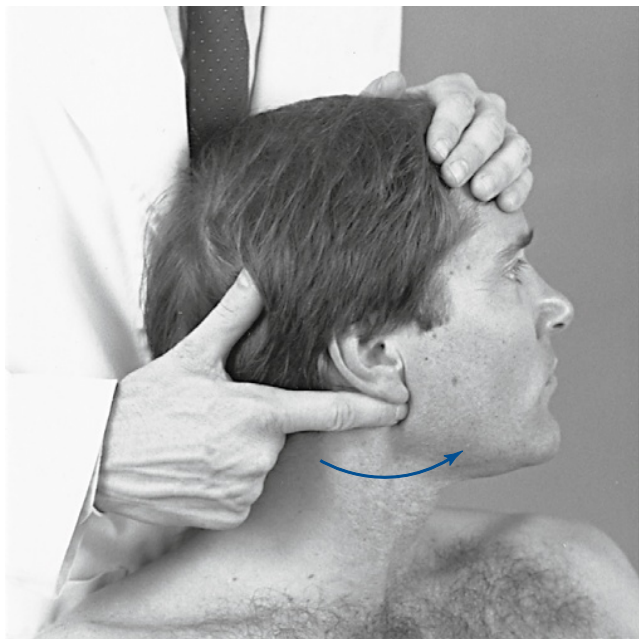


Figure 5-60 Palpation for extension movement of the right atlantooccipital articulations.

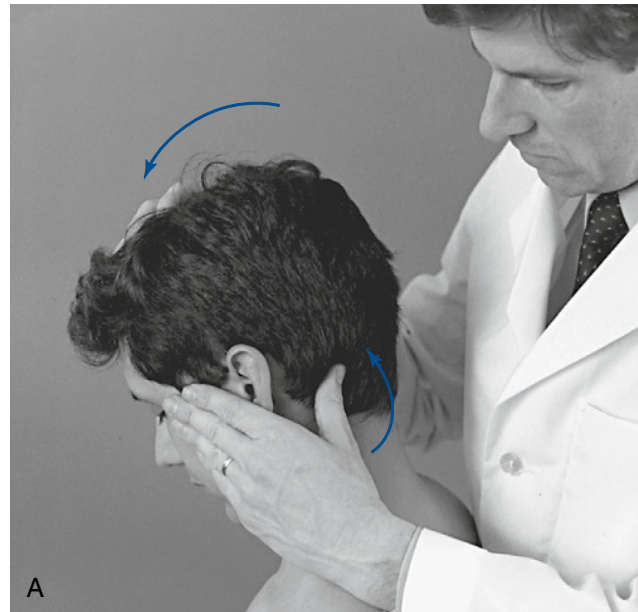


5-62

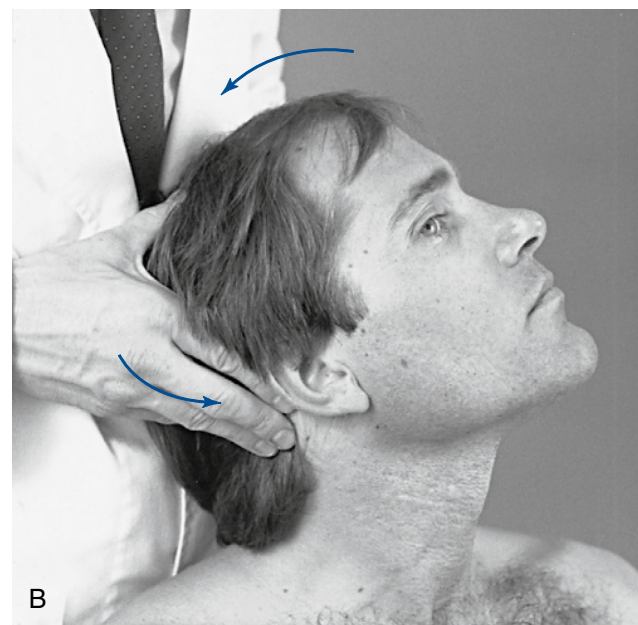
Figure 5-62 Palpation of left rotation at the atlantooccipital articulation.

For extension, the contacts are established over the mastoid and posterior occiput, with pressure applied forward and downward (Figure 5-61). Flexion end play has a firmer quality because it is limited by the strong posterior neck muscles.

C0–1 Rotation (P-A glide). The index finger is placed between the mandibular ramus and the anterior tip of the atlas transverse process. The head is passively rotated away from the side of contact. The gap between the mandibular ramus and the atlas



A



B



5-61A, B

Figure 5-61 End-play evaluation of the atlantooccipital articulation. **A**, Flexion. **B**, Extension.

transverse process opens on the side opposite rotation and close on the side of rotation. Occipital rotation is limited and occurs at the end of cervical rotation (Figure 5-62).

C0–1 Lateral Flexion. Lateral flexion is evaluated by placing the index finger between the inferior tip of the mastoid process and the atlas transverse process (Figure 5-63). This interspace is difficult to locate because of its small size and the overlying musculature. The head is laterally flexed away from the side of contact, and the gap between the mastoid and atlas transverse should open on the side opposite the lateral flexion. End play is evaluated on the side of lateral flexion for medial glide. To evaluate medial glide, the doctor contacts the posterior lateral aspect of the occiput with the lateral surface of the index finger (or fingertips of the index and middle fingers). The patient's head is

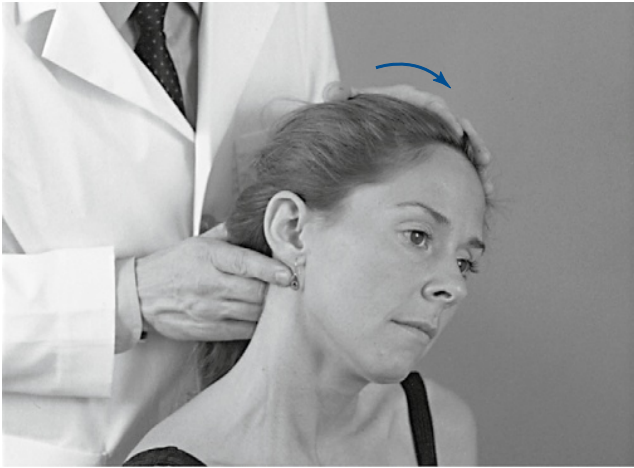


Figure 5-63 Palpation of left lateral flexion at the atlanto-occipital articulation.

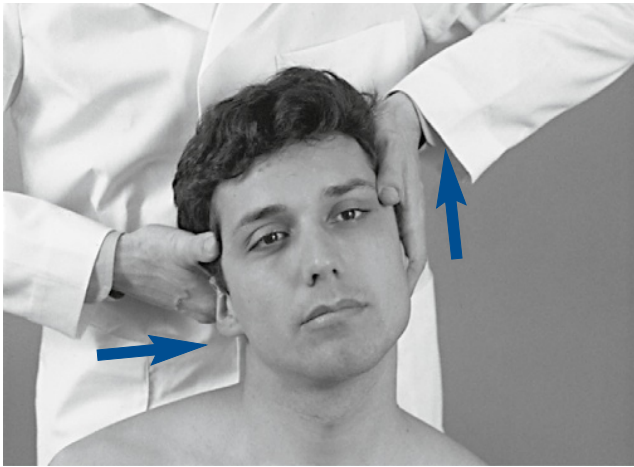


Figure 5-64 Palpation of right lateral flexion end play of the atlanto-occipital articulation. The doctor's right hand applies medial pressure while the left hand distracts superiorly.

laterally flexed toward the side of contact while the doctor springs medially at the end ROM (Figure 5-64).

Segmental Range of Motion and End Play (C1–2). C1–2 Rotation (*Posterior-to-Anterior Glide*). To evaluate atlantoaxial rotation, contact the posterior lateral aspect of the transverse process of the atlas and axis overlapping the C1–2 intertransverse space with the palmar surfaces of the middle and index fingers. The contacts are established on the side opposite cervical rotation, and the head is laterally flexed a few degrees toward the contact and passively rotated away from the side of contact (Figure 5-65). The doctor should palpate anterior rotation of C1 and separation of the C1–2 intertransverse space on the side opposite rotation (side of contact). At the end of passive rotation, evaluate end play by applying forward overpressure against the transverse process of C1. The atlantoaxial articulation lacks a strong interlaminar ligament and has a loose joint capsule, allowing for a comparatively flexible end play.

C1–2 Medial Glide. Establish an index contact on the lateral surface of the atlas transverse process. The patient's head is laterally flexed toward the side of contact while medial springing pressure



Figure 5-65 Palpation of left rotation at the atlantoaxial joint and anterior-to-posterior glide of the right C1 articular pillar.



Figure 5-66 Palpation of right-to-left medial glide of C1.

is applied against the atlas transverse process (Figure 5-66). Lateral flexion at the atlas is limited; this procedure is designed to assess the small degree of medial glide that should be present, not the active range of lateral flexion.

C1–2 Flexion and Extension. Establish bilateral contacts over the C1–2 articulation. The structures are deep and difficult to directly palpate. They are identified by a sense of fullness through the soft tissues. Use the index and middle fingers on one side and a thumb contact on the other. The patient's head is flexed and extended at the C1–2 articulation. Palpate for posteroinferior (PI) glide of the atlas during extension and AS glide during flexion (Figure 5-67). At the end of passive motion, evaluate end play by springing antero-superiorly for flexion and antero-inferiorly for extension. The end play is elastic but firm compared with rotation end play.

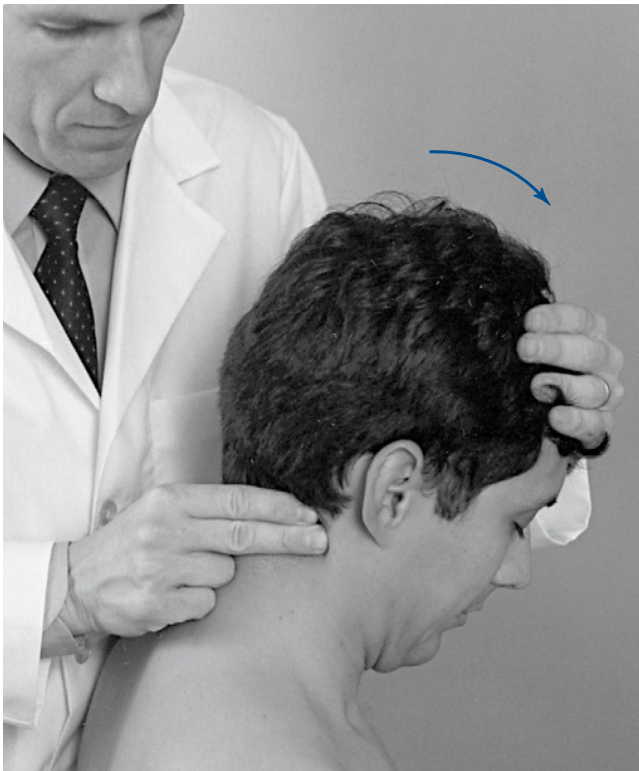


Figure 5-67 Palpation of flexion at the atlantoaxial articulation.

Segmental Range of Motion and End Play (C2–C7). The lower cervical spine may be evaluated with the patient in the sitting or supine position. In the sitting position, the doctor controls movement by contacting the patient's forehead or the crown of the patient's head. In the supine position, passive movement is controlled by cupping the patient's contralateral occiput and mastoid.

Rotation (Posterior-to-Anterior Glide). Evaluate movement by placing the palmar surface of the index or index and middle fingers over the articular pillars. With the patient in the sitting position, use either an upright (palm up) or reverse (palm down) hand contact method (Figure 5-68). Establish palpation contacts on the posterior surface of the articular pillars on the side opposite cervical rotation. The patient's head is passively rotated away from the side of contact. The superior pillar should move forward relative to those below, and the soft tissues should elongate under the contact. With full rotation you should note a stair-stepping effect from the lower to upper cervical spine. At the end of passive motion, evaluate end play by springing from the posterior to the anterior, along the facet planes, normally encountering firm and elastic but giving end play.

Rotation (Anterior-to-Posterior Glide). When evaluating this motion with the patient in the sitting position, stand behind the patient, opposite the side of contact. Establish a soft contact with the ventral surface of the index and middle fingers over the anterolateral (AL) surface of the articular pillars. Take care to avoid excessive pressure over the anterior neurovascular structures. The stabilization hand contacts the patient's forehead or the top of the patient's head. The patient's head is laterally flexed away and rotated toward the side of contact to induce A-P gliding and gapping of the articulations under the area of contact. In addition

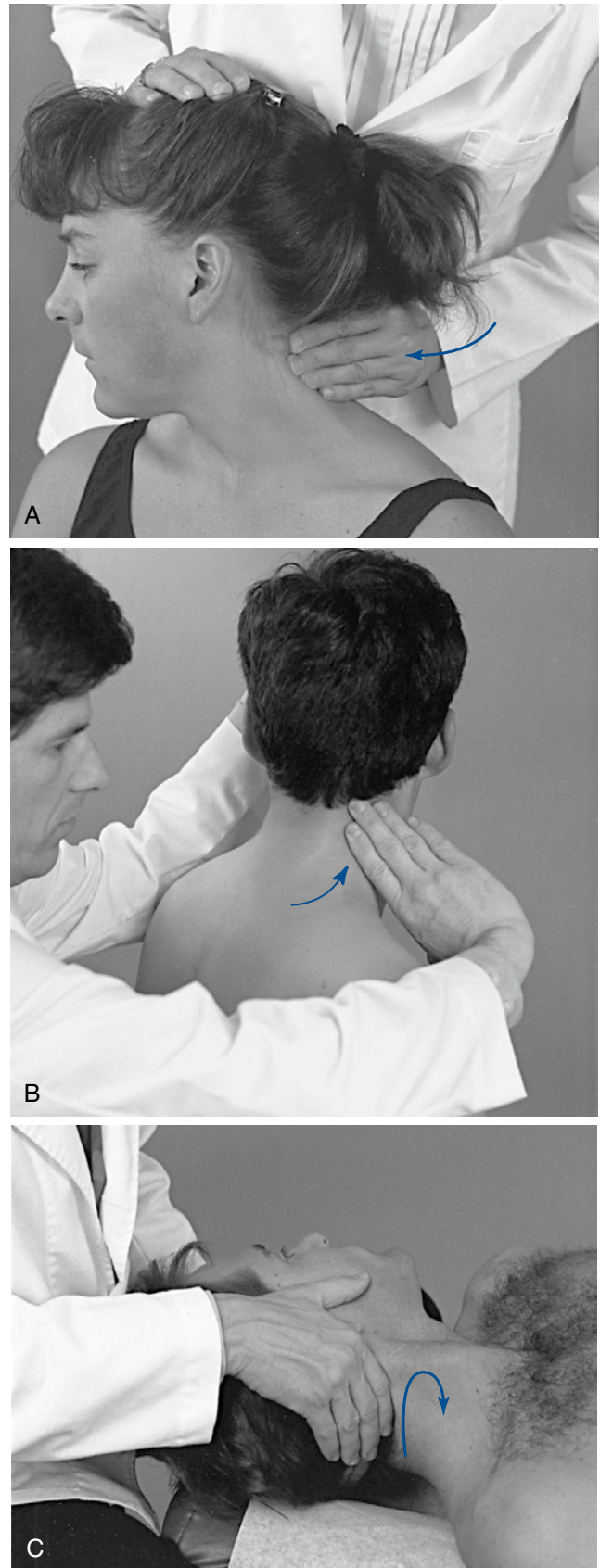


Figure 5-68 Palpation of rotation and posterior-to-anterior glide at the C3–4 articulation. **A**, Right rotation with the palm down. **B**, Left rotation with the palm up. **C**, Left rotation in the supine position.

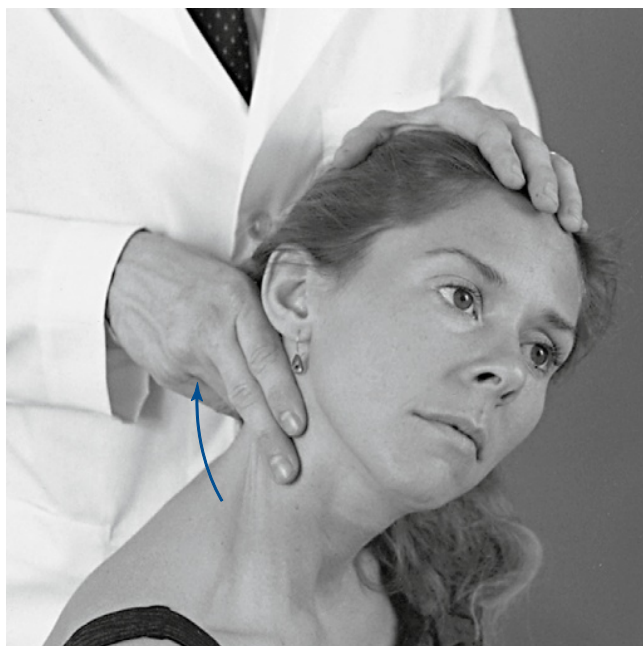


Figure 5-69 Palpation of right rotation and anterior-to-posterior glide at the right C2–3 articulation in the seated position.



Figure 5-70 Palpation of right rotation and anterior-to-posterior glide at the right C2–3 articulation in the supine position. Doctor's right thumb establishes the palpation contact over the right C2–3 articulation.

to the A-P palpation vector (VEC), a slight inferior-to-superior (I-S) orientation should be maintained so that the palpation VEC is at a 90-degree angle to the facet plane (Figure 5-69). Evaluate end-play motion by springing from the anterior to the posterior along the same palpation VEC. You may also perform this method with the patient in the supine position by establishing a soft anterior lateral pillar contact with the thumb of your hand corresponding to the side of palpation (Figure 5-70).

C2–C7 Lateral Flexion. To assess lateral flexion, establish segmental contacts over the articular pillars slightly posterior to the midcoronal plane. If the contacts are placed too far anteriorly, they

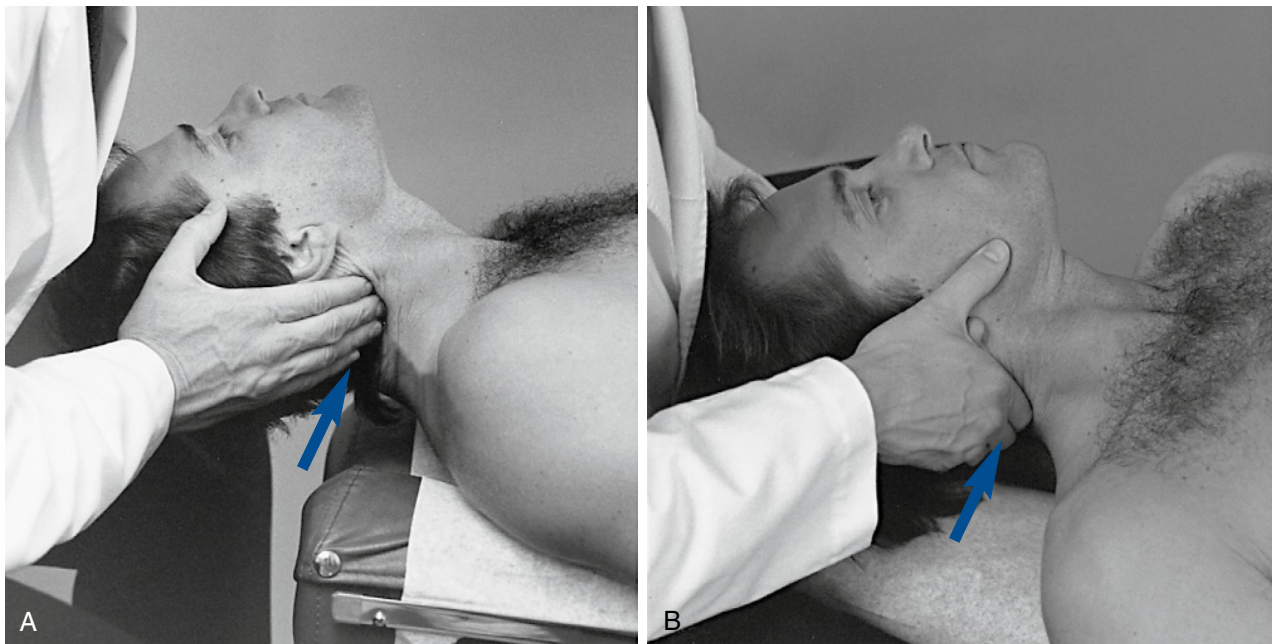


Figure 5-71 Palpation of right lateral flexion of the C5–6 joint.

can become uncomfortable to the patient. Segmental contacts may be established unilateral or bilaterally. They may be established with the index and middle fingers of one or both hands or with the fingers and thumb of the same hand. When using unilateral fingertip contacts, stand to the side opposite the contact and change palpation hands and sides as you evaluate movement to each side (Figure 5-71). With the patient in the supine position, kneel at the head of the table and use bilateral fingertip or index contacts (Figure 5-72).

During the assessment of lateral flexion, palpate bending of the articular pillar on the side of lateral flexion and elongation of the soft tissues on the side opposite the lateral flexion (see Figure 5-71). At the end of passive motion, evaluate end play by applying additional overpressure by pushing toward the midline from the side of lateral flexion (concave toward convex). The VEC should incorporate an inferior inclination to avoid compressing the soft tissues. The end-play quality for lateral flexion is similar to that of rotation—firm but giving and elastic.

C2–C7 Flexion and Extension. To evaluate segmental flexion and extension, establish bilateral or unilateral contacts over the posterior articular pillars. Establish the segmental contacts with the fingertips or with the fingertips and thumb of the same hand (Figure 5-73). During extension, palpate PI gliding of the articular pillars. During flexion, palpate AS gliding of the articular pillars. Evaluate flexion end play by applying additional overpressure in an AS direction, and evaluate extension by applying additional overpressure through the palpation hand in an anterior direction. Extension movement is perceived as anterior glide and an increase in the cervical lordosis and flexion as posterior movement and reversal of cervical lordosis. The quality of end play for flexion is more resistant as a result of strong posterior neck muscles.



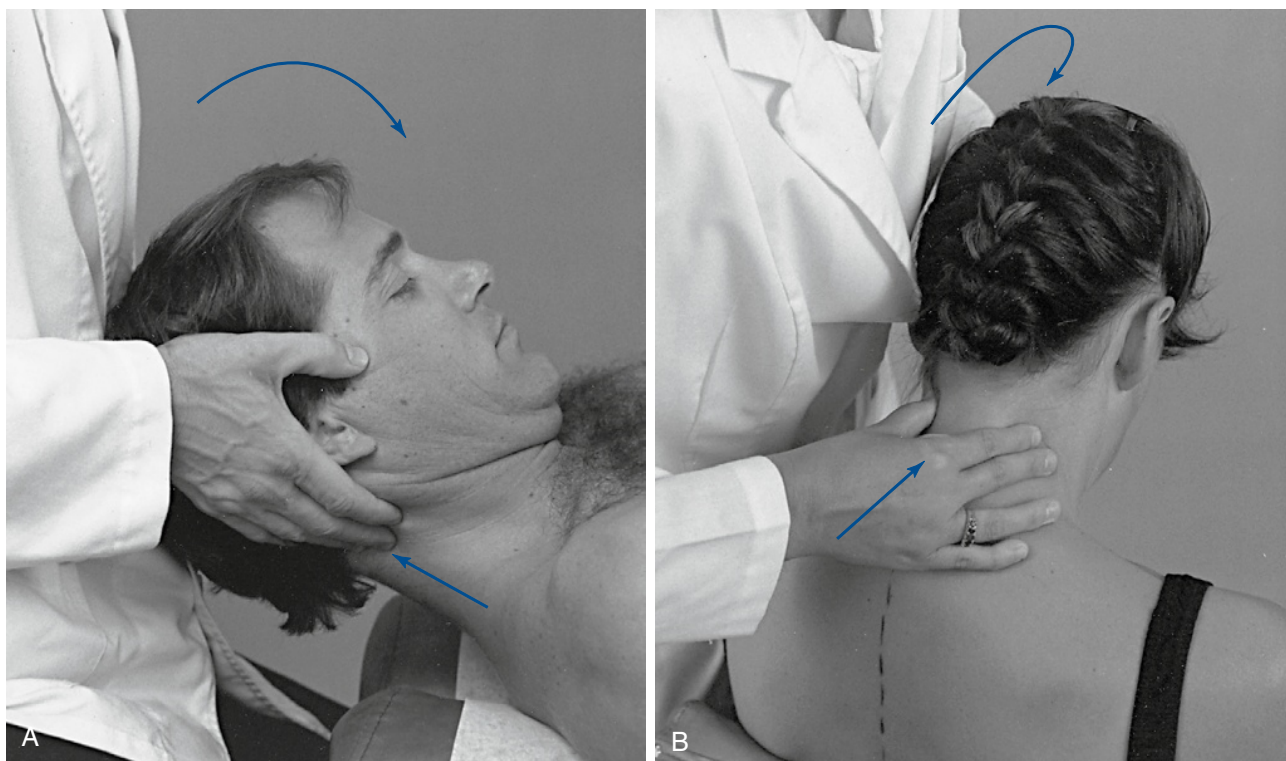
5-72B

Figure 5-72 Palpation of right lateral flexion of C5–C6 in the supine position with a fingertip contact (A) and an index contact (B) of the right C5–C6 articulation.

OVERVIEW OF CERVICAL SPINE ADJUSTMENTS

The cervical spine is flexible and composed of small structures. It is easy to overpower the neck, so caution must be used in the delivery of cervical adjustments. Adjustments of the cervical spine are performed with the patient in sitting, prone, and supine positions. Most techniques involve adjustive positions that produce movement of head and motion segments in the direction of joint

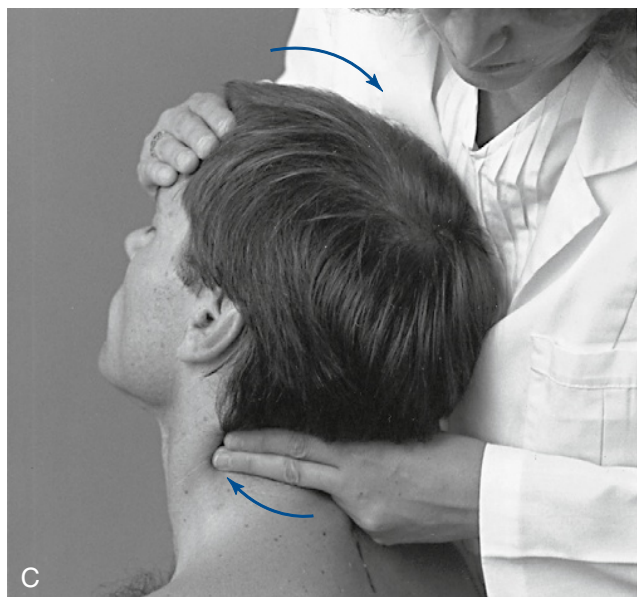
restriction and adjustment. Therefore the majority of the adjustments presented (assisted methods) are applied to develop tension in the motion segments inferior to the level of segmental contact. Resisted methods are used less frequently. When resisted methods are applied, they are typically used in the treatment of rotational dysfunction. Resisted cervical or thoracocervical adjustments are applied to develop maximal tension in the motion segments superior to the level of established contact.



5-73B

Figure 5-73 Palpation of cervical flexion at the C3–4 motion segment in the supine position (A) and the seated position (B).

(Continued)



5-73C

Figure 5-73—Cont'd C, Palpation of cervical extension at the C3–4 articulation.

Rotational Dysfunction

Rotational dysfunction of the cervical spine has been postulated to result from loss of anterior glide of the facets on the side opposite the direction of rotation restriction (side of posterior body rotation) or posterior movement and gapping on the side of rotational restriction (Figure 5-74). The side and site of fixation is assessed by determining the side of subjective and palpable discomfort and comparing the end-play quality of P-A glide on one side to the A-P glide on the other.

Dysfunction may be treated with assisted methods by contacting the posterior pillar of the superior vertebra on the side of posterior body rotation (side opposite the rotation restriction). In the lower cervical spine, the adjustive thrust would be directed anteriorly (Figure 5-75).

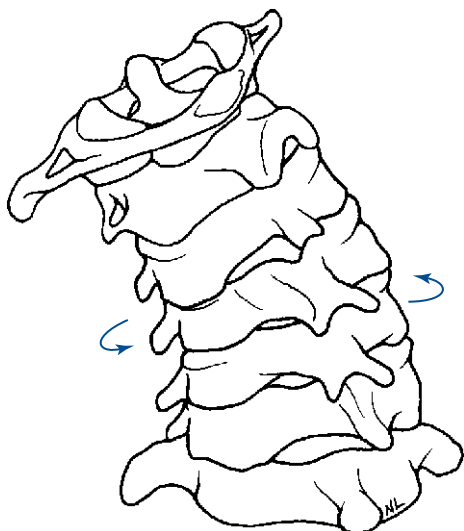
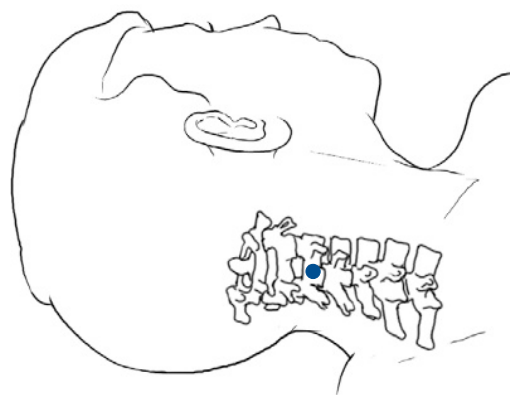


Figure 5-74 Posterior view of the cervical spine, illustrating left rotation characterized by anterosuperior glide of the right facet joints and posteroinferior glide of the left facet joints.



5-75

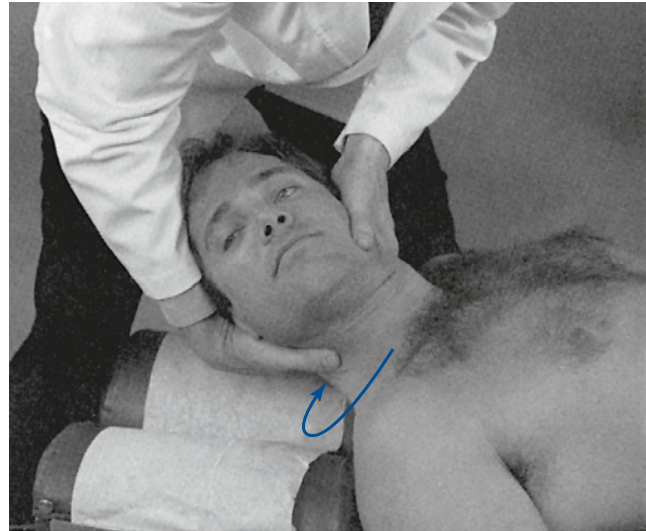
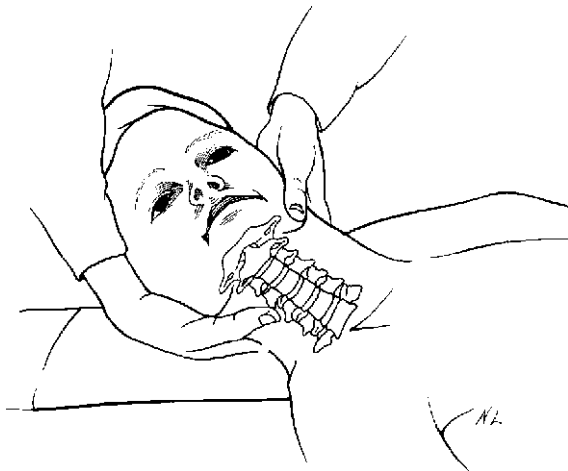
Figure 5-75 Index contact applied to the right C3 articular pillar (*dot*), with adjustive force directed anterosuperiorly (*arrow*) to induce left rotation of the right C3–4 motion segment.

Methods directed at inducing posterior glide and gapping on the side of restricted rotation may be treated with either assisted or resisted methods. In both techniques, the cervical spine is laterally flexed away from the side of contact to lock the contralateral joints and distract the joint to be adjusted. With assisted methods, the contacts are established on the ipsilateral-anterolateral pillar of the superior vertebra on the side of rotational restriction (side opposite the posterior body rotation). The adjustive thrust is directed in a posterior direction (Figure 5-76). When resisted methods are applied, the contacts are established on the spinous process of the inferior vertebra on the side opposite the rotation restriction (inferior vertebra on the side of posterior body rotation). The adjustive thrust is directed medially through the spinous contact, resisted by the counter-rotational positioning of the patient's head (Figure 5-77).

Lateral Flexion Dysfunction

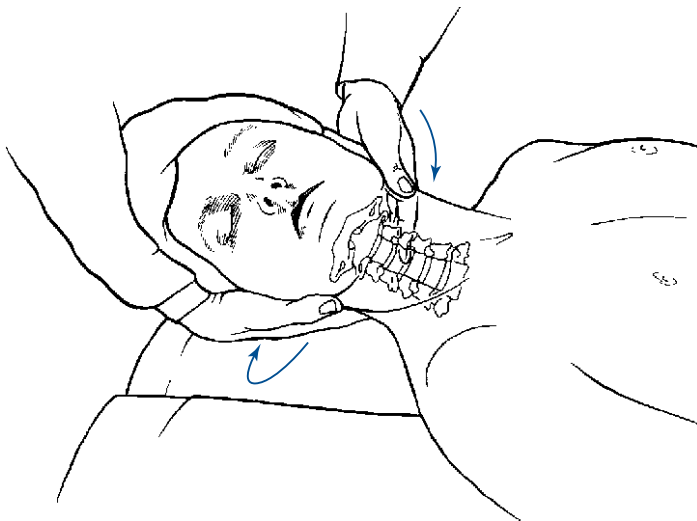
Lateral flexion dysfunction of the cervical spine may result from a loss of inferior glide and approximation of the joints on the side of lateral flexion dysfunction or a loss of contralateral superior glide on the side opposite lateral flexion restriction (Figure 5-78). The exception is the atlantoaxial articulation, which has horizontal facet planes and very limited lateral flexion. Determination of sites and direction of restriction are assessed by end feel evaluation.

Lateral flexion dysfunction is typically treated with assisted methods. In the lower cervical spine, lateral flexion is induced by contacting the articular pillar of the superior vertebrae on the side of lateral flexion restriction and applying adjustive thrusts



5-76

Figure 5-76 Assisted method. Thumb contact established over the right anterolateral pillar of C4, with adjustive force directed posteriorly (arrow) to induce right rotation and gapping in the right C4–5 articulation.



5-77

Figure 5-77 Resisted method. Index contact established on the left spinous process of C4, with adjustive force directed medially to induce right rotation and gapping in the right C4–5 articulation.

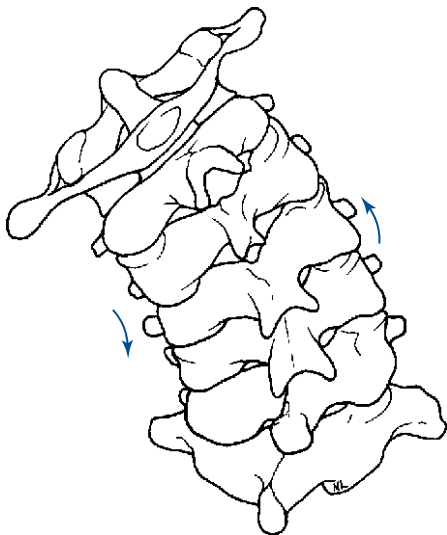


Figure 5-78 Posterior view of the cervical spine, illustrating left lateral flexion and superior glide of the right facet joints and inferior glide of the left facet joints.

medially and inferiorly along the facet planes (Figure 5-79, *A*). Techniques directed at inducing unilateral long-axis distraction of the posterior joints may also be applied to treat restrictions in lateral flexion by inducing distraction in the affected joints (Figure 5-79, *B*).

Lateral flexion restrictions in the atlanto-occipital (C0–C1) joint are distinctive because of the unique anatomy. Methods applied to induce lateral flexion movement in C0–C1 can be applied with contacts established on the ipsilateral or contralateral side of lateral flexion restriction (Figure 5-80, *A–C*).

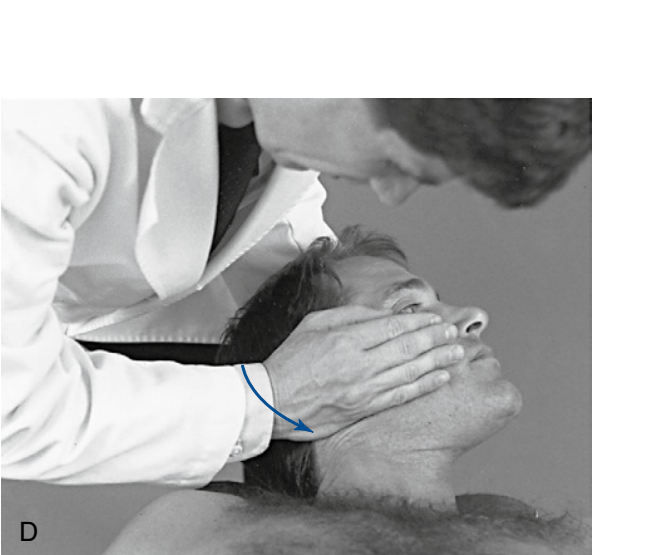
Flexion and Extension Dysfunction

Flexion restrictions (extension malpositions) may be treated with methods that induce gliding distraction in the facet joints. Many of the methods described for treating lateral flexion restrictions and rotational restrictions induce movements that may effectively induce this movement. Adjustments that induce long-axis distraction may also alleviate restrictions in flexion by inducing joint distraction (see Figure 5-79, *B*). Prone methods are also described for cervical flexion



5-79A

Figure 5-79 **A** Index contact applied to the left posterolateral articular pillar of C3, with adjustive force directed medioinferiorly (*arrow*) to induce left lateral flexion of the C3-4 motion segment. **B**, Adjustment applied to induce long-axis distraction in the left C2-3 articulation.



5-80A

Figure 5-80 **A**, Hypothenar contact applied to the left inferior aspect of the occiput to distract the left atlanto-occipital articulation. **B**, Thumb contact applied to the left inferior aspect of the occiput to distract the left atlanto-occipital articulation. **C**, Hypothenar contact applied to the left lateral aspect of the occiput to induce left lateral flexion of C0-C1. **D**, Hypothenar contact applied to the right inferior aspect of the occiput to extend the C0-C1 articulation.

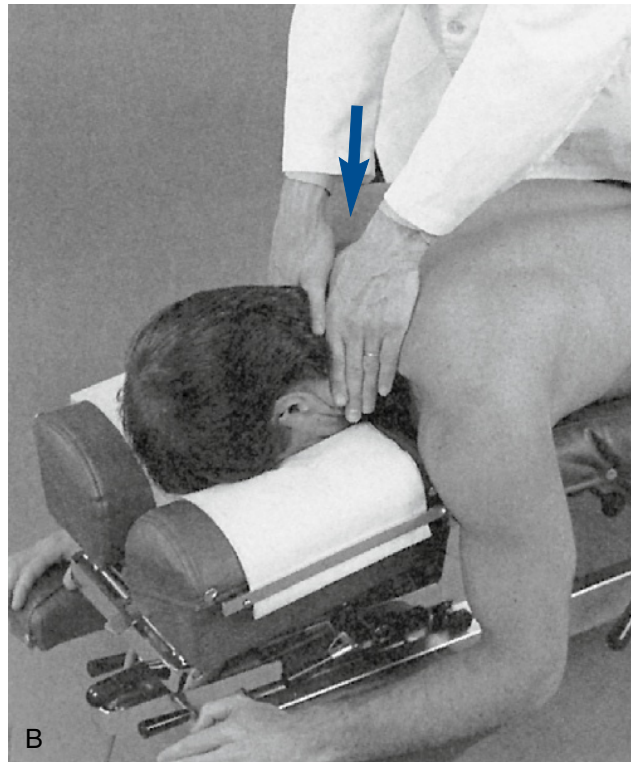
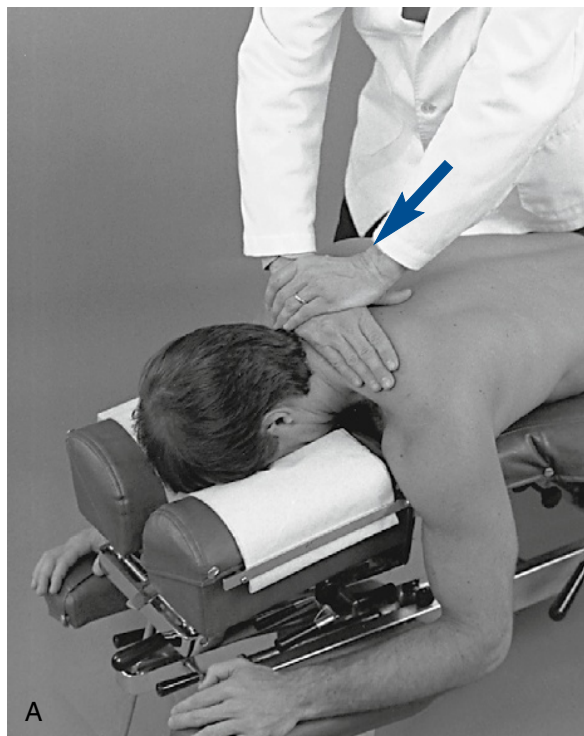


Figure 5-81 A, Adjustment applied to midcervical spine to induce flexion with a posterior-to-anterior (P-A) and inferior-to-superior (I-S) vector. B, Adjustment applied to midcervical spine to induce extension with P-A vector.

restrictions (Figure 5-81, *A*) but it is questionable whether segmental flexion can be induced with a prone stationary position and a posterior to anterior thrust.

Cervical extension induces motion that stretches the capsule and its periarticular tissue in directions not specifically addressed with rotation or lateral flexion adjustments. Therefore, extension restrictions (flexion malpositions) are more effectively addressed by adjustive techniques and VECs directed specifically for this dysfunction. When applying prone methods establish contacts in the midline over the spinous process or both articular pillars (see Figure 5-81, *B*), prestress the joint into extension and deliver a thrust anteriorly. When applying extension adjustments, use extreme caution to avoid excessive extension and joint compression. The thrust must be shallow and gentle.

Atlanto-occipital restrictions in flexion (extension malposition) are treated with methods directed at inducing postero-superior glide or long-axis distraction of the occipital condyle (Figure 5-80, *A* and *B*). Restrictions in extension (flexion malposition) are treated with methods directed at inducing antero-inferior glide of the occipital condyle (Figure 5-80, *D*). The thrust is directed mainly in the sagittal plane, with limited cervical rotation and some segmental lateral flexion to isolate the joint.

All adjustive techniques described in Chapters 5 and 6 are structured around the abbreviations and symbols presented in Box 5-3. The adjustive techniques in this chapter have been given names that are based on the involved joint or region, patient position, contact used by the clinician, body part contacted, and any necessary additional information (e.g., push, pull, with distraction, etc.), as well as the induced joint movement. These

BOX 5-3

Abbreviations Used in Illustrating Technique

THE FOLLOWING ABBREVIATIONS ARE USED THROUGHOUT THE CHAPTER:

IND	Indications
PP	Patient positioning
DP	Doctor positioning
SCP	Segmental contact point on patient
CP	Contact point
IH	Indifferent hand
VEC	Vector
P	Procedure
→	Arrows on photographs indicate direction of force.
Δ	Triangles on photographs indicate stabilization.

names follow the patterns used by the U.S. National Board of Chiropractic Examiners and are designed to be helpful in the teaching and testing for competence of the procedures (see Boxes 5-4–5-10).

UPPER CERVICAL SPINE ADJUSTMENTS (BOX 5-4)

Supine

Hypothenar/Occiput Lift (Figure 5-82)

IND: Restricted flexion, C0-1. Extension malposition, C0-1. Loss of long-axis distraction, C0-1.

BOX 5-4 Upper Cervical Adjustments

- Supine
 - Hypothenar/occiput lift (Figure 5-82)
 - Hypothenar/occiput push (Figure 5-83)
 - Calcaneal/zygomatic push (Figure 5-84)
 - Index/atlas push (Figure 5-85)
- Sitting
 - Calcaneal/zygomatic pull (Figure 5-86)
 - Index/occiput lift (Figure 5-87)
 - Index/occiput push (Figure 5-88)
 - Index/atlas push (Figures 5-89 and 5-90)
 - Digit/atlas pull (Figure 5-91)
- Prone
 - Thenar/occiput push: distraction (Figure 5-92)
 - Thenar/occiput push: extension (Figure 5-93)



Figure 5-82 Hypothenar contact applied to the left inferior aspect of the occiput to flex or distract the left atlanto-occipital articulation.

PP: The patient lies supine, with the doctor supporting the patient's head off of the end of the table and turned away from the side of dysfunction.

DP: Stand at the head of the table, facing cephalad, on the side of the adjustive contact in a low fencer stance, with weight shifted toward the superior leg.

CP: Hypothenar of your caudal hand, with fingers pointing vertically and resting on the skull. You may use an optional thumb contact.

SCP: Inferior edge of the occiput, medial to the mastoid.

IH: Your IH and fingers wrap around the patient's chin while your forearm supports the patient's head.

VEC: I-S.

P: Establish the contacts and rotate the patient's head away from the side of adjustive contact. Apply preadjustive long-axis distraction by leaning your body weight headward. At tension, deliver a shallow, vertically directed thrust superiorly through the contact hand and body. Take care to minimize rotational tension to the upper cervical spine.

Hypothenar/Occiput Push (Figure 5-83)

IND: Restricted rotation, lateral flexion, or extension, C0–1. Rotation, lateral flexion, or flexion malpositions, C0–1.

PP: The patient lies supine with the head off of the end of the table, supported by the doctor and turned away from the side of dysfunction.



Figure 5-83 **A**, Hypothenar contact applied to the right inferior aspect of the occiput to extend the C0–1 motion segment. **B**, Hypothenar contact applied to the left lateral aspect of the occiput to left laterally flex the C0–1 motion segment. **C**, Hypothenar contact applied to the left posterolateral aspect of the occiput to right rotate the left C0–1 motion segment.

DP: Stand at the head of the table on the side of the adjustive contact, angled 45 to 90 degrees to the patient.

CP: Hypothenar of the hand corresponding to the side of segmental contact (e.g., your right hand establishes the contact when contacting the right occiput). The contact hand is arched to cup over the patient's ear, with fingers resting on the angle of the jaw. Index or thenar contacts may be used as alternatives to the hypothenar contact.

SCP: Occiput (posterior supramastoid groove), just posterior to the ear.

IH: Your IH supports and cradles the patient's head, with fingers running along the base of the occiput.

VEC: P-A, S-I, and L-M to induce extension. L-M and S-I to induce lateral flexion.

P: Establish the contacts and laterally flex the head toward the side of contact while rotating it away. The degree of associated occipital extension or lateral flexion depends on the dysfunction being treated. After you establish joint tension, generate a thrust your shoulder along the desired VEC. Take care to minimize full rotational tension to the upper cervical spine.

Extension, C0–1 (see Figure 5-83, *A*): When inducing extension or ipsilateral anterior glide, laterally flex the occiput toward the side of contact and prestress into extension.

Lateral flexion, C0–1 (Figure 5-83, *B*): When inducing lateral flexion, limit extension of the occiput and induce lateral flexion toward the side of adjustive contact.

Rotation, C0–1 (Figure 5-83, *C*): When inducing occipital rotation or ipsilateral anterior glide, rotate the patient's head away from the side of adjustive contact. Avoid full rotational tension when treating rotational restrictions in the upper cervical spine. Rotational movement between the occiput and the atlas is very limited, and it is not necessary to develop full cervical rotation to develop tension at the C0-1 articulation. The incorporation of slight lateral flexion toward the side of contact aids in developing earlier rotational tension.

Calcaneal/Zygomatic Push (Figure 5-84)

IND: Lateral flexion restrictions and malpositions, C0–1.

PP: The patient lies supine, with the head rotated away from the side of contact and lateral flexion restriction.

DP: Stand at the side of the table behind the patient's head in a square stance.

CP: Calcaneal contact (heel of the hand) of the caudal hand, with the fingers pointing toward the vertex of the skull.

SCP: Zygomatic arch.

IH: Your cephalad hand cups the down-side ear with the palm while your fingers wrap around the occiput and upper cervical vertebra.



Figure 5-84 Hypothenar (calcaneal) contact applied over the right zygomatic arch to right laterally flex the atlanto-occipital motion segment.

VEC: L-M.

P: Apply L-M pressure against the zygomatic arch as your IH exerts superior traction against the down-side occiput. At tension, deliver an impulse thrust through both arms, creating a scooping action and L-M movement. Take care to minimize full rotational tension to the upper cervical spine.

Index/Atlas Push (Figure 5-85)

IND: Rotation, lateral flexion restrictions/malpositions, C1–2. Extension restriction (flexion malposition), C1–2.

PP: The patient lies supine.

DP: Stand at the head of table on the side of the adjustive contact, angled 45 to 90 degrees to the patient.

CP: Proximal ventrolateral surface of the index finger of your hand corresponding to the side of segmental contact. Your thumb rests on the patient's cheek while the remaining fingers support the contact and cup the base of the occiput.

SCP: Lateral aspect of the transverse process of the atlas for inducing lateral flexion. Posterior aspect of the transverse process for inducing rotation or coupled extension.

IH: Your IH cradles the patient's head and supports the contralateral occiput.

VEC: P-A, with clockwise or counterclockwise rotation to induce rotation. P-A to induce ipsilateral extension. Medial-to-lateral (M-L) to induce lateral flexion.

P: Rotate the patient's head away from the side of dysfunction and establish the contact. The degree of additional rotation, extension, or lateral flexion depends on the dysfunction being treated. After joint tension is established, generate an impulse thrust along the desired VEC.

Rotation (Figure 5-85, *A*): When treating rotational dysfunction, rotate the patient's head away from and slightly laterally flex the patient's head toward the side of adjustive contact. At tension, deliver a rotational impulse thrust through your wrist and forearms. Avoid full rotational tension with extension when treating rotation restrictions in the upper cervical spine. Rotational tension may be achieved earlier in the arc of motion by inducing slight lateral flexion toward the side of contact.

Lateral flexion (Figure 5-85, *B*): When treating restrictions in lateral flexion, contact the lateral surface of the atlas transverse, minimize rotation of the cervical spine, and thrust laterally to medially.

Extension (Figure 5-85, *C*): When treating coupled extension restrictions, minimize the rotation of the cervical spine while prestressing the joint into extension. Establish contact over the posterolateral mass and deliver a thrust anteriorly by inducing shoulder flexion.

Sitting

Calcaneal/Zygomatic Pull (Figure 5-86)

IND: Restricted flexion, C0–1. Extension misalignment, C0–1.

PP: The patient is seated.

DP: Stand behind the patient with a rolled towel or foam block between your torso and the patient's cervical spine. This is intended to maintain the cervical curve and support the cervical segments during the thrust.

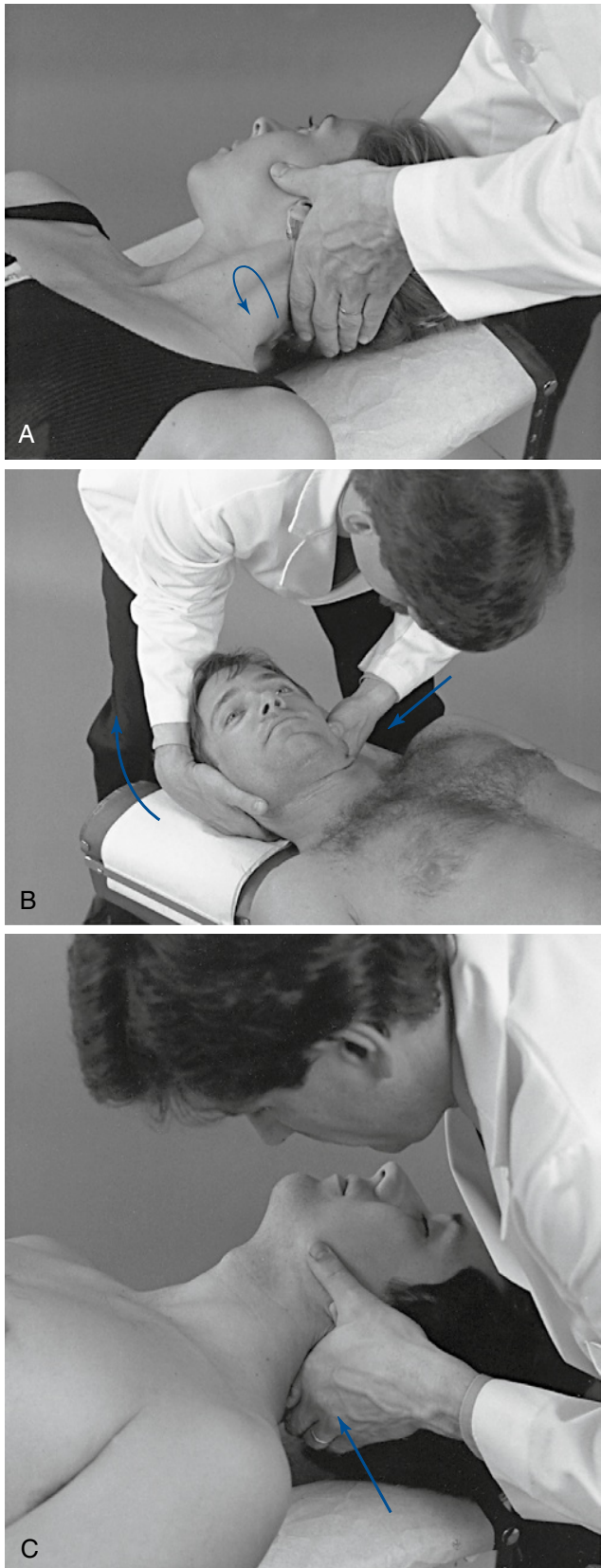


Figure 5-85 **A**, Index contact applied to the left atlas transverse process to right rotate the C1–2 motion segment. **B**, Index contact applied to the left atlas transverse process to left laterally flex the C1–2 motion segment. **C**, Index contact applied to the posterior aspect of the left atlas transverse process to extend the left C1–2 motion segment.



Figure 5-86 Bilateral calcaneal zygoma contacts applied to the zygomatic arches to induce flexion in the C0–1 motion segment.

CP: Calcaneal contact of both hands, with the fingers pointing cephalic (arching over the patient's eyes). Alternatively, reinforced pisiform contacts can be established over the glabellar region.

SCP: Superior aspect of the zygoma bilaterally.

IH: Same as the contact hand.

VEC: S–I.

P: Establish the contacts by compressing the patient's head against your body and applying long-axis distraction through the legs. Apply preadjustive tension in flexion, and then deliver an A–P and S–I thrust equally through both hands.

Index/Occipital Lift (Figure 5-87)

IND: Restricted flexion, lateral flexion, or loss of long-axis distraction, C0–1. Extension or lateral flexion malpositions, C0–1.

PP: The patient sits in a cervical chair, with the head turned away from the side of contact and resting against your chest.

DP: Stand behind the patient, slightly toward the side of cervical rotation.

CP: Proximal palmar surface of the middle finger of the hand corresponding to the side of head rotation (e.g., your left hand

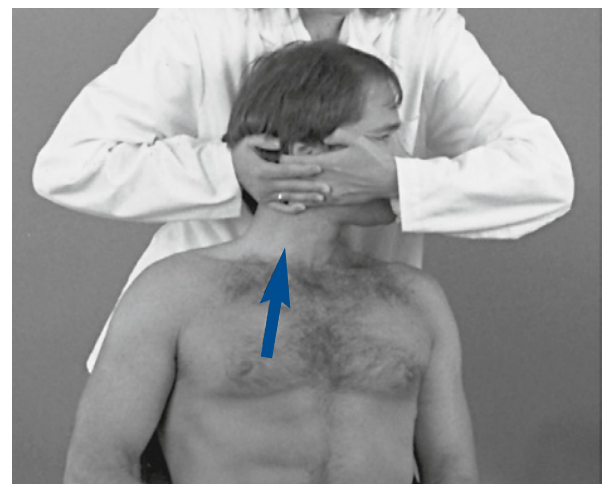


Figure 5-87 Middle finger contact applied to the right lateral and inferior aspect of the occiput to distract the right atlanto-occipital articulation.

establishes the contact on the right occiput when the patient's head is rotated to the left).

SCP: Inferior border of occiput and lateral border of the mastoid process on the side of dysfunction.

IH: Reinforces the contact hand and stabilizes the patient's head against your chest.

VEC: I-S for flexion or long-axis distraction. L-M and I-S for lateral flexion dysfunction.

P: Place the patient in the sitting position and rotate the patient's head away from the side of contact. Reach around the patient's face to contact the dysfunctional joint (a pillow may be used to cushion the patient's head against your chest). Develop preadjustive joint tension by tractioning vertically with arms and legs.

Long-axis distraction: To induce long-axis distraction or occipital flexion, thrust headward with a lifting impulse generated through the arms and legs.

Lateral flexion: To induce lateral flexion, accentuate bending of the patient's head away from you and thrust laterally to medially through the contact arms while maintaining long-axis distraction. Take care to minimize full rotational tension to the upper cervical spine.

Index/Occiput Push (Figure 5-88)

IND: Restricted rotation, lateral flexion, or extension, C0–C1. Rotation, lateral flexion, or flexion malpositions, C0–C1.

PP: The patient sits relaxed in a cervical chair.

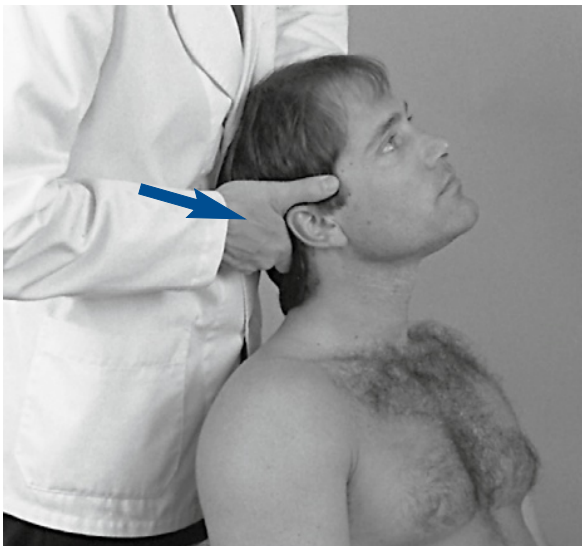
DP: Stand behind the patient, toward the side of segmental contact.

CP: Ventrolateral surface of the index finger of the hand corresponding to the side of segmental contact. The palm is turned up, with wrist straight. The forearm is approximately 45 degrees to the patient, with the remaining fingers cupping the lower occiput.

SCP: Occiput, supramastoid groove on the side of the lesion.

IH: Cups the patient's head and supports the contralateral occiput.

VEC: P-A, S-I, and L-M to induce extension. L-M, S-I, and P-A to induce lateral flexion.



5-88

Figure 5-88 Index contact applied to the right posterior and inferior aspect of the occiput to extend the atlanto-occipital motion segment.

P: Establish stabilization and segmental contact points (SCPs), keeping contact arm angled approximately 45 degrees to the patient's shoulders. Laterally flex the patient's head toward the side of contact, with slight rotation of the head away. The degree of comparative extension and lateral flexion depends on the direction of C0–1 restricted movement. When inducing extension direct the adjustive VEC more anteriorly, and when inducing lateral flexion direct the thrust more medially. Take care to minimize rotational tension to the upper cervical spine.

Index/Atlas Push (Figures 5-89 and 5-90)

IND: Restricted rotation, lateral flexion, or extension, C1–2. Rotation, lateral flexion, or flexion malposition, C1–2.

PP: The patient sits relaxed in a cervical chair.

DP: Stand behind the patient, toward the side of segmental contact.

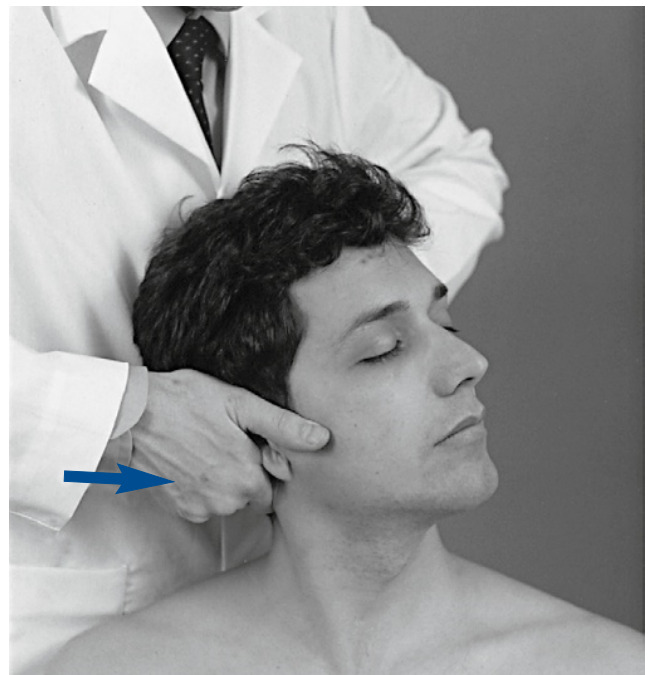
CP: Proximal ventral surface of the index finger of the hand corresponding to the side of segmental contact. The palm is turned up, with the wrist straight. The forearm is approximately 90 degrees to the patient, with the remaining fingers cupping the lower occiput.

SCP: Atlas transverse process: Lateral aspect for inducing lateral flexion. Posterior aspect for inducing rotation and extension.

IH: Cradles the patient's head by cupping the patient's ear and inferior occipital rim.

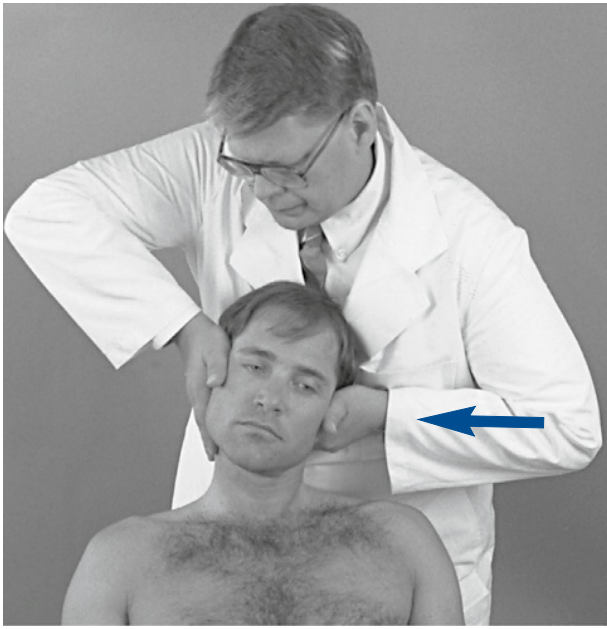
VEC: P-A, with clockwise or counterclockwise rotation to induce rotation. P-A to induce extension. M-L to induce lateral flexion.

P: Rotate the patient's head away from the side of dysfunction and establish the contacts. The degree of additional rotation, extension, or lateral flexion depends on the dysfunction being treated. After joint tension is established, generate a thrust with your shoulder along the desired VEC.



5-89

Figure 5-89 Index contact applied to the posterior aspect of the right transverse process of the atlas to induce left rotation at C1–2.



5-90

Figure 5-90 Index contact applied to the left atlas transverse process to left laterally flex C1–2.

Rotation (see Figure 5-89): When treating rotational dysfunction, rotate the patient's head away from and flex it slightly laterally toward the side of adjustive contact. At tension, deliver a rotational impulse thrust through the wrist and forearms. Minimize full rotational tension with extension by inducing slight lateral flexion toward the side of contact.

Lateral flexion (see Figure 5-90): When treating lateral flexion dysfunction, minimize rotation of the cervical spine and thrust laterally to medially by adducting your shoulder.

Extension: When inducing extension minimize rotation of the cervical spine while prestressing the joint into extension. Establish the contact over the posterior lateral mass and thrust anteriorly by inducing shoulder flexion.

Digit/Atlas Pull (Figure 5-91)

IND: Restricted rotation, C1–2. Rotational malposition, C1–2.

PP: The patient sits relaxed in a cervical chair.

DP: Stand, facing the patient on the side opposite the segmental contact.

CP: Palmar surface of the middle finger of the hand corresponding to the side of segmental contact. The thenar of the contact hand rests on the cheek of the patient.

SCP: Posterior aspect of atlas transverse.

IH: With fingers running vertically, stabilizes the patient's head by supporting the contralateral occiput and temporal region.

VEC: P-A, with clockwise or counterclockwise rotation.

P: Rotate the patient's head away from and slightly laterally flex it toward the side of adjustive contact. Induce rotation and ipsilateral anterior glide by developing a pulling impulse thrust through the contact hand by quickly extending the shoulder. (The same principles for minimizing extension and rotational tension in the upper cervical spine apply here also.)



5-91

Figure 5-91 Middle finger contact applied to the posterior aspect of the left atlas transverse process to induce right rotation at C1–2.

Prone

Thenar/Occiput Push: Distraction (Figure 5-92)

IND: Restricted flexion, C0–1. Loss of long-axis distraction, C0–1. Extension malposition, C0–1.

PP: The patient lies prone, with the head placed in slight flexion.

DP: Stand on either side of the patient in a fencer stance, caudal to the contact, facing cephalad.

CP: Thenar eminence of both hands.

SCP: Establish the contacts bilaterally on the inferior aspect of the occiput, medial to the mastoid.

VEC: I-S, P-A.

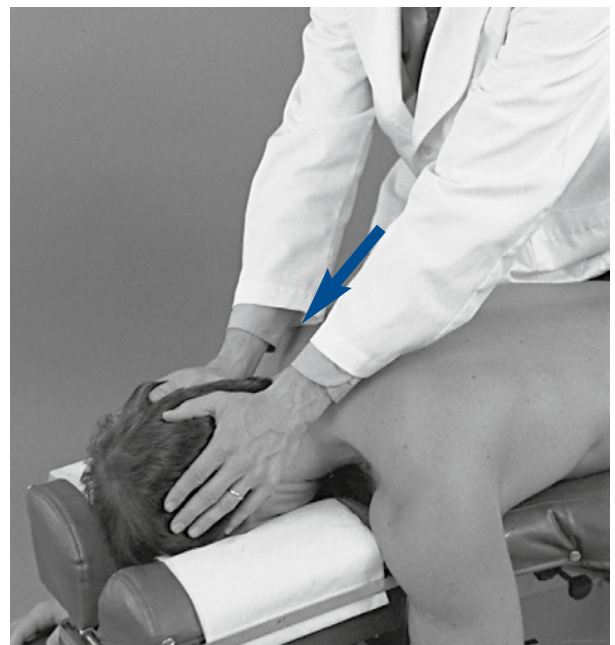


Figure 5-92 Bilateral thenar contacts applied to the posteroinferior aspect of the occiput to flex the atlanto-occipital motion segment.

P: Center your body over the patient in low fencer position, caudal to the contacts, and traction headward. Tension may be developed through both contacts or centered to one side. After joint tension is reached, deliver a cephalically directed thrust through your arms and trunk. The impulse may be directed at one or both articulations. This adjustment may be performed on a drop table or performed as a mobilization procedure.

Thenar/Occiput Push: Extension (Figure 5-93)

IND: Restricted extension, flexion malposition, C0–1.

PP: The patient lies prone, with the head placed in slight extension.

DP: Stand on either side of the patient in a fencer stance, facing cephalically, centered over the contacts.

CP: Thenar eminence of both hands.

SCP: Establish the contacts bilaterally on the posterior occiput, at or above the level of the superior nuchal line.

VEC: P-A

P: Center your body over the patient and deliver an impulse thrust anteriorly. Tension may be developed through both contacts or centered to one side. This adjustment may be performed on a drop table or performed as a mobilization procedure.



Figure 5-93 Bilateral thenar contacts applied to the posterior aspect of the occiput to extend the atlanto-occipital motion segment.

LOWER CERVICAL SPINE ADJUSTMENTS (BOX 5-5)

BOX 5-5 Lower Cervical Adjustments

- Supine
 - Index/pillar push (Figure 5-94)
 - Index/spinous push (Figure 5-95)
 - Thumb/pillar push (Figure 5-96)
 - Thumb/pillar pull (Figure 5-97)
 - Digit/pillar pull (Figure 5-98)
 - Hypothenar/pillar push (Figure 5-99)
- Sitting
 - Digit/pillar pull (Figure 5-100)
 - Index/pillar push (Figure 5-101)
 - Index/spinous push (Figure 5-102)
 - Hypothenar/pillar push (Figure 5-103)
- Prone
 - Index/pillar/push (Figure 5-104)
 - Hypothenar/spinous push (Figure 5-105)
 - Bilateral index/pillar push (Figure 5-106)

Supine

Index/Pillar Push (Figure 5-94)

IND: Restricted rotation, lateral flexion, or extension, C2–C7. Rotation, lateral flexion, or flexion malpositions, C2–C7.

PP: The patient lies supine.

DP: Stand at the head of the table on the side of the adjustive contact, angled 45 to 90 degrees to the patient.

CP: Ventrolateral surface of the index finger of the hand corresponding to the side of segmental contact. The thumb or thenar rests on the patient's cheek as the remaining fingers reinforce the contact.

Use the proximal surface of the index finger in upper cervical segments and the distal surface in the lower cervical segments.

SCP: Posterior articular pillar of superior vertebrae.

IH: Cradles the patient's head and supports the contralateral occiput and upper cervical spine.

VEC: P-A with clockwise or counterclockwise rotation to induce rotation M-L and S-I to induce lateral flexion.

P: Rotate the patient's head away from the side of dysfunction and establish the adjustive contact. The degree of additional rotation, extension, or lateral flexion depends on the dysfunction being treated.

Rotation (Figure 5-94, A): Establish the adjustive contact on the superior articular pillar, rotate the patient's head away while laterally flexing it toward the side of contact. Lateral flexion is incorporated in the positioning of this adjustment to induce unphysiologic movement and approximate the joints above the contact. The degree of lateral flexion should not be excessive or it may lead to compression and locking of the joints to be distracted. At tension, deliver the thrust through the wrists and forearms in a clockwise or counterclockwise direction along the planes of the facet joint.

Lateral flexion (Figure 5-94, B): To induce lateral flexion, laterally flex the head toward the side of contact while minimizing rotation, thrust medioinferiorly.

Extension: To induce segmental extension, prestress the involved joint into extension, contact the posterior pillar, and thrust anteriorly.

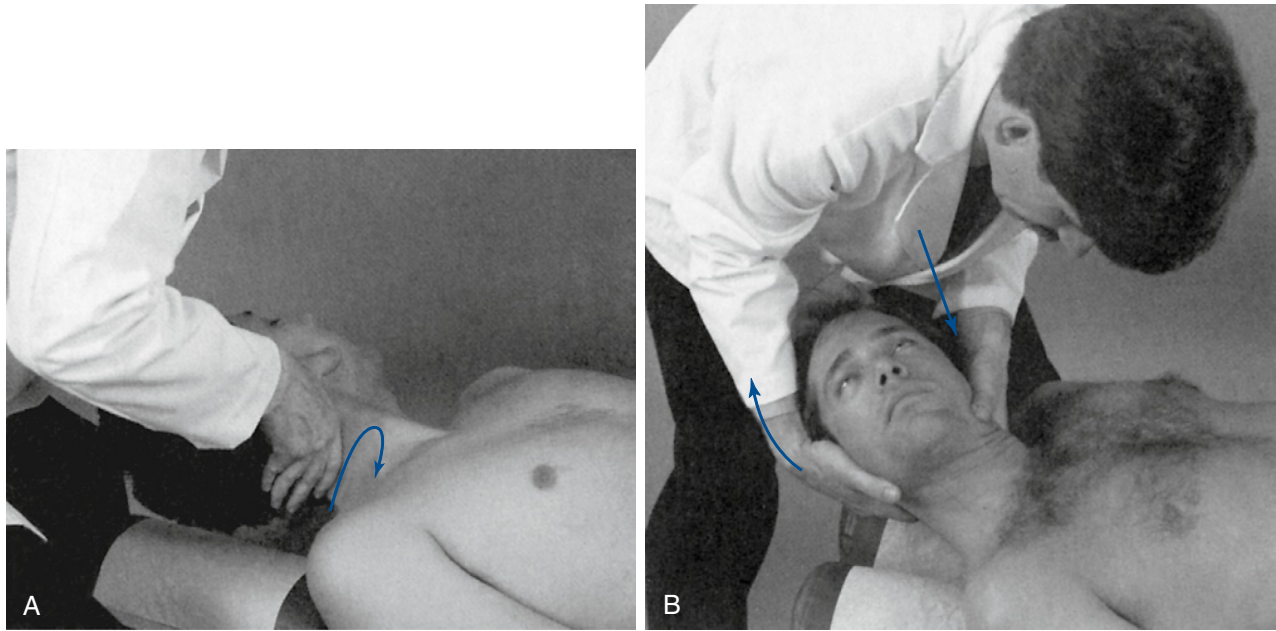
Index/Spinous Push (Figure 5-95)

IND: Restricted rotation or lateral flexion, C2–T3. Rotation or lateral flexion malpositions, C2–T3.

PP: The patient lies supine.

DP: Stand at the head of table on the side of the adjustive contact, angled 45 to 90 degrees to the patient.

CP: Ventrolateral surface of the index finger of the hand corresponding to the side of segmental contact, with the remaining fingers reinforcing the contact.



5-94A, B

Figure 5-94 A, Index contact applied to the posterior aspect of the right C2 articular pillar to induce left rotation of C2–3. B, Index contact applied to the lateral aspect of the C3 articular pillar to left laterally flex C3–4.



5-95C

Figure 5-95 A, Index contact applied to the lateral aspect of the C6 spinous process to left rotate. B, Index contact applied to the left lateral aspect of the C6 spinous process to left rotate and/or to left laterally flex the C6–7 motion segment. C, Resisted method. Index contact applied to the left lateral aspect of the C6 spinous process to right rotate the C6–7 motion segment.

SCP: Lateral surface of the spinous process (Figure 5-95, A).

IH: Cradles the patient's head and supports the contralateral occiput and upper cervical spine. Support and control of the patient's head may be enhanced by placing the arm of your contact hand against the patient's forehead and gripping it between the arm and forearm.

VEC: L-M and S-I

P: Rotate the patient's head slightly away from you and establish a contact on the lateral surface of the spinous process (Figure 5-95, A). The degree of additional rotation or lateral flexion depends on the dysfunction being treated. After joint tension is established, generate a thrust with your shoulder along the desired VEC.

Rotation (Figure 5-95, B): To induce rotation with an assisted contact (superior vertebra), use a more neutral patient position. Establish the contact on the superior spinous process on the side of rotational restriction and rotate the patient's head away from the side of contact. The head should be rotated only far enough to rest it in your IH. At tension, deliver a thrust primarily through the contact hand.

Rotational dysfunction may also be treated with resisted methods. The resisted adjustive approach is designed to induce rotation and gapping in the facet joint contralateral and superior to the side of adjustive contact. When a resisted method is used, the adjustive contact is established on the lateral surface of the spinous process of the inferior vertebra on the side opposite the rotation restriction. Preadjustive tension is developed by rotating the neck in the direction of restriction as counterpressure is applied against the spinous process. Lateral flexion is also induced toward the side of contact to distract the contralateral facet joints and block the ipsilateral facet joints (Figure 5-95, C). To deliver the impulse, counterthrust with both hands. The contact hand thrusts medially by inducing adduction of the shoulder. The IH induces counter-rotation by supinating the forearm.

Lateral Flexion: Establish the adjustive contact over the superior vertebra on the side of lateral flexion restriction. Laterally flex the patient's head toward the side of adjustive contact and deliver an impulse thrust medial, and anteroinferiorly through the segmental contact.

Thumb/Pillar Push (Figure 5-96)

IND: Rotational restrictions and malpositions, C2–7.

PP: The patient lies supine.

DP: Stand at the head of the table on the side of the adjustive contact, angled approximately 90 degrees to the patient.

CP: Palmar surface of the thumb of the hand corresponding to the side of segmental contact. The palm is turned down, with fingers resting on the patient's cheek.

SCP: PL pillar of the superior vertebra.

IH: Your IH cradles the patient's head and supports the contralateral occiput and upper cervical spine.

VEC: P-A, with slight I-S inclination and clockwise or counter-clockwise rotation.

P: After establishing contact, rotate the patient's head away and laterally flex it toward the side of contact. Lateral flexion is

incorporated in the positioning of this adjustment to induce approximation of the joints above the contact level. The degree of lateral flexion should not be excessive or it may lead to compression and locking of the joint to be distracted. The degree of lateral flexion necessary to isolate the lower cervical segments increases in a caudal direction. At tension, direct an impulse thrust anteriorly by inducing rotation through your shoulder.

Thumb/Pillar Pull (Figure 5-97)

IND: Restricted rotation or combined restricted rotation and opposite-side lateral flexion, C2–C7. Rotation and lateral flexion malpositions, C2–C7.

PP: The patient lies supine.

DP: Stand at the head of the table, opposite the side of the adjustive contact, angled approximately 45 degrees to the patient.

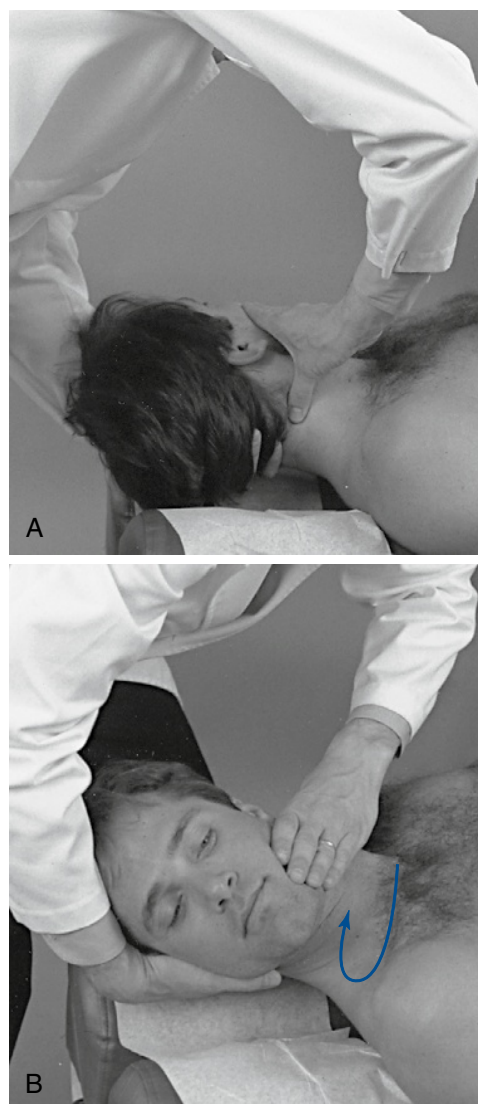


Figure 5-96 Thumb pillar posterior technique.

A, Thumb contact applied to the posterior aspect of the right C3 articular pillar. **B,** Procedure shown from the other side, illustrating the treatment of a right rotation restriction at C3–4.

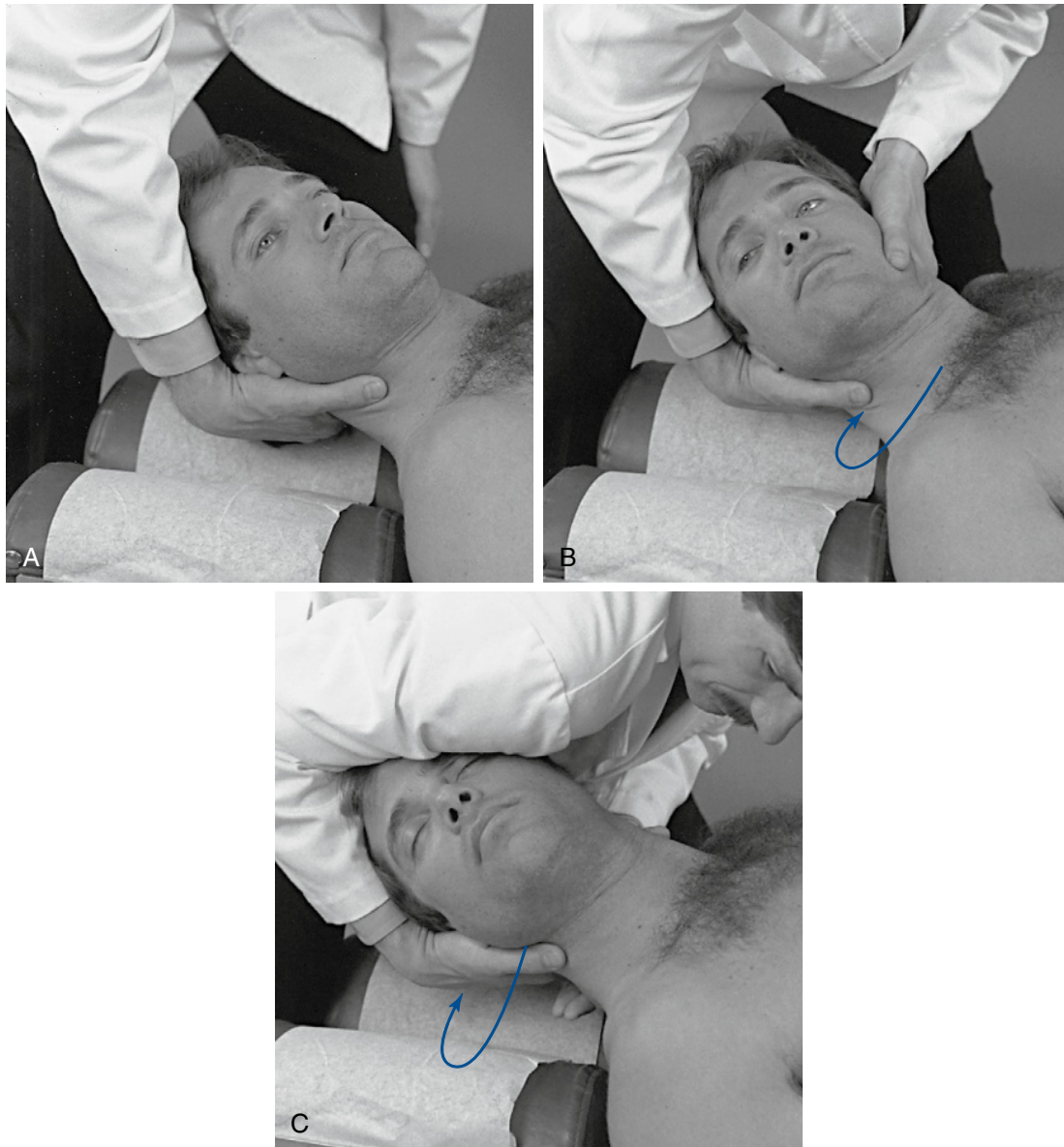


Figure 5-97 Thumb pillar anterior technique. **A**, Thumb contact applied over the anterolateral aspect of the right C2 articular pillar. **B**, Demonstration of procedure in the treatment of a right rotation restriction, applied to induce gapping in the right C2–3 articulation. **C**, Counterthrust technique, with contacts applied to the anterolateral aspect of the right C2 articular pillar and the left lateral aspect of the C3 spinous process. Note support for contacts with the doctor's shoulder.

CP: Palmar surface of the thumb of the hand corresponding to the side of segmental contact. The palm is turned up, with the fingers and the palm of the contact hand supporting the occiput and the upper cervical spine.

SCP: Anterolateral pillar of the superior vertebra (Figure 5-97, *A*).

IH: Your IH cups the ear, supporting the contralateral occiput (Figure 5-97, *B*). Support of the patient's head may be improved by placing the arm of your contact hand against the patient's forehead and gripping it between the arm and forearm (Figure 5-97, *C*).

VEC: A-P, with slight I-S inclination, inducing clockwise or counterclockwise rotation.

P: After establishing the contacts, rotate the patient's head toward and laterally flex it away from the side of adjustive contact. At tension, direct an impulse thrust posteriorly by inducing adduction of your shoulder and supination of the forearm. This adjustment is applied to induce rotation and ipsilateral joint gapping at the articulation below the contact level (Figure 5-97, *B*).

This adjustment may be combined with the index spinous adjustment to induce a counter thrust (push/pull). In this scenario, the IH contacts the spinous process of the lower vertebrae on the contralateral side. At tension, both hands thrust toward the midline to induce counter-rotation (Figure 5-97, *C*).



Figure 5-98 Index contact applied to the right articular pillar of C4 to induce left rotation in the C4–5 motion segment.

Digit/Pillar Pull (Figure 5-98)

IND: Rotational restriction and malpositions, C2–C7.

PP: The patient lies supine

DP: Stand at the head of the table, opposite the side of the adjustive contact

CP: Palmar surface of the middle finger of the hand corresponding to the side of segmental contact, with the palm resting on the patient's cheek.

SCP: Articular pillar of superior vertebrae.

IH: With fingers running horizontally, the IH stabilizes the patient's head by supporting the contralateral cheek, occiput, and temporal region.

VEC: P-A, with slight I-S inclination and clockwise or counterclockwise rotation.

P: Rotate the patient's head away from the side of contact and laterally flex it slightly toward the side of contact. Rest the patient's head in the IH. At tension, direct a pulling impulse thrust anteriorly along the facet planes by inducing shoulder extension.

Hypothenar/Pillar Push (Figure 5-99)

IND: Restrictions in rotation, lateral flexion, or long-axis distraction, C2–C7. Malpositions in rotation and lateral flexion and decreased interosseous spacing, C2–C7.

PP: The patient lies supine, head rotated, with side of contact up.

DP: Stand at the side of the table, behind the patient's head.

CP: Pisiform-hypothenar contact of the caudad hand, with the wrist in extension.

SCP: Articular pillar: posterior aspect for rotational dysfunction, lateral aspect for long-axis dysfunction.

IH: The cephalad hand grasps the patient's chin, allowing the head to rest on your forearm.

VEC: P-A for rotation, I-S for long-axis distraction

P: The IH provides cephalad traction. At tension, deliver a cephalad thrust for a long-axis distraction and an anterior thrust to induce rotation.

Sitting

Digit/Pillar Pull (Figure 5-100)

IND: Rotational restriction and malpositions, C2–C7.

PP: The patient sits relaxed in a cervical chair.

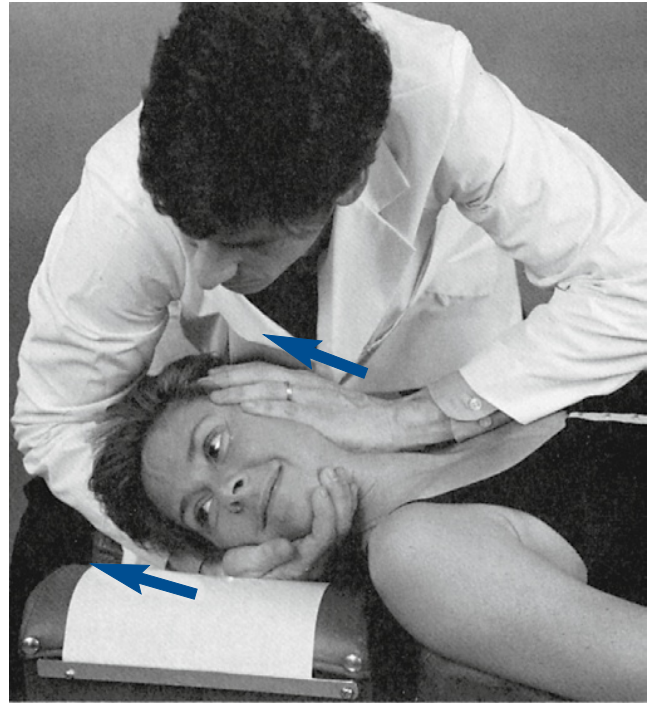


Figure 5-99 Hypothenar contact applied to the left lateral aspect of the cervical articular pillars to induce long-axis distraction.

DP: Stand, facing the patient on the side opposite the segmental contact.

CP: Palmar surface of the middle finger of the hand corresponding to the side of segmental contact, with the palm resting on the patient's cheek.

SCP: Articular pillar of the superior vertebra.

IH: With the fingers running vertically, the IH stabilizes the patient's head by supporting the contralateral occiput and temporal region.

VEC: P-A, with slight I-S inclination and clockwise or counterclockwise rotation.

P: Rotate the patient's head away from the side of contact and laterally flex it slightly toward the side of contact. At tension, direct a pulling impulse thrust anteriorly along the facet planes by inducing shoulder extension.

Index/Pillar Push (Figure 5-101)

IND: Restricted rotation, lateral flexion, or extension, C2–C7. Rotation, lateral flexion, or flexion malpositions, C2–C7.

PP: The patient sits relaxed in a cervical chair.

DP: Stand behind the patient, toward the side of segmental contact.

CP: Index finger of hand corresponding to the side of segmental contact. The palm is turned up, with the thumb and thenar resting on the patient's cheek. In the upper cervical spine, establish the contact toward the proximal surface of the index finger and toward the distal surface in the lower cervical spine.

SCP: Articular pillar of the superior vertebra.

IH: With fingers pointing down, the hand and fingers stabilize the opposing occiput and cheek.



Figure 5-100 Digital contact applied to the left C4 articular pillar to right rotate the C4–5 motion segment.

VEC: P-A, with clockwise or counterclockwise torque to induce rotation (Figure 5-101, *A*), P-A to induce extension, and L-M to induce lateral flexion (Figure 5-101, *B*).

P: Place the patient in a cervical chair, establish SCPs, and rotate the patient's head away from and slightly laterally flex it toward the side of adjustive contact. To induce rotation, lateral flexion, or extension, apply the same adjustive VECs presented for the supine index pillar adjustment.

Index/Spinous Push (Figure 5-102)

IND: Restricted rotation or combined rotation and lateral flexion restriction, C2–T3. Rotational or lateral flexion malpositions, C2–T3.

PP: The patient sits relaxed in a cervical chair.

DP: Stand behind the patient, toward the side of segmental contact.

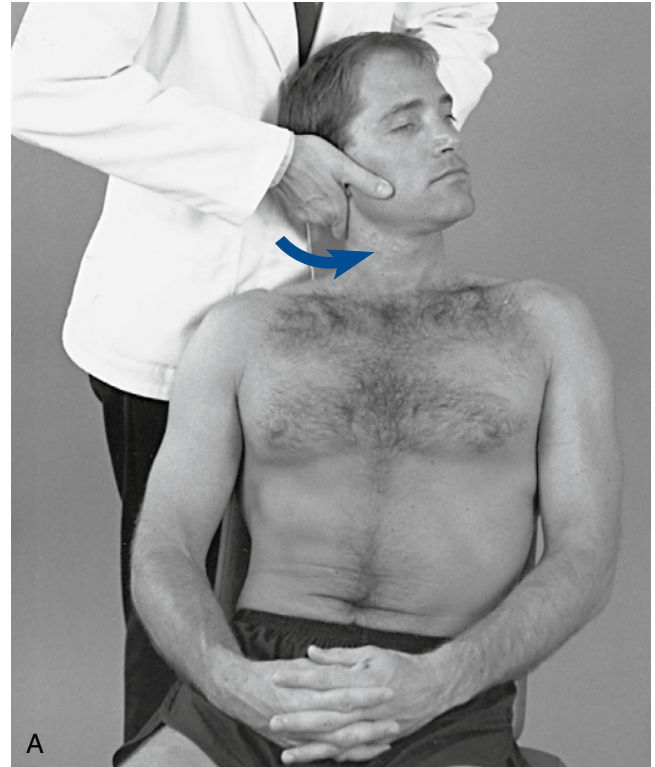
CP: Index finger of hand corresponding to the side of segmental contact. The palm is turned up, with the thumb resting on the patient's cheek.

SCP: Lateral aspect of the spinous process.

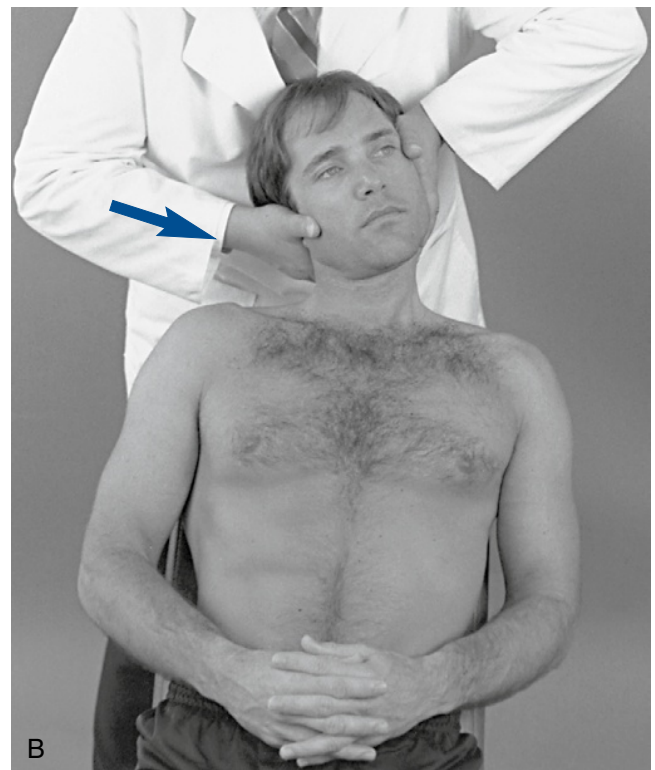
IH: With fingers pointing down, the hand and fingers stabilize the opposing occiput and cheek.

VEC: P-A and L-M.

P: Place the patient in a cervical chair, establish SCPs, and rotate the patient's head away from and slightly laterally flex it toward the side of adjustive contact. At tension, an impulse thrust is delivered anteriorly, medially, and inferiorly.



A



B



Figure 5-101 **A**, Index contact applied to the right articular pillar to left rotate the C3–4 motion segment. **B**, Index contact applied to the right lateral aspect of the C3 articular pillar to right laterally flex the C3–4 motion segment.

Hypothenar/Pillar Push (Figure 5-103)

IND: Restricted rotation or combined restricted rotation and opposite-side lateral flexion, C2–C7. Rotation and lateral flexion malpositions, C2–C7.



Figure 5-102 Index contact applied to the right lateral aspect of the C4 spinous process to right laterally flex or right rotate the C4–5 motion segment.



Figure 5-103 Hypothenar contact applied to the anterolateral aspect of the right C4 articular pillar to induce right rotation.

PP: The patient sits relaxed in a cervical chair.

DP: Stand in front of the patient, toward the side of contact.

CP: Hypothenar of hand corresponding to the side of adjustive contact. The fingers of the contact hand extend obliquely vertically to provide stabilizing support to the patient's head.

SCP: Anterolateral pillar of superior vertebra.

IH: With fingers running vertically, the IH stabilizes the contralateral upper cervical spine and occiput.

VEC: A-P and slightly I-S.

P: Stand in a fencer stance on the side of adjustive contact and establish a broad, fleshy hypothenar contact over the anterolateral articular pillar. Rotate the patient's head toward and laterally flex it away from the side of adjustive contact. At tension, direct an impulse thrust perpendicular to the facet plane by thrusting posterosuperiorly through your shoulder. Maintain slight vertical traction through both hands during the delivery of the adjustment. This adjustment is applied to induce A-P rotation and ipsilateral joint gapping at the articulation below the contact.

Prone

Index/Pillar Push (Figure 5-104)

IND: Restricted rotation or lateral flexion, C2–C7. Malpositions in rotation or lateral flexion, C2–C7.

PP: The patient lies in the prone position, with the headrest lowered to induce slight thoracocervical flexion.

DP: Stand in a fencer stance on either side of table, facing cephalad.

CP: Index finger (lateral aspect) of the hand corresponding to the side of the adjustive contact. The wrist is held in ulnar deviation, with the fingers pointing to the floor and the thumb resting on the posterior cervical soft tissues (Figure 5-104, A).

SCP: Posterior aspect of the articular pillar of the superior vertebra (Figure 5-104, A).

IH: A thumb-web contact is established at the inferior rim of the occiput while the palm and fingers contact the cheek and side of the face.

VEC: P-A and slightly I-S for rotational restrictions. P-A and superior-to-inferior (S-I) for lateral flexion restrictions.

P: The IH tractions the head cephalically while laterally flexing it toward the contact and slightly rotating it away from the side of adjustive contact (e.g., for left-sided contact, induce left lateral flexion and right rotation).

Rotation: To induce rotation, contact the posterior pillar on the side opposite the rotational restriction and rotate the head in the direction of joint restriction.

Lateral flexion: To induce lateral flexion, contact the articular pillar on the side of lateral flexion restriction and laterally flex the neck toward the side of contact. At tension, thrust medio-anteroinferiorly (Figure 5-104, B).

Hypothenar/Spinous Push (Figure 5-105)

IND: Flexion or extension restrictions or malpositions, C2–C7.

PP: The patient lies prone, with the patient's neck flexed and headpiece slightly lowered for flexion restrictions (Figure 5-105) and extended for extension restrictions.

DP: Stand in a fencer stance, facing cephalad.

SCP: Spinous process.

IH: The IH reinforces the contact hand with fingers pointing vertically.

VEC: P-A.

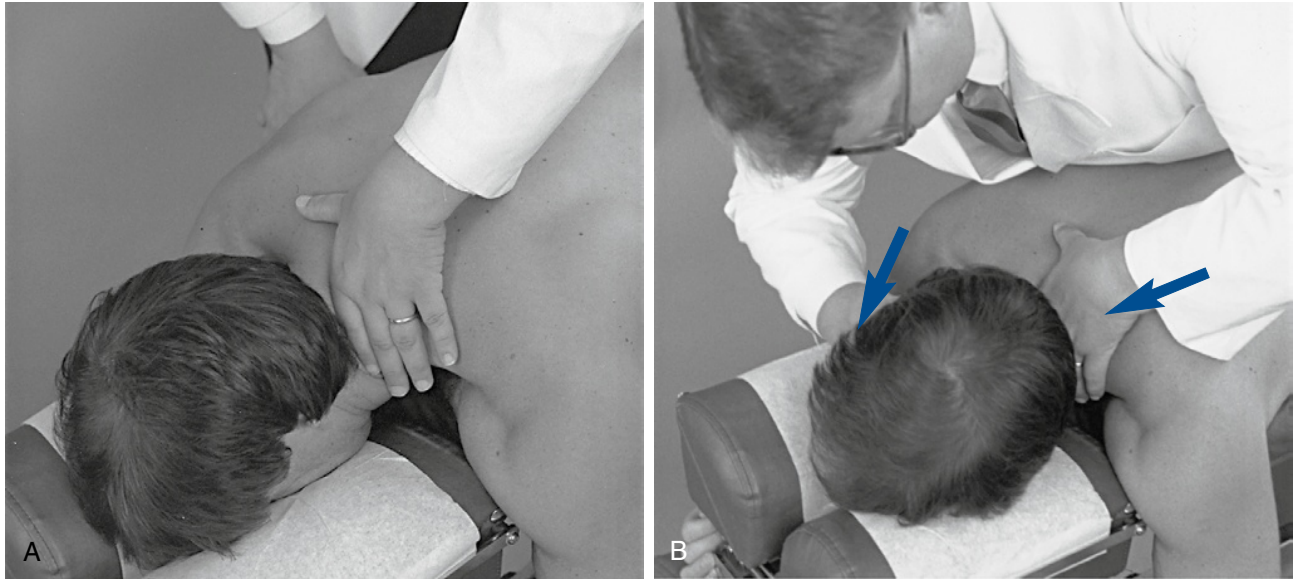


Figure 5-104 Index pillar prone technique. **A**, Index contact established over the posterior aspect of the left C5 articular process. **B**, Procedure shown to induce left lateral flexion and right rotate at the left C5–6 motion segment.

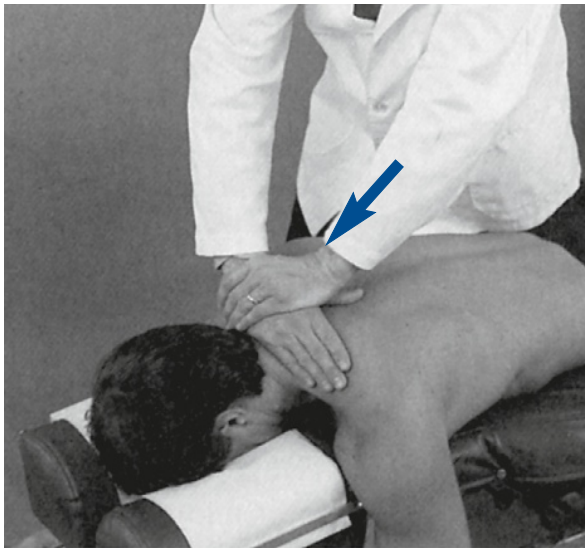


Figure 5-105 Hypothenar contact applied to the C6 spinous process to flex the C6–7 motion segment.

P: When treating flexion dysfunction, position the patient's cervical spine into flexion and deliver a P-A and cephalically directed impulse.

When treating extension dysfunction, position the patient's cervical spine in a slightly extended position and center your body over the contact. Extension is induced by delivering an impulse thrust anteriorly. When performing this adjustment, use extra caution to avoid excessive depth and hyperextension of the neck.

These adjustments can be performed on a drop table. A drop mechanism that allows downward and forward movement may provide a mechanical advantage over a straight downward drop by minimizing the compression and providing axial distraction.

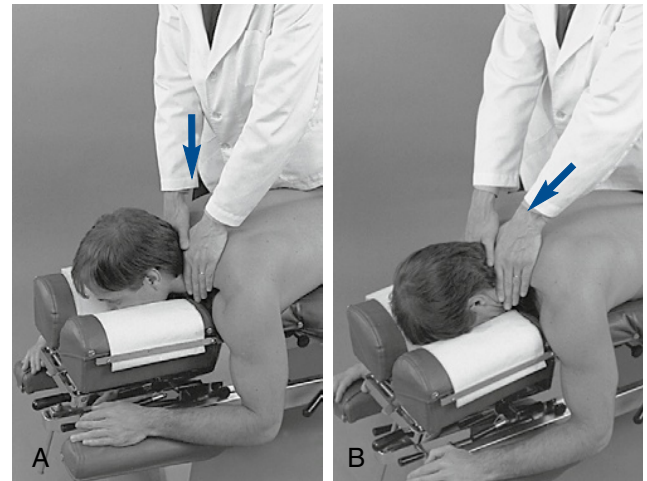


Figure 5-106 Bilateral index contacts established over the posterior aspect of the C4 articular pillars to extend (**A**) or flex (**B**) the C4–5 motion segment.

Bilateral Index/Pillar Push (Figure 5-106)

IND: Restricted extension C2–C7. Flexion malpositions, C2–C7.

PP: The patient lies prone.

DP: Stand in a fencer stance, facing cephalad, with the center of gravity over the contact.

CP: Proximal index of both hands, with thumbs crossing at the midline.

SCP: Posterior pillars.

VEC: P-A.

P: When treating flexion dysfunction, position the patient's cervical spine into flexion and deliver a P-A and cephalically directed impulse. It is unlikely that prone patient positions are the most effective option for inducing cervical flexion.

When treating extension dysfunction, position the patient's cervical spine in a slightly extended position and center your body over the contact. Extension is induced by delivering an

impulse thrust anteriorly. When performing this adjustment, use extra caution to avoid excessive depth and hyperextension of the neck.

These adjustments can be performed on a drop table. A drop mechanism that allows downward and forward movement may provide a mechanical advantage over a straight downward drop by minimizing the compression and providing axial distraction.

THORACIC SPINE

In the thoracic spine, protection and function of the thoracic viscera take precedence over intersegmental spinal mobility. Although the limiting anatomic structures make the thoracic spine the least mobile part of the spinal column, the small movements that do occur within the functional units are still significant. Although more clinical attention has been focused around the cervical and lumbar regions, the thoracic region is an area that must be considered important because of the possible mechanical changes that may result in effects to the elements of the autonomic nervous system. Furthermore, the addition of the articulations for the ribs makes the thoracic region an exceptional structure. Finally, this region seems to be prone to chronic postural problems affecting sections of the thoracic spine and the supporting soft tissues (myofascial pain syndromes).

FUNCTIONAL ANATOMY

The body of the typical thoracic vertebra (T2–T8) is heart-shaped, with both the A-P and side-to-side dimensions of equal length (Figure 5-107). The anterior surface of the body is convex from side to side, and the posterior surface is deeply concave. Both the superior and inferior surfaces of the body are flat, with a ring around the margin for attachment of the IVD. The pedicles of thoracic vertebrae are short and have inferior vertebral notches deeper and larger than in any other part of the spine. The laminae are short, broad, thick, and overlapping. The spinous processes are long and slender, with a triangular shape in cross-section. They point obliquely downward, overlapping in the midthoracic spine and limiting extension

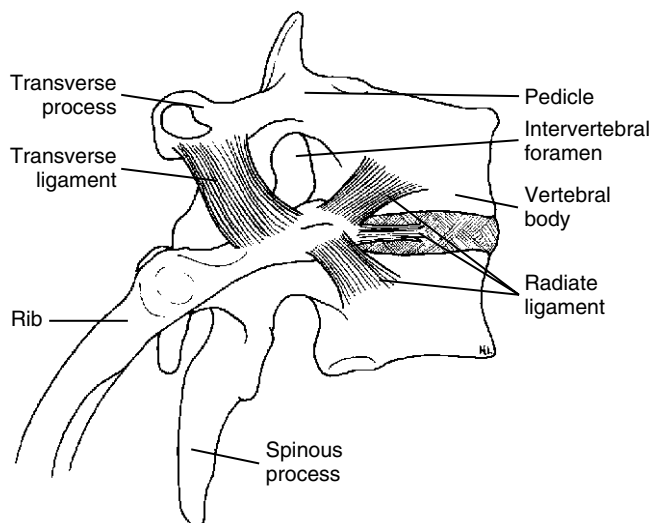


Figure 5-107 Typical thoracic motion segment.

movement. The transverse processes arise from behind the superior articular processes. They are thick, strong, and relatively long, with a concave facet on the anterior side. The intervertebral foramina in this region are essentially circular in shape and fairly small when compared with other areas of the spine (see Figure 5-107).

The articular facets form an angle of approximately 60 degrees from the transverse toward the coronal plane and 20 degrees from the coronal toward the sagittal plane (Figure 5-108). The inferior articular process arises from the laminae to face inferomedioanteriorly. The superior articular process arises from near the lamina-pedicle junction to face superolaterally and posteriorly. The inferior articular process lies posterior to the superior articular process of the vertebra below.

The IVDs are comparatively shallow in the thoracic spine. The disc height-to-body height ratio is 1:5, making it the smallest ratio in the spine (Figure 5-109). This low ratio contributes to the decreased flexibility of the thoracic spine. The nucleus is also more centrally located within the annulus of the thoracic disc than it is in either of the other two spinal regions.

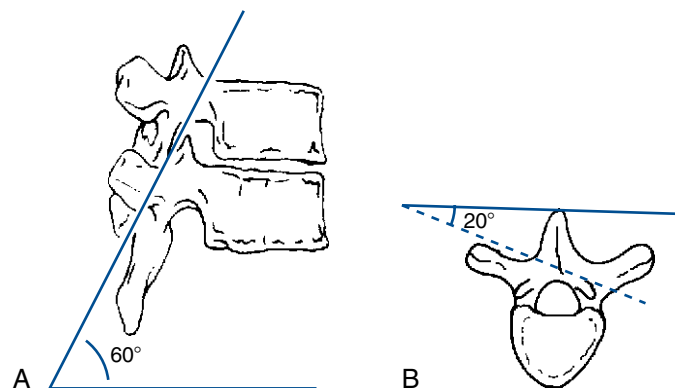


Figure 5-108 The thoracic facet planes.

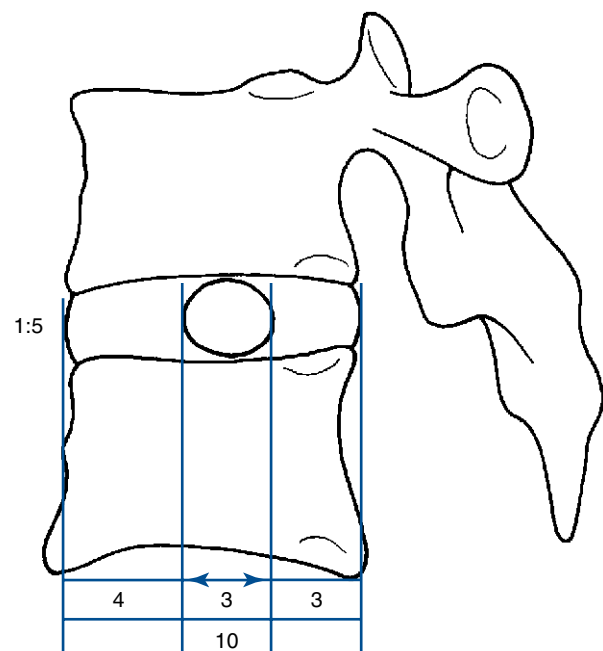


Figure 5-109 The location of the nucleus and the disc height-to-body height ratio in the thoracic spine.

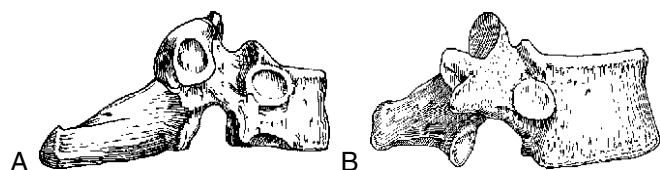


Figure 5-110 Structure of the atypical thoracic vertebra. **A**, T1 vertebra. **B**, T12 vertebra.

One special feature of thoracic vertebrae is the presence of costovertebral and costotransverse joints, which form the articulations for the ribs (see Figure 5-107). The costovertebral joints (demifacets) are located on either side of the vertebral body to form an articulation with the heads of the ribs. The costotransverse joints are located on the anterior aspects of the transverse processes to articulate with the tubercles of the ribs.

The thoracic atypical vertebrae include T1 and T9 to T12 (Figure 5-110). The vertebral body of T1 resembles that of C7 and possesses a whole facet for articulation with the first rib. The T9 vertebra may have no demifacets below, or it may have two demifacets on either side (in which case, the T10 vertebra will have demifacets only at the superior aspect). T10 has one full rib facet located partly on the body of the vertebra and partly on the tubercle. The T11 segment has complete costal facets, but no facets on the transverse processes for the rib tubercle. This vertebra also begins to take on characteristics of a lumbar vertebra. The spinous process is short and almost completely horizontal. T12 has complete facets on the vertebra for articulation with the ribs, but otherwise resembles a lumbar vertebra. The inferior articulating surfaces of T12 are convex and are directed laterally and anteriorly in the sagittal plane, like those in the lumbar spine. Superior, inferior, and lateral tubercles (see Figure 5-110) replace the transverse processes.

THORACIC CURVE

The thoracic spine forms a kyphotic curve of less than 55 degrees,¹⁸ with an accepted range of 20 to 50 degrees^{19,20} and an average of 45 degrees⁶ (Figure 5-111). It is a structural curve present from birth and maintained by the wedge-shaped vertebral bodies that are approximately 2 mm higher posteriorly. The thoracic curve begins at T1–2 and extends down to T12, with the T6–7 disc space as the apex.²¹

Alterations in the thoracic curve can be anatomic or postural. A change in the primary thoracic curve is likely to produce a change in the secondary curves in the cervical and lumbar spine. The lumbar curve tends to increase, and the cervical curve decreases or shifts forward, creating a cervical “poking” posture. This postural syndrome of forward head positioning and rounded and forward shoulders is often associated with chronic stretch weakness of the middle and lower trapezius muscles. The chronic strain to the posterior back and neck muscles can induce local myofascial pain syndromes and headaches.²² As the thoracic kyphosis increases, it crowds the thoracic viscera, interfering with normal physiologic functioning.

Juvenile kyphosis (Scheuermann disease) and osteoporosis also result in an increased thoracic kyphosis. In juvenile kyphosis the wedge shape of the vertebral body is exaggerated, but the cause remains inconclusive. Theories of the pathogenesis include aseptic necrosis, endplate

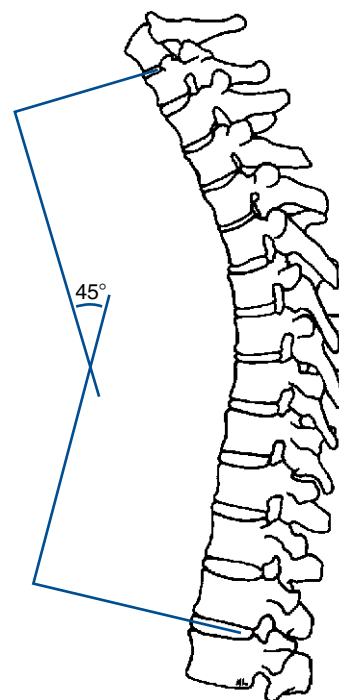


Figure 5-111 The location of the nucleus and the disc measurement of the thoracic curve.

fracture, infection, endocrine abnormalities, trabecular deficiencies, vitamin deficiencies, fluoride toxicity, and mechanical factors.²³

Osteoporosis reduces the number and size of trabeculae in the vertebral body, diminishing the axial loading stretch and resulting in compression fractures, which accentuate the kyphotic curve. Dietary deficiencies, malabsorption syndromes, steroid use, and endocrine disorders have been implicated as causal factors of osteoporosis.²⁴

RANGE AND PATTERNS OF MOTION

Of the three cardinal planes of movement, sagittal plane movement of flexion and extension is the most restricted. Rotation and lateral flexion demonstrate nearly equal movement, with each exhibiting nearly twice as much movement as flexion and extension (Table 5-4; see Figure 5-24).

Movement in the upper thoracic spine is generally less than in the lower. The exception is rotation, which decreases dramatically in the lower thoracic segments as the facet facings become more sagittal.⁵ The instantaneous axis of movement for the thoracic spine, like other spinal regions, remains somewhat tentative.²⁵ Using fresh cadaveric specimens, Panjabi and associates²⁵ determined the likely sites for flexion and extension, lateral flexion, and rotation (Figure 5-112).

Flexion and Extension

Combined flexion and extension in the thoracic spine averages approximately 6 degrees per motion segment, demonstrating a cephalocaudal increase in flexibility. Movement averages 4 degrees in the upper thoracic spine, 6 degrees in the middle thoracic spine, and 12 degrees in the lower two thoracic segments.⁵ Extension is more limited than flexion because of the impaction of the articular processes and spinous processes.

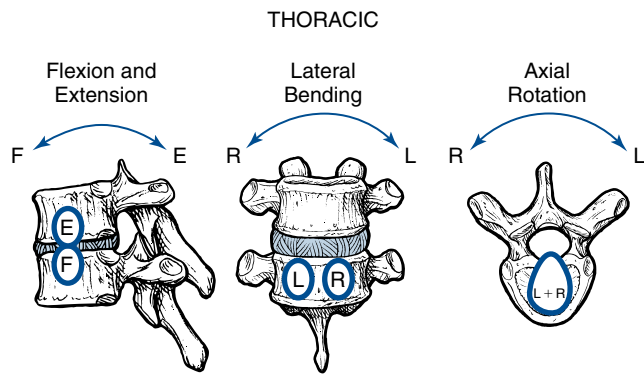


Figure 5-112 Instantaneous axis of rotation for flexion and extension (A), lateral flexion (B), and axial rotation (C) in a thoracic segment. (From White AA, Panjabi MM: *Clinical biomechanics of the spine*, ed 2, Philadelphia, 1990, JB Lippincott.)

TABLE 5-4 Average Range of Motion for the Thoracic Spine

Vertebra	Combined Flexion and Extension	One-Side Lateral Flexion	One-Side Axial Rotation
T1–2	4	5	9
T2–3	4	6	8
T3–4	4	5	8
T4–5	4	6	8
T5–6	4	6	8
T6–7	5	6	7
T7–8	6	6	7
T8–9	6	6	9
T9–10	6	6	4
T10–11	9	7	2
T11–12	12	9	2
T12–L1	12	8	2

Modified from White AA, Panjabi MM: *Clinical biomechanics of the spine*, ed 2, Philadelphia, 1990, JB Lippincott.

Thoracic flexion and extension combine sagittal plane rotation with slight sagittal plane translation. The degree of combined translation is minimal and uniform throughout the thoracic spine.⁵ During flexion the articular facets glide apart as the IVD opens posteriorly. During extension, the facet joints and posterior disc approximate (Figure 5-113).

Lateral Flexion

Lateral flexion averages approximately 6 degrees to each side, with the lower two segments averaging 7 to 9 degrees. Lateral flexion is coupled with axial rotation throughout the thoracic spine. This is especially apparent in the upper thoracic spine, where the pattern duplicates that of the cervical spine. The coupling is such that lateral flexion and rotation occur to the same side (e.g., body rotation to the concavity and spinous deviation to the convexity)^{5,26} (Figure 5-114). In the middle and lower thoracic spine, the coupling is less distinct and may occur in either direction (Figure 5-115).

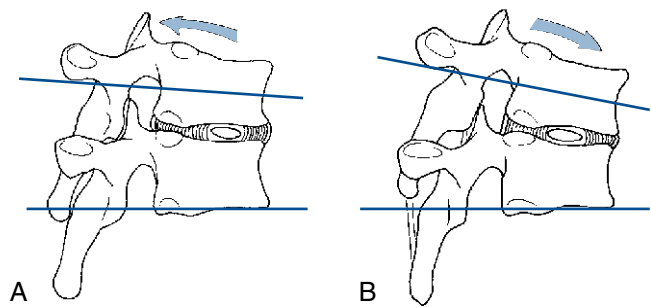


Figure 5-113 Extension (A) and flexion (B) of a thoracic segment.

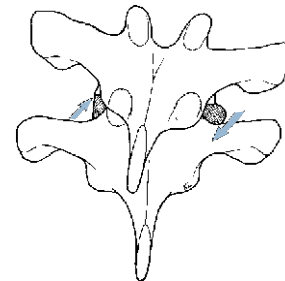


Figure 5-114 Lateral flexion of an upper thoracic segment, showing coupling movement in rotation and lateral flexion to the same side. This pattern is the same as that of the cervical spine.

It is often assumed, however, that the lower thoracic segments have a tendency to follow the coupling pattern of the lumbar spine. The lumbar pattern is opposite that of the cervical and upper thoracic segments and incorporates lateral flexion with coupled axial rotation in the opposite direction^{27,28} (see Figure 5-115). White and Panjabi⁵ point out, however, that coupling patterns still remain controversial, and doctors must guard against any strong conclusions.

During lateral flexion, the IVD and facet joints approximate on the side of lateral flexion and separate on the side opposite lateral flexion (see Figure 5-114). In the upper thoracic spine the inferior articular facets also glide medially relative to the superior articular facet on the side of lateral flexion and laterally on the side opposite lateral flexion. This is a result of the strong coupled axial rotation in the upper thoracic spine.

Rotation

Segmental axial rotation averages 8 to 9 degrees in the upper thoracic spine (Figure 5-116). Rotational motion decreases slightly in the middle thoracic spine and drops off dramatically to approximately 2 degrees in the lower two or three thoracic segments.⁵ The marked decrease in rotational mobility in the lower segments no doubt reflects the transition from coronal plane facets to sagittal plane facets.

Rotational movements in the thoracic spine are also coupled with lateral flexion. In the upper thoracic spine, rotation is coupled with same-side lateral flexion. This leads to MI gliding of the inferior facet relative to the superior facet on the side of trunk rotation and LS gliding of the inferior facet on the side opposite trunk rotation. The coupling is not as marked in the lower segments as it is in the upper segments.⁶ This may occur because the facets of the lower thoracic spine become more sagittal in their orientation.

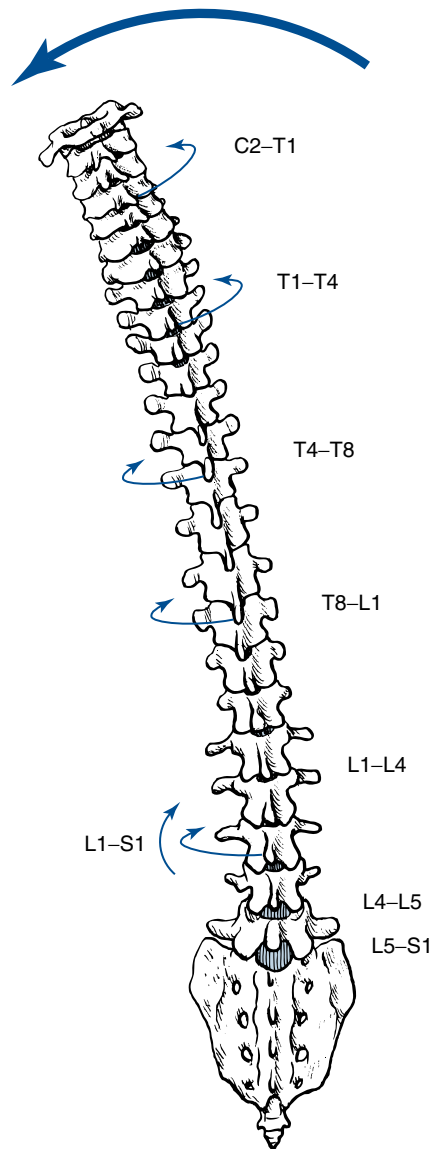


Figure 5-115 Coupled relationship of lateral flexion and axial rotation throughout the spine. The cervical and upper thoracic regions have lateral flexion coupled with ipsilateral rotation, and the lumbar spine and lower thoracic regions have lateral flexion coupled with contralateral rotation. (From White AA, Panjabi MM: *Clinical biomechanics of the spine*, ed 2, Philadelphia, 1990, JB Lippincott.)

KINETICS OF THE THORACIC SPINE

The same principles of concentric and eccentric muscle activity discussed for the cervical spine apply to the trunk. Nonsegmental muscles can induce movement of the entire thoracic spine or act segmentally. They include the erector spinae, rectus abdominis, quadratus lumborum, and abdominal obliques. The segmental muscles that influence each thoracic motion segment include multifidi, interspinalis, intertransversarii (small in the thoracic segments), and rotatores.

Flexion is initiated by concentric activity of the rectus abdominis and controlled or limited by eccentric activity of the erector spinae. Flexion is further limited by the elastic limits of the

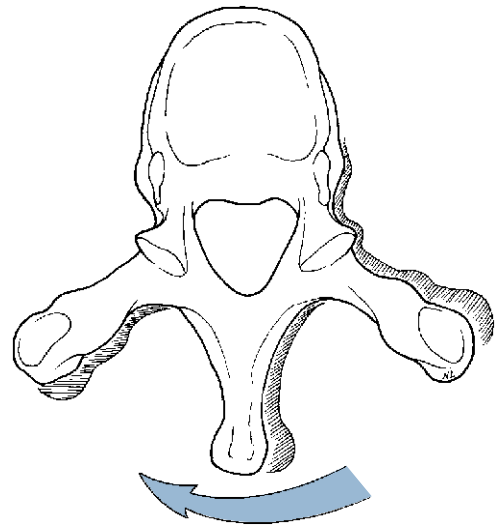


Figure 5-116 Horizontal view, illustrating right rotation of the superior vertebra (*light*) relative to the inferior vertebra (*dark*).

myofascial tissue, ligamentum flavum, interspinous ligament, supraspinous ligament, PLL, capsular ligaments, posterior IVD, and bony impact of the vertebral bodies.

Extension is initiated by concentric activity of the erector spinae and controlled or limited by eccentric activity of the rectus abdominis. Extension is mainly limited by the bony impact of the spinous and articular processes, but the ALL, anterior IVD, and elastic limits of myofascial tissue also contribute.

Lateral flexion is initiated by concentric activity of ipsilateral erector spinae and quadratus lumborum and controlled or limited by contralateral eccentric activity of the same muscles. Further limiting of lateral flexion movement occurs through impact of the articular facets, contralateral capsular, ligamentum flavum, intertransverse ligament, and elastic limits of contralateral segmental and nonsegmental muscles.

Rotation is initiated by concentric activity of ipsilateral erector spinae, multifidus, and rotatores and controlled or limited by concentric and eccentric activity of the abdominal obliques and erector spinae. Rotation is further limited by the articular capsules, interspinous ligament, supraspinous ligament, ligamentum flavum, bony impact of the articular facets, and elastic limits of bilateral segmental and nonsegmental muscles.

FUNCTIONAL ANATOMY AND BIOMECHANICS OF THE RIB CAGE

The rib articulations can be divided into two groups, one connecting the heads of the ribs to the vertebral body (costovertebral joints) and one connecting the necks and tubercles of the ribs with the transverse process (costotransverse joints). The costovertebral joints of ribs 1 and 10 to 12 articulate with a single vertebral body. In the remaining costovertebral joints, the rib heads articulate with adjacent vertebral bodies. A facet on the posterior tubercle of the ribs and a corresponding facet on ribs 1 to 10 form the costotransverse articulations. Ribs 11 and 12 do not have costotransverse articulations.

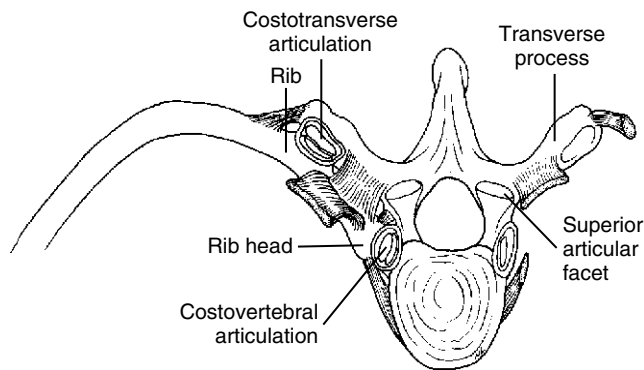


Figure 5-117 Axial view of a thoracic vertebra with rib attachments.

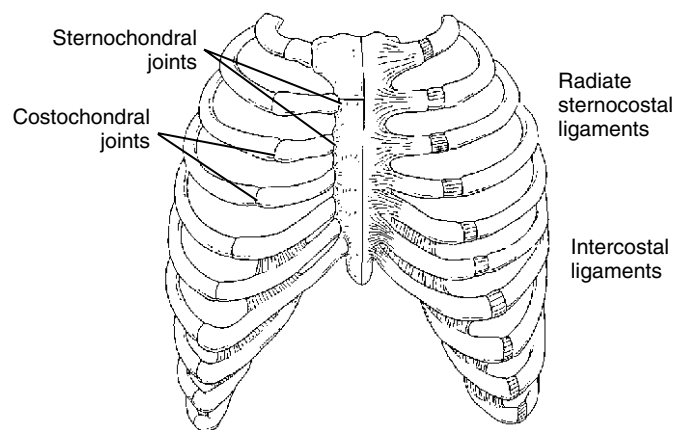


Figure 5-118 Anterior attachments of the ribs to the sternum: 2 to 7 directly and 8 to 10 indirectly via costocartilage.

The articulations that are formed between the vertebral and costovertebral bodies and the transverse and costotransverse processes are each tightly secured by ligaments (Figure 5-117). Both of these articulations are true synovial joints. The costotransverse articulation is surrounded by a joint capsule, with further strength from the costotransverse ligaments. The costovertebral articulations have a single capsular ligament surrounding the two demi-facet articulations, which are further strengthened by the radiate ligament.

These synovial joints are prone to the same pathologic conditions that affect other synovial joints, including the subluxation and dysfunction complex. Furthermore, the ribs play an integral part in the normal activity of the thoracic functional unit and should be a significant consideration in evaluation for thoracic dysfunction.

Anteriorly, the first seven ribs connect to the sternum directly, and the eighth, ninth, and tenth ribs attach indirectly via the costocartilage (Figure 5-118). The eleventh and twelfth ribs are free floating, with no anterior attachment. The anterior articulations move mainly because of the elastic quality of the costocartilage. Calcification and subsequent decreases in movement can occur with age.

Movements of the Rib Cage with Spinal Movements

The ribs influence movement of the individual thoracic vertebrae, and the rib cage influences the movement of the entire thoracic spine. With flexion and extension, the ribs move correspondingly

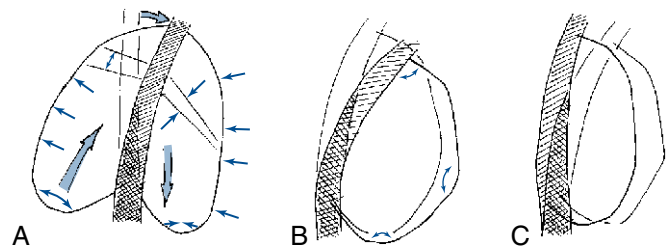


Figure 5-119 The effects of lateral flexion (A), flexion (B), and extension (C) on the shape of the rib cage.

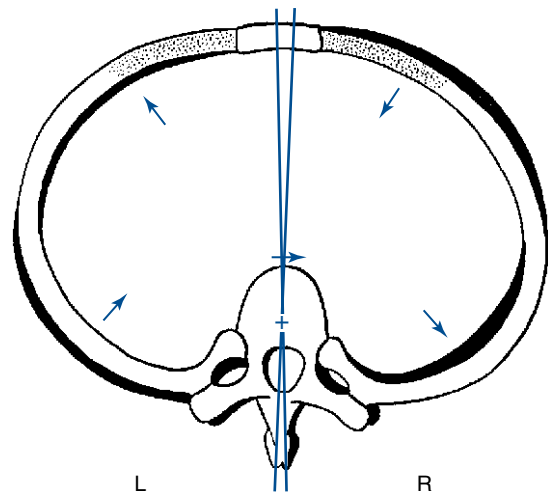


Figure 5-120 The effects of right rotation of a thoracic vertebra on its associated rib, leading to accentuation of the posterior concavity of the rib on the side of vertebral rotation and flattening of the posterior concavity of the rib on the opposite side. (From Kapandji IA. In: *The physiology of the joints*, ed 2, vol 3, Edinburgh, 1974, Churchill Livingstone.)

with the thoracic spine, resulting in the posterior intercostal spaces opening up with flexion and closing with extension. The entire rib cage must flatten superiorly and inferiorly, increasing the sternal angle, for flexion of the thoracic spine to take place. The converse is true for extension (Figure 5-119). A similar relationship occurs with lateral flexion as the rib cage is depressed on the side of lateral flexion. Furthermore, the lateral intercostal spaces open on the convex side and close on the concave side. With thoracic rotation, the rib angle is accentuated on the side of posterior trunk rotation, and flattening of the rib angle occurs on the side of anterior trunk rotation (Figure 5-120).

Movements of the Rib Cage with Respiration

Individually and collectively, the ribs undergo two main types of motion during respiration. These movements are commonly referred to as *bucket handle* and *pump handle* movements.

Bucket-handle movement increases the transverse diameter of the rib cage by elevating the rib and its costochondral arch (Figure 5-121). Bucket-handle movement is greater in the lower thoracic spine, where the relatively flat tubercular facets of the ribs and corresponding articular facets of the transverse processes allow the rib to ride up and down against the transverse process. The lower ribs may therefore roll around an axis connecting the costovertebral and sternochondral joints. This allows for elevation

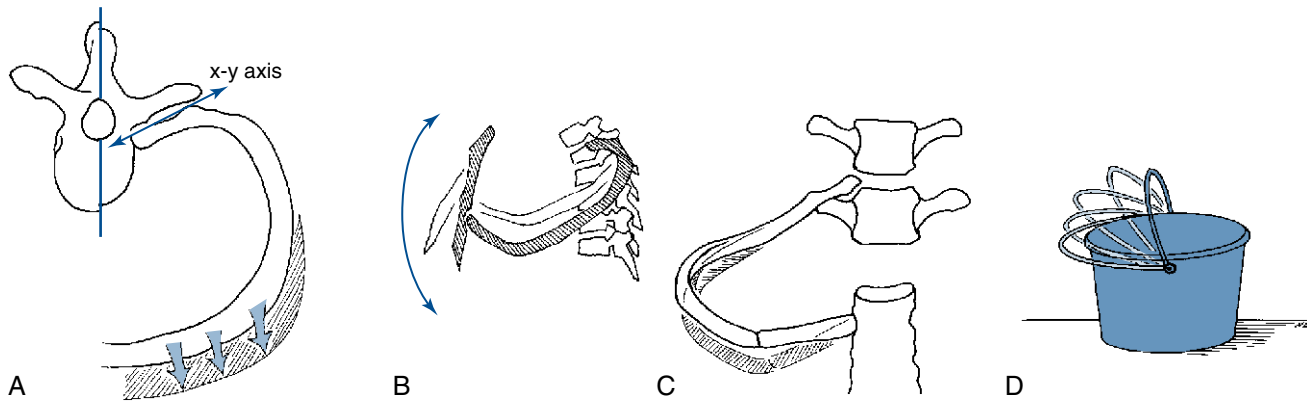


Figure 5-121 Movements of the ribs. **A**, Axial view of pump-handle movement, with the rib rotating around the x-y axis, elevating the rib in front. **B**, Lateral view, demonstrating elevation of the rib and anterior-to-posterior expansion of the rib cage. **C**, Bucket-handle movement, illustrating transverse expansion of the rib cage.

and depression of the ribs with respiration and a movement that simulates the rolling movement of a bucket handle when it is elevated on its hinges.²⁹

Pump-handle movement increases the A-P diameter of the rib cage. It occurs more in the upper rib cage than in the lower and results from the elevation of the anterior aspect of the rib cage with the upward and forward movement of the sternum. In contrast to the lower ribs, the tubercular facets of the upper ribs are situated in deep cup-shaped sockets on the transverse processes. Therefore, the rib is free to move only along the axis that connects the costovertebral and costotransverse joints. With inspiration, the rib head rolls downward, elevating the anterior end of the rib like the handle of a pump²⁸ (see Figure 5-121).

Kinetics of Respiration

During quiet respiration, thoracic mobility is minimal because the diaphragm is the main muscle of respiration. The intercostal muscles are slightly active to supply tension, and the quadratus lumborum fixes the twelfth rib to provide a stable attachment. However, during forced respiration, the external intercostal muscles become active to elevate the ribs and receive secondary help as needed from the scaleni, pectoralis minor, serratus anterior, and iliocostalis cervicis.

Expiration is usually a passive process resulting from the elastic tension produced in the ribs, costocartilage, and pulmonary parenchyma. Forced expiration is produced by the internal intercostal muscles, which receive secondary help from the abdominal muscles, iliocostalis lumborum, longissimus, and quadratus lumborum. The activity of the expiratory muscles is also used to perform the Valsalva maneuver, increasing intra-abdominal pressure.

Upper rib dysfunction is theorized to be associated with ribs fixated in a superior (flexed) position, as a result of the pull of the iliocostalis cervicis, longissimus cervicis, scalenes, and serratus posterior and superior muscles. Similarly, because of the effects of the longissimus thoracis, iliocostalis lumborum, quadratus lumborum, and serratus posterior and inferior muscles, the lower ribs tend to be pulled and fixated inferiorly. However, the iliocostalis thoracis muscle may produce the opposite movement in each area, depressing the upper ribs and raising the lower ribs.

FUNCTIONAL ANATOMY AND CHARACTERISTICS OF THE TRANSITIONAL AREAS

The thoracocervical (C6–T3) and thoracolumbar (T10–L1) segments form a transition between the thoracic spine and the cervical and lumbar regions. Hence, some characteristics or activities are shared by both regions and some are unique to each region.

Thoracocervical Junction (C6–T3)

The notable structural changes in this segment include spinous processes that become more elongated, point caudally, and lose the bifid characteristic of the cervical spine. Furthermore, there are no uncinate processes or transverse foramen. The upper thoracic segments include costotransverse and costovertebral articulations, as well as an increased slope to the articular facets.

Because of the distal attachment of the cervical muscles, including the splenius, longissimus, and semispinalis cervicis, as well as the semispinalis capitis muscles, cervical spine movements involve the upper thoracic spine. The ribs in this area provide stability but also decrease motion. Movements in all directions are decreased between C6 and T3, but the coupled movements in this area are the same as for the typical cervical region (e.g., lateral flexion is coupled with rotation to the same side).

The significance of this area is twofold. First, this area is structurally and functionally related to the neurovascular structures of the upper extremities, because this area forms the thoracic outlet (Figure 5-122). Second, the thoracocervical junction has been deemed a difficult area to apply manipulative therapy. This reputation has been established because of the necessary structural characteristics for a transition from the most mobile area of the spine to the area that is significantly less mobile, as well as the external characteristics of distribution of body fat (dowager hump) and the shoulder and scapular muscles.

Thoracolumbar Junction (T10–L1)

The thoracolumbar transition area is similar to the thoracocervical junction in that it must serve to join an area of greater mobility with one of lesser mobility, as well as change from a primary (kyphotic) curve to a secondary (lordotic) curve. The most

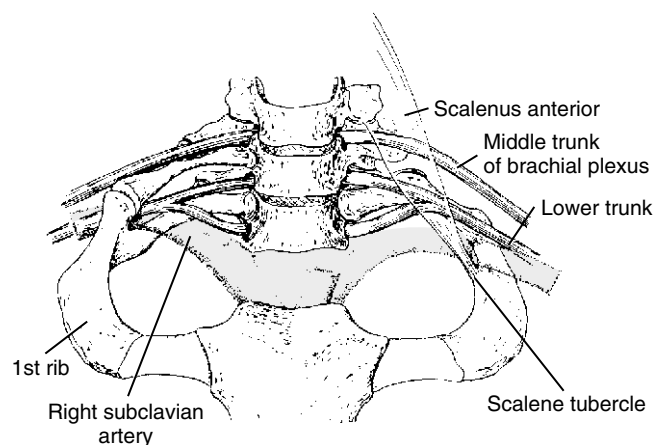


Figure 5-122 The cervicothoracic junction and its relationship to the neurovascular bundle. (From Grieve GP: *Common vertebral joint problems*, ed 2, Edinburgh, 1988, Churchill Livingstone.)

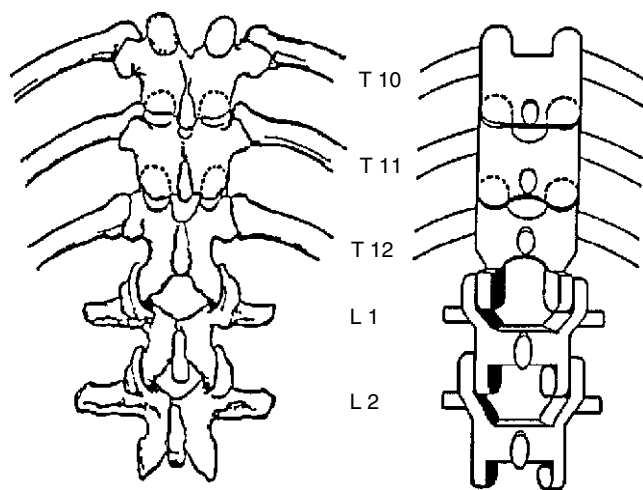


Figure 5-123 The thoracolumbar transition is characterized by a change in facet planes from coronal to sagittal.

significant structural characteristic in this area is the change from the coronal facet plane in the thoracic spine to the sagittal plane facets in the lumbar spine (Figure 5-123). This transition, although typically thought to occur at the T12–L1 segment, has been shown to occur at any of the segments between T10 and L1. Davis³⁰ reported the change was found to occur most commonly at the T11–12 level (Table 5-5).

Of further clinical importance is the distribution of the lateral branches of the posterior primary rami of the spinal roots of T12–L2. These nerves form the cluneal nerves and innervate the skin and superficial structures of the upper posterolateral buttock, posterior iliac crest, and groin area (Figure 5-124). Dysfunction within the lower thoracic segments may refer pain into these regions and be mistaken for disorders of the lumbosacral or sacroiliac regions, which also commonly refer pain to these zones. Maigne³¹ believes this syndrome can account for up to 60% of chronic and acute backache, generally considered the result of lumbar or sacral joint changes.

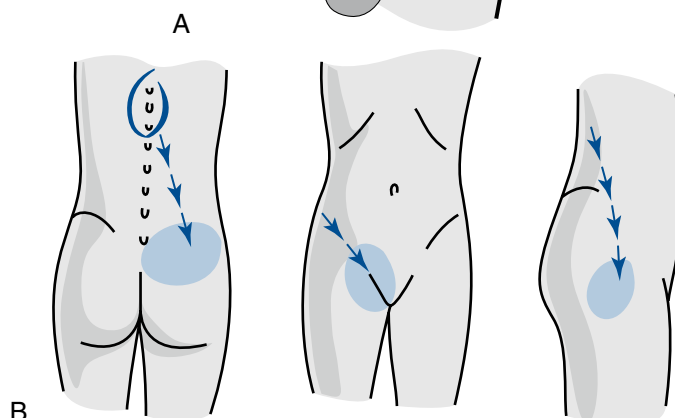
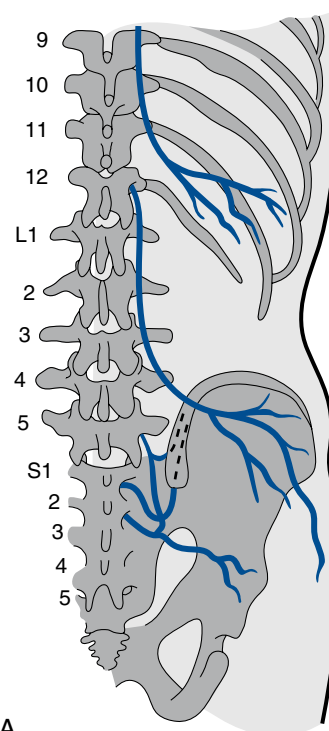


Figure 5-124 The course of the cluneal nerves (A) and possible distribution of pain findings and sensory changes (B). (A from Grieve GP: *Common vertebral joint problems*, ed 2, Edinburgh, 1988, Churchill Livingstone. B from Basmajian JV: *Manipulation, traction and massage*, ed 3, Baltimore, 1985, Williams & Wilkins.)

TABLE 5-5 Frequency of Lower Thoracic Segments Demonstrating Transition from Coronal Facets to Sagittal Facets

Segment	Percentage
T10–11	7.46
T11–12	68.66
T12–L1	23.88
Total	100.00

EVALUATION OF THE THORACIC SPINE

Observation

Before palpation is begun, a visual examination should be made to observe for any deviations in posture or symmetry. Postural syndromes that may predispose the patient to spinal dysfunction and pain are common in the thoracic spine and should not be overlooked. Idiopathic scoliosis has its greatest expression in the thoracic spine, and any noted curvatures should be assessed for flexibility.

Alignment in the coronal plane is evaluated by observing the orientation of the spinous processes, symmetry of paraspinal soft tissues, and contours of the rib cage. The alignment of the shoulders and angles of the scapula should be observed and compared relative to the iliac crests. Sagittal plane alignment is assessed by observing the status of the thoracic curve and noting the position of the gravity line. Orientation of the trunk in the transverse plane is noted by looking at the shoulders and vertebral borders of the scapula for any winging (Figure 5-125).

Global motion of the thoracic spine is typically not separated from lumbar movements. Both are measured and recorded as movements of the entire trunk. However, when desired, regional movements of the thoracic spine may be assessed with the inclinometric measuring methods previously described in Chapter 3 (see Figure 3-10). Table 5-6 presents the average global ROMs for the thoracic spine.

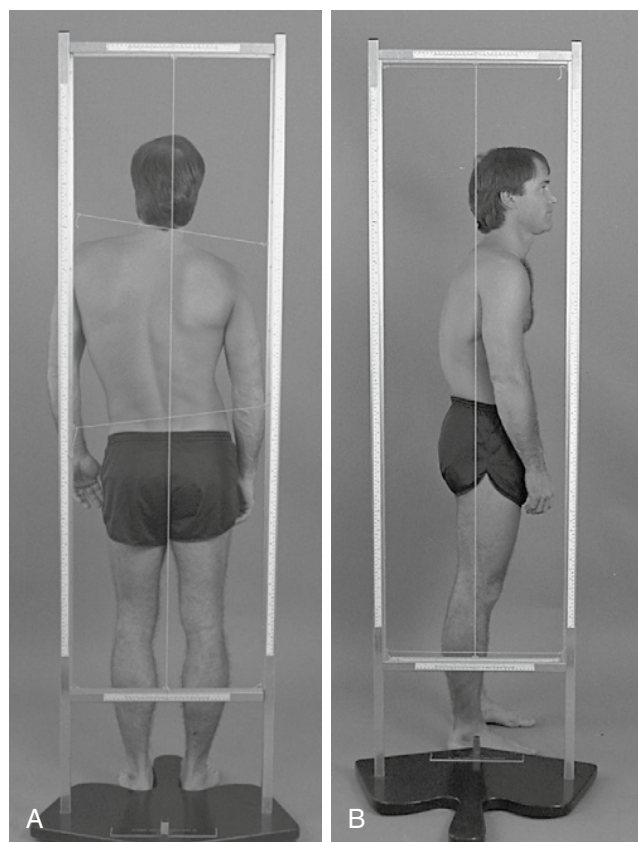


Figure 5-125 Postural evaluation. **A**, Posterior plumb line, demonstrating shoulder and pelvic unleveling creating a C-shaped scoliosis. **B**, Lateral plumb line, demonstrating a round back deformity with an anterior shift in the gravitational line.

TABLE 5-6 Global Range of Motion for the Thoracic Spine

Flexion	25–45 degrees
Extension	25–45 degrees
One-Side Lateral Flexion	20–40 degrees
One-Side Rotation	30–45 degrees

Static Palpation

Static palpation of the spine and posterior chest wall is commonly performed with the patient in the prone position. During the evaluation, stand to the side of the patient and accommodate the patient by bending at the knees, hips, and waist. Palpation typically begins with an assessment of superficial temperature and sensitivity, followed by the assessment of consistency and mobility of the dermal layer and muscular layer. Palpation of bony landmarks incorporates a scanning assessment of contour, tenderness, and alignment of the spinous processes, transverse processes, rib angles, interspinous spaces, and intercostal spaces. In addition, the alignment of the scapula and its borders and angles is customarily included in the evaluation of the thoracic spine.

Potential tenderness and alignment of the spinous processes, interspinous spaces, and transverse processes are assessed with unilateral or bilateral fingertip contacts (Figures 5-126 and 5-127). Paraspinal muscle tone is evaluated by applying bilateral contacts with the palmar surfaces of the fingers or thumbs to explore for areas of tenderness and altered muscle tone and texture (see Figure 5-127).

Rib alignment and tenderness is assessed by palpating along the rib angles with the fingertips or thumb. Palpation may be conducted with the patient in the sitting or prone position. The sitting evaluation has the advantage of being able to induce trunk rotation to accentuate the ribs for palpation. When rib alignment is assessed in the sitting position, the patient is asked to cross the arms over the chest and flex slightly forward. The doctor then sits or stands beside the patient and rotates the patient forward on the side to be palpated (Figure 5-128).

The rib angles should be uniform in prominence and not tender. A tender or distinctly palpable lump that stands out in relation to the adjacent ribs may indicate rib dysfunction. Take care to differentiate a prominent rib from a myofascial trigger point (the former is bony and immobile; the latter softer and more mobile). The pain associated with costotransverse dysfunction is often accentuated with respiration and may radiate to the anterior chest wall. Dysfunction of the costosternal junction may also be present with or without posterior dysfunction. Evaluate the anterior chest wall for myofascial or joint dysfunction when the patient complains of pain of the posterior or anterior chest wall.

Motion Palpation

Joint Play. The thoracic spine should be scanned for sites of painful or abnormal JP with the patient sitting or prone. Areas of elicited abnormality should be further assessed with

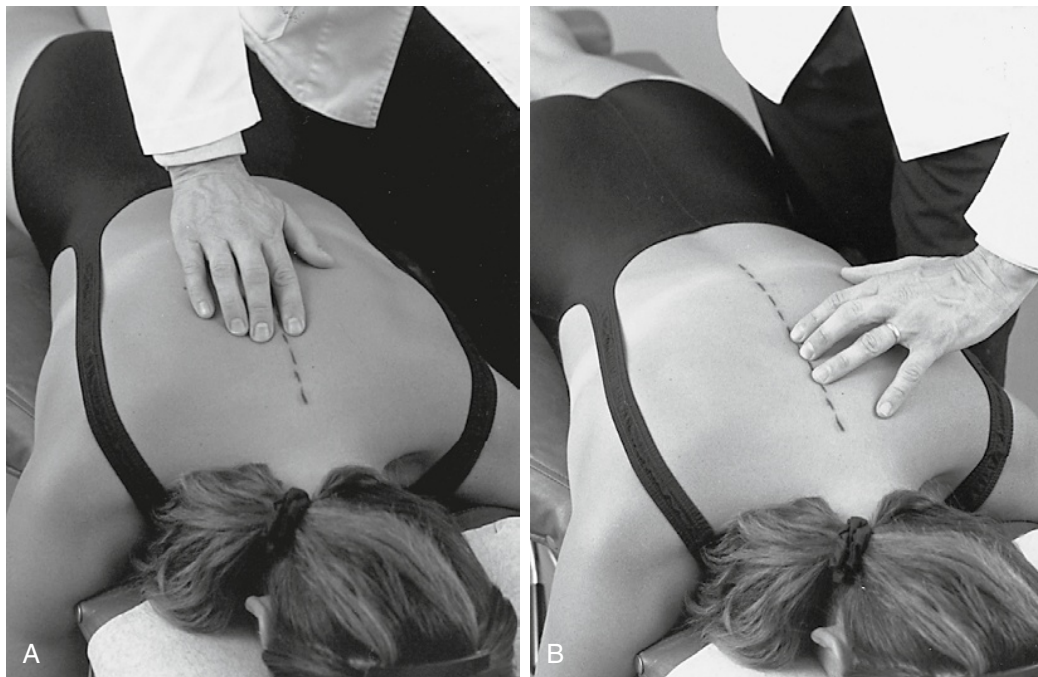


Figure 5-126 Palpation of rotational alignment and sensitivity of thoracic spinous processes (A) and interspinous spaces and sensitivity (B).

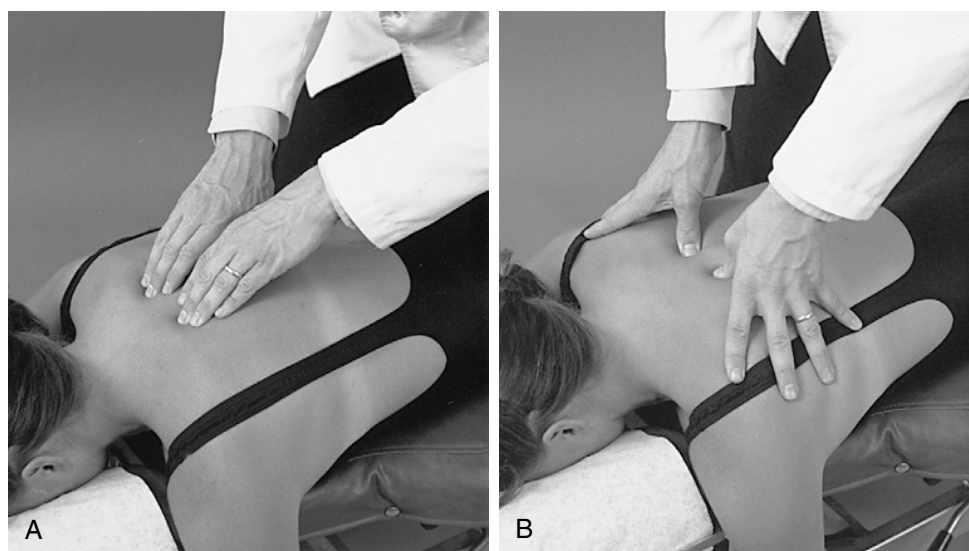


Figure 5-127 Palpation of transverse process alignment and paraspinal soft tissue tone, texture, and sensitivity using fingertip contacts (A) and thumb contacts (B).

counter-rotational JP procedures. With the patient in the prone position, P-A glide is assessed by establishing contacts over the spinous process or bilaterally over the transverse processes. P-A pressure is gradually applied and a springing motion is created. During P-A JP assessment, a subtle pain-free gliding and recoil should be felt at each level tested (Figures 5-129 and 5-130).

To further isolate the specific level of pain and possible dysfunction, counter-rotational JP and provocation testing may be applied. To perform this procedure, place thumbs on opposing sides of adjacent spinous processes and apply springing pressure

toward the midline (Figure 5-131). This procedure is less giving than A-P glide, and a perceptible decrease in movement is encountered when pressure is applied to adjacent vertebrae. Pain elicited at one level and not at adjacent levels helps localize sites of possible dysfunction.

If desired, this procedure may be performed with the contacts located over the transverse processes instead of the spinous processes. Precise location of landmarks is more difficult with this method, but it may provide an acceptable alternative in circumstances in which the patient's spinous processes are tender to light palpation (Figure 5-132).



Figure 5-128 Palpation of the right T6 rib angle.

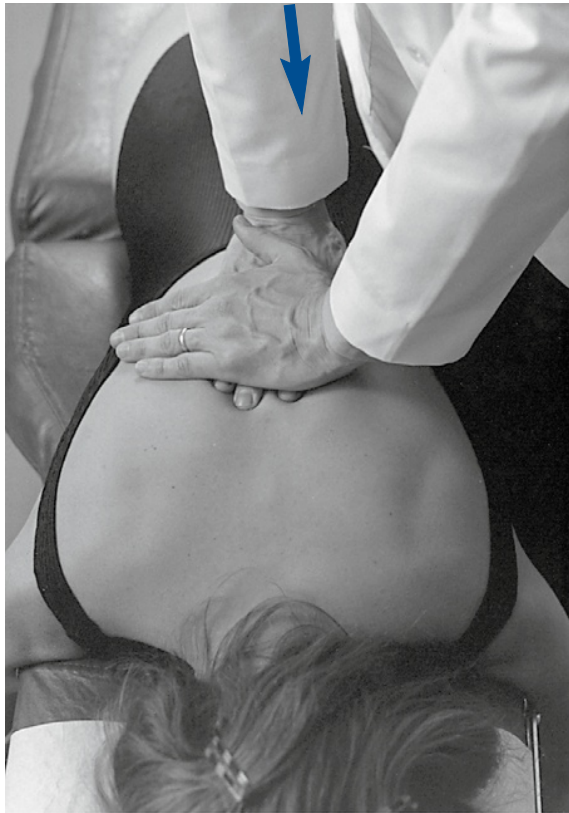


Figure 5-129 Prone midthoracic joint play evaluation, using bilateral fingertip contacts over the transverse processes and applying a posterior-to-anterior vector of force.



Figure 5-130 Prone midthoracic joint play evaluation, using bilateral thenar contacts over the transverse processes and applying a posterior-to-anterior vector of force.

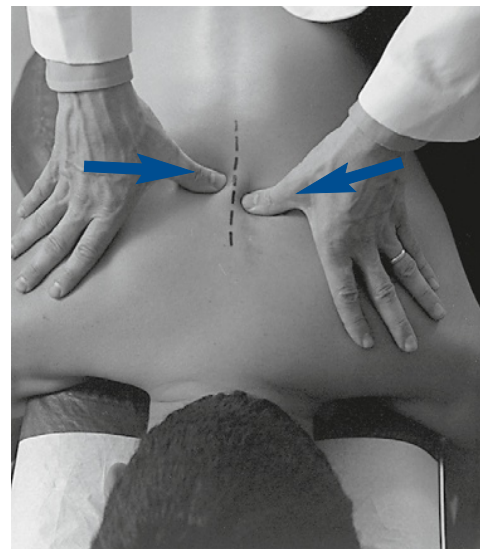


Figure 5-131 Counter-rotational joint play evaluation for left rotational movement of T7 relative to T8. Opposing forces are directed toward the midline through a contact established on the right side of the T8 and the left side of the T7 spinous process.

Segmental Motion Palpation and End Play. Movement is typically evaluated with the patient in the sitting position, with arms flexed and folded across the chest so that the hands can grasp the opposing shoulders. The doctor's position may be sitting behind or standing beside the patient; the standing position is usually preferred when the upper thoracic segments are evaluated. Movement is controlled through contacts on the patient's shoulders for the middle and lower thoracic segments or on the crown of the patient's head when the upper



Figure 5-132 Counter-rotational joint play evaluation for right rotational movement of T5 relative to T6. Opposing forces are directed posteriorly to anteriorly over the right T6 transverse process and left transverse process of the T5.

thoracic segments are evaluated (Figure 5-133). IH contacts on the patient's head are to be avoided in patients with cervical complaints.

Rotation. The segmental contact is established against the lateral surface of the adjacent spinous process on the side of induced rotation. The palpating thumb is placed so that the

pad spans the interspinous space. The support hand reaches around the front of the patient and grasps the opposite flexed arm or shoulder and rotates the patient's trunk toward the side of contact (see Figure 5-133). During normal rotation, the superior spinous process should be palpated, rotating away from the spinous process below. Movement should occur in the direction of trunk rotation. If separation is not noted and adjacent spinous processes move together, segmental restriction should be suspected.

To assess end play, additional overpressure is applied through the contact and IH at the end of passive motion. Firm elastic but giving motion should be encountered. Contacts may be established against the superior spinous process on the side of induced rotation (see Figure 5-133) or over the transverse process and posterior joint on the side opposite the induced rotation (Figure 5-134).

Lateral Flexion. To assess lateral flexion, a segmental contact against the lateral surface of the adjacent spinous processes is established on the side of induced lateral flexion. The doctor either sits or stands behind the patient toward the side of induced lateral flexion. If a sitting position is selected, movement is guided by placing the forearm across the patient's shoulders (Figure 5-135). In the standing position, movement is directed by placing the doctor's hand on the patient's shoulder on the side of induced lateral flexion (Figure 5-136). In the lower thoracic spine, slight patient flexion is produced to accentuate the spinous process and reduce coupled rotation.

Movement is induced by asking the patient to bend to the side as downward pressure is applied through the indifferent arm. As bending is being actively produced, medial pressure is applied through the contact hand to help accentuate the bending at the site of palpation. The indifferent arm and contact arm work together to isolate the site of lateral flexion by adjusting the amount of

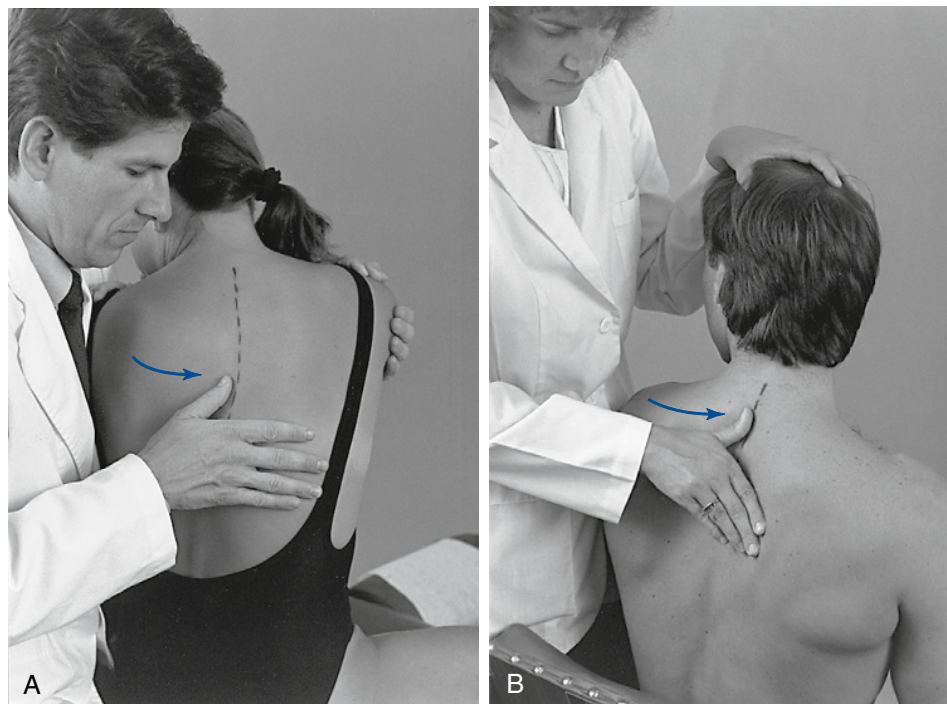


Figure 5-133 Palpation of left rotational movement at the T7–8 level (A) and the T2–3 level (B), using a thumb contact across the left interspinous space.



Figure 5-134 Palpation of left rotational end play over the T7–8 articulation.

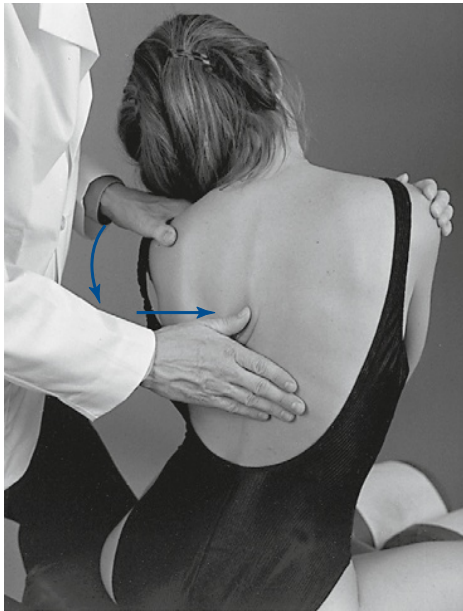


Figure 5-136 Palpation of left lateral flexion at the T6–7 level, with the doctor standing.

applied downward pressure and tilting of the patient. During this movement, the spinous process should be felt to shift toward the opposite side (convex side) while the spine bends smoothly around the contact point (CP). End play is evaluated by applying additional overpressure at the end ROM.

Flexion and Extension. The segmental contacts are established over the interspinous spaces with the doctor's fingertips or thumb. When flexion and extension in the upper thoracic spine are evaluated, movement is guided by placing the IH on the crown of the patient's head (Figure 5-137).



Figure 5-135 Palpation of left lateral flexion movement at T6–7, using a thumb contact across the left T6–7 interspace, with the doctor seated.

When evaluating flexion or extension in the middle to lower thoracic segments, ask the patient to overlap or interlace his or her fingers behind the neck. To evaluate flexion, place your indifferent forearm across the patient's shoulder, or grasp the patient's flexed elbows to help guide movement. To evaluate extension, place your forearm beneath the patient's flexed arms and apply a lifting action to assist in the development of extension (Figure 5-138). To assess motion, actively or passively flex and extend the spine. Take care to place the apex of bending at the level of palpation. During flexion, the interspinous spaces should open symmetrically, and during extension, they should approximate.

Flexion end play is assessed by maintaining palpatory contact with the inferior aspect of the superior spinous process while gentle downward pressure is applied through a forearm contact on the patient's shoulders. Extension end play is evaluated by pushing anteriorly after full extension has been reached. Extension end play is inhibited by the impact of the spinous processes and has a more rigid quality than flexion, lateral flexion, or rotation.

Rib Motion Palpation

Ribs 3 to 12. The evaluation of rib mobility incorporates an assessment of bucket-handle movement and costotransverse end play. End play is assessed by placing the patient in the sitting position and inducing slight flexion, lateral flexion, and rotation of the patient forward on the side of palpation. The palpation contact is established with the doctor's thumb or fingertips over the rib angle, just lateral to costotransverse articulation. The doctor reaches around with the nonpalpating hand to grasp the patient's shoulder and induce rotation. The patient is rotated and the rib is stressed posteriorly to anteriorly at the end of rotation (Figure 5-139). A rib that remains distinctly prominent and provides firm resistance relative to adjacent segments indicates rib dysfunction.

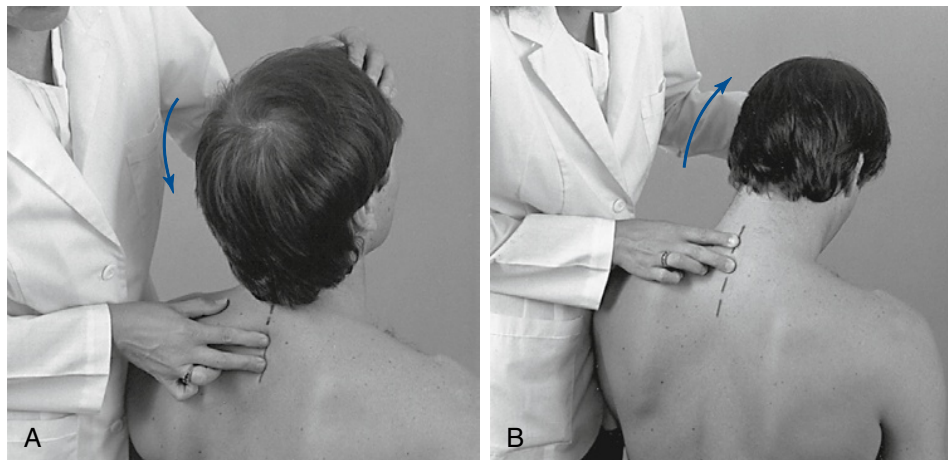


Figure 5-137 Palpation of extension movement (A) and flexion movement (B) in the upper thoracic spine, using fingertip contacts in the interspinous spaces.

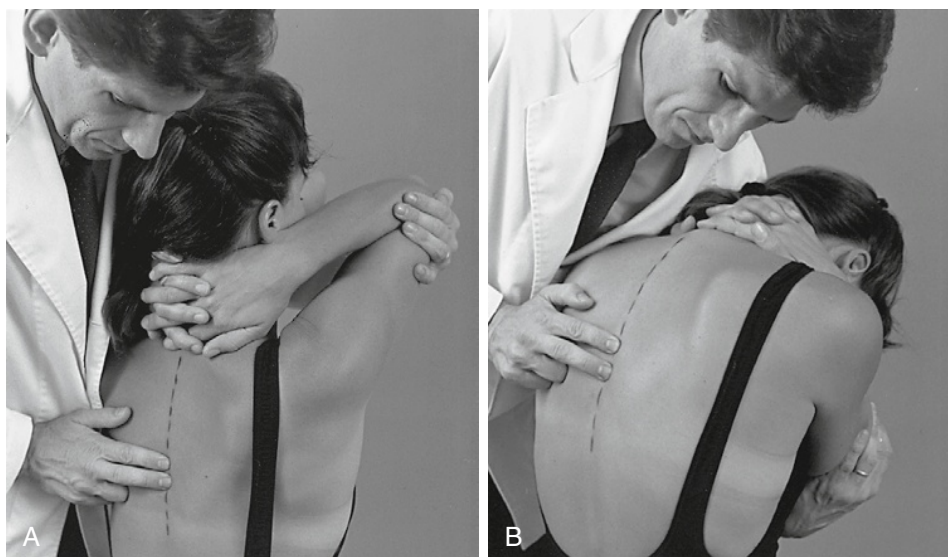


Figure 5-138 Palpation of extension movement (A) and flexion movement (B) in the middle thoracic spine, using fingertip contacts in the interspinous spaces.

To assess bucket-handle motion, the doctor places his or her fingertips in the intercostal spaces at the midaxillary line. The doctor's indifferent arm is placed across the patient's shoulders, and the patient is laterally flexed toward and away from the side of contact (Figure 5-140). The intercostal spaces should open with lateral flexion away from and close with lateral flexion toward the contacts. Absence of symmetric opening and closing may indicate dysfunction at the costotransverse joint or stiffness in the intercostal soft tissues.

Ribs 1 and 2. To evaluate mobility of the upper two ribs, place the patient in a sitting position and contact the PS portion of the first or second rib with the fingertips. The IH grasps the crown of the patient's head, rotates it away from the side being palpated, and laterally flexes and extends the head toward the side being palpated (Figure 5-141). During this motion the rib should drop inferiorly and seem to disappear. Dysfunction should be suspected if the rib remains prominent and immobile during the passive movements of the head.

Anterior Rib Dysfunction. To evaluate movement of the costosternal joints and anterior intercostal spaces, place the patient in the sitting position and stand behind the patient while contacting the intercostal spaces just lateral to the sternum. Take care to avoid contact with the breasts of female patients. Flex the patient's elbow and shoulder on the side of palpation and grasp the elbow (Figure 5-142). Move the patient's flexed arm into further flexion and palpate for opening of the intercostal spaces (e.g., the superior rib should move cranially in relation to the inferior rib).

OVERVIEW OF THORACIC SPINE ADJUSTMENTS

Prone Adjustments

Prone thoracic adjustments are characteristically direct short-level methods (Figure 5-143). They have the advantage of providing effective and specific access to points of contact while allowing

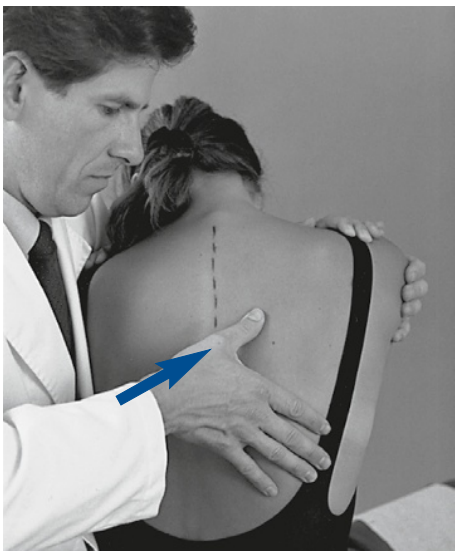


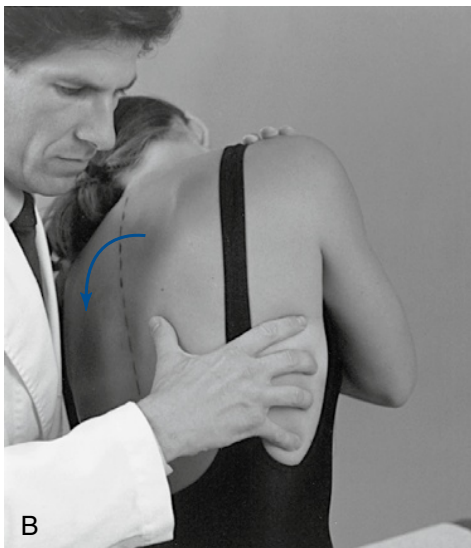
Figure 5-139 Palpation of posterior-to-anterior end play of the right T7 rib articulation, using a thumb contact over the right T7 rib angle.



Figure 5-141 Palpation of the first rib, using a fingertip contact over the superior aspect of the angle of the right first rib.



A



B

Figure 5-140 Palpation for bucket handle rib movement. **A**, Starting position, with fingers in the intercostal spaces in the midaxillary line. **B**, Left lateral flexion movement to evaluate opening of the right intercostal spaces.



Figure 5-142 Palpation of right anterior rib mobility, using a fingertip contact in the right anterior intercostal spaces.



Figure 5-143 Prone unilateral hypothenar transverse adjustment.

for positions that maximize posterior to anterior incorporation of the doctor's body weight in directing and delivering adjustive thrusts.

Although they are commonly delivered with the patient in a relatively neutral position, it is possible to modify prone positioning to induce positions that assist in the development of pre-adjustive tension and the desired movement. Elevation of the thoracolumbar section of an articulating table or Dutchman roll may be used to develop segmental flexion, and the thoracolumbar section may be lowered to induce extension. Placing the patient on his or her forearms on an adjustive bench can also be used to induce more preadjustive segmental extension. Lateral flexion may be induced by bending the patient to the side or by side-bending a flexion table. Rotation of the trunk is not practical on most adjusting tables, although some rotation may be induced with rotation of the pelvic section of flexion tables.

After the patient is appropriately positioned and the contacts are established, the doctor reduces articular slack by transferring additional body weight into the contact. At tension, the adjustive thrust may be generated solely through the arms but more frequently incorporates a combined body-drop and arm thrust.

Knee-Chest Adjustments

Knee-chest adjustments are similar to prone thoracic adjustments (Figure 5-144). In most cases, the only difference is the modification in positioning the patient must undergo on the knee-chest table. Knee-chest adjustments may be applied to any region of the thoracic spine and under many of the same circumstances as prone thoracic adjustments. However, they are probably most effectively applied in the treatment of lower thoracic extension restrictions (flexion malpositions). The knee-chest table does not restrict thoracolumbar extension and therefore allows the doctor to maximize movement into extension. Although it does provide for maximal extension, it also makes the patient vulnerable to hyperextension. Consequently, the doctor must be skilled in this procedure and apply it only with shallow, gentle, nonrecoil thrusts.



Figure 5-144 Knee-chest hypothenar spinous adjustment.

Sitting Adjustments

Sitting thoracic adjustments afford the doctor the opportunity to modify patient position in the development of preadjustive tension (Figure 5-145). They are typically applied as assisted adjustments with the adjustive contact established on the superior vertebra. The IH contacts the anterior forearm to assist in the development of appropriate trunk rotation. At tension, both hands thrust to induce motion in the direction of restriction to induce distraction at the motion segments below the level of contact. They are most commonly applied for rotational restrictions in the middle to lower thoracic spine but may be applied for combined rotation and lateral flexion restrictions.

Supine Adjustments

The effectiveness of supine adjustive techniques is a controversial topic within chiropractic. A number of chiropractic colleges have limited or no instruction in supine techniques, and a significant percentage of practicing chiropractors also object to their application. The basis for this position appears to be related to the contention that supine techniques are less specific and therefore less effective. Unfortunately, this contention has led to a lack of investigation and understanding of the appropriate application of supine techniques. We believe supine techniques should not be dismissed out of hand. They can be effective if applied in the proper circumstances, and they should be considered for incorporation in the management of thoracic dysfunction. Supine techniques allow the doctor the opportunity to significantly modify patient position. Predisposing patients into the direction of desired movement restriction may be helpful in producing the desired adjustive movement and effect. Supine techniques also use the patient's body weight to assist the doctor in developing pre-adjustive tension and adjustive force. Their major disadvantage is access to posterior spinal CPs and the close physical contact that is generally required between the doctor and patient.

One of the major distinctions between prone and supine adjustive techniques is the activity of the posterior spinal contacts. In supine techniques, the posterior contact is customarily passive and provides a fulcrum point for localizing the site of preadjustive tension and reactive adjustive force.

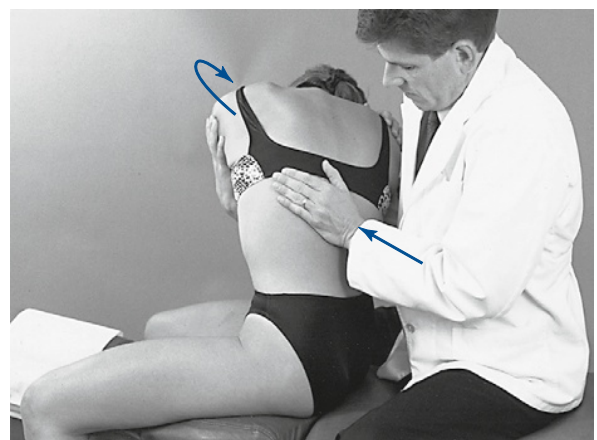


Figure 5-145 Sitting thoracic hypothenar transverse adjustment.

The adjustive impulse in supine technique is generated by thrusting with the weight of the doctor's torso through the patient toward the posterior contact (Figure 5-146). The adjustive thrust accelerates the patient toward the posterior contact and generates a force back toward the patient's spine as the posterior contact meets the firm resistance of the adjustive table.

Supine adjustive techniques also involve a potential component of axial traction applied during the development of preadjustive tension and delivery of an adjustive impulse. Long-axis traction may aid in the distraction of the posterior joints and is helpful in minimizing unnecessary compression to the patient's rib cage. The doctor generates this force by incorporating a small headward (I-S) orientation and movement as he or she develops preadjustive tension.

The positions of the posterior hand contacts vary depending on the area of application and the dysfunction being treated. The optional hand positions (Figure 5-147) and the appropriate application of each are discussed under each specific adjustment. When applying a bilateral transverse contact, the spinous processes rest in the midline of the doctor's cupped hand or clenched fist as the thenar and phalanxes contact each side of the patient's spine over the transverse process. Care must be taken to ensure the contacts are established medial to the rib angles and equally balanced. When developing contacts in the lower thoracic spine, it is important to place the hand in a more vertical position to bridge the distance between the table and the patient.

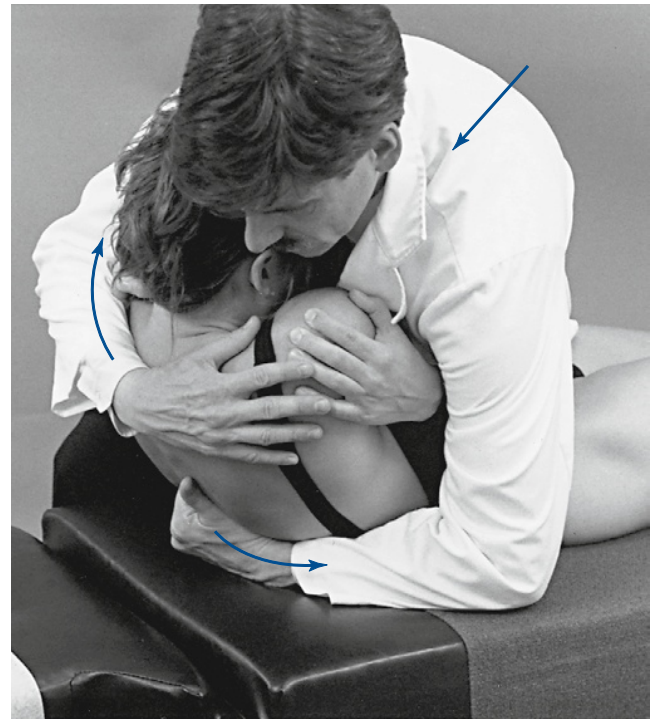


Figure 5-146 Supine thoracic adjustment, using a clenched fist for the posterior contact.

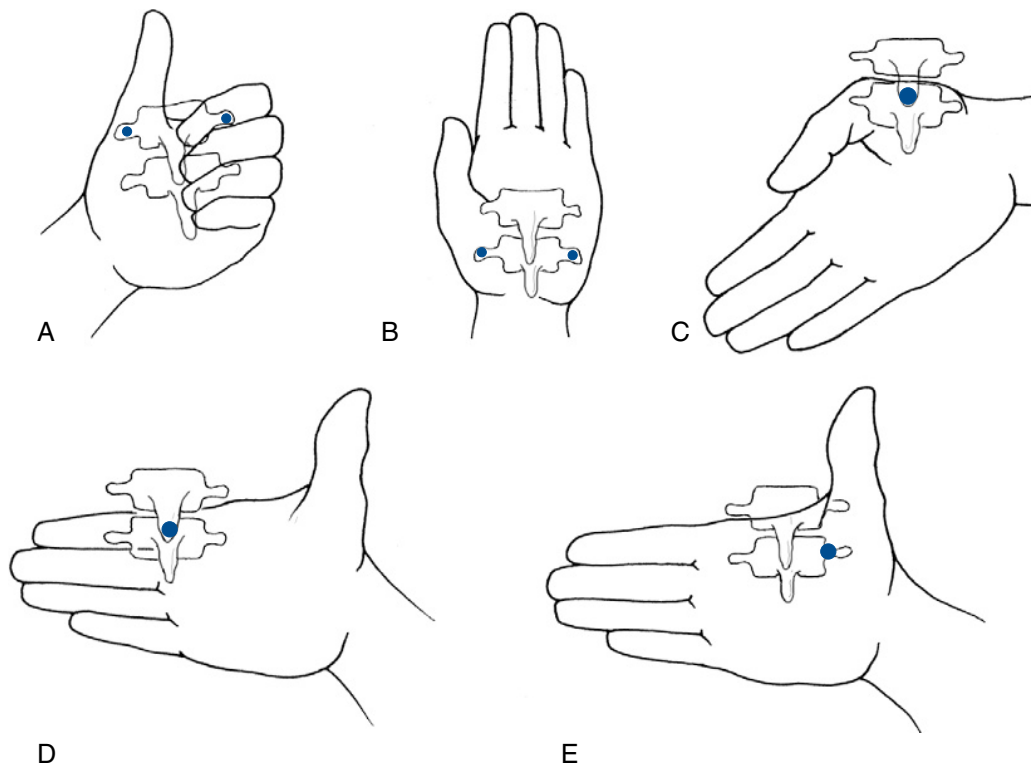


Figure 5-147 Optional hand positions for supine thoracic adjusting. **A**, Clenched fist, where the thenar and index contacts are established bilaterally over the vertebral transverse processes. **B**, Open palm, where the thenar and hypothenar contacts are established bilaterally over the vertebral transverse processes. **C**, Open palm, where the thenar contact is established against the inferior tip of the spinous process. **D**, Open palm, where the index contact is established against the inferior tip of the spinous process. **E**, Open palm, where the thenar contact is unilaterally established over the vertebral transverse process.

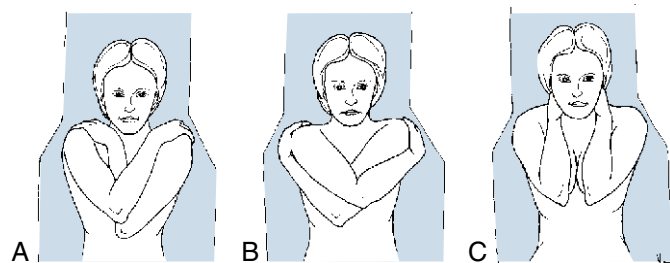


Figure 5-148 Optional patient arm positions in the supine thoracic adjustment. **A**, Right arm crossed over left. **B**, Crossed-arm position to separate the scapula and decrease posterior-to-anterior depth of torso. **C**, Pump-handle position.

Supine adjustive techniques also allow for a variety of optional patient arm placements (Figure 5-148). The positioning of the patient's arms is mainly a matter of doctor discretion. However, when crossing the patient's arms across the chest, it is important to consider both patient and doctor comfort. To reduce the stress to the patient's anterior chest or breasts, a small sternal roll may be placed between the patient's crossed arms. To lessen the pressure against the doctor's upper abdomen or chest, a rectangular pillow may be placed between the patient's crossed arms and the doctor.

When using crossed-arm positions in large patients, it is helpful to cross the arms in a manner that decreases the combined A-P diameter of the patient's thorax. Positions that cross only one arm over the chest or cross the opposing forearm beneath the other (Figure 5-148, *B*) decrease the A-P distance. Positions that interlace crossed arms (Figure 5-148, *A*) tend to produce more padding for the patient's anterior chest but tend to increase the A-P distance from the patient's forearms to the table.

Standing Adjustments

Standing thoracic adjustments use the same mechanical principles as supine thoracic adjustments (Figure 5-149). More significantly, they provide positions that allow the doctor to use the strength of his or her legs in developing preadjustive tension and the adjustive impulse. When applying standing methods, it is important to direct the adjustive force in an A-P and I-S direction to avoid uncomfortable compression of the patient's upper abdomen. Standing adjustments may be difficult to perform in acute patients who cannot withstand weight-bearing and are impractical in situations in which there are large discrepancies in height between doctor and patient.

Rotational Adjustments

Rotational dysfunction of the thoracic spine may result from decreased mobility in the posterior joints and associated soft tissues on one side or both sides of the involved motion segment (Figure 5-150). The side and site of fixation are assessed by comparing each side for subjective and palpatory pain, soft tissue texture asymmetry, and end-play quality.

Rotational dysfunction may be treated with prone, supine, standing, or sitting adjustive methods. Prone methods most commonly use assisted contacts, but methods that apply counterthrust contacts are also frequently used. Resisted methods are not commonly applied in prone patient positions.



Figure 5-149 The standing thoracic adjustment.

Assisted methods are applied with the doctor establishing contacts over the transverse process or spinous process of the superior segments (Figures 5-151 and 5-152). Resisted methods use contacts applied over the transverse or spinous process of the inferior segments (Figures 5-153 and 5-154). Counterthrust methods use contacts applied over the adjacent transverse process (Figure 5-155). When using bilateral contacts, the doctor has the option of making one hand active and one hand passive or of making both hands active. Counterthrust methods use active thrusts through both CPs. Counterstabilizing methods use one active hand and one countersupporting and nonthrusting contact.

When rotational dysfunction is treated, it may be more effective to use bilateral contacts on adjacent vertebral segments as compared with unilateral contacts (see Figure 5-155). This should help isolate the adjustive forces to the site of fixation and reduce tension to adjacent joints.

Prone resisted adjustive methods are commonly applied in the upper thoracic spine (C7–T2). During the application of these methods, the cervical spine is slightly laterally flexed away from and rotated toward the direction of restriction (see Figure 5-153).

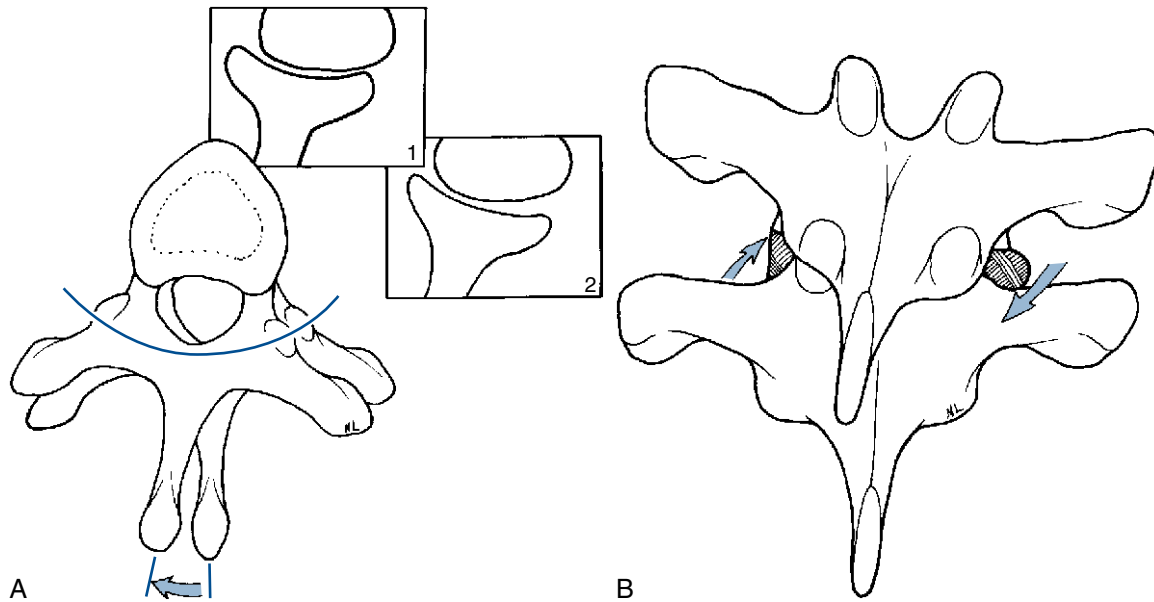


Figure 5-150 A, Transverse view of right rotation at T5–6, showing gliding movement of the left articulation (*Box 1*) and gliding and end-play gaping of the right articulation (*Box 2*). B, Coronal view, illustrating the coupled right lateral flexion associated with right rotation at T5–6, with superior glide of the left T5 articular surface relative to T6 and inferior glide of the right T5 articular surface relative to T6.

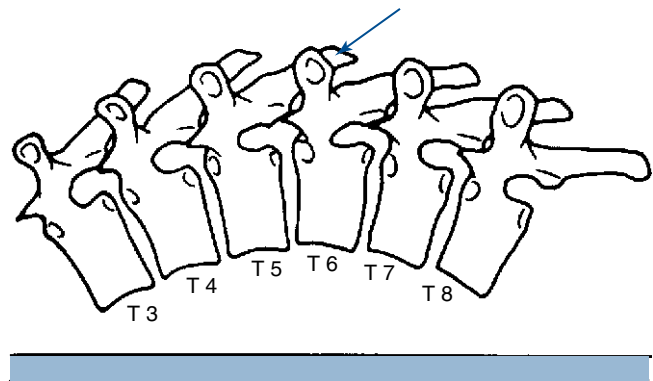


Figure 5-151 Hypothenar transverse contact applied to the right transverse process of T6 to induce left rotation or left lateral flexion of the T6–7 motion segment.

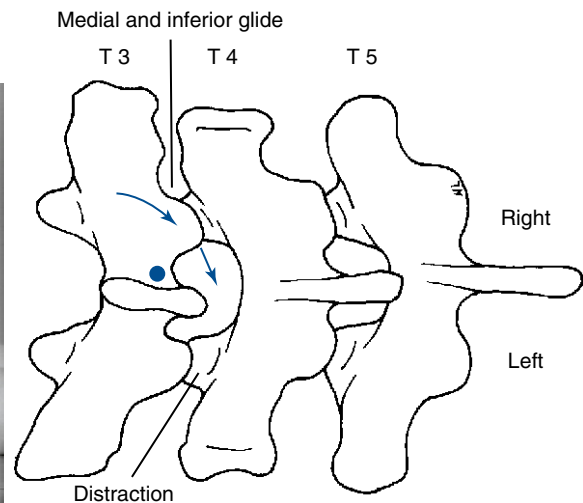
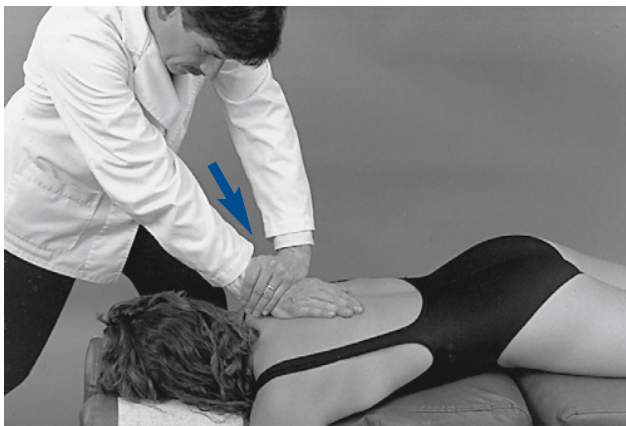
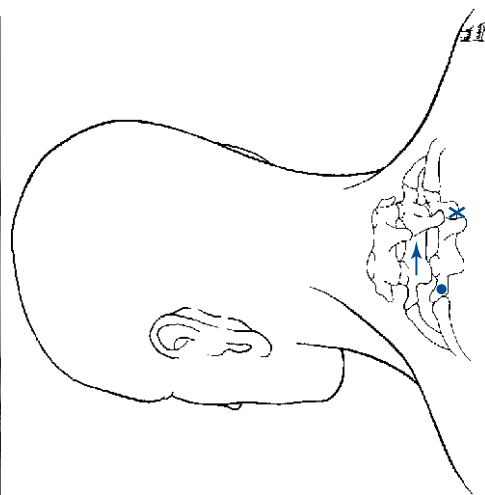
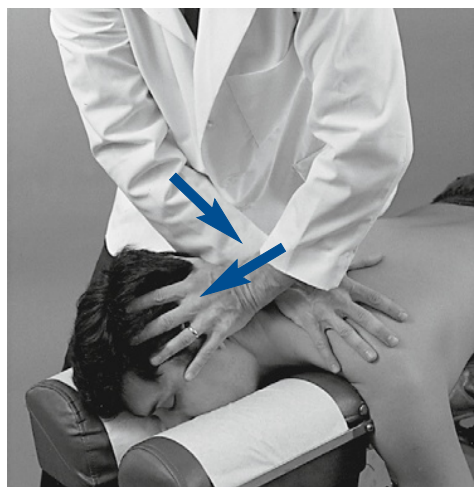


Figure 5-152 Unilateral hypothenar contact applied to the right lateral surface of the T3 spinous process (*dot*) to induce right rotation or right lateral flexion in the T3–4 motion segment. *Arrows* indicate direction of adjustive thrust.



5-153

Figure 5-153 Resisted method. Hypothenar contact applied to the left transverse process of T2 (*dot*) or right spinous process of T2 (*x*), resisted by counter-rotation and lateral flexion of the segments above. Depicted is a procedure for treatment of a left rotation and/or coupled right lateral flexion restriction at T1–2 with distraction of the left T1–2 articulation. *Arrows* indicate the direction of motion induced during the application of the procedure.

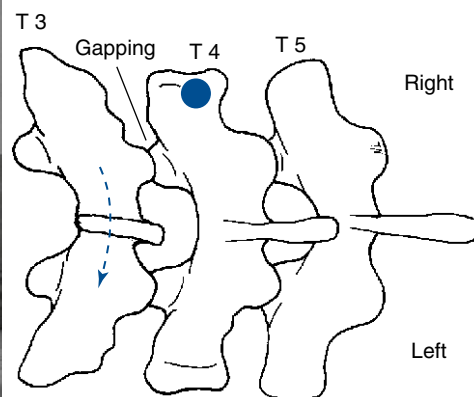
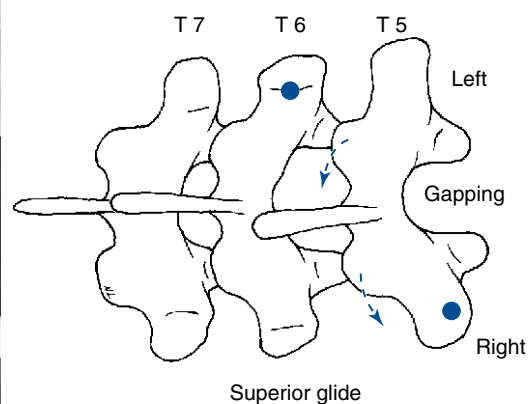


Figure 5-154 Resisted unilateral hypothenar contact applied to the right transverse process of T4 (*dot*) to induce gapping in the right T3–4 articulation. The adjustive force (*solid arrow*) is directed posteriorly. The broken arrow and position of T3 illustrates the relative movement generated between T3 and T4. It does not reflect any starting malpositioned state of T3. This procedure is not commonly applied.



5-155

Figure 5-155 Crossed bilateral contacts applied to the T5–6 motion segment to induce left rotation. The left hypothenar contact is established over the left transverse process of T6 (*dot*) and the right thenar contact is established over the right transverse process of T5. *Solid arrows* illustrated in the picture indicate direction of adjustive force, and the *broken arrows* illustrated in the diagram indicate the motion induced in the T5–6 motion segment during the application of the procedure.

When applying resisted methods with transverse process contacts, the contacts are established on the side of rotational restriction. When applying resisted methods with spinous process contacts, the contacts are established on the side opposite the rotational restriction (see Figure 5-153). The inferior vertebral contact applies counterpressure to induce pretension in the joints superior to the level of contact. At tension, the adjustive thrust is delivered through the contact hand as the IH applies countertraction headward. For example, if the doctor is treating a restriction in left rotation at the T1–2 motion segment with a resisted approach, the contact is established over the left transverse process of T2 or the right lateral surface of the T2 spinous process. The superior segments and head are rotated into left rotation, and maximal tension should be generated in the motion segments superior to the point of adjustive contact (T2) (see Figure 5-153).

When using sitting patient positions in the treatment of rotational dysfunction, it is customary to use assisted methods to aid in the development of trunk rotation. In all assisted methods, the adjustive contact is established over the superior vertebra, and the thrust is directed to induce distraction in the joint below the contact (Figure 5-156).

When applying supine adjustments in the treatment of rotational dysfunction, either assisted or resisted methods may be used. Assisted methods are applied to induce rotation at the segments below the level of contact. Maintaining the patient in a position of segmental flexion may assist the doctor in distracting the joints below the level of contact (Figure 5-157). With resisted methods, the doctor establishes a thenar contact over the transverse process of the vertebra inferior to the level of dysfunction. The contact is established on the side of fixation with the patient's shoulders

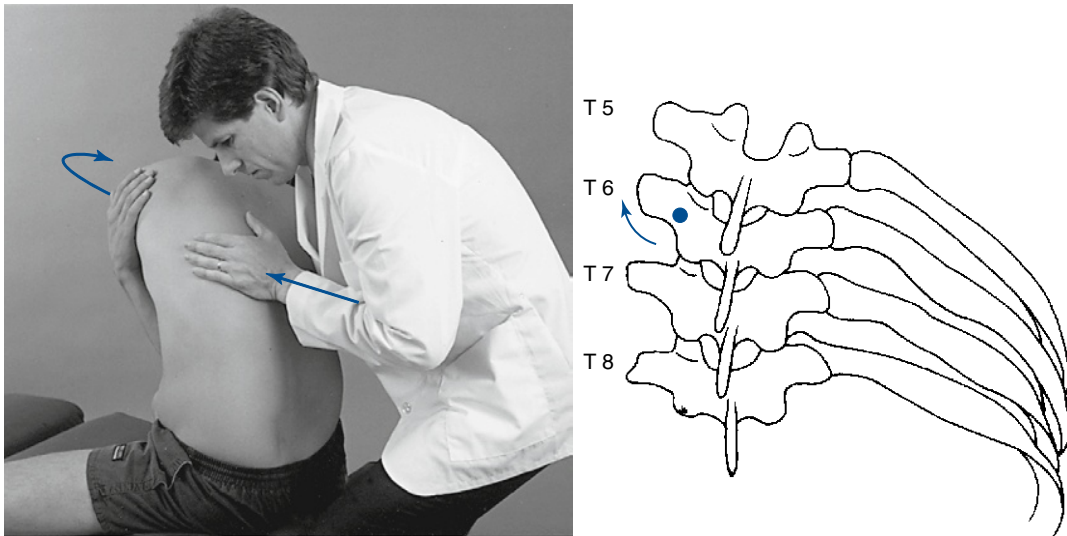


Figure 5-156 Unilateral hypothenar contact applied to the left T6 transverse process (*dot*) to induce right rotation or right lateral flexion.

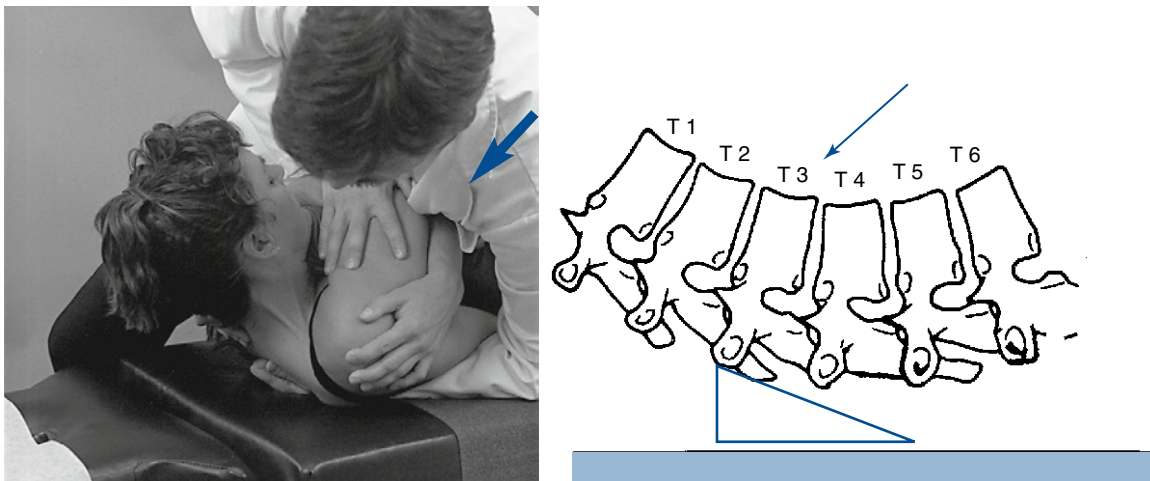


Figure 5-157 Assisted method. Unilateral thenar contact applied to the right transverse process of T3 to induce left rotation or left lateral flexion in the T3–4 motion segment. Wedge illustrates the placement of the hand and thenar, and arrow illustrates the direction of adjustive vector through the doctor's trunk.

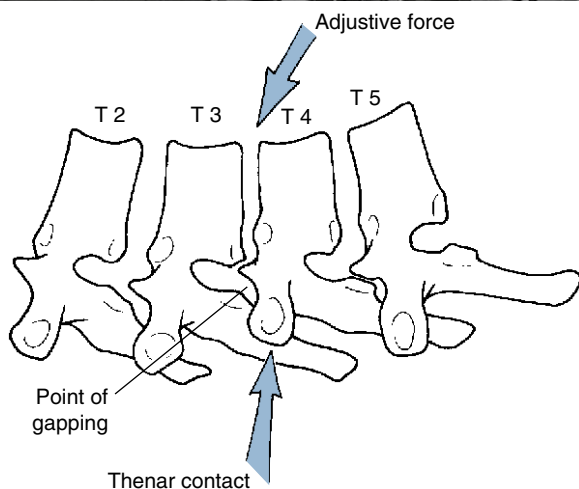
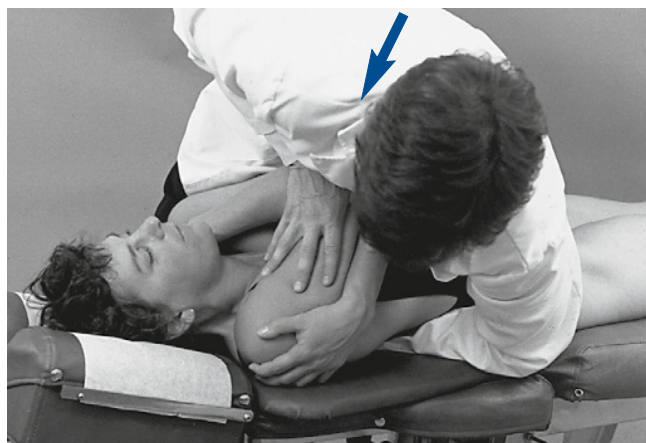


Figure 5-158 Resisted method. Unilateral thenar contact applied to the right transverse process of T4 to induce right rotation and gapping in the right T3–4 articulation.

rotated toward the side of contact (Figure 5-158). The contact provides a block and fulcrum point to induce rotation in the joints above the contact. The adjustive thrust is directed through the doctor's trunk toward the table to induce rotation and gapping in the direction of trunk rotation at the segments above the contact level (see Figure 5-158).

Lateral Flexion Adjustments

Lateral flexion dysfunction in the thoracic spine may result from a loss of inferior glide and approximation of the facet joints on the side of lateral flexion restriction or loss of opening on the side opposite the lateral flexion restriction (Figure 5-159). When treating lateral flexion restrictions in the prone position, the doctor may establish a unilateral transverse process contact on the superior vertebra on the side opposite the lateral flexion restriction and thrust P-A and superiorly (Figure 5-160) or on the side of lateral flexion restriction and thrust P-A and inferiorly. Lateral flexion dysfunction may also be treated in the prone position with bilateral transverse contacts. When applying this method, the contacts are established on each side of the superior vertebra. The contact hand on the side of lateral flexion

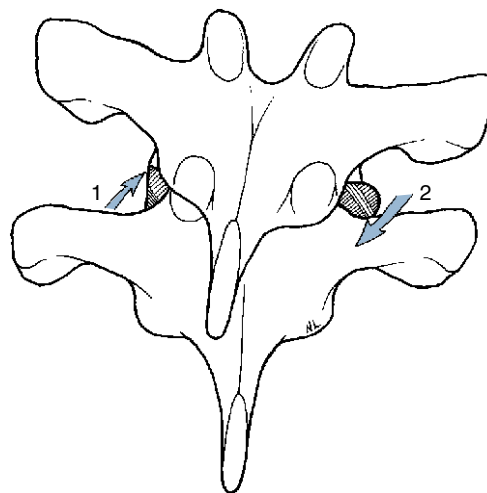


Figure 5-159 Coronal view of right lateral flexion showing gliding distraction of the left articular surfaces (1) and gliding approximation of the right articular surfaces (2).

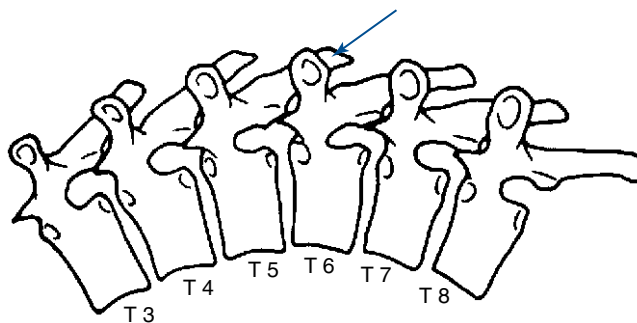
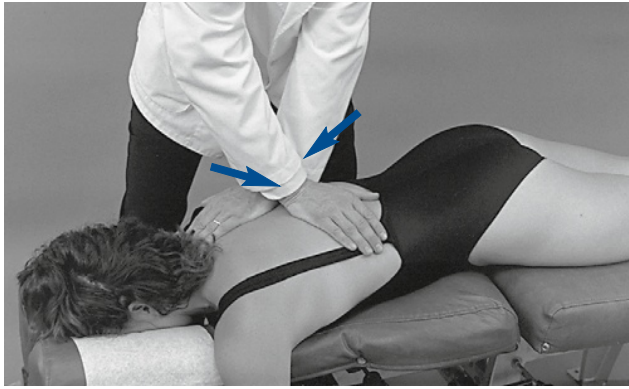


Figure 5-160 Hypothenar transverse contact applied to the right transverse process of T6 to induce right lateral flexion of the T6–T7 motion segment.

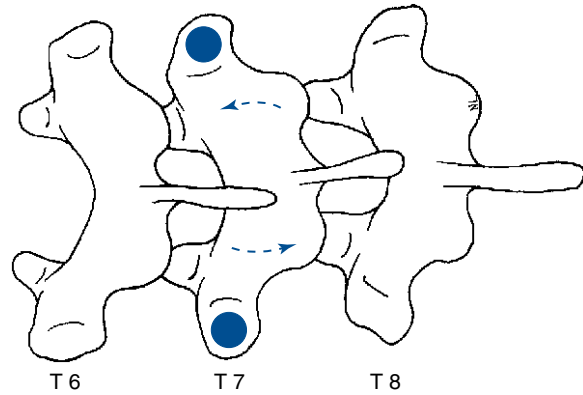
restriction drives anteriorly and inferiorly to induce inferior glide while the other drives anteriorly and superiorly to induce superior glide (Figure 5-161).

When using spinous contacts in the prone position (Figure 5-152), the doctor establishes the contacts on the side of lateral flexion restriction. The thrust is delivered anteriorly and medially toward the midline to induce closure of the facets and disc on the side of lateral flexion restriction.



5-161

Figure 5-161 Bilateral hypothenar contacts applied to the transverse processes of T7 (dots) to induce left lateral flexion at the T7–8 joint.



In prone adjustive methods, with the patient positioned in a neutral position, it is unlikely that the doctor's hand contacts can establish enough tension (grip) with the underlying structures to induce segmental lateral flexion.³² This suggests that using approaches and patient positions capable of prestressing the spine in the direction of desired spinal movement may be preferable.

In prone *upper* thoracic spine adjustments, the doctor may choose to use a resisted method. With resisted methods the contact is established on the lower thoracic segment, and preadjustive tension is developed by tractioning the cervical segments on the side of adjustive contact (Figure 5-153). At tension, an adjustive thrust is directed anteroinferiorly with the contact hand as countertension is directed through the indifferent contact. This method is designed to separate and distract the joints above the SCP.

If lateral flexion dysfunction is treated in the sitting, standing, or supine position, assisted patient positions are commonly used. The contacts are established on the transverse process of the superior vertebra on the side opposite the lateral flexion restriction, and the patient is laterally flexed in the direction of restriction. In the sitting position, the thrust is directed anteriorly and superiorly (Figure 5-156); in the supine and standing positions, the thrust is directed through the trunk posteriorly and superiorly. In the supine and standing positions, the patient must be maintained in a flexed position to assist in the distraction of the involved joint (Figure 5-157).

Flexion and Extension Adjustments

Flexion and extension dysfunction may be treated with prone, knee-chest, supine, or standing patient positions. Flexion restrictions produce a loss of gliding distraction in the posterior joints, and extension restrictions produce a loss of inferior glide and approximation in the posterior joints (Figure 5-162).

To induce flexion in the prone position and distraction of the posterior joints, the doctor commonly establishes contacts against the transverse processes or spinous process of the superior vertebra and directs an adjustive VEC anteriorly and superiorly

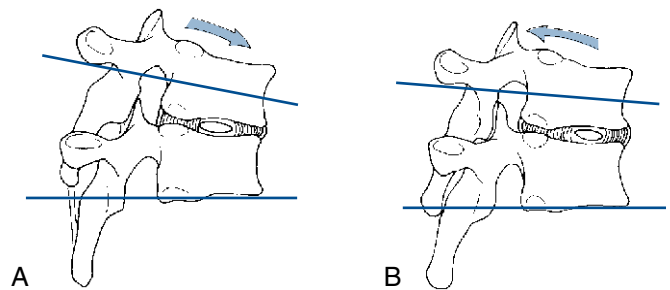


Figure 5-162 Sagittal view of the middle thoracic segments in flexion with separation and gliding distraction of the facet joints (A) and extension with gliding approximation of the facet joints (B).



5-163

Figure 5-163 Assisted bilateral thenar contacts applied to induce flexion of T7–8 motion segment.

(Figure 5-163). In the upper or lower thoracic spine, where superior vertebral contacts may be hard to maintain, the doctor may contact the inferior vertebra of the involved motion segment. With this method, the doctor faces caudally, and the adjustive thrust is directed anteroinferiorly to induce separation superior to the contact (Figure 5-164). In prone adjustive methods, with the patient positioned in a neutral position, it is unlikely that



Figure 5-164 Assisted bilateral thenar contacts applied to T8 to induce flexion of the T7–8 motion segment.

the doctor's hand contacts can establish enough tension (grip) with the underlying structures to induce segmental flexion.³² This suggests that using approaches and patient positions capable of prestressing the spine in the direction of desired spinal movement may be preferable.

Supine adjustive postures may be more effective in the treatment of flexion restrictions by allowing for the production and maintenance of spinal flexion. The adjustive contacts may be established on either the superior or inferior vertebra of the involved motion segment while the patient is maintained in a position of segmental flexion. Superior (assisted) vertebral contacts are designed to distract the motion segments inferior to the level of the contact hand (Figure 5-165). Inferior vertebral contacts are designed to distract the motion segments superior to the level of contact.

With superior vertebral contacts, the doctor establishes contacts with an I-S tissue pull, and an S-I tissue pull is used with inferior vertebral contacts. To develop preadjustive tension, the doctor leans into the patient directing the patient's trunk into the posterior contacts. At tension, the adjustive thrust is delivered posteriorly through the doctor's torso toward the site of contact (see Figure 5-165).

To induce extension in the prone or knee-chest positions, the doctor establishes contacts over the transverse process or spinous process of the superior vertebra of the dysfunctional joint. The doctor's center of gravity is commonly positioned over the contacts with the adjustive VEC directed anteriorly to induce extension (Figure 5-166). In the upper or lower thoracic spine the doctor may face caudally and direct the VEC slightly inferiorly to assist in the development of tension and extension.

To induce extension in the supine or standing patient positions, the contacts are typically established over the inferior vertebral segments. The joint to be adjusted is bent into extension over the top of the posterior contact, and the doctor

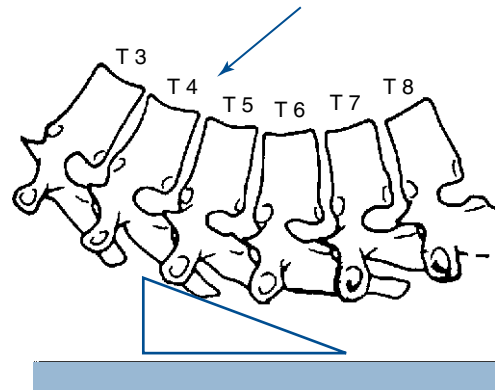
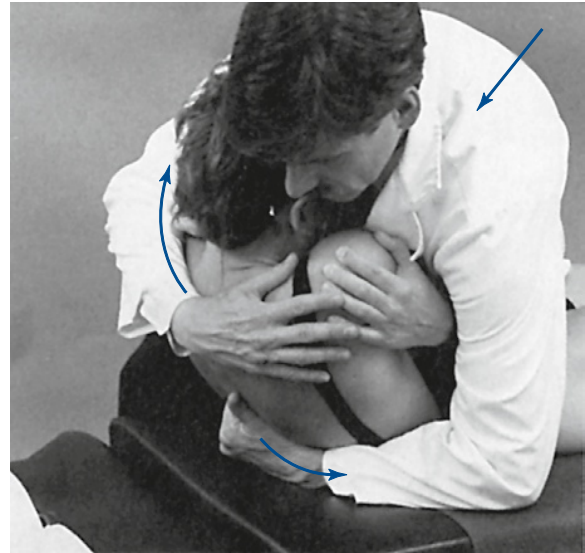


Figure 5-165 Assisted method, with bilateral contacts established on the transverse processes of T5 to induce flexion at the T5–6 motion segment.

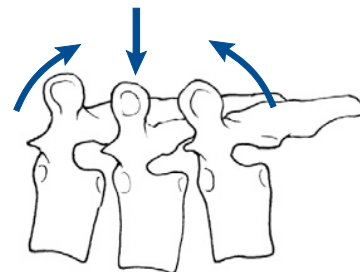
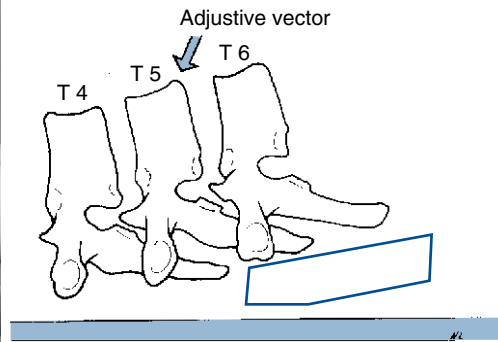


Figure 5-166 Bilateral thenar contacts applied over the T7 transverse processes (*center arrow*), with the adjustive vector directed posteriorly to anteriorly. The posterior-to-anterior thrust generates anterior translation of T7 and extension of T7–8 and T6–7 motion segments.

thrusts posteriorly, using body weight to induce distraction in the joints above the contact (Figure 5-167). This adjustment should also incorporate some long-axis distraction to help decompress the joint. Straight P-A thrusts may unnecessarily compress the rib cage.



5-167

Figure 5-167 Bilateral contacts applied to the transverse processes of T6 to induce extension at T5–6. Block indicates placement of hand under T6 transverse processes and segments below.

OVERVIEW OF RIB ADJUSTMENTS

Prone Rib Adjustments

Prone rib adjustments are usually direct short-lever or combined short- and long-lever methods, incorporating specific but broader contacts. They are delivered with the patient in a relatively neutral position; the doctor's body weight is used in the development of preadjustive tension. Broader contacts are used so that the thrust does not focus the force of the adjustment over a small area of the rib. The rib is more fragile than the vertebral TPs and is therefore more easily injured.

Sitting Rib Adjustments

Sitting rib adjustments, like sitting thoracic adjustments, afford the doctor the opportunity to induce rotation or lateral flexion in the development of preadjustive tension. The adjustive contacts are established on the rib angle just lateral to the transverse process and are applied as assisted adjustments. The IH contacts the anterior forearm to assist in the development of appropriate trunk rotation. At tension, the doctor thrusts to induce distraction at the costotransverse articulation.

Supine Rib Adjustments

Supine rib adjustments can be very effective in the treatment of rib dysfunction and should be considered for incorporation in the management of rib dysfunction. The adjustive impulse in supine rib techniques is generated by thrusting with the weight of the doctor's torso through the patient toward the posterior contact. The posterior contact is established just medial to the rib angle with the doctor's thenar process. The positioning of the patient's arms is mainly a matter of doctor discretion and patient comfort.

Gapping Adjustments of the Costotransverse Articulation. The primary adjustive movement generated at the costotransverse articulation is likely to be one of gapping. This movement is generated as the rib is distracted from its transverse process articulation. This movement is most effectively produced by applying a P-A force against the rib lateral to the transverse process (Figure 5-168).

To induce costotransverse gapping in the prone position, the doctor establishes a contact over the posterior aspect of the rib angle

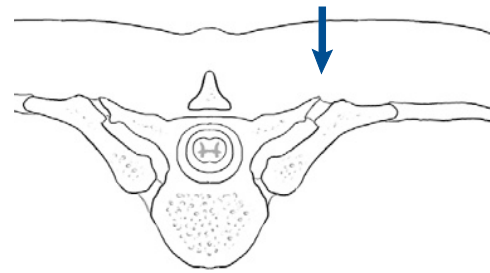


Figure 5-168 Illustration of gapping at the costotransverse articulation induced by a posterior-to-anterior adjustive force.

just lateral to the transverse process. The adjustive VEC is directed posteriorly to anteriorly to depress the rib anteriorly and induce separation at the costotransverse articulation (see Figure 5-192).

To induce gapping in the supine position, the contact is established at the same location, and the doctor accelerates his or her body weight toward the posterior contact (see Figure 5-187). The acceleration of the doctor's body weight through the patient's torso is designed to accelerate the patient into the doctor's posterior contact. The doctor's posterior contact, which is fixed against the adjusting table, produces a reactive force anteriorly against the rib angle, producing gapping at the costotransverse articulation.

Bucket-Handle Rib Adjustments. The side-posture position is most effective for inducing bucket-handle movements. The patient lies over a roll to develop preadjustive tension, and a contact is applied in the intercostal space against the superior or inferior aspect of the rib in the midaxillary line. With the doctor facing cephalad, the contacted rib is elevated; with the doctor facing caudal, the rib is depressed (see Figure 5-195).

THORACIC ADJUSTMENTS

THORACOCERVICAL ADJUSTMENTS (BOX 5-6)

Prone

Thumb/Spinous Push (Figure 5-169)

IND: Restricted rotation or lateral flexion, C6–T3. Rotation, lateral flexion, or combined rotation and lateral flexion malpositions, C6–T3.

BOX 5-6 Thoracocervical Adjustments

- Prone
 - Thumb/spinous push (Figure 5-169)
 - Hypothenar/transverse push (Figure 5-170)
 - Bilateral/thenar and hypothenar/transverse push (Figure 5-171)
- Sitting
 - Thumb/spinous push (Figure 5-172)
- Side-posture
 - Thumb/spinous push (Figure 5-173)

PP: The patient lies prone, with the headpiece lowered below horizontal to produce slight flexion in the thoracocervical spine.

DP: Stand in a low fencer stance on either side of the patient, facing cephaladly. The forward leg approximates the level of the patient's head, and your body weight is centered over the midline of the patient.

CP: Distal palmar surface of the thumb. The thumb is partially abducted and locked, with the fingers resting on the patient's trapezius. When standing on the side of adjustive contact, your caudal hand establishes the contact (Figure 5-169, *A*). A fleshy hypothenar contact may be substituted for the thumb contact (Figure 5-169, *B*). When standing on the side opposite the adjustive contact, the cephalic hand establishes the contact.

SCP: Lateral surface of the spinous process.

IH: Your IH supports the upper cervical spine as the fingers contact the inferior occiput.

VEC: L-M, with slight P-A angulation to maintain the segmental contact.

P: Lightly establish contacts and develop preadjustive tension. Deliver an impulse thrust through the contact and the IH. The impulse generated through the IH is shallow; take care not to excessively rotate or laterally flex the cervical spine.

Rotation: Thoracocervical rotational dysfunction may be treated with assisted or resisted patient positions. When applying assisted positions, establish the contact on the superior spinous process on the side of rotational restriction (side of spinous rotation) (Figure 5-169, *A*). Slightly laterally flex the neck toward the side of contact while slightly rotating it away (e.g., for right-side contact, induce slight right lateral flexion and left rotation). Cervical rotation is minimized to ensure neutral positioning of the thoracocervical spine. The thrust is delivered primarily through the contact hand while the IH produces only modest cephalic distraction.

When using a resisted method, establish the contact on the inferior spinous process on the side opposite the rotational restriction (side opposite the spinous rotation) (Figure 5-169, *B*). Develop preadjustive tension by rotating the patient's head in the direction of joint restriction and laterally flex the head toward the side of adjustive contact. At tension, deliver an impulse counterthrust toward the midline through both hands.

Lateral flexion: When lateral flexion dysfunction is treated, the contact is established on the side of lateral flexion restriction (Figure 5-169, *C*). Preadjustive tension is developed by laterally flexing the patient's head toward the side of contact while inducing minimal contralateral rotation. At tension, an impulse thrust is generated medially through the contact hand while the stabilization hand applies a thrust cephalically.

Hypothenar/Transverse Push (Combination Move and Modified Combination Move) (Figure 5-170)

IND: Restricted rotation and/or coupled lateral flexion, C7–T4. Rotation, lateral flexion, or combined rotation and lateral flexion malpositions, C7–T4.

PP: The patient lies prone, with the headpiece lowered below horizontal to produce slight flexion in the thoracocervical spine.

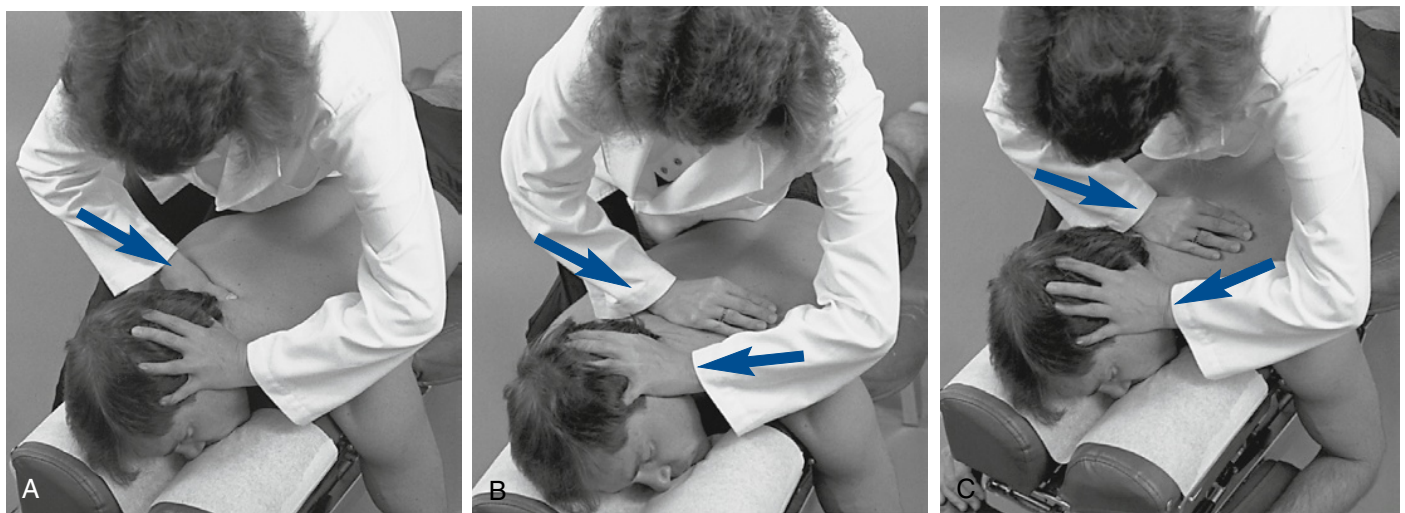
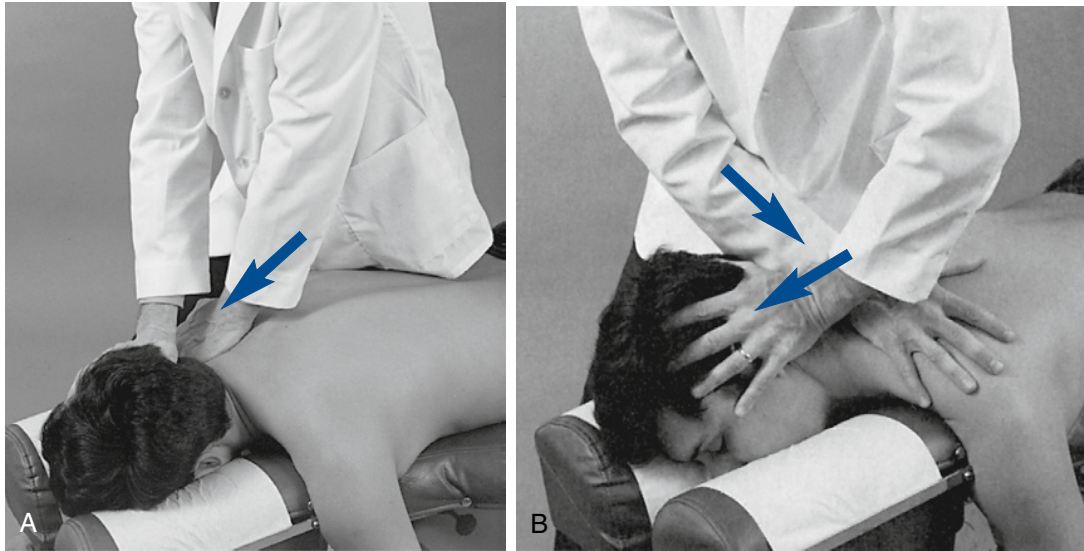


Figure 5-169 **A**, Assisted method, with thumb contact applied to the right lateral aspect of the T2 spinous process to induce right rotation at the T2–3 motion segment. **B**, Resisted method with a hypothenar contact applied to the right lateral surface of the T3 spinous process to induce left rotation at the T2–3 joint. **C**, Hypothenar contact applied to the right lateral surface of the T2 spinous process to induce right lateral flexion in the T2–3 motion segment.



5-170A, B

Figure 5-170 **A**, Assisted position, with hypothenar contact applied to the right T1 transverse process to induce left lateral flexion or left rotation the T1-2 motion segment. **B**, Resisted method, with hypothenar contact applied to the left T2 transverse process to induce left rotation or right lateral flexion of the T1-2 motion segment.

DP: Stand in a fencer stance, facing cephalad. Your forward leg approximates the level of the patient's head, and your upper body weight should be centered over the contact.

CP: Hypothenar (pisiform) of arched hand. When standing on the side of adjustive contact (combination move), the caudal hand establishes the vertebral contact (see Figure 5-170, *A*). When standing on the side opposite the adjustive contact (modified combination move), the cephalic hand establishes the vertebral contact (see Figure 5-170, *B*).

SCP: Transverse process.

IH: The IH supports the upper cervical spine as the fingers contact the inferior occiput.

VEC: P-A, with same-side contacts (see Figure 5-170, *A*). P-A and S-I, with opposite-side contacts (see Figure 5-170, *B*).

P: Lightly establish the contacts and gently rotate and traction the patient's head by producing ipsilateral rotation and contralateral lateral flexion. For example, with a right-side contact, induce left lateral flexion and right rotation of the cervical spine. At tension, deliver a thrust through the contact and IHs. The impulse imparted through the IH is shallow; take care not to excessively rotate or laterally flex the cervical spine. A body-drop thrust typically assists the impulse.

Rotation: Rotational dysfunction may be treated with assisted or resisted methods. With assisted patient positions, the doctor typically stands on the side of adjustive contact (see Figure 5-170, *A*). Establish the segmental contact on the superior vertebra of the dysfunctional motion segment on the side of posterior body rotation (side opposite the rotational restriction). Develop preadjustive tension by leaning anteriorly with the weight of your torso as the IH induces slight lateral flexion away from the side of contact. Deliver the thrust anteriorly to distract the joint below the contact.

With resisted methods, the doctor typically stands on the side opposite the adjustive contact and establishes a contact on the inferior vertebra on the side opposite the side of posterior

vertebral body rotation (see Figure 5-170, *B*). Develop preadjustive tension by rotating the patient's head in the direction of restriction as you apply counterpressure against the transverse process contact. At tension, both arms counterthrust to induce distraction of the articulation superior to the contact.

Lateral flexion: Lateral flexion dysfunction may be treated with assisted or resisted methods. In both methods, axial rotation is minimized, and lateral flexion and gliding distraction are stressed. In the assisted method, the doctor typically stands on the side of adjustive contact, establishes a contact on the superior vertebra, and thrusts anteriorly and superiorly (see Figure 5-170, *A*). Prestressing the joint in the direction of desired lateral flexion may assist in the production of lateral flexion. It is unlikely that this method can induce lateral flexion without also producing coupled rotation.

In the resisted method, the doctor typically stands on the side opposite the adjustive contact, establishes a contact on the inferior vertebra, and thrusts anteriorly and inferiorly in a direction that opposes the thrust generated with the IH (see Figure 5-170, *B*).

Bilateral/Thenar and Hypothenar/Transverse Push (Figure 5-171)

IND: Restricted extension, T1–T4. Flexion malpositions, T1–T4.

PP: The patient lies prone, with the headpiece lowered below horizontal for flexion restrictions and neutral for extension restrictions.

DP: Stand at the head of the table, facing caudad.

CP: Bilateral thenar contacts running parallel to the spine. (Bilateral knife-edge contacts can also be used.)

SCP: Transverse processes of superior vertebra.

VEC: P-A and S-I (see Figure 5-171).

P: Establish hypothenar contacts with an S-I tissue pull and develop joint tension by transferring additional body weight into the contacts. At tension, deliver a thrust through the arms and body.

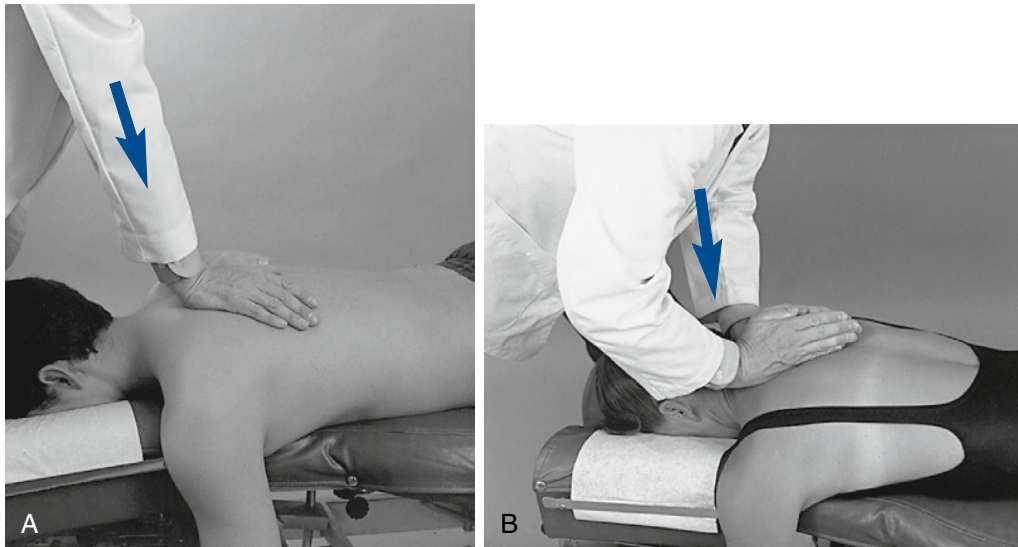


Figure 5-171 (A) Bilateral thenar transverse method, applied with an assisted contact and posterior-to-anterior vector to induce extension. (B) Bilateral hypothenar (knife edge) method, applied with an assisted contact and P-A and S-I vector to induce extension.

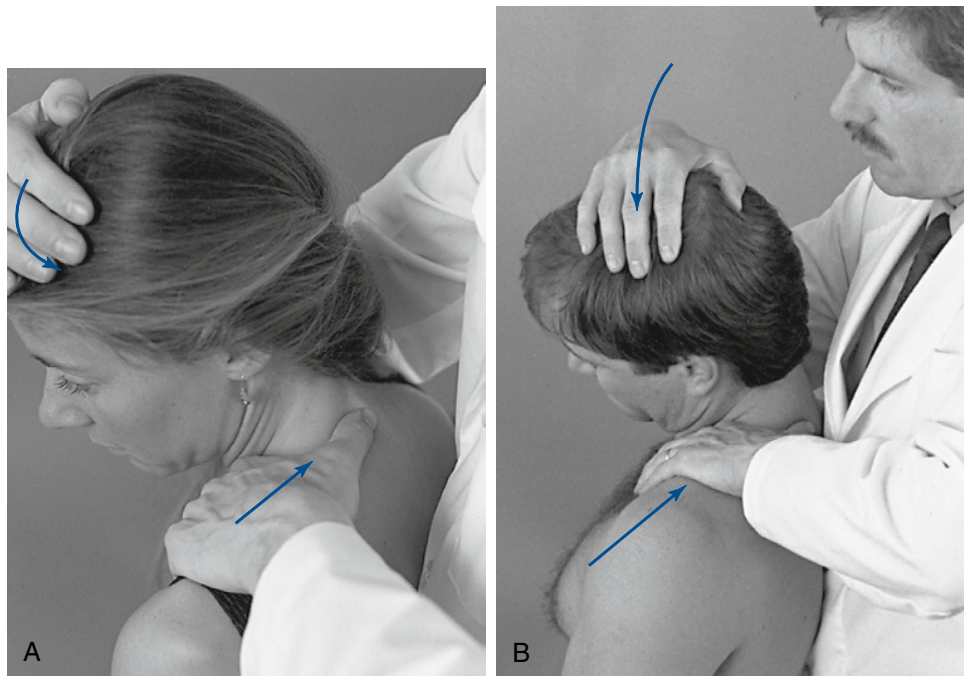


Figure 5-172 A, Assisted method, with a thumb contact applied to the left side of the spinous process of T1 to induce left rotation of T1–2. B, A thumb contact applied to the left side of the spinous process of T1 to induce left lateral flexion of T1–2.

Sitting

Thumb/Spinous Push (Figure 5-172)

IND: Restricted rotation or lateral flexion, C6–T3. Rotation, lateral flexion, or combined rotation and lateral flexion malpositions, C6–T3.

PP: The patient sits relaxed in a cervical chair.

DP: Stand behind the patient, slightly toward the side of spinous contact.

CP: Thumb of contact hand, with palm rotated down.

SCP: Lateral surface of the spinous process.

IH: The contralateral hand contacts the top of the patient's head while the forearm supports the lateral head and face.

VEC: L-M.

P: Establish the contacts and circumduct the patient's head toward the side of spinous contact. At tension, deliver an L-M impulse thrust through the contact hand.

Rotation: Rotational dysfunction may be treated with either assisted or resisted methods. When treating rotational restrictions with an assisted method, contact the superior spinous process on the side of rotational restriction (side opposite body rotation) and rotate the patient's head in the direction of restriction (see Figure 5-172, A). Generate the adjustive thrust by thrusting toward the midline primarily with the contact arm.

When using a resisted method, contact the inferior spinous process on the side opposite the rotational restriction (side of body rotation of superior segment). Rotate the head and segments above in the direction of restriction. At tension, deliver a thrust by thrusting toward the midline through both arms. The greater proportion of the adjustive force is delivered by the contact arm.

Lateral flexion: When treating lateral flexion dysfunction, establish the contact on the superior vertebra on the side of lateral flexion restriction. The patient's neck is laterally flexed in the direction of restriction. At tension, direct an impulse thrust toward the midline through the contact arm. The contact thrust is reinforced by a shallow distractive force delivered with the IH (see Figure 5-172, *B*).

Side Posture

Thumb/Spinous Push (Figure 5-173)

IND: Restricted rotation or lateral flexion, C6–T3. Rotation, lateral flexion, or combined rotation and lateral flexion malpositions, C6–T3.

PP: Place the patient in side posture, with the spine in a neutral position and the patient's head supported in your cephalic hand.

DP: Stand in front of the patient in a square stance.

CP: Thumb or thenar of caudal hand.

SCP: Lateral surface of the spinous process.

IH: The cephalic hand and forearm cradle the patient's cervical spine and head.

VEC: L–M.

P: Stand in front of the patient and lean over to establish the indifferent and segmental contacts. The contacts must be soft and fleshy or they become uncomfortable to the patient. At tension, deliver an impulse thrust laterally to medially through the contact hand.

Lateral flexion: When treating lateral flexion dysfunction, establish the contact on the superior vertebra on the side of lateral flexion restriction. The patient's neck is laterally flexed in the direction of restriction. At tension, direct a thrust toward the

midline through the contact arm. The contact thrust is reinforced by a shallow distractive force delivered with the IH (see Figure 5-173, *A* and *B*).

Rotation: Contact the spinous process on the side of deviation (side of rotational restriction) and rotate the patient's head in the direction of restriction. At tension, a thrust is directed toward the midline. The thrust is reinforced by a shallow rotational pull through the IH.

THORACIC ADJUSTMENTS (BOX 5-7)

BOX 5-7

Thoracic Adjustments

- Prone
 - Bilateral thenar/transverse push (Figure 5-174)
 - Bilateral hypothenar/transverse push (crossed bilateral) (Figure 5-175)
 - Unilateral hypothenar/spinous push (Figure 5-176)
 - Unilateral hypothenar/transverse push (Figure 5-177)
 - Hypothenar spinous crossed thenar/transverse push (Figure 5-178)
- Knee chest
 - Hypothenar/spinous push (Figure 5-179)
 - Hypothenar/transverse and bilateral hypothenar/transverse push (Figure 5-180)
- Supine
 - Supine thoracic opposite-side thenar/transverse drop (Figure 5-181)
 - Supine thoracic same-side thenar/transverse drop (Figure 5-182)
 - Supine thoracic pump handle (opposite or same-side) (Figure 5-183)
- Sitting
 - Hypothenar/transverse pull (Figure 5-184)
- Standing
 - Thenar/transverse push (Figure 5-185)
 - Long-axis distraction (Figure 5-186)

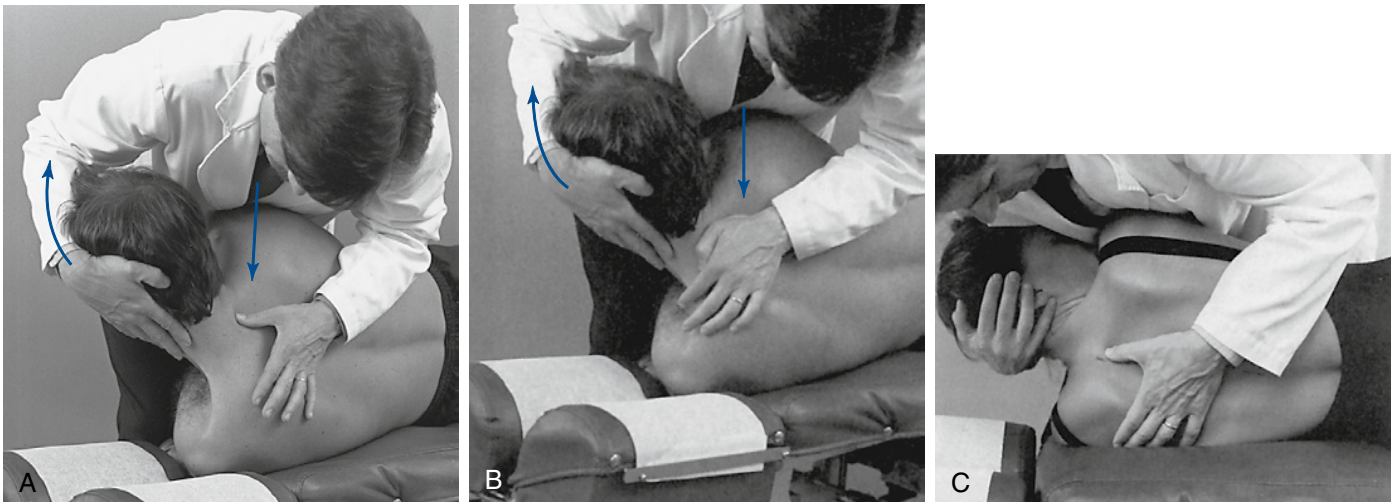


Figure 5-173 Thumb (*A*) or thenar (*B*) contact applied to the right lateral aspect of the C7 spinous process to induce right lateral flexion at the C7–T1 motion segment. *C*, Thumb contact applied to the right lateral aspect of the T1 spinous process to induce right rotation of the T1–2 motion segment.

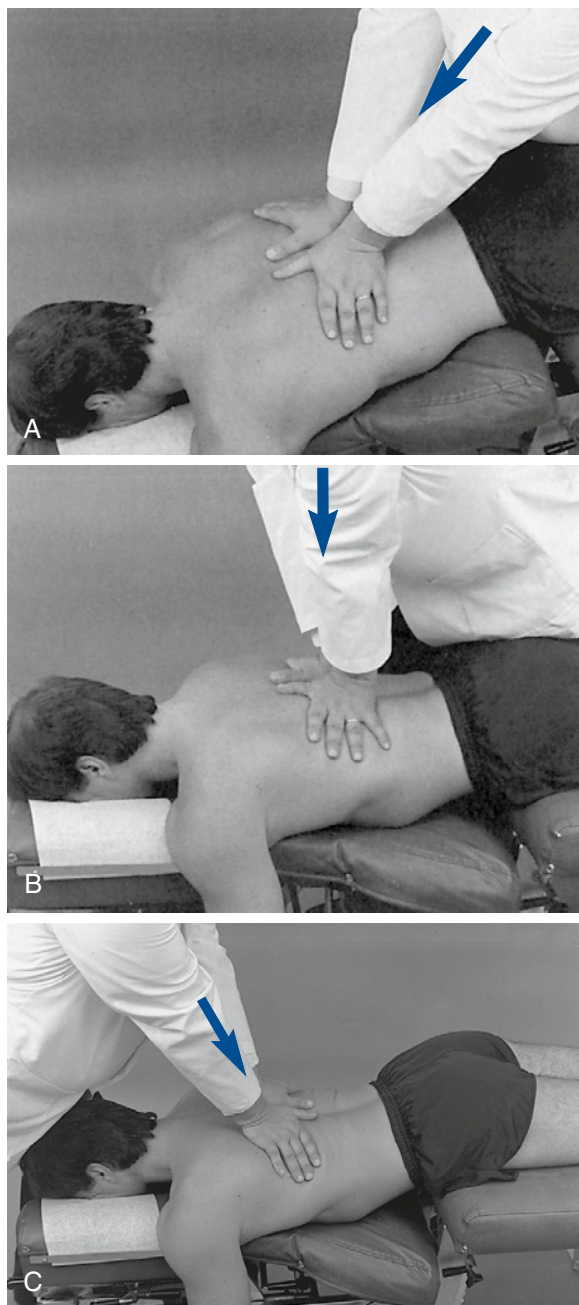


Figure 5-174 Bilateral thenar contacts applied to the T8 transverse processes to induce flexion (A) and extension (B) of the T8-9 motion segment. C, Alternate method to create extension, with the doctor facing caudad.

Prone

Bilateral Thenar/Transverse Push (Figure 5-174)

IND: Restricted flexion, extension, lateral flexion, or rotation, T4–T12. Flexion, extension, lateral flexion, or rotation malpositions, T4–T12.

PP: The patient lies prone. Prestressing spinal joints in the direction of desired adjustive movement may assist the doctor in inducing the desired motion. To provide added flexion, a small roll may be placed under the patient's chest. To provide added extension, the thoracic piece may be lowered anteriorly or the patient may place his or her flexed arms and forearms under the chest.

DP: Stand in a fencer stance on either side of the patient.

CP: Bilateral thenar contacts parallel to the spine, with fingers fanned and running medially to laterally.

SCP: Transverse processes.

VEC: P-A and I-S to induce flexion, lateral flexion, or rotation (see Figure 5-174, A). P-A to induce extension or rotation (see Figure 5-174, B).

P: Establish bilateral thenar contacts and develop joint tension by transferring additional body weight into the contacts while tractioning the superficial tissue in the direction of the adjustive VEC. When using a VEC that is predominantly P-A, the doctor may use either an I-S or S-I tissue pull. The choice depends on the region adjusted and the doctor's preference. At tension, a thrust is delivered through the arms, trunk, and body.

Flexion: To induce flexion, establish the contacts over the superior vertebra and deliver the thrust anteriorly and superiorly through both contacts (see Figure 5-174, A). Placing a roll under the level of adjustive contact may increase flexion pre-adjustive tension.

Extension: To induce extension, establish the contacts over the superior vertebra and deliver the thrust anteriorly through both contacts (see Figure 5-174, B and C). To increase preadjustive tension in extension, the patient may raise his or her torso off the table by rising up on the forearms or by lowering the thoracolumbar section of an articulating table.

Lateral flexion: To induce lateral flexion, establish bilateral contacts over the superior vertebra but deliver the adjustive thrust unilaterally. The thrust is delivered anteriorly and superiorly through the contact established on the side opposite the lateral flexion restriction. It is unlikely that this method can induce lateral flexion without inducing coupled rotation.

Rotation: To induce rotation, establish contacts over the superior or inferior vertebra. With superior vertebral contacts, deliver the thrust anteriorly on the side of posterior body rotation (side opposite the rotation restriction). With an inferior vertebra contact, deliver the thrust anteriorly on the side opposite the posterior body rotation (side of rotational restriction). Inferior vertebra contacts (resisted method) are designed to induce gapping of the posterior joints above the site of contact. Inferior vertebra contacts have not been traditionally used in this manner (see Figure 5-154).

Bilateral Hypothenar/Transverse Push (Crossed Bilateral) (Figure 5-175)

IND: Restricted flexion, lateral flexion, or rotation, T4–T12. Extension, lateral flexion, or rotation malpositions, T4–T12.

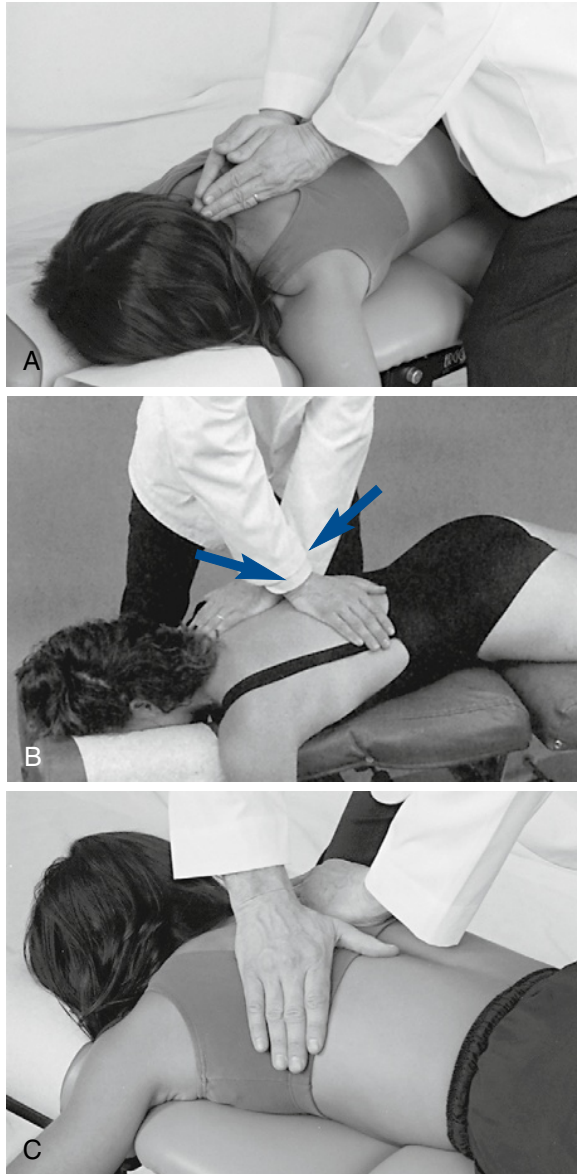
PP: The patient lies prone. Prestressing spinal joints in the direction of desired adjustive movement may assist the doctor in inducing the desired motion. To provide added flexion, a small roll may be placed under the patient's chest.

DP: Stand in a fencer, modified fencer, or square stance, depending on the restriction treated. Stand on either side of the patient.

CP: Bilateral hypothenar (pisiform) contacts. A thenar contact may be substituted for the crossing hypothenar contact

SCP: Transverse processes.

VEC: P-A (see following discussion).



5-175B

Figure 5-175 A, Bilateral hypothenar contacts applied to the transverse processes of T6 to induce flexion at T6–7. B, Crossed bilateral hypothenar contacts applied to the right T6 transverse process and the left T7 transverse process to induce left rotation of the T6–7 motion segment. C, Crossed bilateral hypothenar/thenar contacts applied to right and left transverse processes of T6 to induce left lateral flexion of the T6–7 motion segment.

P: Remove superficial tissue slack and establish contacts on the transverse processes. At tension, deliver a thrust through the arms, trunk, and body.

Flexion: To induce flexion, stand in a fencer stance, facing cephalad, and establish contacts on the superior vertebra with your hands on the edge and your fingers running parallel to the spine. At tension, deliver a thrust anteriorly and superiorly through both contacts (see Figure 5-175, A). Placing a roll under the level of adjustive contact may increase flexion preadjustive tension.

Rotation: When treating rotational dysfunction, stand in a modified fencer or square stance. Establish bilateral hypothenar contacts;

hands are arched and arms cross to contact both sides of the spine. The caudal hand contacts the superior vertebra on the side of posterior body rotation (side opposite the rotational restriction). The cephalic hand contacts the contralateral side (Figure 5-175, B). The hand reaching across the spine may develop a broad stabilizing contact or a contact over the contralateral inferior vertebra. A thenar contact may be substituted for the hypothenar contact on the crossed-hand contact.

Develop preadjustive tension by leaning anteriorly into the contacts and tractioning the hands apart. At tension, deliver a thrust anteriorly with the caudal hand while the cephalic hand stabilizes the contralateral structures or counterthrusts anteriorly on the contralateral inferior vertebra (see Figure 5-175, B).

Lateral flexion: When inducing lateral flexion, establish the segmental contacts bilaterally on the transverse process of the same vertebra. Deliver the thrust through both hands. One hand thrusts anteriorly and superiorly while the other thrusts anterior and inferiorly (see Figure 5-175, C). It is unlikely that segmental lateral flexion can be induced with the patient in a neutral prone position.³² Prestressing the patient into lateral flexion may increase the potential for producing lateral flexion.

Unilateral Hypothenar/Spinous Push (Figure 5-176)

IND: Restricted flexion, extension, lateral flexion, or rotation, T4–T12. Extension, flexion, rotation, or lateral flexion malpositions, T4–T12.

PP: The patient lies prone. Prestressing spinal joints in the direction of desired adjustive movement may assist the doctor in inducing the desired motion. To provide added flexion, a small roll may be placed under the patient's chest. To provide added extension, the thoracic section of an articulated adjusting table may be lowered anteriorly.

DP: Stand in a fencer, modified fencer, or square stance, depending on the dysfunction being treated.

CP: Midhypothenar.

SCP: Spinous process.

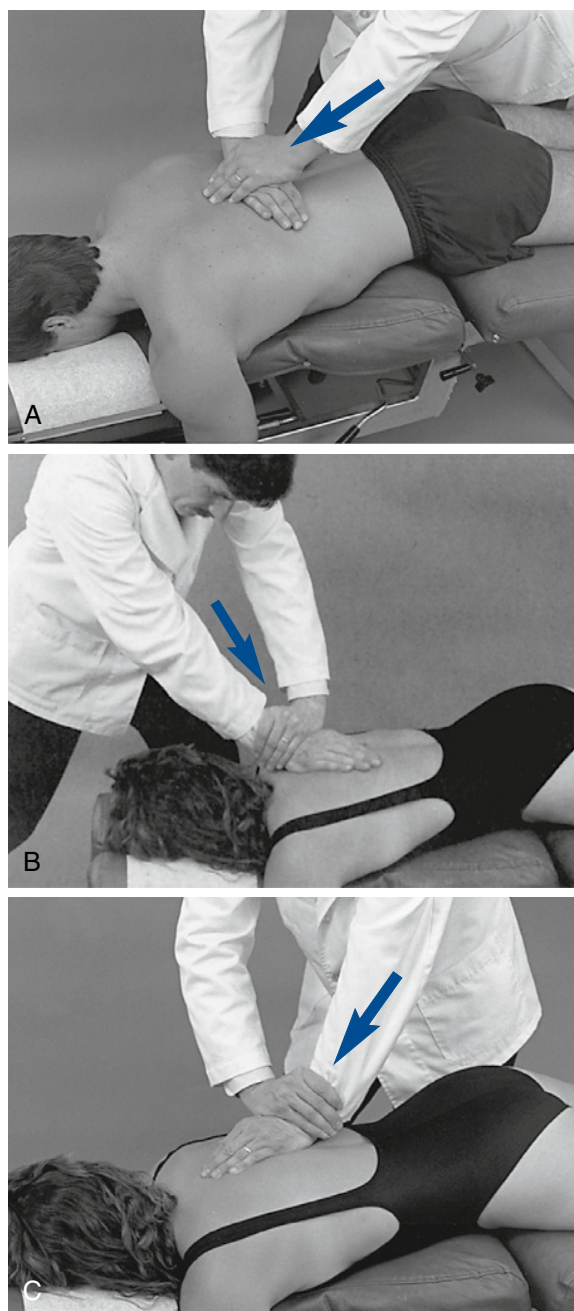
IH: Your IH supports your contact hand on the dorsal surface, with the fingers wrapped around the wrist.

VEC: P-A for extension restrictions, P-A and I-S for flexion restrictions. L-M, S-I, and P-A for rotation or lateral flexion restrictions.

P: Remove superficial tissue slack and establish a fleshy hypothenar contact against the spinous process. Develop preadjustive tension by transferring additional body weight into the contact. At tension, deliver a thrust through the arms, trunk, and body.

Flexion: Stand in a fencer stance, facing cephalad on either side of the patient. Establish the adjustive contact by sliding the midhypothenar contact superiorly onto the inferior tip of the superior spinous process (see Figure 5-176, A), with your center of gravity oriented inferior to the level of adjustive contact. At tension, thrust anteriorly and superiorly.

Extension: Stand in a fencer stance on either side of the patient. Establish a midhypothenar contact over the spinous process. Orient your center of gravity over the dysfunctional joint. At tension, thrust anteriorly.



5-176B

Figure 5-176 A, Reinforced midline hypotenar

contact applied to the inferior aspect of the spinous process of T7 to induce flexion at T7–T8. B, Hypotenar spinous contact applied to the right lateral surface of the T3 spinous process to induce right rotation and right lateral flexion at the T3–4 motion segment. C, Hypotenar spinous contact applied to the right lateral surface of the T8 spinous process to induce right rotation and left lateral flexion of the T8–9 motion segment.

Lateral flexion or rotation: Stand in a fencer stance or square stance on the side of adjustive contact. Establish the adjustive contact by sliding medially onto the lateral surface of the superior spinous process on the side of rotation or lateral flexion restriction (side of spinous rotation). The contact must be fleshy or it may be painful to the patient. While developing the contact, apply a slight clockwise or counterclockwise torquing movement to traction the tissue. This leaves your

fingers oriented at an angle of approximately 45 degrees to the long axis of the spine (see Figure 5-176, B).

To induce lateral flexion coupled with same-side rotation, the cephalic hand is used as the contact. Stand in a square stance or modified fencer stance and face caudally (see Figure 5-176, B). This adjustment is commonly applied in the treatment of coupled restrictions in rotation and lateral flexion to the same side (PRS or PLS listings)

To induce lateral flexion coupled with opposite-side rotation (PRI or PLI listings), the caudal hand establishes the contact and you stand in a square stance or modified fencer stance, facing cephalically (see Figure 5-176, C). At tension, deliver an adjustive thrust anteromedially and superiorly. Spinous contact methods are not commonly used for treating opposite-side restrictions in rotation and lateral flexion. It is unlikely that prone spinous contacts can induce segmental rotation or lateral flexion with the patient in a neutral prone position.³² Prestressing the patient into lateral flexion may increase the potential for the desired motions.

Unilateral Hypotenar/Transverse Push (Figure 5-177)

IND: Restricted rotation or lateral flexion, T4–T12. Rotation or lateral flexion malpositions, T4–T12.

PP: The patient lies prone.

DP: Stand in a fencer stance or square stance on the side of adjustive contact.

CP: Hypotenar (pisiform) of caudal hand, with hand arched and fingers running parallel to the spine.

SCP: Transverse process.

IH: Your IH supports your contact hand on the dorsal surface, with the fingers wrapped around the wrist.

VEC: P-A, coupled with an I-S or S-I VEC, depending on the dysfunction treated and the vertebra contacted.

P: Remove superficial tissue slack and establish transverse process contacts. Develop preadjustive tension by transferring additional body weight into the contacts. At tension, deliver a thrust through the arms, trunk, and body.

Rotation: When treating rotational dysfunction, you may establish contacts on the superior or inferior vertebra of the dysfunctional motion segment. When contacting the superior vertebra, establish the contact on the side opposite the rotational restriction (side of posterior body rotation) (see Figure 5-177, A). When contacting the inferior vertebra, establish the contact on the side of rotational restriction (side opposite posterior body rotation) (see Figure 5-177, C). The inferior vertebral contact (resisted method) is applied to induce more tension and gapping in the joint superior to the contact. The method is not commonly applied.

Lateral flexion: When treating lateral flexion dysfunction, establish the contact over the superior vertebra. When contacting the transverse process on the side opposite the lateral flexion restriction, stand in a fencer stance, facing cephalically. Establish the contact with the caudal hand and deliver the adjustive thrust anteriorly and superiorly (see Figure 5-177, A). When contacting the side of lateral flexion restriction, stand in a square or fencer stance and establish the contact with the cephalic hand. Deliver the thrust anteriorly and inferiorly (see Figure 5-177, B). It is unlikely that segmental lateral

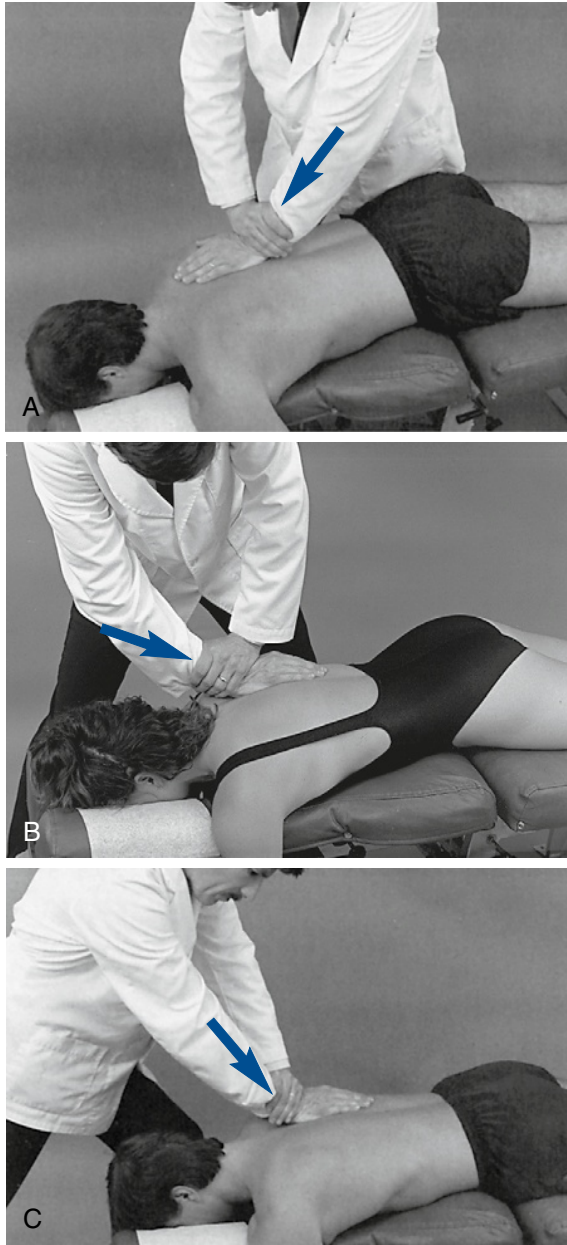


Figure 5-177 **A**, Assisted hypothenar contact applied to the right T5 transverse process to induce left rotation or left lateral flexion at the T5–6 motion segment. **B**, Assisted hypothenar contact applied to the right transverse process of T5 to induce right lateral flexion at T5–6 motion segment. **C**, Resisted hypothenar contact applied to the right transverse process of T5 to induce right rotation and gapping in the right T4–5 articulation.

flexion can be induced with the patient in a neutral prone position.³² Prestressing the patient into lateral flexion may increase the potential for producing lateral flexion.

Hypothenar Spinous Crossed Thenar/Transverse Push (Figure 5-178)

IND: Restricted rotation and or coupled lateral flexion, T4–T12. Rotation or coupled lateral flexion malpositions, T4–T12.

PP: The patient lies prone.

DP: Stand in a modified fencer stance or square stance on side of spinous contact.

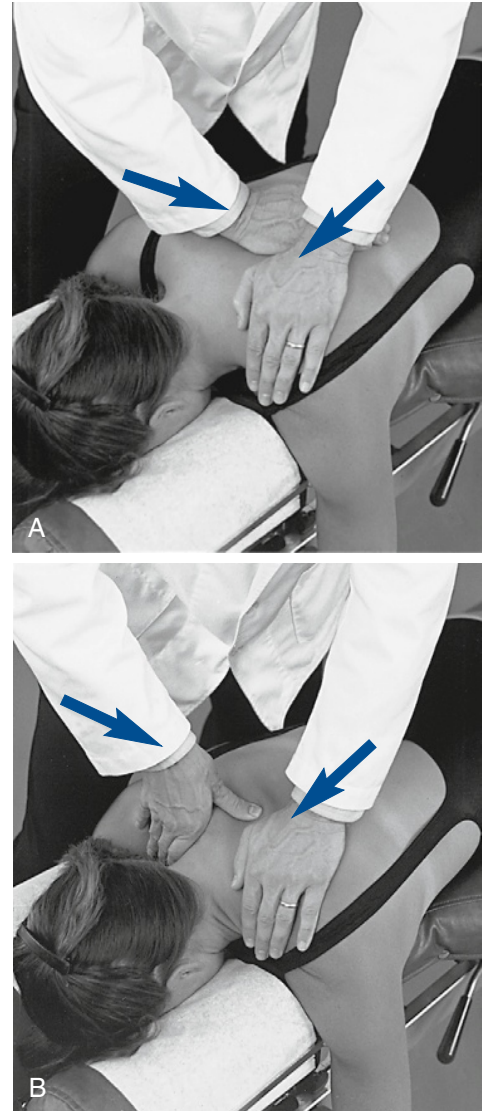


Figure 5-178 Hypothenar (**A**) or thumb (**B**) contact applied to the right lateral surface of the T4 spinous process and a thenar contact applied to the left T4 transverse process to induce right rotation or right lateral flexion at the T4–5 motion segment.

CP: Hypothenar (pisiform) (see Figure 5-178, *A*) or thumb (see Figure 5-178, *B*) of the cephalic hand and thenar of the caudal hand.

SCP: Lateral surface of the spinous process and transverse process of the corresponding vertebra.

VEC: P-A, L-M, and S-I, with hand contacting the spinous process. P-A and I-S, with transverse process contact.

P: Remove superficial tissue slack by sliding the thumb or hypothenar against the lateral surface of the spinous process while sliding the thenar superiorly onto the ipsilateral transverse process. Develop preadjustive tension by transferring additional body weight into the contacts. At tension, deliver a thrust through the arms and body. It is unlikely that segmental lateral flexion can be induced with the patient in a neutral prone position.³² Prestressing the patient into lateral flexion may increase the potential for producing lateral flexion.

Knee-Chest

Hypothenar/Spinous Push (Figure 5-179)

IND: Restricted extension, lateral flexion, or rotation, T4–T12. Flexion, rotation, or lateral flexion malpositions, T4–T12.

PP: Position the patient in the knee-chest position, with the chest support placed so that the patient's thoracic spine is level with or slightly lower than the lumbar spine. The patient's femurs should be angled between 95 and 110 degrees.

DP: Stand at the side of the table in a square stance, typically on the side of the contact. You may also stand in a fencer stance, facing caudally.

CP: Hypothenar.

SCP: Lateral surface of the spinous process.

IH: Your IH supports your contact hand on the dorsal surface, with the fingers wrapped around the wrist.

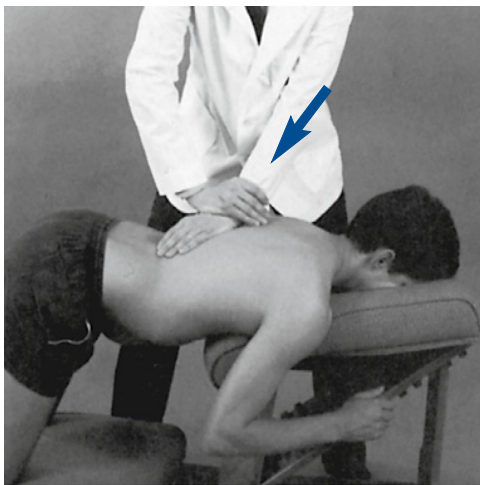


Figure 5-179 Hypothenar contact applied to the left lateral surface of the T8 spinous process to induce extension, left rotation, or left lateral flexion at the T8-9 motion segment, using the knee-chest position.

VEC: P-A for extension restriction or flexion malposition. L-M, S-I, and P-A for rotation or lateral flexion restrictions or malpositions.

P: The IH first raises the patient's abdomen to make the spinous processes more prominent and available for establishing the contacts. Instruct the patient to allow the torso to drop, and at tension, deliver an impulse thrust. Knee-chest tables provide their greatest advantage in assisting the doctor in the application of inducing spinal extension. The potential to induce lateral flexion may be improved by prestressing the patient in the direction of desired lateral flexion. The patient is vulnerable to hyperextension in this position, so the thrust must be shallow and nonrecoiling.

Hypothenar/Transverse and Bilateral Hypothenar/Transverse Push (Figure 5-180)

IND: Restricted extension, lateral flexion, or rotation, T4–T12. Flexion, rotation, or lateral flexion malpositions, T4–T12.

PP: Position the patient in the knee-chest position, with the chest support placed so that the patient's thoracic spine is level with or slightly lower than the lumbar spine. The patient's femurs should be angled between 95 and 110 degrees.

DP: Stand at the side of the table in a square stance, when using unilateral contact. You may also stand in a fencer stance, facing caudally.

CP: Hypothenar (pisiform).

SCP: Transverse process.

IH: With a unilateral contact, the IH supports the contact hand on the dorsal surface, with the fingers wrapped around the wrist. With bilateral contacts, the IH is placed on the opposite side to stabilize or impart an assisting impulse.

VEC: P-A.

P: The IH first raises the patient's abdomen to make the transverse process more prominent and available for establishing the contacts. Instruct the patient to allow the torso to drop, and at tension, deliver an impulse thrust. The patient is vulnerable to hyperextension in this position, so the thrust must be shallow and nonrecoiling. If the IH delivers an assisting thrust, it is directed posteriorly to anteriorly. Knee-chest tables provide their greatest advantage in assisting the doctor

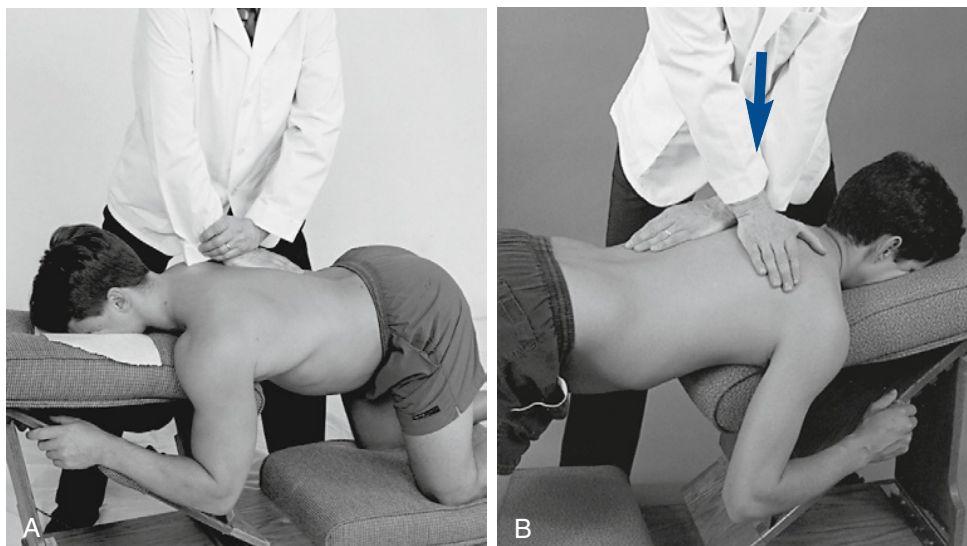


Figure 5-180 **A**, Unilateral hypothenar contact applied to the right T5 transverse process to induce left rotation and or right lateral flexion at the T5–6 motion segment. **B**, Bilateral hypothenar contacts applied to the T5 transverse processes to induce extension at the T5–6 motion segment.

in the application of inducing spinal extension. The potential to induce lateral flexion may be improved by prestressing the patient in the direction of desired lateral flexion.

Supine

Opposite-Side Thenar/Transverse Drop (Figure 5-181)

IND: Restricted flexion, extension, rotation, or lateral flexion, T3–T12. Flexion, extension, rotation, or lateral flexion malpositions, T3–T12.

PP: The patient sits or lies supine, with arms crossed and hands grasping shoulders.

DP: Stand in a modified fencer stance and reach around the patient to establish the posterior contact.

CP: The cupped hand, clenched fist, or index or thenar of the contact hand (see Figure 5-147).

SCP: Bilateral transverse process, unilateral transverse process, depending on the dysfunction being treated.

IH: Your IH contacts the patient's crossed arms or cradles the patient's neck and upper back (see Figure 5-181, *A* and *B*).

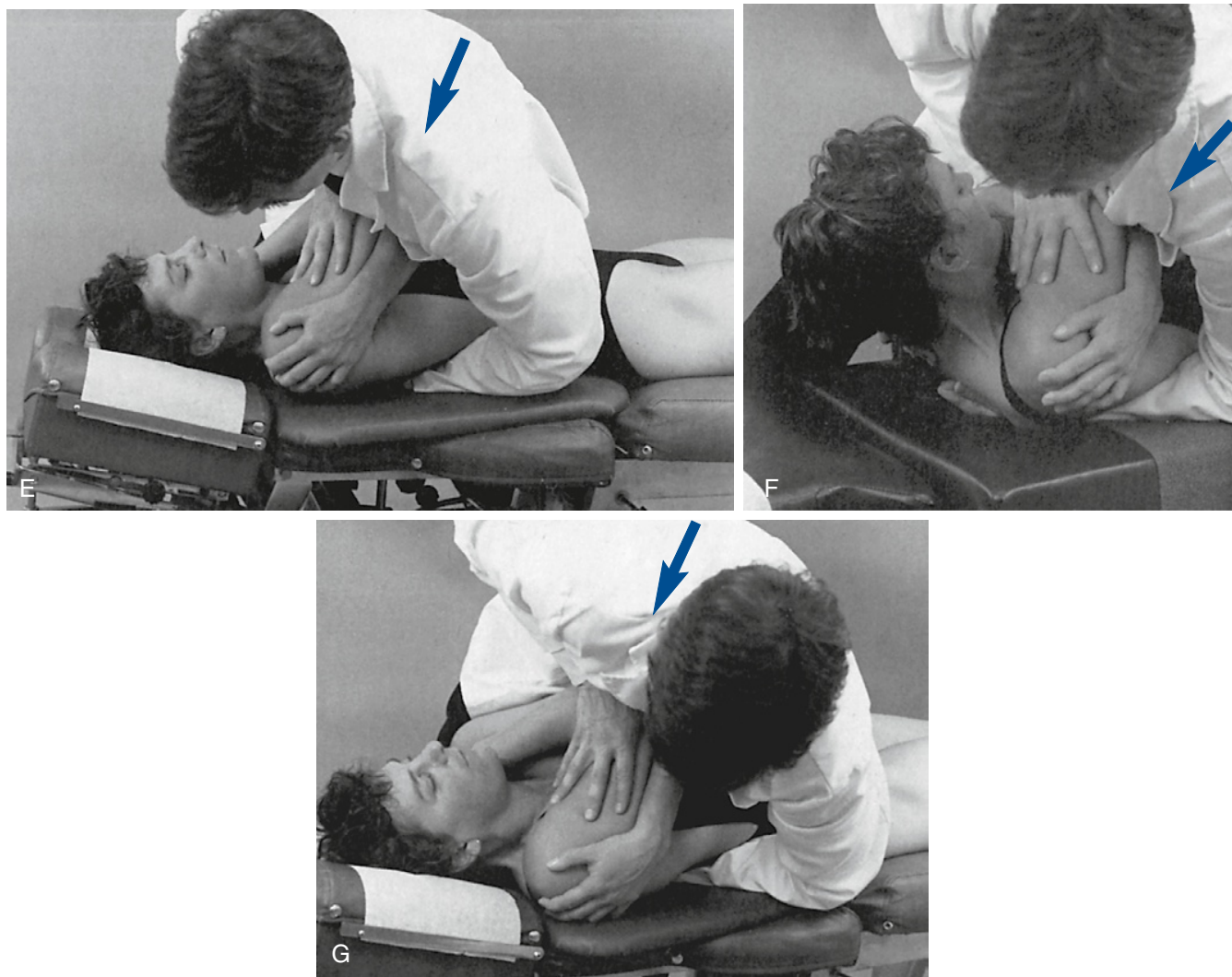
VEC: A-P and I-S through the doctor's torso. A-P for extension or rotational dysfunction.

P: When starting in the supine position, first roll the patient toward you to place the posterior spinal contact (see Figure 5-181, *A*). Slide superiorly to establish a superior vertebral contact and inferiorly to establish an inferior vertebral contact. After the spinal contact is established, return the patient to the



Figure 5-181 Supine thoracic adjustment, using an opposite-side contact, with the patient in a crossed-arm position. **A**, Starting in the supine position. **B**, Starting in the seated position. **C**, Assisted method, using a cradling support and a clenched fist applied to the transverse process of T7 to induce flexion at the T7–8 motion segment (*small arrow indicates direction of tissue pull*). **D**, Resisted method, using a crossed-shoulder support and a clenched fist applied to the transverse process of T8 to induce flexion at the T7–8 motion segment (*small arrow indicates direction of tissue pull*).

(Continued)



5-181E, G

Figure 5-181–Cont’d **E**, Clench-fist contact applied to the T8 transverse process to induce extension at the T7–8 motion segment. **F**, Assisted thenar contact established on the right T3 transverse process to induce left rotation of the right T3–4 articulation. **G**, Resisted thenar contact applied to the right T4 transverse process to induce right rotation and gapping of the right T3–4 articulation.

supine position and initiate the IH contacts (see Figure 5-181, *C* to *E*). During this process, it is important that the patient is rolled onto the contact hand with minimal pressure. Undue pressure exerted against the anterior contacts can lead to painful compression against your posterior contact.

Develop preadjustive tension by adding progressive compression and traction through your trunk and anterior contacts. At tension, deliver a short-amplitude, moderate-velocity body-drop thrust, generated primarily through your trunk and lower extremities. When applying supine adjustive techniques, it is important to avoid straight compression to the trunk and rib cage. This is accomplished by applying slight headward traction during the development of tension.

Supine thoracic adjustments are also commonly started with the patient in a sitting position. This is particularly helpful when adjusting the lower thoracic spine, when adjusting large patients, or when rolling the patient onto the contact is too painful (Figure 5-181, *B*).

Flexion: When treating flexion restrictions (extension malpositions), the patient is maintained in a position of segmental flexion (see Figure 5-181, *C* and *D*). Place the adjustive contacts bilaterally on the transverse processes or in the midline, against the spinous process. Establish the transverse contacts with your cupped hand or clenched fist.

When using an assisted method, establish the contact on the transverse process of the superior vertebra of the dysfunctional motion segment (see Figure 5-181, *C*). At tension, deliver the thrust posteriorly and superiorly through the trunk, legs, and posterior contact.

When using a resisted method, establish the contact on the transverse process of the inferior vertebra (see Figure 5-181, *D*). Apply downward counterpressure through the contact to oppose the adjustive thrust, which is directed posteriorly and superiorly through the trunk and legs.

Extension: When treating extension restrictions (flexion malpositions), establish the contact bilaterally on the transverse

processes of the inferior vertebra of the dysfunctional motion segment. Develop preadjustive tension by inducing segmental extension and deliver the adjustive thrust posteriorly (Figure 5-181, *E*).

Rotation: To produce rotation you may establish unilateral thenar contacts on either the superior or inferior vertebra of the involved motion segment. When using a superior vertebral contact (assisted method), establish the contact on the transverse process on the side opposite the rotation restriction (side of posterior body rotation). Maintain the patient in a flexed position and direct the thrust posteriorly (see Figure 5-181, *F*).

When using an inferior vertebral contact (resisted method), establish the contact on the transverse side of the rotational restriction (side opposite posterior body rotation). For example, when treating a right rotation restriction at T3–4 (left body rotation), the contact would be established on the right T4 transverse process. During the development of preadjustive tension, the patient is rolled farther toward the side of the posterior contact (see Figure 5-181, *G*). The adjustive thrust is directed posteriorly. The inferior vertebral contact (right T4 contact) is applied to induce gapping in the facet joint ipsilateral and superior to the contact.

Lateral flexion: Lateral flexion dysfunction is typically treated by establishing unilateral contacts on the side opposite the lateral flexion restriction. When using assisted patient positions, contact the superior vertebra, induce flexion and lateral flexion away from the side of contact and thrust posteriorly through the trunk and legs.

When using resisted methods, contact the inferior vertebra, laterally flex the patient away, and apply downward counterpressure with the contact to oppose the adjustive thrust. This method may be applied to treat combined restrictions in rotation and opposite-side lateral flexion.

Same-Side Thenar/Transverse Drop, Crossed Arm (Figure 5-182)

IND: Restricted flexion, extension, rotation, or lateral flexion, T3–T12. Flexion, extension, rotation, or lateral flexion malpositions, T3–T12.



Figure 5-182 Supine thoracic adjustment, using a crossed-arm patient position and a same-side contact applied to a midthoracic segment to induce extension.

PP: Ask the patient to sit or lie supine, with arms crossed and hands grasping shoulders.

DP: Stand in a fencer stance on the side of adjustive contact.

CP: The cupped hand, clenched fist, or thenar of the contact hand.

SCP: Bilateral transverse process, or unilateral transverse process, depending on the restriction being treated.

IH: Use your IH to contact the patient's crossed arms or cradle the patient's neck and upper back.

VEC: A-P through the doctor's torso.

P: Stand on the side of the established contact and instruct the patient to cross his or her arms. Roll the patient away from you and establish the posterior contact. Then roll the patient back into position and contact the patient's crossed arms or cradle the patient's neck and shoulders (see Figure 5-182). Progressive compression to remove soft tissue slack is followed by a moderate-velocity, short-amplitude body-drop thrust.

The specific considerations for flexion, extension, and rotational restrictions are the same as previously mentioned in the adjustments described in Figure 5-181, *C* to *G*. Other than personal preference, this method is commonly applied in the treatment of larger patients or if less contact is desired between the doctor and patient.

Thenar/Transverse Drop, Pump Handle (Opposite or Same-Side) (Figure 5-183)

IND: Restricted flexion, rotation, or lateral flexion, T3–T12. Flexion, extension, rotation, or lateral flexion malpositions, T3–T12.

PP: The patient may begin in either the sitting or supine position, with elbows flexed and fingers interlocked or overlapping behind neck.

DP: Stand in a fencer stance on either side of the patient.

CP: The cupped hand, clenched fist, or thenar of your contact hand.

SCP: Bilateral transverse process, or unilateral transverse process, depending on the dysfunction treated.

IH: Either cradle the patient's neck and upper back with the IH and arm or support the patient by leaning across the patient's forearm with the forearm and upper abdomen (see Figure 5-183, *A* and *B* [opposite-side method]; *C* and *D* [same-side method]).

VEC: A-P and I-S to produce flexion or lateral flexion. A-P for extension or rotation.

P: Stand on either side of the patient and establish the posterior contacts. Your IH and forearm contact the patient's forearms or reach around to cradle the patient's neck and upper back. The development of preadjustive tension and the delivery of the adjustive thrust are identical to those previously described.

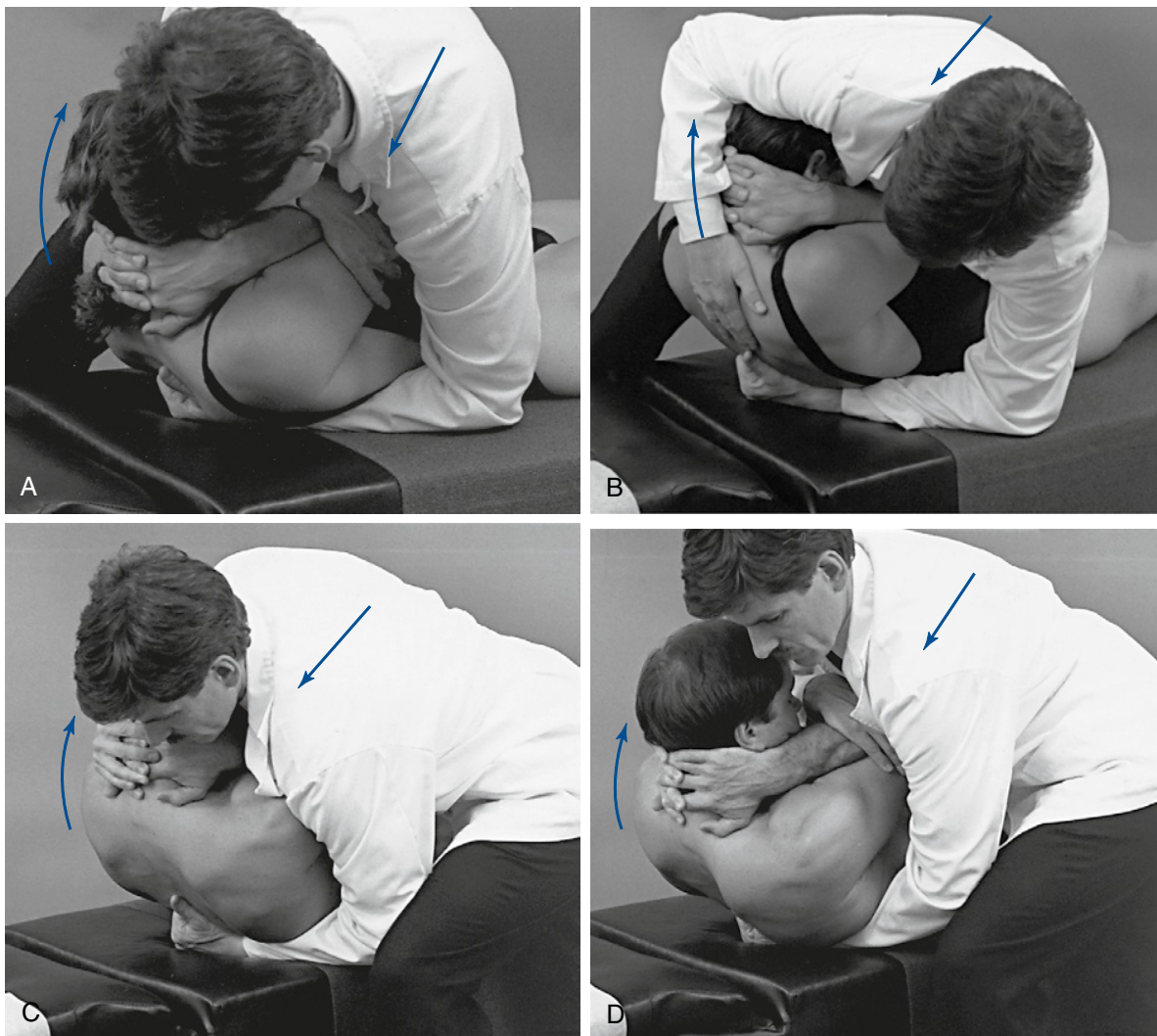
The specific considerations for flexion, and rotational dysfunction are the same as previously mentioned in supine adjustments. The pump handle position is especially helpful in inducing flexed patient postures and is not frequently used when inducing extension.

Sitting

Hypothenar/Transverse Pull (Figure 5-184)

IND: Restricted rotation or coupled lateral flexion, T5–T12. Rotation or lateral flexion malpositions, T5–T12.

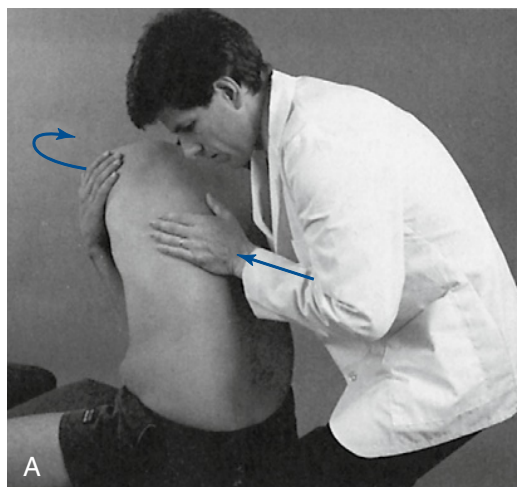
PP: The patient sits with legs straddling the adjusting bench. The patient's arms are crossed with the hands grasping the shoulders.



5-183B

Figure 5-183 Supine thoracic adjustments, using patient pump-handle position to assist in the development of flexion.

A, Opposite-side method, with the doctor assisting the production of flexion with a forearm contact across the patient's flexed forearms. **B**, Opposite-side method, with doctor cradling the patient. **C**, Same-side method, with cradling support and a pump-handle position. **D**, Same-side method, with the doctor assisting the production of flexion with a forearm contact across the patient's flexed forearms.



5-184A

Figure 5-184 **A**, Sitting assisted hypothenar transverse contact applied to the left T6 transverse process to induce right rotation and right lateral flexion of the T6-7 motion segment. **B**, Sitting assisted hypothenar spinous contact applied to the left lateral surface of the T6 spinous to induce left rotation and right lateral flexion of the T6-7 motion segment.

B, Sitting assisted hypothenar spinous contact applied to the left lateral surface of the T6 spinous to induce left rotation and right lateral flexion of the T6-7 motion segment.

DP: Sit or stand behind the patient.

CP: Hypothenar or thenar of hand corresponding to the side of contact.

SCP: Transverse or spinous process of superior vertebra.

IH: Your IH and arm reach around the patient to contact the opposing forearm. The IH is active in producing adjustive force

VEC: Pulling rotatory force generated by doctor's anterior arm contact and torso.

P: Ask the patient to sit and cross his or her arms. Position yourself behind the patient in either a standing or seated position (see Figure 5-184). Preadjustive tension is typically developed by flexing, laterally flexing, and rotating the patient in the direction of joint restriction (assisted method). Once tension is established, deliver an impulse thrust by inducing a pulling and twisting thrust generated through your indifferent arm trunk and posterior contacts. The direction of induced lateral flexion and the point of adjustive contact depend on the dysfunction being treated.

Rotation: When treating rotational dysfunction, you may establish contacts on the spinous process or transverse process. When using a transverse process contact, establish the contact on the superior vertebra on the side opposite the rotation restriction (side of posterior body rotation) (see Figure 5-184, *A*). To use a spinous process contact, slide medially and establish a fleshy mid-hypothenar contact on the lateral surface of the spinous process on the side of rotational restriction (side of spinous rotation) (see Figure 5-184, *B*). Laterally flexing the patient away from the side of contact is commonly incorporated to increase distraction in the joint on the side of contact.

Lateral flexion: Pure lateral flexion cannot be effectively produced with sitting thoracic adjusting methods; some degree of rotation is a product of sitting thoracic methods. Patients who cannot tolerate moderate rotation of the spine are not good candidates for sitting thoracic methods.

To treat lateral flexion dysfunction, contact the transverse process of the superior vertebra on the side opposite the lateral flexion restriction (Figure 5-184, *A*). Develop preadjustive tension by flexing and laterally flexing the patient away from the side of contact.

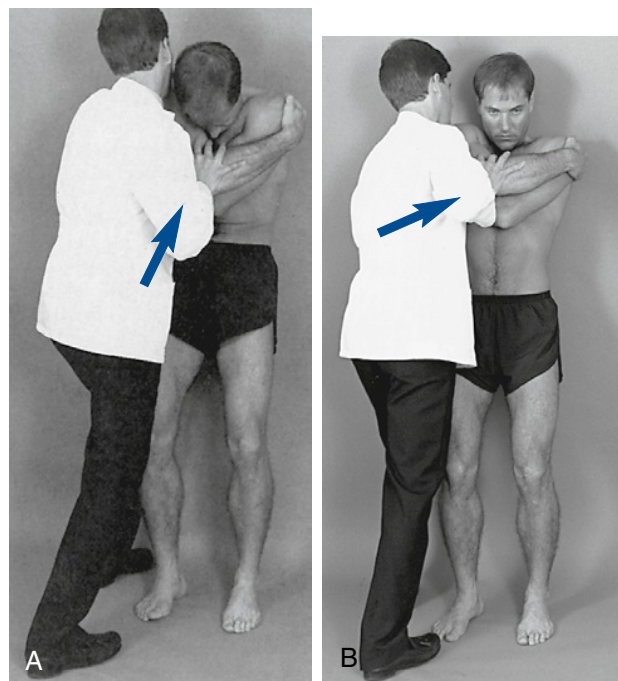
Combined rotation and lateral flexion: Sitting thoracic adjustments can also be applied to treat combined restrictions in rotation and lateral flexion. Transverse process contacts are more commonly applied with restrictions in rotation and same-side lateral flexion, and spinous process contacts may be more effective in treating restrictions in rotation and opposite-side lateral flexion. Figure 5-184, *A*, demonstrates treatment of a rotation and same-side lateral flexion restriction, and Figure 5-184, *B*, demonstrates rotation and opposite-side lateral flexion.

Standing

Thenar/Transverse Push (Figure 5-185)

IND: Restricted flexion, extension, rotation, or lateral flexion, T3–T12. Flexion, extension, rotation, or lateral flexion malpositions, T3–T12.

PP: The patient stands, leaning against a wall, with feet shoulder-width apart. The arms are crossed, with the hands grasping the shoulders.



5-185B

Figure 5-185 Standing thoracic adjustment for a midthoracic segment. *A*, Against a wall to induce flexion. *B*, Against a wall to induce extension.

DP: Stand on either side of the patient in a fencer stance, with your medial leg posterior and body angled about 45 degrees to the patient.

CP: Your cupped hand or fist or the open palm of your outside hand reaches behind the patient to contact the patient posteriorly. Your hand must be cushioned from the wall by using a padded wall board or placing a pad between your hand and the wall.

SCP: Transverse or spinous process of involved vertebra.

IH: Your IH contacts the patient's crossed arms.

VEC: A-P and I-S

P: Stand to the side of adjustive contact, rotate the patient away, and establish the posterior contact. Develop preadjustive tension by leaning into the patient while applying vertical traction. As tension is developed, it is important to produce some long-axis traction by pushing cephalically through your legs and arms. At tension, deliver an impulse thrust through your trunk and lower extremities.

Extension: With extension restrictions (flexion malpositions), contact the inferior vertebra, extend the motion segment, and deliver the thrust posteriorly (see Figure 5-185, *B*).

Flexion: When treating flexion restrictions (extension malpositions), the superior vertebra of the dysfunctional motion segment is typically contacted. Maintain the patient in a position of flexion and deliver the thrust posteriorly and superiorly (see Figure 5-185, *A*). This is the most difficult dysfunction to treat with standing thoracic methods because flexed postures roll the patient's spine away from the doctor's posterior contact. This can be minimized, to some degree, by having the patient push his or her buttocks away from the wall.

Rotation: When treating rotation dysfunction, adjustive contacts may be established on the superior or inferior vertebra of the dysfunctional motion segment. When using a superior verte-

bral contact (assisted method), establish the contact on the side opposite the rotation restriction (side of posterior body rotation). Position the patient in slight flexion and deliver a thrust posteriorly and superiorly.

When using an inferior vertebra contact (resisted method), establish the contact on the side of rotational restriction (side opposite the posterior body rotation) and deliver a thrust posteriorly. This adjustment may also be used for rib dysfunction by moving the contact lateral to the angle of the involved rib.

Lateral flexion: Lateral flexion dysfunction is typically treated by establishing unilateral contacts on the side opposite the lateral flexion restriction. When using assisted patient positions, contact the superior vertebra and induce flexion and lateral flexion away from the side of contact. At tension, thrust posteriorly and superiorly through the trunk and legs toward the posterior contact.

When using resisted methods, contact the inferior vertebra and laterally flex the patient away from the side of contact. Apply inferior counterpressure with the posterior contact to oppose the anterior and superiorly directed adjustive thrust. This method may be applied to treat combined rotation and opposite-side lateral flexion dysfunction. It is unlikely that this method can induce lateral flexion without the production of coupled rotation.



Figure 5-186 Standing thoracic adjustment for a midthoracic segment, with the doctor standing behind to induce flexion and long-axis distraction.

Thoracic Long-Axis Distraction (Figure 5-186)

IND: Restricted flexion and long-axis distraction, T3–T12. Extension malpositions, T3–T12.

PP: The patient stands with feet at least 10 inches apart (more if the patient is taller than the doctor), with hands interlaced behind the neck and elbows together or arms crossed over the chest.

DP: Stand behind the patient in a fencer stance, placing your forward leg between the patient's legs. Your anterior chest may be padded with a sternal roll or small pillow.

CP: A true segmental contact is not established on the back, but the sternal angle of the doctor is placed over the region to be distracted.

SCP: Over the spinous processes of the dysfunctional region.

VEC: A-P and I-S.

P: Grasp the patient's forearms, stressing the patient's thoracic spine into slight flexion. At tension, pull posteriorly and superiorly through the patient's arms.

This procedure is applied to develop regional distraction, although a sternal block can be used to make the contact more specific. It can be applied as a thrust or nonthrust mobilization procedure. The thrust is gentle and shallow and generated by the doctor pulling posteriorly and superiorly through the contacts on the patient's elbows.

RIB ADJUSTMENTS (BOX 5-8)

BOX 5-8 Rib Adjustments

- Supine
 - Thenar/costal drop (Figure 5-187)
 - Index/costal push (Figure 5-188)
- Prone
 - Hypothenar/costal push (Figure 5-189)
 - Modified hypothenar (thenar)/costal push (Figure 5-190)
 - Index/costal push (Figure 5-191)
 - Hypothenar costal/push (Figure 5-192)
 - Iliac hypothenar/costal push (Figure 5-193)
 - Covered-thumb/costal push (Figure 5-194)
- Side posture
 - Web/costal push (Figure 5-195)
- Sitting
 - Index/costal push (Figure 5-196)
 - Hypothenar (thenar)/costal push (Figure 5-197)
- Costosternal Adjustments
 - Supine covered thumb/costosternal push (Figure 5-198)
 - Sitting hypothenar/costosternal pull (Figure 5-199)

Supine

Thenar/Costal Drop (Figure 5-187)

IND: Rib dysfunction, R2–R12.

PP: The patient lies supine, with arms crossed and the hands grasping the shoulders. Patient arm placement is at the doctor's



Figure 5-187 Supine rib adjustment, using a thenar contact over the right third rib starting in the supine position (**A**) and an anterior-to-posterior thrust through the doctor's torso to induce gapping in the right costotransverse articulation (**B**). **C**, Thenar contact applied over the inferior margin of the right eighth rib, starting in the seated position. **D**, Anterior-to-posterior thrust through the doctor's torso to induce gapping in the right eighth costotransverse articulation.

discretion. When treating lower rib fixations, the patient typically begins in the seated position.

DP: Stand in a modified fencer stance on either side of the patient.

CP: Thenar eminence.

SCP: Just medial to the rib angle.

IH: Your IH contacts the patient's crossed arms or cradles the patient's neck and upper back.

VEC: A-P.

P: Stand in a low fencer stance on either side of the patient. Access the contact by rolling the patient toward you or placing him or her in a sitting position (see Figure 5-187).

At tension, direct a moderate-velocity thrust toward the posterior contact established on the rib. Acceleration of the doctor's body weight toward the posterior contact accelerates the patient's torso toward the doctor's posterior contact, which in turn exerts a P-A force against the rib contact. The reactive P-A force generated against the rib is designed to accelerate the rib forward and induce gapping in the costotransverse joint.

In the treatment of lower rib dysfunction, the patient is typically started in the sitting position (see Figure 5-187, *B*). The patient is maintained in some degree of thoracolumbar flexion, and the contact hand is held in a more vertical (bridged) contact to establish tension in the lower thoracic spine (see Figure 5-187, *D*).

Index/Costal Push (Figure 5-188)**IND:** Dysfunction of the first rib.**PP:** The patient lies supine.**DP:** Stand at the head of the table, facing caudad.**CP:** Index contact of the hand corresponding to the side of contact.**SCP:** Angle of first rib.**IH:** Your IH cups the patient's ear with the palm while the index and middle fingers support the upper cervical spine**VEC:** S-I and L-M.**P:** To establish the contact, the IH raises the patient's head to about a 45-degree angle and then extends the head backward over the index contact on the first rib. The head is then rotated

about 20 degrees away from the contact and slightly laterally flexed over the contact. At tension, an impulse thrust is delivered superiorly to inferiorly and laterally to medially.

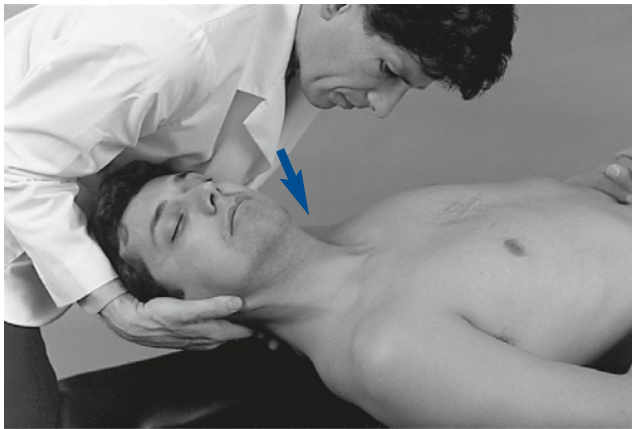
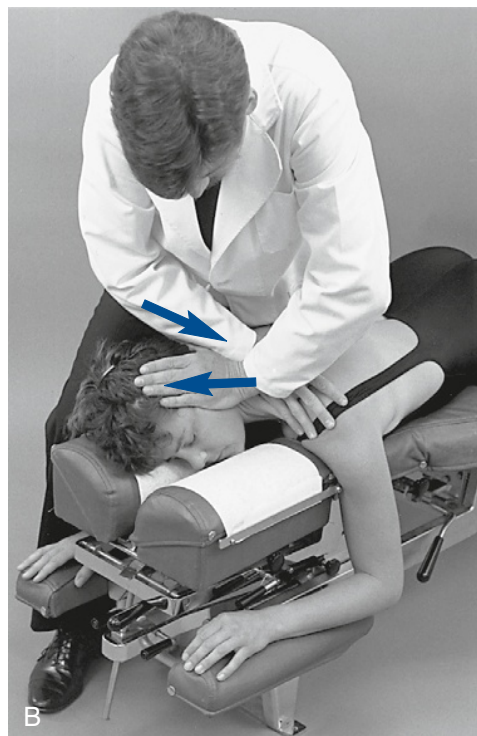
Prone Upper**Hypothenar/Costal Push** (Figure 5-189)**IND:** Rib dysfunction, R1–R4.**PP:** The patient lies prone, with the headpiece lowered below horizontal to produce slight flexion in the thoracocervical spine. (The patient's head rests on the anterolateral cheek.)**DP:** Stand in a fencer stance on either side of the patient, facing cephalad. Your superior leg approximates the level of the patient's head and your upper body weight is centered over the contact.**CP:** Hypothenar of arched hand. When standing on the same side of adjustive contact (combination move), the hypothenar of your caudal hand establishes the contact (see Figure 5-189, A). When standing on the side opposite the adjustive contact (modified combination move), the hypothenar of your cephalic hand establishes the contact (see Figure 5-189, B).**SCP:** Rib angle.**IH:** The IH supports the upper cervical spine as the fingers contact the inferior occiput.**VEC:** P-A.**P:** Place the patient in the prone position and establish the adjustive contacts. Develop preadjustive tension by transferring body weight into the contact while rotating the patient's head toward and laterally flexing it away from the contact. At tension, deliver an impulse thrust through the contact and indifferent arms. The impulse delivered through the contact hand is typically assisted by a body-drop thrust. The impulse imparted through the IH**Figure 5-188** Index contact established over the superior aspect of the angle of the left first rib to distract the left T1 costotransverse articulation.**A****B****Figure 5-189** Hypothenar contact established on the superior margin of the angle of the left first rib applied to distract the left costotransverse articulation. **A**, Doctor stands on the same side as the contact. **B**, Doctor stands on the opposite side of the contact.



Figure 5-190 Hypothenar contact established over the second rib to induce distraction of the left second costotransverse articulation.

is shallow, and care should be taken not to excessively rotate or laterally flex the cervical spine. To maximize distractive tension in the soft tissues or intercostal tissues superior to the contact, apply caudal pressure against the rib angle.

Modified Hypothenar (Thenar)/Costal Push (Figure 5-190)

IND: Rib dysfunction, R1–R2.

PP: The patient lies prone, with the headpiece lowered below horizontal to produce slight flexion in the thoracocervical spine. (The patient's head rests on the anterolateral aspect of the cheek.)

DP: Stand in a fencer stance at the head of the adjustive bench, facing caudad.

CP: Hypothenar of hand corresponding to the side of contact. The arm is straight, with the elbow locked.

SCP: Posterior superior angle of the first or second rib.

IH: Your IH cups the patient's ipsilateral ear while the fingers rest against the lateral face.

VEC: P-A and S-I.

P: After establishing the contacts, the patient's head is laterally flexed away and rotated toward the contact. At tension, an impulse thrust is delivered through the contact hand while a simultaneous counterdistraction force is delivered through the IH. The adjustive force is delivered primarily with a body-drop thrust.

Index/Costal Push (Figure 5-191)

IND: Rib dysfunction, R1.

PP: The patient lies prone. (The patient's head rests on the anterolateral aspect of the cheek.)

DP: Stand in a low fencer stance, facing cephalad, on the side of rib dysfunction, slightly headward of the adjustive contact.

CP: Index finger of hand corresponding to the side of contact. The wrist is straight and locked, with arm approximately 45 degrees to vertical plane.

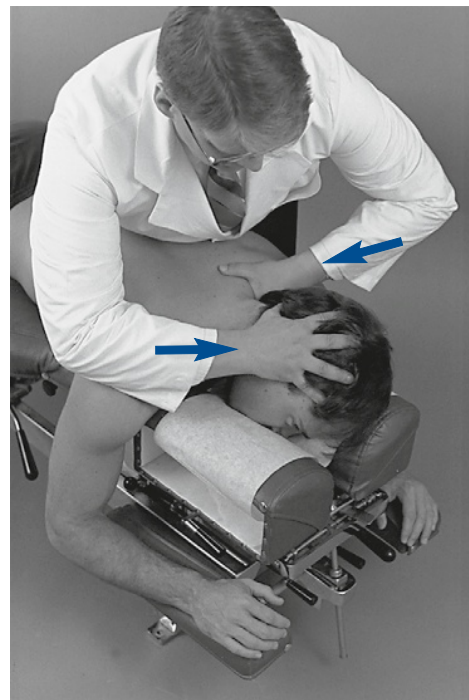


Figure 5-191 Index contact established over the superior angle of the left first rib to induce distraction of the left costotransverse articulation.

SCP: Posterior superior angle of the first rib.

IH: Your IH cups the patient's contralateral inferior occiput and lateral skull.

VEC: S-I, L-M, and P-A.

P: After establishing the contacts, laterally flex the patient's head toward the contact while rotating it away (e.g., with the contact established on the patient's left first rib, the patient's head is left laterally flexed and right rotated). At tension, deliver an impulse thrust through the contact hand while a simultaneous counterdistraction force is delivered through the IH.

Prone

Hypothenar/Costal Push (Figure 5-192)

IND: Rib dysfunction, R3–R10.

PP: The patient lies prone, ideally on a table with a brachial cut-out to induce scapular abduction.

DP: The doctor stands in a fencer stance when using unilateral contacts and in a square stance when using bilateral contacts.

CP: Hypothenar of the cephalic hand in the upper thoracic spine and the caudal hand in the lower thoracic spine.

SCP: Rib angle.

IH: Your IH supports the contact hand or develops a broad stabilizing contact on the contralateral rib cage.

VEC: P-A.

P: Place the patient in the prone position and establish the adjustive contacts. Develop preadjustive tension by transferring body weight into the contact. At tension, deliver an impulse thrust through the contact arm, assisted by a body-drop thrust.

When treating upper rib dysfunction thrust anteriorly and inferiorly (see Figure 5-192, *A*). When treating lower rib dysfunction thrust anteriorly and superiorly (see Figure 5-192, *B*).

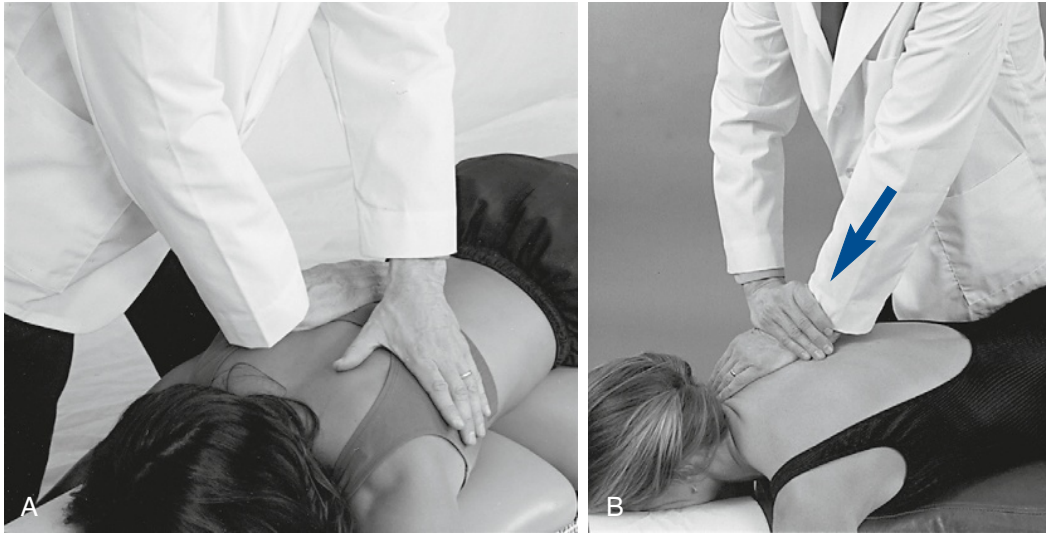


Figure 5-192 **A**, Hypothenar contact applied to the superior margin of the right sixth rib angle to induce gapping of the right sixth costotransverse articulation. Crossed contact applies gentle countersupport without imparting a thrust. **B**, Hypothenar contact applied to the inferior margin of the right fourth rib angle to induce gapping of the right fourth costotransverse articulation.

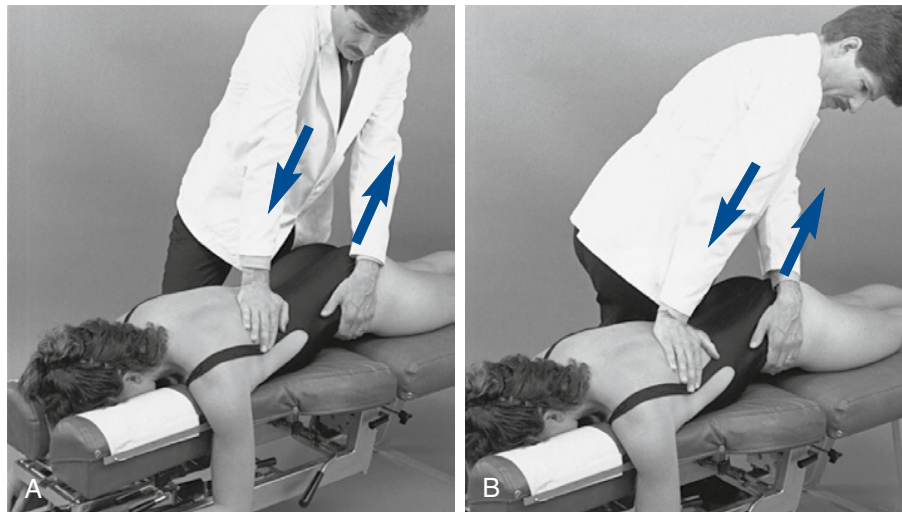


Figure 5-193 Hypothenar contact applied to the inferior margin of the left ninth rib angle to induce distraction of the left ninth costotransverse articulation. **A**, Doctor in a square stance. **B**, Doctor in modified square stance, facing caudally.

Iliac Hypothenar/Costal Push (Figure 5-193)

IND: Rib dysfunction, R7–R12.

PP: The patient lies prone. To provide added flexion, a small roll may be placed under the patient's upper abdomen.

DP: Stand in a square stance (see Figure 5-193, *A*) or a modified fencer stance (see Figure 5-193, *B*) on the side opposite the adjustive contact.

CP: Hypothenar of cephalic hand.

SCP: Rib angle.

IH: The fingers of your inferior hand reach around to grasp the anterior ilium (anterosuperior iliac spine [ASIS]) on the side of adjustive contact.

VEC: P-A, I-S, and L-M.

P: Establish contacts on the rib angle and anterior ilium and induce preadjustive tension by lifting and tractioning inferi-

orly against the ilium as the weight of your trunk is transferred anteriorly and superiorly against the contact. The counterdistraction tension induced through the ilium is not marked, and the patient's pelvis should not be rotated off the table more than 1 to 2 inches. Deliver the thrust with an impulse and body-drop thrust through the contact hand. A thumb-thenar contact can be placed in the intercostal space laterally to influence bucket-handle movements.

Covered-Thumb/Costal Push (Figure 5-194)

IND: Rib dysfunction, R3–R12.

PP: The patient lies in the prone position.

DP: Stand on side opposite of contact, facing cephalad for I-S VEC or caudad for S-I VEC.

CP: The cephalad hand's thumb-thenar contact follows the rib contour.



Figure 5-194 Covered-thumb contact applied to the left fifth rib angle to induce distraction of the left fifth costotransverse articulation.

SCP: Angle of the rib just lateral to the transverse process.

IH: Place the caudad hand's pisiform-hypothenar contact over contact hand's thumbnail, with the fingers wrapped around the wrist.

VEC: P-A and either S-I or I-S.

P: Lean in with your body weight to establish joint and deliver a straight-arm body-drop thrust.

Side-Posture

Web/Costal Push (Figure 5-195)

IND: Rib dysfunction, R2–R10 (bucket-handle dysfunction or intercostal distraction).

PP: The patient lies with the dysfunctional side up and arm abducted over the head. A roll may be used to induce lateral flexion.

DP: Stand behind the patient in a fencer stance, inferior to the contact.

CP: Web contact of the outside hand.

SCP: Inferior margin of the superior rib at the midaxillary line.

IH: The IH supports the contact hand on the dorsal surface, with the fingers wrapped around the wrist.

VEC: I-S and L-M.

P: Establish the adjustive contact by sliding the web of the contact hand onto the superior rib of the dysfunctional intercostal space. Develop preadjustive tension by leaning headward. At tension, deliver a shallow impulse thrust superiorly and medially to separate the intercostal space. Take care to avoid excessive medial pressure to the rib cage. This procedure may be used to mobilize the intercostal space by applying a slow stretch instead of an adjustive thrust.

Sitting

Index/Costal Push (Figure 5-196)

IND: Rib dysfunction, R1.



Figure 5-195 Web contact applied to the inferior margin of the right seventh rib in the midaxillary line to induce separation in the intercostal space between the seventh and eighth ribs.

PP: The patient sits relaxed in a cervical chair.

DP: Stand behind the patient, toward the side of rib dysfunction.

CP: Index finger of the hand corresponding to the side of dysfunction. The wrist is locked, with the arm angled approximately 45 degrees to the horizontal plane.

SCP: Posterior superior angle of the first rib.

IH: The IH grips the top of the patient's head while the forearm rests against the patient's contralateral skull.

VEC: S-I, L-M, and P-A.



Figure 5-196 Index contact established along the superior margin of the right first rib to induce distraction and inferior glide in the right first costotransverse articulation.

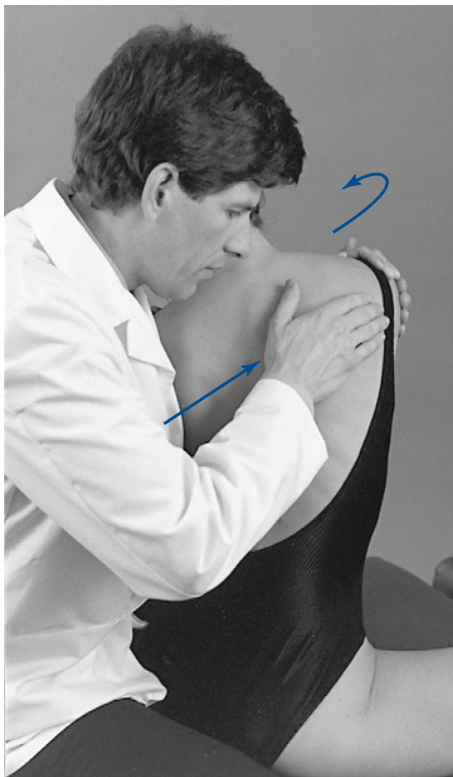


Figure 5-197 Hypothenar contact applied to the right sixth rib angle to induce distraction in the right fifth costotransverse articulation.

P: Lightly establish the contacts and circumduct the patient's head toward the side of rib dysfunction. At tension, deliver an impulse through the shoulder of the contact hand while simultaneously delivering a shallow distraction force through the IH.

Hypothenar (Thenar)/Costal Push (Figure 5-197)

IND: Rib dysfunction, R4–R12.

PP: The patient sits with legs straddling the adjusting bench. The arms are crossed, with the hands grasping the shoulders.

DP: Sit or stand behind the patient.

CP: Hypothenar or thenar of hand corresponding to the side of contact.

SCP: Just medial to the rib angle.

IH: Your IH and arm reach around the patient to contact the opposing forearm.

VEC: P-A and L-M.

P: Preadjustive tension is typically developed by flexing, laterally flexing, and rotating the patient forward on the side of contact. Once tension is established, deliver an impulse thrust through the contact hand, assisted by a pulling thrust generated through your indifferent arm and trunk.

COSTOSTERNAL ADJUSTMENTS

Supine

Covered-Thumb/Costosternal Push (Figure 5-198)

IND: Anterior rib dysfunction, R2–R6.

PP: The patient lies supine, with both arms resting on the table.

DP: Stand on the side opposite the adjustive contact.



Figure 5-198 Covered-thumb contact over the right anterior sixth rib to induce distraction in the right sixth costosternal articulation.

CP: Thumb of caudal hand.

SCP: Anterior rib just lateral to costosternal junction.

IH: Palm of superior hand covering the thumb and dorsum of the contact hand.

VEC: M-L and slightly P-A.

P: Slide laterally onto contact with the thumb and reinforce the contact with the IH. Deliver a shallow and gentle impulse thrust, emphasizing a lateral VEC to avoid compression of the rib cage. When applying this procedure to female patients, it is important to ensure that the breast tissue is distracted away from the doctor's contact hand and that the patient is properly draped.

Sitting

Hypothenar/Costosternal Pull (Figure 5-199)

IND: Anterior rib dysfunction, R2–R6.

PP: The patient sits with legs straddling the adjusting bench and arms relaxed in lap.

DP: Sit behind the patient.

CP: Hypothenar of the hand corresponding to the side of adjustive contact.

SCP: Anterior rib just lateral to costosternal junction.

IH: The palm of your superior hand reinforces the dorsum of the contact hand.

VEC: M-L.

P: Slide laterally onto contact with the hypothenar of the contact hand and reinforce the contact with the palm of the IH. Develop distractive tension by tractioning laterally and rotating the patient toward the side of adjustive contact. At tension, deliver a shallow impulse through both arms and the trunk. Avoid excessive anterior compression of the rib cage by inducing rotation and distraction during the adjustive thrust. When applying this procedure to female patients it is important to ensure that the breast tissue is distracted away from the doctor's contact hand and that the patient is properly draped.



Figure 5-199 Hypothernar contact over the left third anterior rib to induce distraction in the third costosternal articulation.

LUMBAR SPINE

The most important characteristic of the lumbar spine is that it must bear tremendous loads created by body weight that interact with forces generated by lifting and other activities involving powerful muscle actions. In addition to bearing formidable loads, the lumbar spine is largely responsible for trunk mobility, thereby placing significant mechanical demands on this region.

FUNCTIONAL ANATOMY

The typical lumbar vertebra is a large kidney-shaped structure designed to carry the heavy loads imposed by upright posture. It is wider from side to side than from anterior to posterior. The anterior surface of the body is convex from side to side, and the posterior surface is concave from superior to inferior and from side to side. The superior and inferior surfaces range from flat to slightly concave (Figure 5-200).

L5 is considered an atypical lumbar vertebra. It has the largest circumference of all vertebrae, and its body is thicker at its anterior aspect than at its posterior aspect, although the overall thickness of the body is somewhat less than the bodies of the superior lumbar vertebrae. The transverse processes are short and thick; the spinous process is shorter and more rounded than the other lumbar vertebrae, and the superior articulating processes are directed more posteriorly and less medially. The inferior articulating processes are farther apart and are oriented more in the coronal plane, compared with the normal sagittal orientation of the remaining lumbar vertebrae.

The lumbar pedicles originate from the upper part of the vertebra and extend horizontally and posteriorly; the pedicles are short and strong. The superior vertebral notch is shallow,

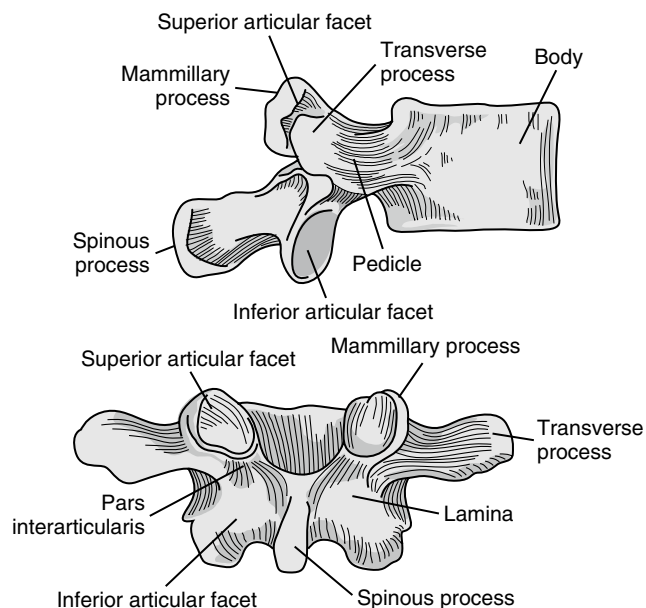


Figure 5-200 Posterior view (A) and side view (B) of a lumbar segment. (From Dupuis PR, Kirkaldy-Willis WH. In Cruess RL, Rennie WRJ, eds: *Adult orthopaedics*, New York, 1984, Churchill Livingstone.)

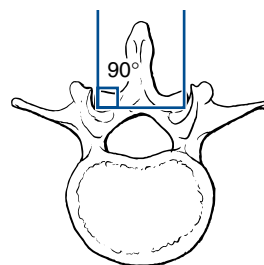


Figure 5-201 Lumbar facet planes. (From White AA, Panjabi MM: *Clinical biomechanics of the spine*, ed 2, Philadelphia, 1990, JB Lippincott.)

and the inferior vertebral notch is deep. The lumbar laminae are short, broad, and strong and run in a vertical plane (see Figure 5-200).

The thick and broad spinous processes are hatchet-shaped structures that point straight posteriorly. The transverse processes are long, slender, and flattened on their anterior and posterior surfaces. They originate from the lamina-pedicle junction and are considered to be quite frail. L3 has the longest of the lumbar transverse processes.

The articular processes are also large, thick, and strong. The superior articular processes are concave and face posteromedially, and the inferior articular processes are convex and face anteriorly and laterally. The superior articular processes are wider apart and lie outside the inferior articular processes. The mammillary processes are located on the superior and posterior edge of the superior articular process.

The lumbar facets lie primarily in the sagittal plane but become more coronal at the lumbosacral junction (Figure 5-201). This facet configuration limits rotational flexibility and allows for greater mobility in flexion and extension (Figure 5-202). The lumbar facets normally carry 18% of axial load and up to 33% in

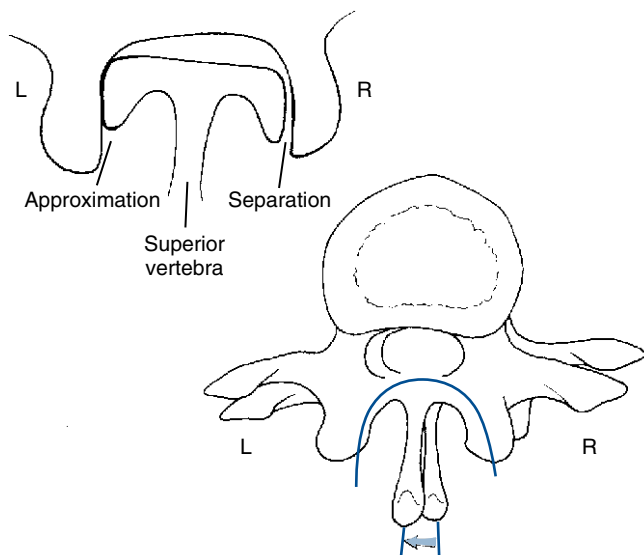


Figure 5-202 Right rotation of a lumbar segment, illustrating how impact of the left facets (compression facet) limits rotation.

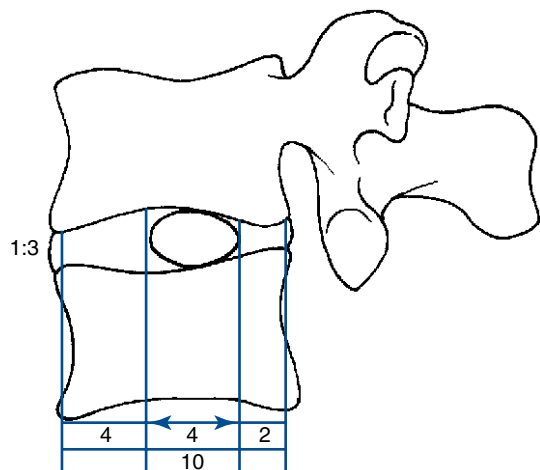


Figure 5-203 Location of the nucleus and disc height-to-body height ratio in the lumbar spine.

extended postures. The facets with their articular capsules provide up to 45% of the torsional strength of the lumbar spine.^{33,34}

The lumbar IVDs are well developed. The nucleus is localized somewhat posteriorly in the disc, and the disc height-to-body height ratio is 1:3 (Figure 5-203). This relationship allows for more movement than the thoracic segments and maintains a significant preload state, giving the disc's greater resistance to axial compressive forces.

The lumbar spinal canal contains, supports, and protects the distal portion of the lumbar enlargement of the spinal cord proximally (conus medularis) and the cauda equina with spinal nerves distally. This portion of the central nervous system is ensheathed in three meninges and tethered to the coccyx by the filum terminale. Because the cord itself ends at the level of L2, the nerve roots (NRs) continue down the spinal canal as the cauda equina. The NRs exit the dura slightly above the foraminal opening, causing their course to be more oblique and their length to increase (Figure 5-204).

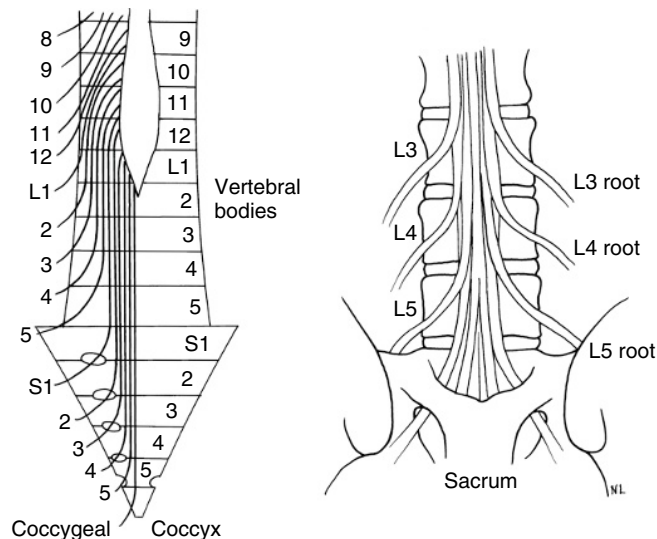


Figure 5-204 Course of the lumbar nerve roots.

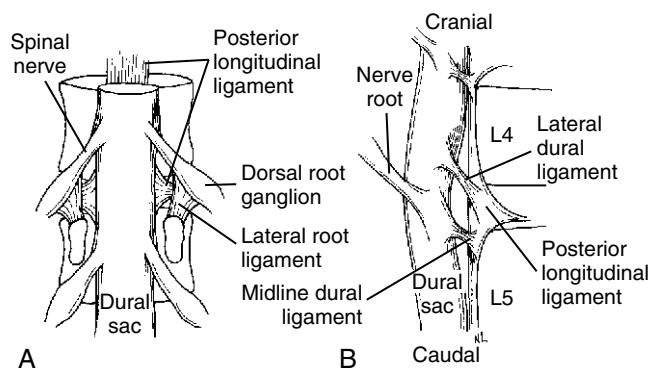


Figure 5-205 Hoffmann's dural ligaments. **A**, Posterior view. **B**, Lateral view, with the posterior arch removed.

The dural sac and its contents are not freely mobile structures. A series of ligamentous attachments, called *Hoffman ligaments*, define a specific range of movement, thus stabilizing the dural sac within the foraminal canal (Figure 5-205). Although spinal cord movements are limited, the spinal cord does demonstrate flexibility during different movements and activities. When flexing from the neutral position, the length of the spinal canal increases. This is because the instantaneous axis of motion is located in the anterior aspect of the vertebral bodies. Similarly, in extension the canal length decreases. The spinal cord must follow the changes in the canal during these physiologic movements (Figure 5-206). It accomplishes this through the mechanisms of folding and unfolding, as well as elastic deformation. In the neutral position, the cord is folded somewhat like an accordion and has slight tension. During flexion the cord first unfolds and then undergoes elastic deformation. During extension the cord first folds upon itself, then undergoes elastic compression.

LUMBAR CURVE

The secondary lordotic lumbar curve starts to develop when a child is approximately 9 to 12 months of age and beginning to sit up. As the child learns to stand, the curve becomes established,

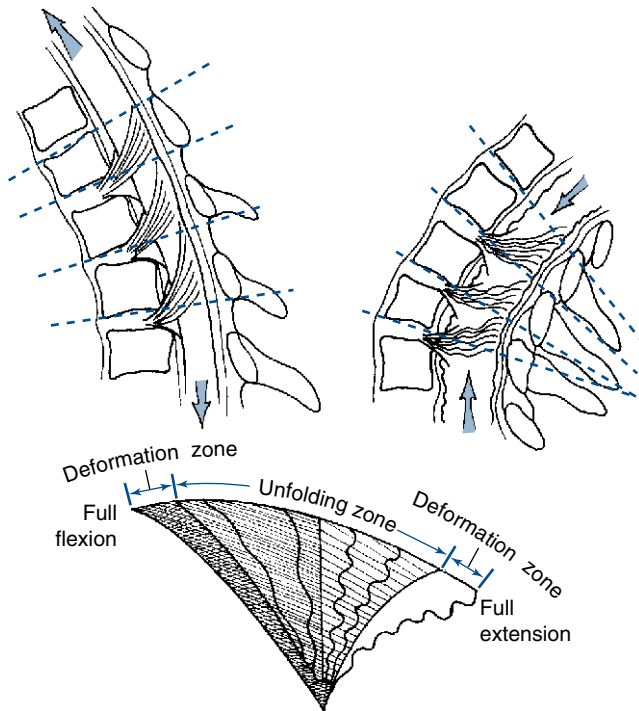


Figure 5-206 Effects of flexion (*top left*) and extension (*top right*) movements on the spinal canal and its contents (cord, meninges, and nerve roots).

usually by 18 months of age. The lumbar lordosis usually begins at the L1–2 level and gradually increases at each level caudal to the sacrum, with the apex of the curve centering around the L3–4 disc.³⁵

Moe and Bratford³⁶ state that the normal lumbar lordosis should be 40 to 60 degrees but fail to define the levels used for measurement. The often ill-defined radiographic image of the superior aspect of the sacrum makes it difficult to use for measuring the lumbar lordosis. When using the inferior aspect of the L5 vertebral body and the superior aspect of the L1 vertebral body, a normal range for the lumbar lordosis is 20 to 60 degrees^{37,38} (Figure 5-207).

In the upright bipedal posture, the lumbar curve, as well as the rest of the spine, is balanced on the sacrum. Therefore, changes in the sacral base angle can influence the depth of the A-P curves in the spine. The sacral base angle increases with an anterior pelvic tilt, resulting in an increase in the lumbar lordosis, which places more weight-bearing responsibility on the facets. The sacral base angle decreases with a posterior pelvic tilt, resulting in a decrease in the lumbar lordosis, placing more weight-bearing responsibility on the disc and reducing the spine's ability to absorb axial compression forces.

RANGE AND PATTERNS OF MOTION

The lumbar spine is significantly more flexible in flexion and extension than any other lumbar movements. Approximately 75% of trunk flexion and extension occurs in the lumbar spine, with approximately twice as much flexion occurring as extension. The first 60 degrees of torso flexion consist of lumbar spine sectional

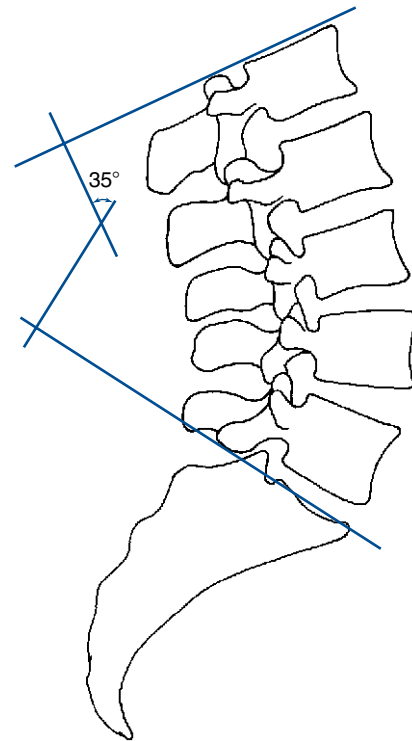


Figure 5-207 Measurement of the lumbar lordosis, showing a 35-degree curve.

TABLE 5-7 Average Segmental ROMs for the Lumbar Spine

Vertebra	Combined Flexion and Extension	One-Side Lateral Flexion	One-Side Axial Rotation
L1–2	12	6	2
L2–3	14	6	2
L3–4	15	8	2
L4–5	16	6	2
L5–S1	17	3	1

Modified from White AA, Panjabi MM: *Clinical biomechanics of the spine*, ed 2, Philadelphia, 1990, JB Lippincott.

flexion as the pelvis is stabilized by the gluteal and hamstring muscles. After the lumbar flexion, the pelvis begins to flex, producing an additional 30 degrees of motion. In contrast, lumbar lateral flexion exhibits only moderate mobility, and axial rotation is quite limited. The majority of trunk rotation occurs in the thoracic spine. Table 5-7 and Figure 5-24 identify lumbar segmental ROM.

Flexion and Extension

Combined segmental flexion and extension in the lumbar spine averages 15 degrees per segment, with motion increasing in an S-I direction.^{5,39} Lumbar flexion and extension combines sagittal plane rotation with an average of 2 to 3 mm of sagittal plane translation in each direction.^{5,40,41} White and Panjabi⁵ have proposed

that 4.5 mm be considered the upper limit for the radiographic investigation of clinical joint instability. Coupling of lateral flexion and rotation with flexion and extension have also been noted,^{28,41} but are considered by White and Panjabi⁵ to be abnormal patterns suggestive of suboptimal muscle control.

The precise location of the IAR for lumbar movements has not been established.⁵ The IAR for flexion and extension is most commonly placed within the IVD of the subjacent vertebrae, with flexion located toward the anterior portion and extension toward the posterior (Figure 5-208). During flexion the vertebra tilts and slides anteriorly as the inferior facets move superiorly and away from the lower vertebra. The disc is compressed anteriorly and stretched posteriorly. During extension the facets approximate one another, and the ALL, anterior portion of the joint capsule, and anterior portion of the disc are stretched (Figure 5-209).

Lateral Flexion

Segmental lateral flexion averages approximately 6 degrees to each side. Movement is about the same for each segment, with the exception of the lumbosacral joint, which demonstrates approximately half the movement.^{5,39} Lateral flexion in the lumbar spine is coupled with opposite-side rotation (e.g., body rotation to the

convexity and spinous deviation to the concavity). This leads to a pattern in which the spinous processes end up pointing in the same direction as the lateral flexion (Figure 5-210).^{5,42} This pattern is opposite to that in the cervical and upper thoracic spine (see Figures 5-116 and 5-117).

There are several theoretic factors producing coupled rotation during lateral flexion. One important factor is the principle that it is not possible to bend a curved rod without producing some rotation. The second force acting on the spine to produce coupled rotation is a product of eccentric muscle activity. Lateral bending is controlled mainly by eccentric activity of the quadratus lumborum, which inserts posteriorly to the normal axis of motion. The normal axis is located in the posterior one third of the disc. Therefore, normal muscular activity leads to posterior rotation of the vertebral bodies on the side of convexity and rotation of the spinous processes to the side of concavity.

The IAR for lateral flexion is placed within the subadjacent disc space.⁵ For left lateral flexion, the axis is located on the right side, and for right lateral flexion, on the left (see Figure 5-208). During lateral flexion, the vertebra tilts and slides toward the concave side, producing a smooth, continuous arc. The facets approximate on the concave side and separate on the convex side. The disc is compressed on the concave side and stretched on the convex side. The ligamentum flavum, intertransverse ligament, and capsular ligaments are stretched on the convex side.

Grice⁴³ and Cassidy⁴⁴ have studied the coupling movements for lateral flexion and have proposed classifying segmental lateral flexion into one of four commonly encountered patterns (Figure 5-211). In addition, various pathomechanical theories have been proposed as possible explanations for each abnormal pattern.

The first pattern, type I movement, exhibits the normal pattern of coupling in which lateral flexion is associated with axial rotation to the opposite side. This produces a pattern in which posterior body rotation occurs on the side opposite the lateral flexion and in which the spinous processes rotate toward the side of lateral flexion. Purportedly, this pattern is represented when normal motor control is exerted through eccentric unilateral

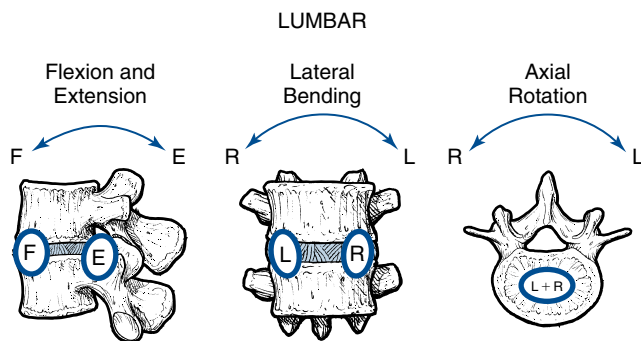


Figure 5-208 Approximate locations for the instantaneous axes of rotation for the 6 degrees of freedom in the lumbar segments. (From White AA, Panjabi MM: *Clinical biomechanics of the spine*, ed 2, Philadelphia, 1990, JB Lippincott.)

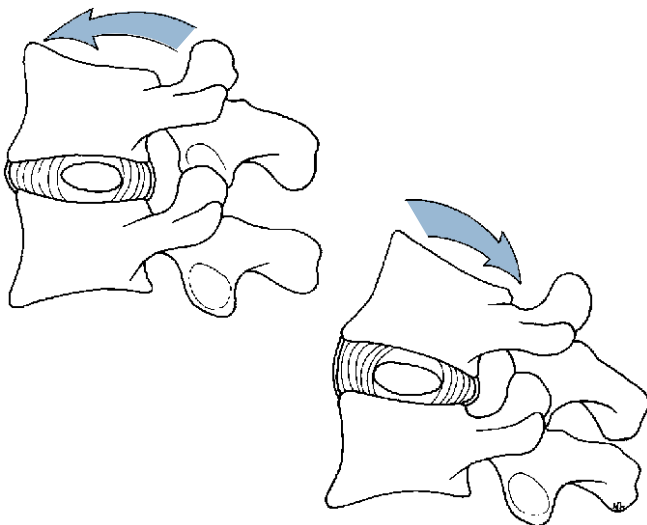


Figure 5-209 Flexion and extension movements of a lumbar segment.

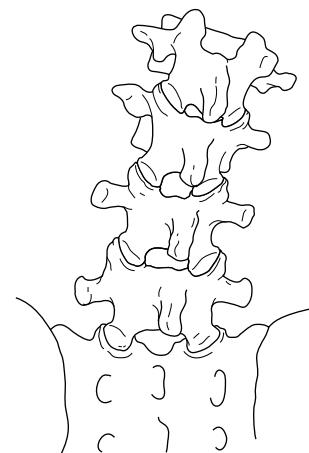


Figure 5-210 Coupling pattern of lateral flexion with contralateral rotation in the lumbar spine.

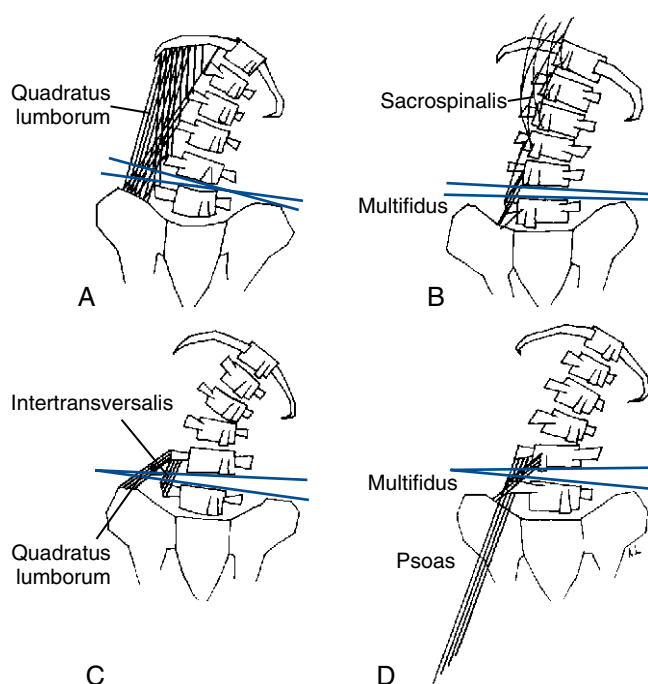


Figure 5-211 A, Type I movement of coupled lateral flexion with contralateral rotation under the control of the quadratus lumborum muscle. B, Type II movement of coupled lateral flexion with ipsilateral rotation under the control of the sacrospinalis and multifidus muscles. This pattern may be sectional, as shown here, or segmental. C, Type III movement, consisting of segmental aberrant lateral flexion because of faulty disc mechanics or quadratus lumborum and intertransversalis muscles. D, Type IV movement, consisting of segmental aberrant lateral flexion and rotation because of faulty disc mechanics or psoas and multifidus muscles.

contraction of the quadratus lumborum muscle, although synergistic muscles act as a brake to prevent hypermobility. Type I movement may still be abnormal if it is diminished or excessive (see Figure 5-211, A).

Type II motion combines lateral flexion and axial rotation to the same side. This induces posterior body rotation on the side of lateral flexion and rotation of the spinous process to the side opposite the lateral flexion. A muscular imbalance of the sacrospinalis, especially the longissimus and spinalis portions, is proposed as the source of this abnormal pattern. The semilumped sitting posture may also produce this pattern of movement (see Figure 5-211, B).

Type III movement is represented by aberrant segmental lateral flexion and normal coupled rotation. With type III movement, the involved segment demonstrates no lateral flexion or lateral flexion movement in the direction opposite the bending of the trunk. This pattern is theorized to result from faulty disc mechanics or overdominance of the quadratus lumborum or intertransversalis muscles (see Figure 5-211, C).

The last pattern, type IV, is represented by aberrant segmental rotation and lateral flexion. This pattern may also result from faulty disc mechanics or an imbalance in the psoas or multifidus muscles (see Figure 5-211, D).

These patterns are primarily determined by evaluation of lateral bending functional x-ray studies. The use of lateral flexion and flexion-extension movement radiographs has been described

(see Figure 3-23) and recommended for evaluation of segmental motion and instability.⁴³⁻⁶¹ Although functional radiology should be considered an important potential tool in the evaluation of joint dysfunction, its limitations should also be realized. There is evidence to suggest that findings on lateral bending radiographs do not correlate well with back pain and other abnormal clinical findings.^{53,62}

Rotation

Axial rotation is quite limited in the lumbar spine. Segmental ROM is uniform throughout the lumbar segments and averages only 2 degrees per motion segment.^{5,39} The sagittally oriented facet joints act as a significant barrier to rotational mobility. During rotation, the facet joints glide apart on the side of rotation and approximate on the side opposite rotation (see Figure 5-202). The instantaneous axis for axial rotation is placed within the posterior nucleus and annulus¹ (see Figure 5-208).

Rotation of the lumbar spine is also consistently coupled with lateral flexion and slight sagittal plane rotation. The coupled lateral flexion varies between upper and lower lumbar segments. Rotation in the upper three segments (L1–L3) is coupled with opposite-side lateral flexion. Rotation in the lower two segments (L4 and S1) is coupled with same-side lateral flexion.^{28,63} The transitional change in coupling that occurs at the L4–5 motion segment may be of clinical significance in predisposing this level to increased torsional stress, clinical instability, and degenerative change.⁵

The pattern of coupled sagittal plane rotation depends on the starting position of the lumbar spine.²⁸ With the spine in a neutral starting position, the lumbar spine flexes at all levels when rotated. When the spine is rotated from a flexed posture, it has a tendency to extend, and when rotated from an extended posture, it has a tendency to flex. This pattern was also noted for lateral flexion, leading to the generalization that “lateral bending or axial rotation has a tendency to straighten the spine (move it toward a neutral posture) from the flexed as well as the extended postures.”⁵

KINETICS OF THE LUMBAR SPINE

The control of flexion movements is largely a result of the eccentric contraction of the erector spinae (sacrospinalis) muscles (Figure 5-212), although it is initiated by concentric contraction of the psoas and abdominals. The iliopsoas flexes the spine when the femur is fixed, and the abdominal muscles flex the spine when the pelvis is fixed. During the first 60 degrees of flexion, the pelvis is locked by the gluteus maximus and hamstrings, but after 60 degrees, the weight of the trunk overcomes the stabilizing force of the glutei and hamstrings, and the pelvis rotates an additional 30 degrees at the hips. In full flexion, all the muscles are relaxed, except the iliocostalis thoracis, and the trunk is supported by ligaments and passive muscle tension.

The return to neutral is the reverse activity, with the pelvis moving first under the control of the hip extensors, followed by extension of the lumbar spine, controlled by erector spinae muscles. Flexion is limited by the ligamentum flavum, PLL, the posterior aspect of the capsular ligament, and interspinous ligament.

Extension is initiated by concentric contraction of the sacrospinalis. Again, after the initial movement, gravity and eccentric activity of the abdominal muscles become the major controlling

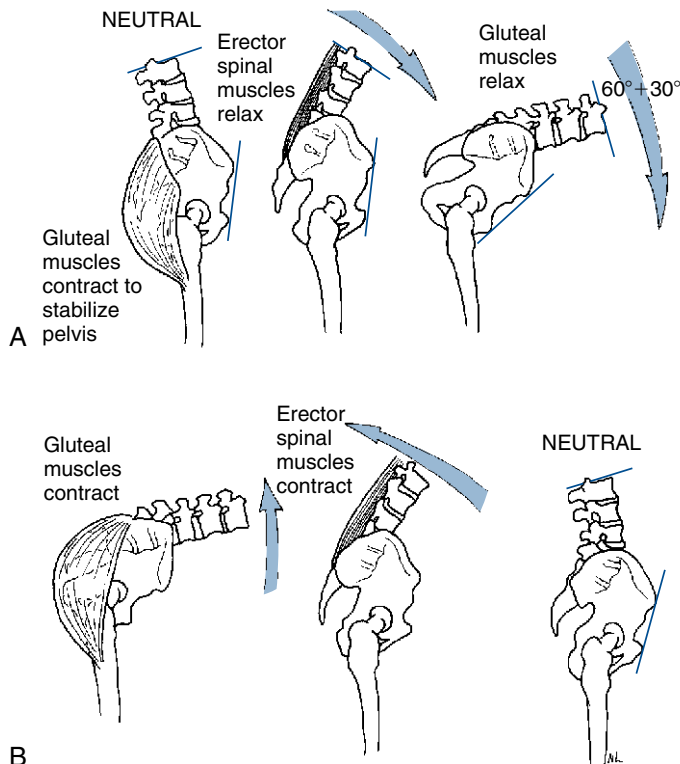


Figure 5-212 Flexion of the trunk. **A**, The first 60 degrees of flexion involve eccentric contraction of the lumbar paraspinal muscles, followed by an additional 30 degrees of hip flexion after relaxation of the gluteal muscles. **B**, In extension, the converse occurs.

forces in extension. Extension is limited by the ALL and anterior annulus and most significantly by bony impact of the spinous processes and articular facets.

Lateral flexion is initiated by concentric contraction of the quadratus lumborum on the ipsilateral side and then immediately controlled by eccentric activity of the contralateral quadratus lumborum. Lateral flexion movement is limited by impact of the articular facets on the side of bending, and the capsular ligaments, ligamentum flavum, intertransverse ligament, and deep lumbar fascia on the contralateral side.

Rotation is initiated by concentric activity of the abdominal obliques and assisted by concentric activity of the short segmental muscles (multifidus and rotatores) on the contralateral side. Rotational movements are controlled or limited by eccentric activity of the ipsilateral multifidus and rotatores (although mainly limited by facet design), as well as the capsular, interspinous, and flaval ligaments. Balancing contraction of the contralateral muscles is important in maintaining the normal instantaneous axis of motion for axial rotation.

EVALUATION OF THE LUMBAR SPINE

Observation

The assessment of lumbar function should begin with an evaluation of lumbopelvic alignment and ROM. The sacral base forms the foundation of the spine, and a functional or structural alteration in the pelvis or lower extremities may alter the alignment of the lumbar spine and segments above. Pelvic and hip alignment

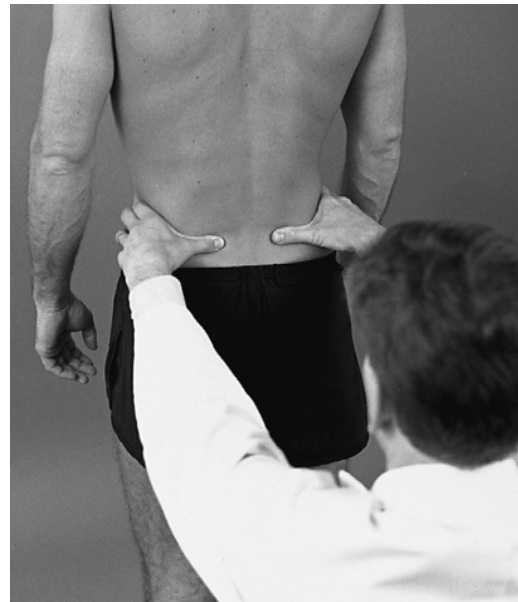


Figure 5-213 Observation of lumbopelvic alignment and posture with palpation of the posterior iliac spines and iliac crests.

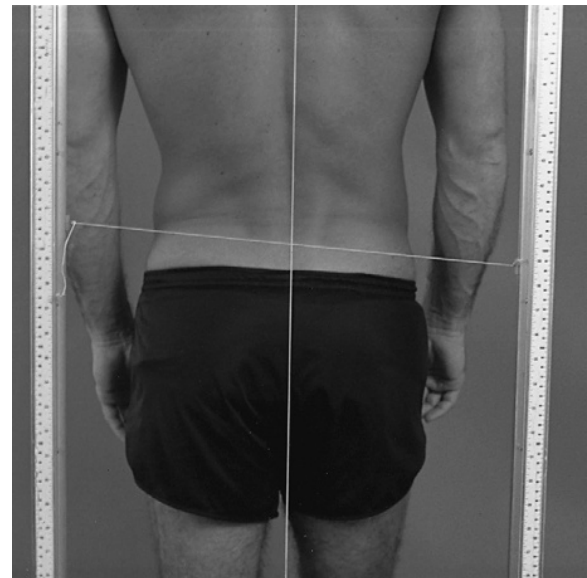


Figure 5-214 Posterior plumb line observation, demonstrating pelvic unleveling low on the right, with a right convex curve in the lumbar spine.

are evaluated by observing the alignment of the gluteal folds, posterior iliac spines (sacral dimples), and iliac crests.

To palpate the alignment of the pelvis, place the thumbs on the posterior iliac spines and the fingertips along the superior margin of the crests (Figure 5-213). Compare each side for symmetry and their orientation to the greater trochanter for possible leg length inequality.

Coronal plane alignment of the lumbar spine is evaluated by observing the orientation of the spinous process, status of the paraspinal muscles, and contours of the waist (Figure 5-214). Scoliotic curvatures in the lumbar spine are frequently represented by muscle asymmetry and increased paraspinal muscle mass on the convex side of scoliosis. Compensatory curvatures are

common in the lumbar spine, and any noted deviations should be followed up with an assessment of leg length.

Sagittal plane orientation of the hips, pelvis, and lumbar spine is evaluated from the side. The lumbosacral angle in large part determines the angle of the lumbar curve and is often mirrored by the positioning of the pelvis. Anterior or posterior tilting of the pelvis usually results from alterations in the hip angle. Anterior pelvic tilt results from bilateral hip flexion, and posterior pelvic tilt results from bilateral hip extension (Figure 5-215).

Anterior pelvic tilt increases the lumbosacral angle and lumbar curve; posterior pelvic tilt reduces the lumbosacral angle and curve. The lumbar curve also alters its angle relative to structural alteration in the thoracic curve. Congenitally straight thoracic curves often lead to straightening of the lumbar and cervical curves. Increased thoracic kyphosis may lead to accentuation of the lumbar and cervical curves.

Lumbopelvic movement should be observed for range and symmetry. Flexion, extension, and lateral flexion are usually assessed with the patient standing. Rotation is more effectively evaluated in the sitting position to fix the pelvis and prevent hip rotation.

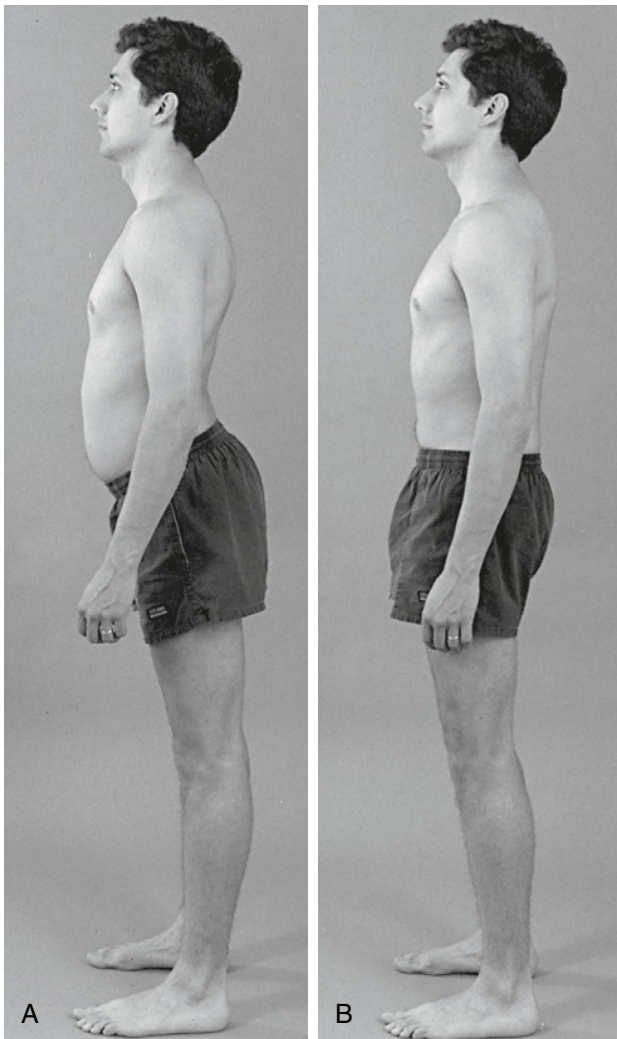


Figure 5-215 Observation of posture from a lateral view, showing an anterior pelvic tilt with lumbar hyperlordosis (A) and a posterior pelvic tilt with lumbar hypolordosis (B).

To assess flexion, the patient bends forward, and any limitations, painful arcs, or alterations in normal sequencing are observed. With normal range, the patient should be able to come within several inches of the floor with the fingertips, and the lumbar curve should reverse (Figure 5-216).

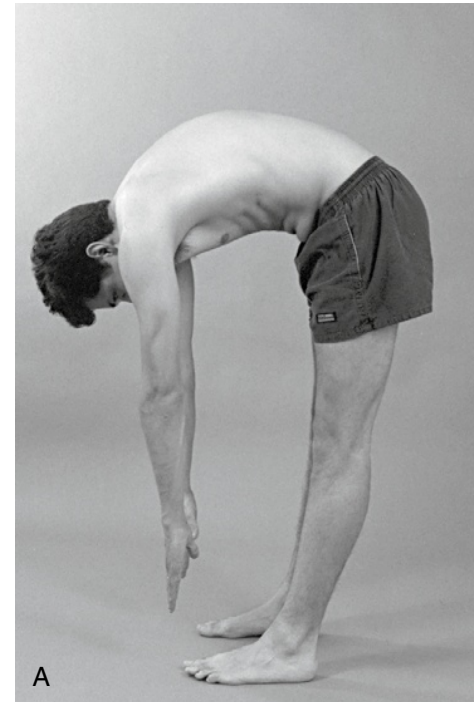


Figure 5-216 Observation of thoracolumbar range of motion. A, Flexion. B, Extension.

(Continued)

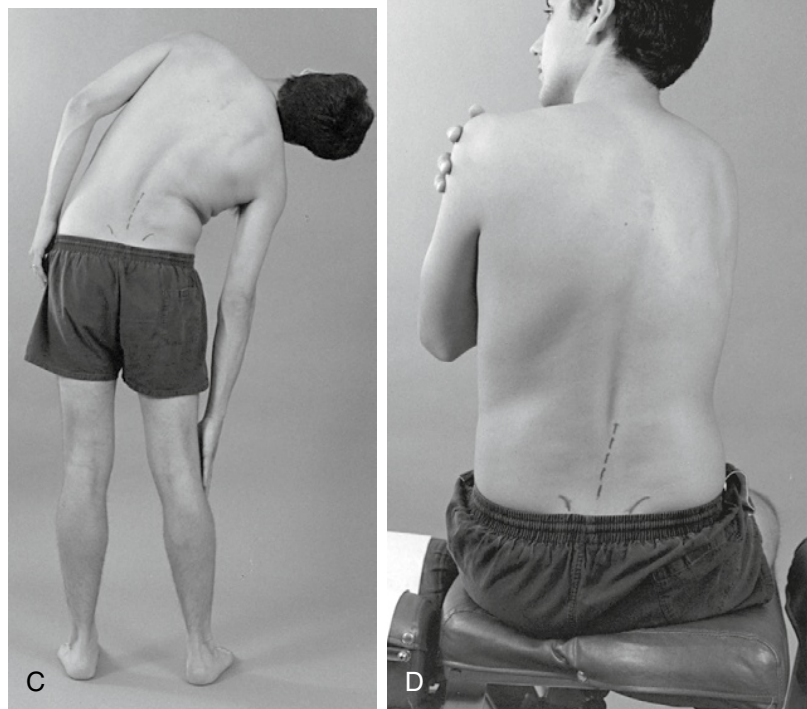


Figure 5-216—Cont'd C, Lateral flexion. D, Rotation.

After the patient returns to neutral, the doctor should stabilize the patient's hips while extension is performed. Extension is significantly more limited than flexion, and mild midline lumbosacral discomfort is commonly associated with full extension.

Lateral bending is evaluated by instructing patients to bend to one side while running their fingers down the lateral surface of the leg. It is important to ensure that the patient does not axially rotate the trunk, bend the knee, or raise the foot off the floor while bending. The movement should be symmetric, and the doctor should record where the patient's fingertips pass relative to the knees. A smooth C curve should be observed to the side of bending. Areas of regional restriction or increased movement may be noted by observing for a sudden break (bent-stick configuration) in integrated movement.

To isolate movements of the lumbar spine from hip and thoracic movements, the doctor may use inclinometric methods and measure movement between the thoracolumbar and lumbosacral regions. Table 5-8 lists the normal ranges for lumbar movement; the methods for measuring movement have been previously described (see Figure 3-10).

TABLE 5-8 Global ROMs for the Lumbar Spine

Flexion	40–60 degrees
Extension	20–35 degrees
One-Side Lateral Flexion	15–25 degrees
One-Side Axial Rotation	5–18 degrees

Static Palpation

To evaluate the bony and soft tissue structures of the lumbar spine, the patient is placed in the prone position and scanned for areas of potential tenderness, misalignment, or asymmetry.

To scan the bony landmarks, use the pads of the fingers or thumbs and palpate the spinous processes, interspinous spaces, and mammillary processes (Figure 5-217). Palpation of the interspinous spaces is enhanced by placing a small roll under the abdomen or elevating the lumbar and pelvic sections of an articulating table. The mammillary processes are not distinctly palpable; rather, the doctor feels for a sense of firmness in the soft tissue as the contact passes over each mammillary process. Rotational prominence of lumbar spinal segments is perceived by a sense of fullness in the muscles over the top of the mammillary processes and not by actual palpation of bony rotation.

Palpation of the lumbar paraspinal soft tissues is conducted by applying bilateral contacts with the palmar surfaces of the fingers or thumbs. Evaluation should incorporate an assessment of tone and texture of the erector spinae, quadratus lumborum, deep segmental muscles, and iliolumbar ligaments. The psoas muscle should also be palpated for tone and tenderness. The psoas is accessible for palpation in the supine position, and the belly becomes more evident by inducing hip flexion on the side of palpation.

Motion Palpation

Joint Play. The lumbar spine may be scanned in the sitting or prone position for sites of painful or restricted JP. Sites of suspected abnormality should be further assessed with specific JP

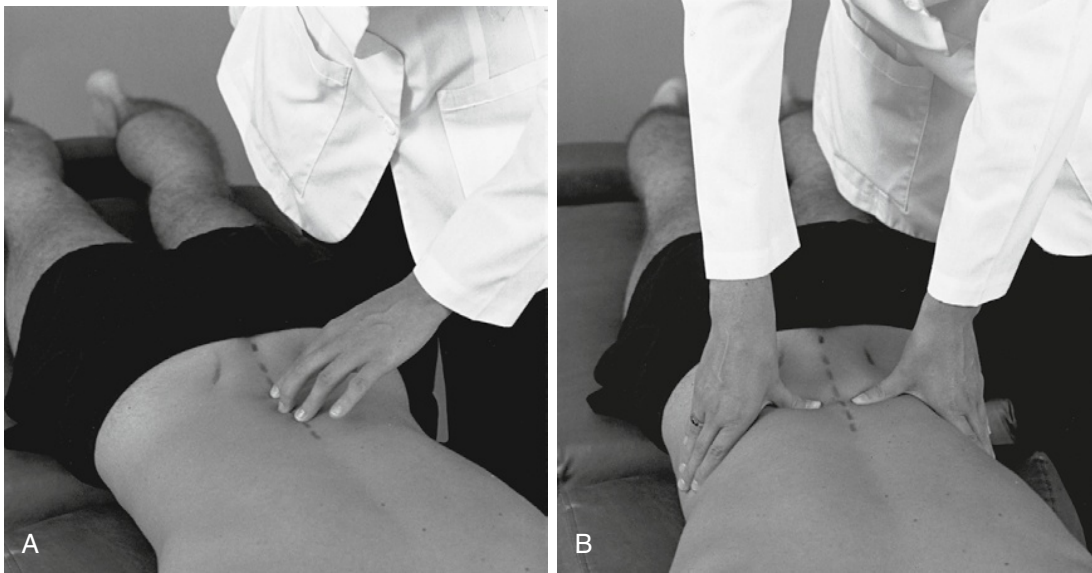


Figure 5-217 Palpation of lumbar interspinous alignment and sensitivity (A) and paraspinal muscle tone, texture, and sensitivity (B).

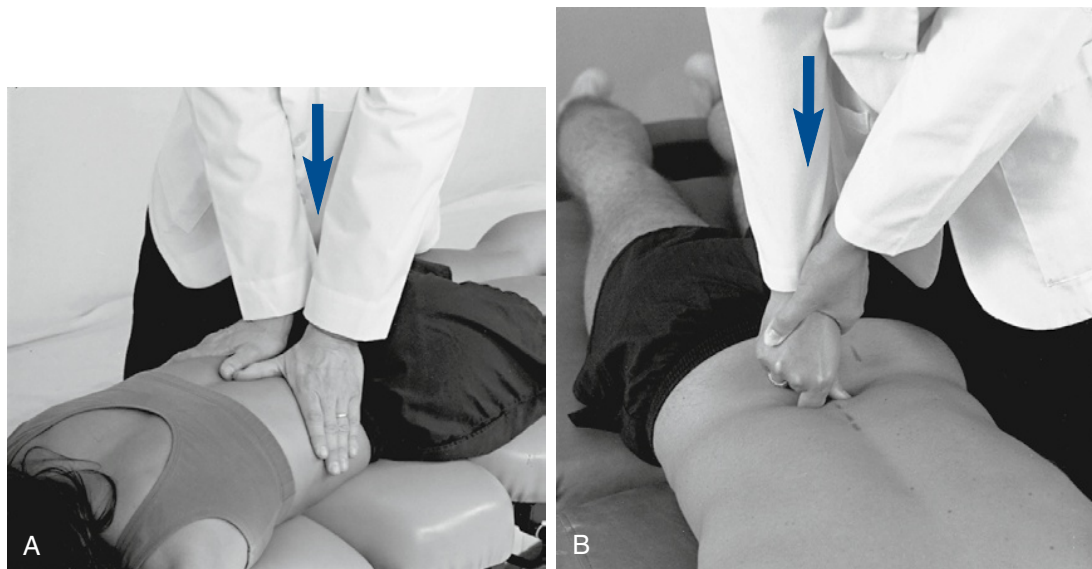


Figure 5-218 Lumbar joint play evaluation for posterior-to-anterior glide. A, Bilateral thenar contacts over the mammillary processes. B, Digit contact over the spinous process.

tests. The same methods applied in the thoracic spine for assessing P-A glide and counter-rotation may be applied to the lumbar spine (Figure 5-218). Additional options include the evaluation of rotation, the assessment of lateral glide, and a side-posture method for evaluating possible instability.

To assess rotational JP, the doctor contacts adjacent spinous processes (Figure 5-219) or the spinous process and the anterior crest of the ilium and applies counter-rotation across the joint. Normal movement is pain-free and is associated with a sense of giving with pressure and recoil when pressure is released.

Lateral glide of individual lumbar motion segments may be evaluated with the patient in the prone position by establishing a thumb contact against the lateral surface of adjacent spinous processes with the cephalic hand while the other hand grasps the patient's anterior and medial thigh (Figure 5-220). Movement is

induced by applying medial pressure against the spinous process while the patient's leg is passively abducted. Normal movement is represented by segmental bending and shifting of the spinous process away from the contact. This procedure can also be performed by moving the pelvic section of a flexion table from side to side.

To evaluate segmental stability in side posture, place the patient on either side, with the upper thigh and knee flexed. Establish a fingertip contact over the spinous processes and interspinous spaces with the cephalic hand and straddle the patient's flexed knee (Figure 5-221). Apply posterior shearing pressure through the patient's knee and gentle anterior pressure through the palpation hand. Feel for gliding between adjacent spinous processes. Excessive posterior glide (translation) of the inferior spinous process relative to the superior spinous process suggests possible clinical joint instability.

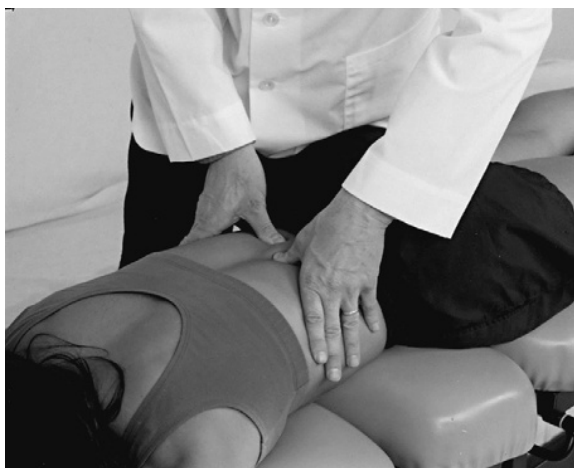


Figure 5-219 Counter-rotational joint play evaluation for left rotational movement of L3 relative to L4. Opposing forces are directed toward the midline through a contact established on the left side of the L3 spinous process and the right side of the L4 spinous process.

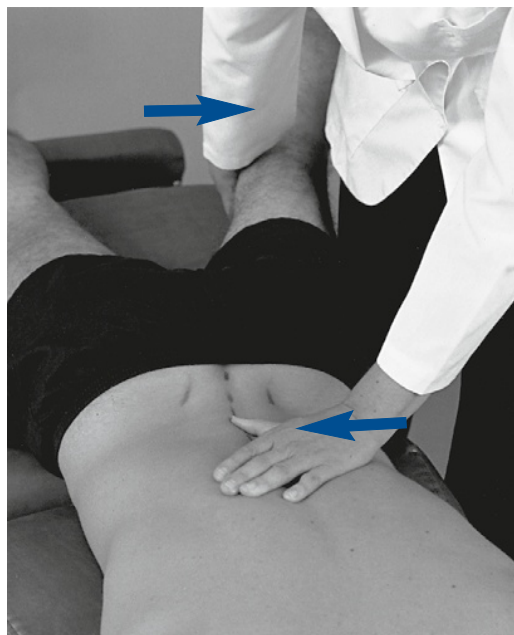


Figure 5-220 Lumbar joint play evaluation for lateral glide in the L3–4 motion segment, using a thumb contact across the left L3–4 interspinous space.

Lumbar Segmental Motion Palpation and End Play. Lumbar segmental motion may be evaluated with the patient in the sitting or side-posture positions. Both postures are effective for assessing lumbar mobility, but side-posture positions do not provide as much freedom for full trunk movement. This is especially true for the evaluation of lumbar lateral flexion and end play.

Sitting Methods. For sitting evaluations, place the patient on an adjusting bench or palpation stool with the arms crossed over the chest. The doctor may sit behind or stand beside the patient. Movement is controlled through contacts on the patient's shoulders.

Rotation. Lumbar segmental rotation is evaluated by establishing a thumb contact against the lateral surface of adjacent spinous

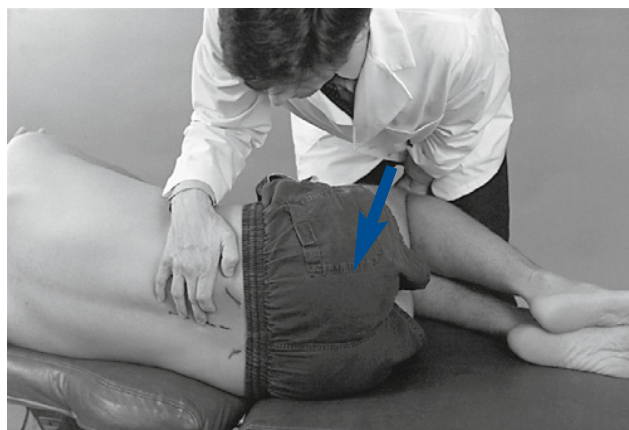


Figure 5-221 Side-posture evaluation for lumbar clinical instability, with the doctor applying an anterior-to-posterior force along the line of the shaft of the patient's femur while palpating for increased translational movement between L3 and L4.

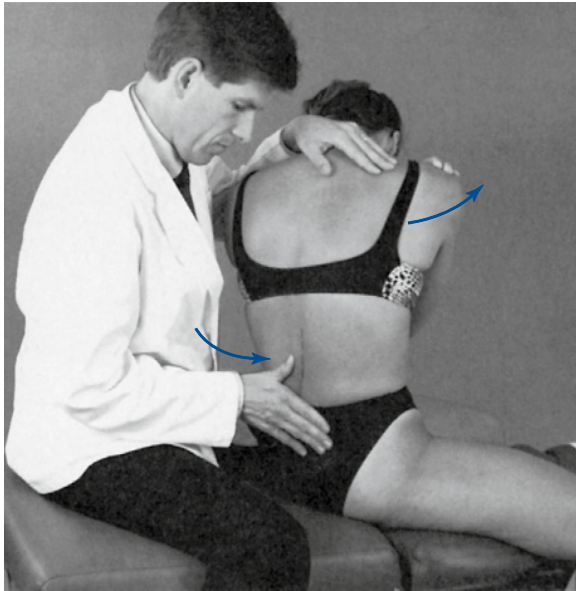
processes. The contact is placed on the side of induced rotation so that the pad spans the interspinous space. The support hand reaches around the front of the patient, hooks the patient's anterior shoulder or grasps the opposite forearm, and rotates the patient's trunk toward the side of contact (Figure 5-222). During normal rotation the doctor should palpate the superior spinous process, rotating away from the spinous process below. Movement should occur in the direction of trunk rotation. If separation is not noted and adjacent spinous processes move together, segmental restriction should be suspected.

Lumbar segmental movement is quite limited in rotation (1 to 2 degrees), and the examiner may not be able to discriminate reductions of movement within these limits. Therefore, the doctor should concentrate on movement of the segment from its neutral position (any shifting between adjacent spinous processes) and on the quality of end-play movement.

Although the lumbar facets do provide a bony obstacle to rotation, there is still some normal give to rotational end play. To assess end play, the doctor applies additional overpressure at the end of passive motion. Contacts may be established against the superior spinous process on the side of induced rotation (Figure 5-222) or over the mammillary process and posterior joint on the side opposite the induced rotation.

Lateral Flexion. To assess lateral flexion, the palpation contact is also established against the lateral surface of adjacent spinous processes. The contact is placed on the side of desired lateral flexion and the patient is asked to bend toward the side of contact (Figure 5-223). The doctor guides movement by applying downward and medial pressure through the shoulder contact and medial pressure through the contact hand. The indifferent arm and contact arm must work together to maximize bending at the site of palpation. To induce lateral flexion in the lumbar spine, the patient's upper trunk must be shifted sufficiently to the side of bending. During the performance of lateral flexion, the doctor should feel the spine bend smoothly around the contact and the spinous process shift toward the opposite side (convex side).

End-play evaluation is conducted by shifting the contact to the superior spinous process and applying additional overpressure at the end ROM. Lateral flexion end play is more elastic than rotation; a firm but giving response should be noted.



5-222

Figure 5-222 Segmental ROM and end-feel evaluation for left rotation at the L2–3 articulation, using a thumb contact across the left L2–3 interspace.



5-223

Figure 5-223 End-feel evaluation for right lateral flexion at the L2–3 motion segment, using a thumb contact across the right lateral aspect of the L2–3 interspace.

Flexion and Extension. Lumbar flexion and extension are evaluated by establishing palpation contacts in the interspinous spaces with the doctor's fingertips or dorsal middle phalanx of the index finger (Figure 5-224). Movement is induced by asking the patient to slouch and arch the lumbar spine. The indifferent arm and palpation hand help guide movement and ensure that the apex of bending is accentuated at the level of palpation. During flexion the interspinous spaces should open, and during extension they should approximate.

Flexion end play is assessed by contacting the superior spinous process with the thumb or fingertips while gentle downward pressure is applied through the doctor's IH and arm against the

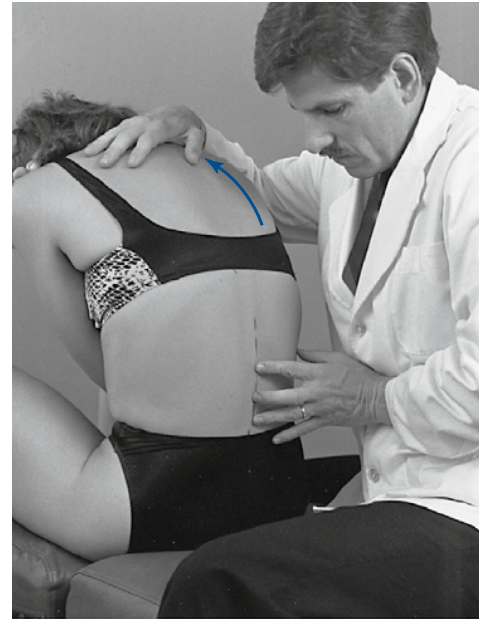


Figure 5-224 Movement evaluation of lumbar flexion, with fingertip contacts in the L2–3 and L3–4 interspace.

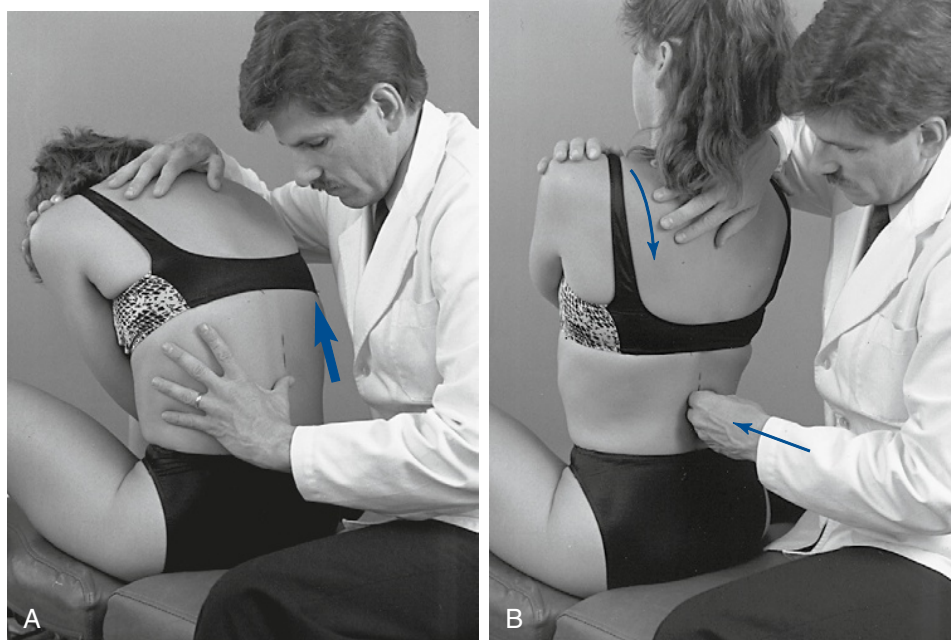
patient's shoulders (Figure 5-225, *A*). To induce extension, push anteriorly against the contact while inducing backward bending (see Figure 5-225, *B*). Extension end play is inhibited by the impact of the spinous processes and has a more rigid quality than flexion.

Side-Posture Methods. To conduct side-posture mobility tests, the patient is placed on his or her side, with the down-side arm crossed over the chest and resting on the opposite shoulder. The down-side leg is extended along the length of the table, and the knee and hip of the opposite leg are flexed. The doctor controls movement through the patient's shoulders and by cradling the patient's flexed leg.

Flexion and Extension. To evaluate flexion and extension, stand facing the patient and cradle the patient's flexed knee between your thighs. Palpate the interspinous space with the caudal hand and grasp the patient's shoulder with the cephalic hand and forearm. Induce flexion and extension by moving the patient's knees superiorly and inferiorly, and feel for opening and closing of the interspinous spaces (Figure 5-226).

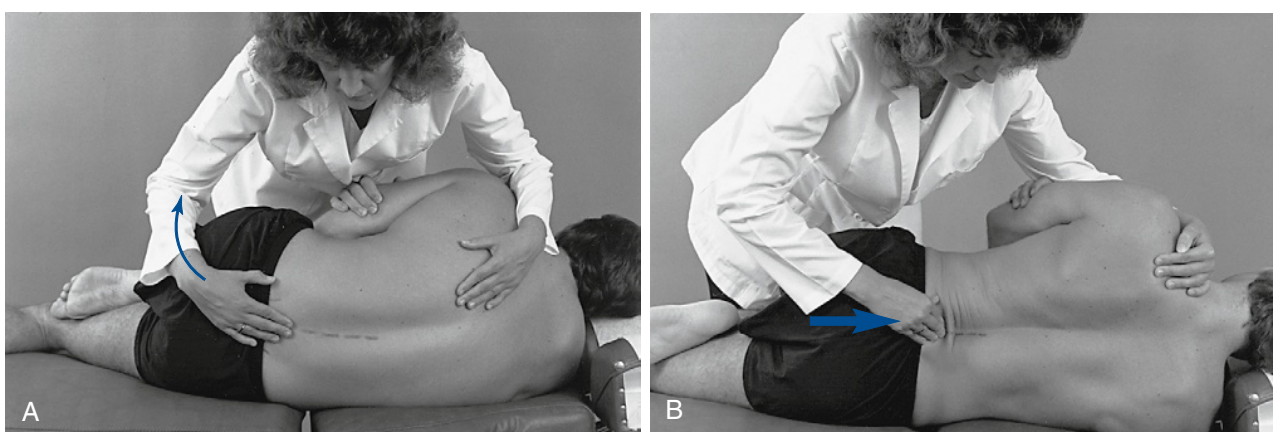
Lateral Flexion. To assess lateral flexion, use the caudal hand to establish a contact on the ischial tuberosity and gluteal soft tissues of the patient's up-side hip. Place the fingertips or thumb of the doctor's cephalic hand on either side of adjacent spinous processes and interspinous space. To induce movement, push headward against the patient's pelvis with the caudal hand while palpating for movement at the interspinous spaces (Figure 5-227). On the concave side of bending, the interspinous spaces should close and open on the convex side. If dysfunction is present, the spinous processes do not move into the doctor's palpating fingers on the convex side (down side) or remain firm and do not glide away if palpating is done from the concave side (up side).

Rotation. Side-posture lumbar rotation may be assessed by inducing movement through the patient's trunk or pelvis. If the patient's trunk is used, the indifferent contacts are established



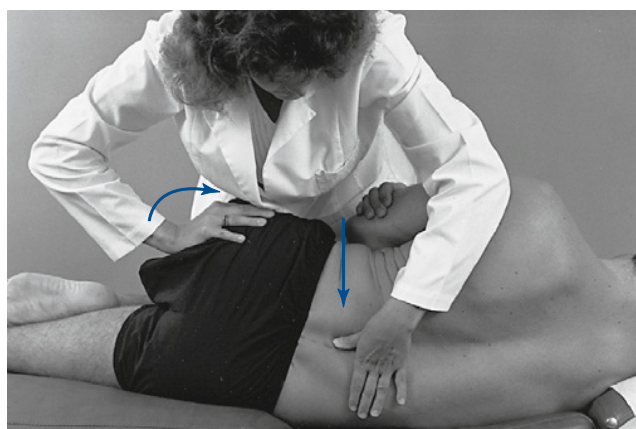
5-225A, B

Figure 5-225 **A**, End-play evaluation for flexion at the L2–3 motion segment, using a thumb contact over the inferior tip of the L2 spinous process. **B**, Lumbar extension end play, with a reinforced interphalangeal joint contact over the L2–3 interspace.



5-226B

Figure 5-226 **A**, Side-posture movement evaluation for flexion. **B**, Extension of the L4–5 motion segment, using a fingertip contact over the L4–5 interspace.



5-227

Figure 5-227 Side-posture movement evaluation for lateral flexion of the L2–3 motion segment, using a thumb contact over the lateral aspect of the L2–3 interspace.

against the patient's up-side shoulder (Figure 5-228). If the pelvis is used, the indifferent contacts are established against the patient's thigh or proximal leg. The patient's torso can be contacted with the doctor's superior hand or forearm (Figure 5-228, *A*). When counterrotating the shoulder and pelvis, slide your forearm between the patient's arm and chest, contact the shoulder, and have the patient grasp the opposite forearm with the up-side hand or the shoulder will not provide enough resistance to induce rotation (Figure 5-228, *B*). To induce movement through the pelvis, contact the patient's up-side thigh with the dorsal surface of the caudal ankle or distal thigh. For both methods, locate the palpation contacts at the desired interspinous space with the middle finger or thumb of the caudal hand.

To induce trunk movement, apply posterior pressure against the patient's shoulder and forearm. To induce pelvic rotation, apply downward pressure against the patient's distal thigh or proximal leg (5-228B). When movement is initiated through the trunk, the



5-228A

Figure 5-228 Side-posture movement evaluation of rotation at the L3–4 motion segment. **A**, Evaluation of right rotation mobility. Caudal hand establishes fingertip contacts over the L3–4 spinous process while the cephalad hand induces posterior trunk rotation. **B**, Evaluation of left rotation, using fingertip contacts over the left lateral surface of the L3 spinous process and right lateral surface of the L4 spinous process with counterrotation of shoulders and pelvis.

superior spinous process rotates away from the inferior spinous process in the direction of trunk rotation (toward the table). When movement is initiated with the pelvis, the inferior spinous process rotates away from the superior spinous process in the direction of pelvic rotation (away from the table).

ADJUSTMENTS OF THE LUMBAR SPINE

Side-Posture Adjustments

Side-posture lumbar adjustments are the most frequently applied adjustments for lumbar spinal dysfunction. They offer freedom of movement to alter patient position and adaptability to methods that improve the doctor's leverage and mechanical advantage. Although they are difficult adjustments to perfect, doctors can reduce frustration by understanding their mechanical principles and effects.

The level of segmental tension is regulated by patient positioning and the degree of induced lumbar flexion, lateral flexion, and the amount of counter-rotation induced between the shoulders and pelvis (Figure 5-229). The direction of adjustive thrust in relation to the direction of torso movement and pelvic movement is also important in localizing the adjustive.

Adjustments that direct the adjustive thrust in the same direction as torso movement but opposite pelvic movement are defined as *assisted adjustments*. Adjustments that direct the adjustive thrust in a direction opposite the torso movement and in the same direction as pelvic movement are defined as *resisted adjustments* (Figure 5-230). Assisted adjustments are applied to develop maximal preadjustive tension in the motion segments inferior to the established contact, and resisted adjustments are applied to develop maximal tension in the motion segments superior to the established contact.

The degree of torso movement is determined by the positioning of the patient's shoulders, and the degree of pelvic movement is determined by the placement of the patient's pelvis. Various

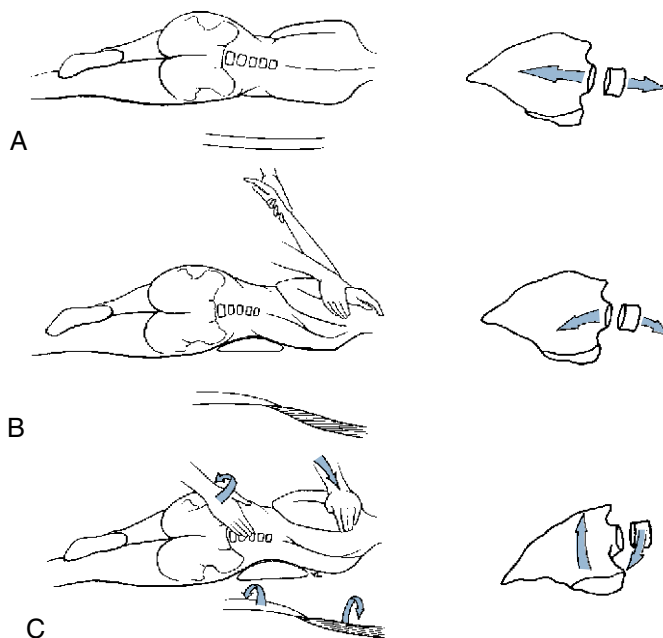


Figure 5-229 Side-posture patient positioning. **A**, Development of segmental flexion and distraction of the posterior joint by flexing the patient's upper knee and hip. **B**, Development of lateral flexion by placing a pillow under the lumbar spine and pulling the patient's shoulder down and forward. **C**, Counter-rotation of the pelvis and shoulders to induce gapping distraction in the up-side (left) facet joint.

methods for positioning and contacting the patient's shoulders and pelvis are pictured in Figures 5-231, 5-232, and 5-233.

Proper use of body weight and leverage is also critical to the effective application of side-posture adjusting. Side-posture adjustments often demand the added force that is produced by incorporating the doctor's body weight in the development of preadjustive tension and adjustive thrusts.⁶⁴

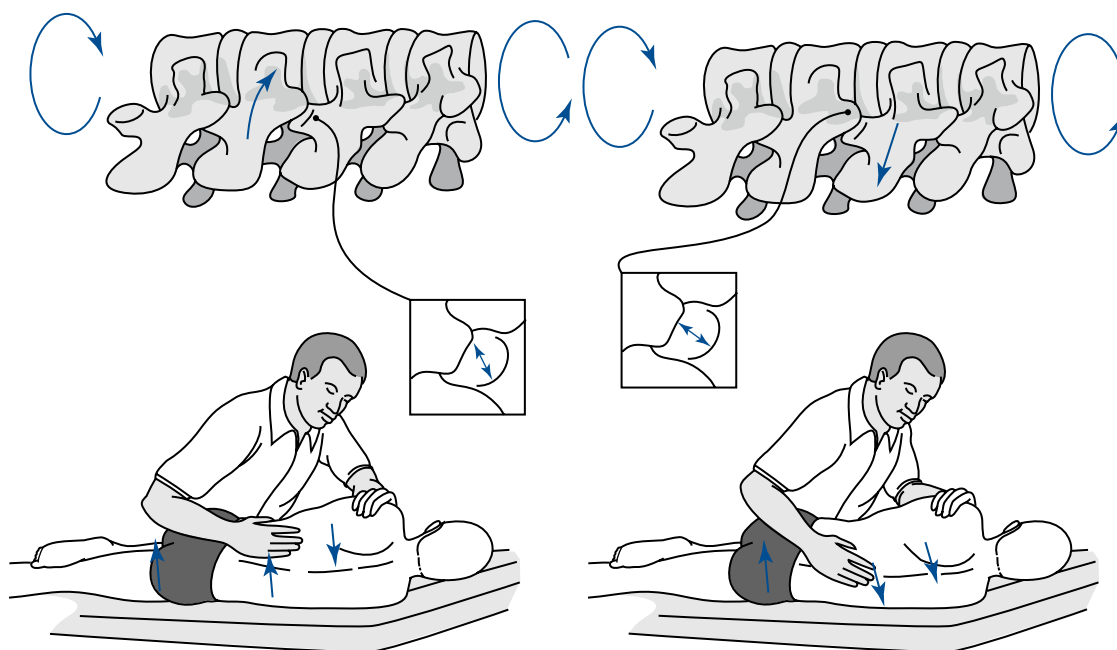


Figure 5-230 Side-posture positioning for rotational dysfunction. **A**, Resisted positioning, with contact applied to the inferior vertebra to induce gapping in the joints superior to the contact. **B**, Assisted positioning, with contact applied to the superior vertebra to induce gapping in the joints inferior to the contact. Arrows indicate adjustable vectors and direction of shoulder and pelvic rotations.

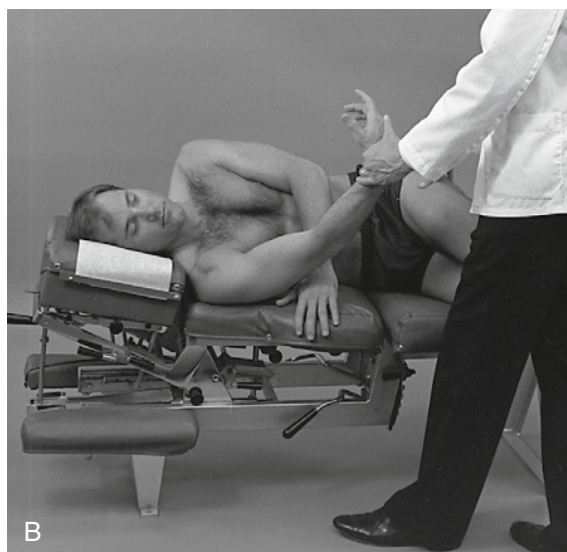
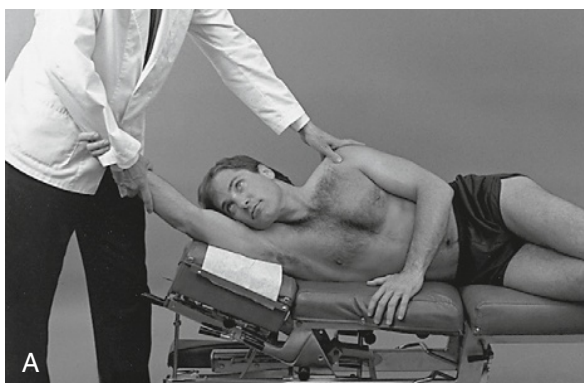


Figure 5-231 Movement of the patient's lower arm to assist in the development of lateral flexion. **A**, Lateral flexion away from the table, induced by pulling the arm headward to place the patient on the lateral surface of the shoulder to produce left lateral flexion (LLF). **B**, Lateral flexion toward the table, induced by pulling the arm footward to place the patient on the posterolateral surface of the shoulder to produce right lateral flexion (RLF). Elevation of the pelvic and lumbar sections or a pillow placed under the lumbar spine will assist in the development of lateral flexion toward the table.



Figure 5-232 Optional patient arm positioning for side-posture adjusting. **A**, Forward positioning of the upper arm applied with flexible individuals to maintain neutral trunk positioning. **B**, Midline positioning of the upper arm to accommodate neutral positions or positions incorporating slight posterior trunk rotation.

(Continued)

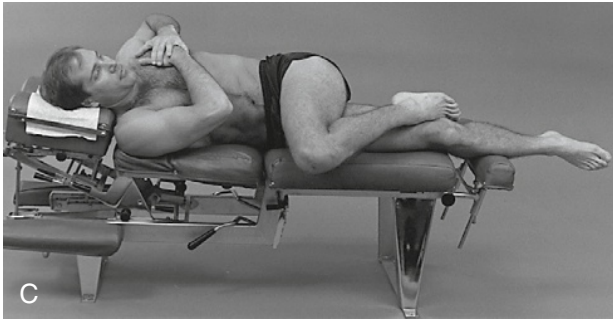
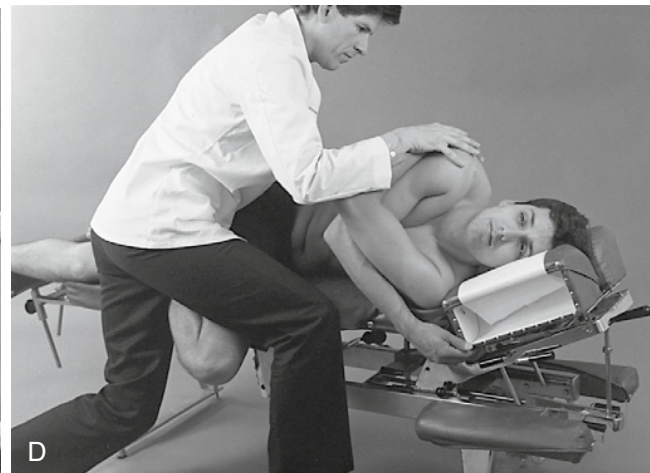
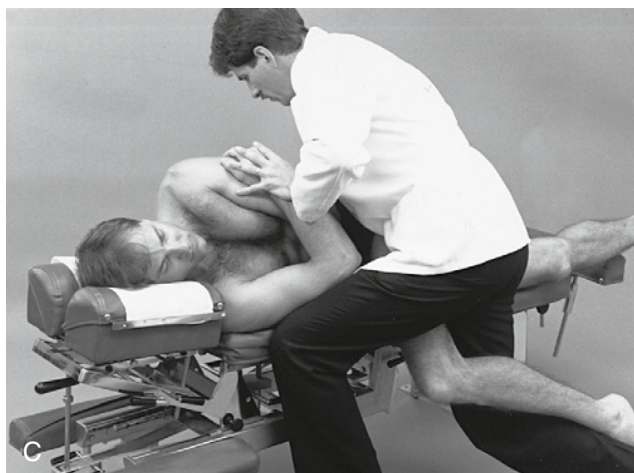
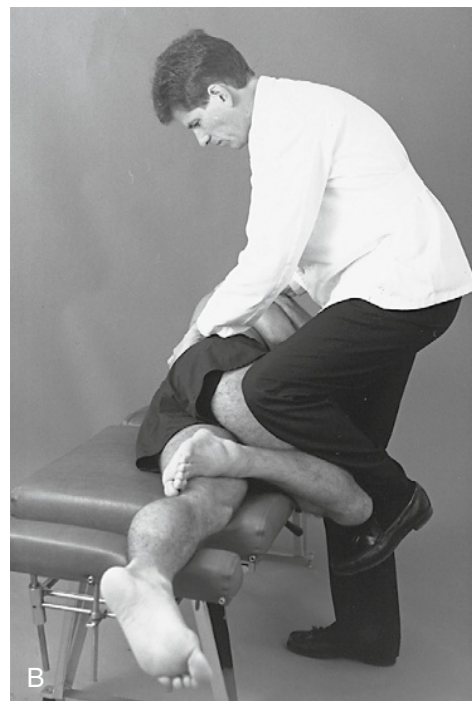


Figure 5-232—Cont'd C, Posterior positioning of the upper arm applied with large patients or specifically to induce posterior movement of the trunk.

Prone and Knee-Chest Adjustments

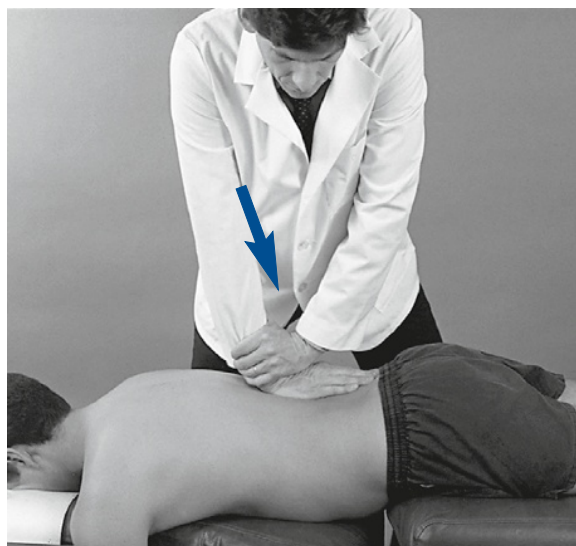
Prone and knee-chest adjustments are applied with specific short-lever contacts (Figure 5-234). They are especially suited to the treatment of extension restrictions or adjusting situations in which it is desirable to minimize rotation. Extension is easily induced in the prone or knee-chest positions, and the lumbar sagittal facet facings do not conflict with anteriorly directed adjustive VECs. The doctor also has the advantage of centering their body over the contact.

Because the knee-chest positions are especially helpful in maximizing lumbar extension, the patient is vulnerable to hyperextension in this position. Therefore, the doctor must be skilled in the application of this procedure and deliver the adjustive thrust in a shallow and nonrecoiling manner.



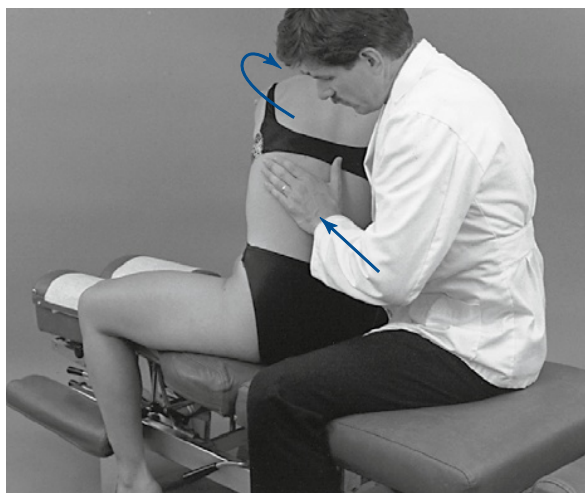
5-233A, B, C

Figure 5-233 Optional leg contacts that may be used in the application of side-posture pelvic and lumbar adjustments. A, Lateral thigh-to-thigh contact. B, Square stance shin-to-knee contact. C, Straddle thigh contact. D, Straddle flexed-knee contact.



5-234

Figure 5-234 Prone unilateral hypothenar mammillary adjustment.



5-235

Figure 5-235 Sitting mammillary push adjustment.

Sitting Lumbar Adjustments

Sitting lumbar adjustments (Figure 5-235) conform to the same mechanical principles previously discussed for sitting thoracic adjustments. They use assisted methods and are applied to develop maximal tension in the motion segments below the level of contact. They are typically applied for lumbar rotation or combined rotation and lateral flexion dysfunction. The most frequent and effective site of application occurs at the thoracolumbar region. Critical to their application is an understanding of the thoracolumbar transition to sagittal facet orientation and the effect this has on axial rotation and facet movements.

Rotational Adjustments

Rotational dysfunction of the lumbar spine theoretically may result from decreased mobility in the posterior joints on the side of rotational restriction or on the side opposite rotational restriction. Although the movements on each side are small, reduced play in

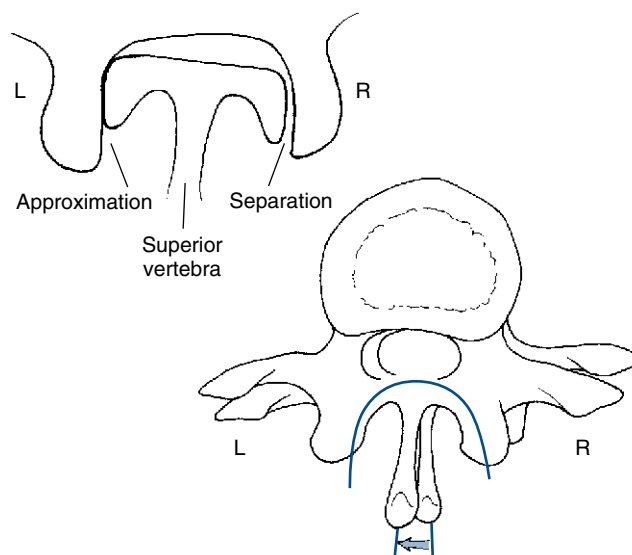


Figure 5-236 Illustration of right rotation, demonstrating gapping of the right articulation and anterior glide and approximation of the left articulation.

either joint is potentially detrimental to joint function. Fixation in the joint on the side of rotational restriction may produce a loss of facet separation (Figure 5-236). Fixation in the joint on the side opposite the rotational restriction (compression facets) may theoretically produce a loss of anterior glide of the inferior facet relative to the superior facet (see Figure 5-236). The facet joints on the side opposite the direction of trunk rotation act as a major barrier to axial rotation. Bony impact of these structures is largely responsible for limiting lumbar rotation. As a result, functional changes in the periarticular soft tissues of these joints are unlikely to significantly affect the range of axial rotation. In contrast, the joints on the side of trunk rotation are not limited by bony impact. Therefore, functional changes in these articulations may have a significantly greater potential to limit joint movement.

Rotational dysfunction leading to a loss of gapping on the side of restricted rotation may be treated with side-posture assisted or resisted methods. With both patient positions, the affected joint is placed up (away from the table) and the patient is flexed, laterally flexed toward the table, and counter-rotated at the level of dysfunction.

With resisted methods, the contact is established on the up-side mammillary process or the down-side of the spinous process of the inferior vertebra. The thrust is delivered in the direction opposite the shoulder rotation. This method is applied to induce rotation and gapping in the facet joints superior to the point of contact (Figure 5-237).

With assisted methods, the contact is established on the spinous process of the superior vertebra and the thrust is delivered in the direction of shoulder rotation. This method is also applied to induce facet gapping, but the point of distraction is directed to the articulations below the contact (Figure 5-238). To incorporate forces applied in each of the previous two methods, apply a combination spinous push-pull adjustment (Figure 5-239).

To induce rotation and facet gapping in the sitting position, use patient postures that are identical to those used with side-posture assisted spinous push adjustments. Contact the lateral surface of

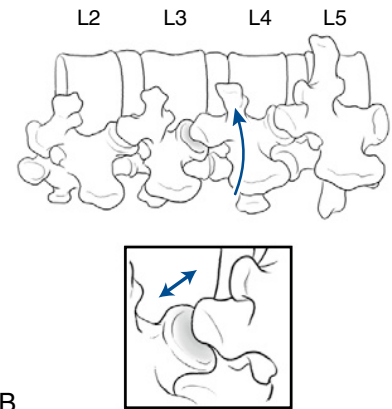
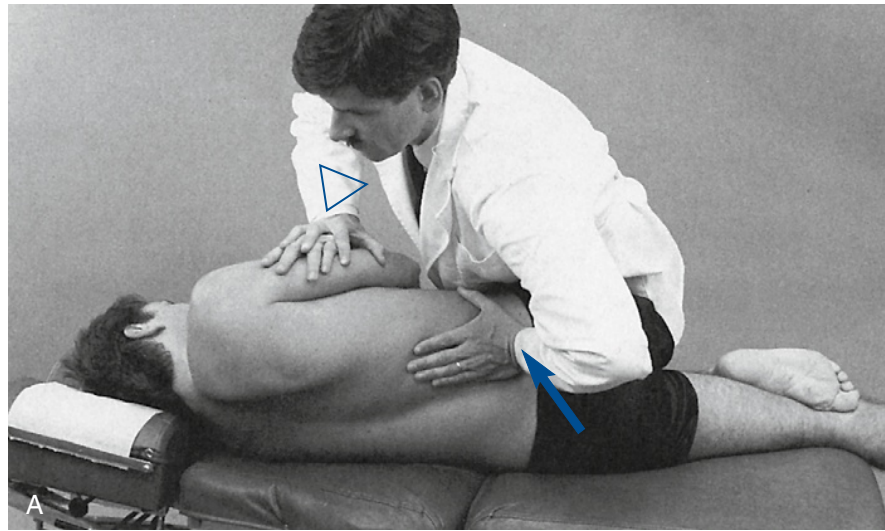


Figure 5-237 Resisted position with a hypothenar contact applied to the right L4 mammillary process to induce right rotation and gapping of the right L3–4 articulation.

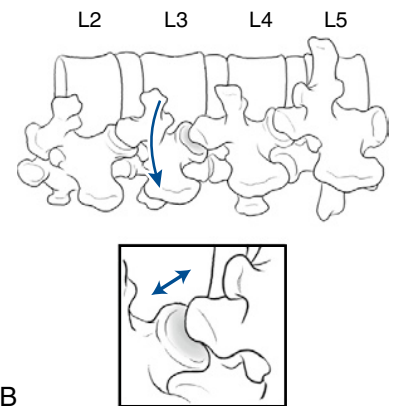
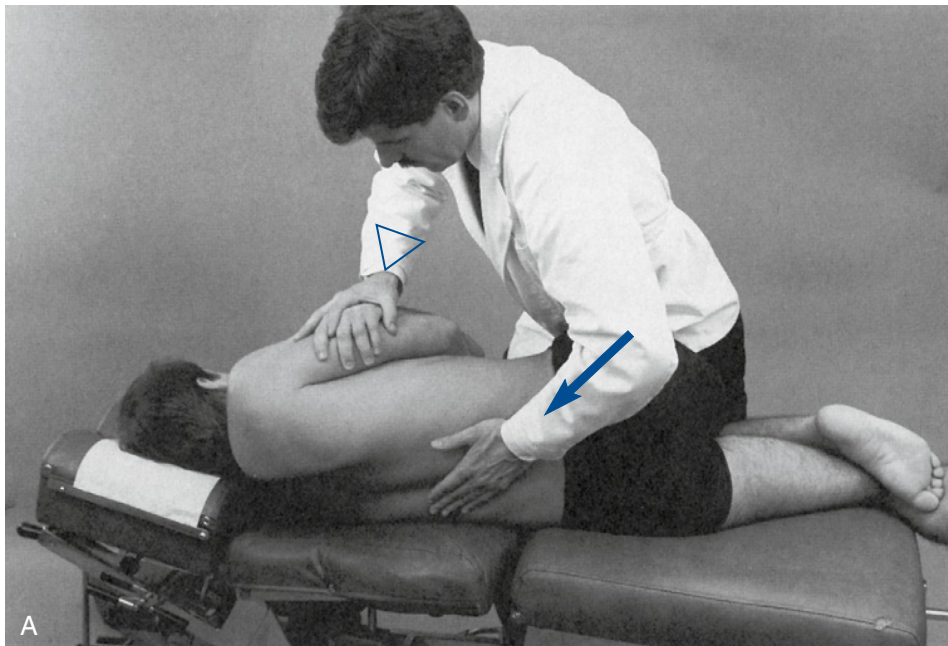


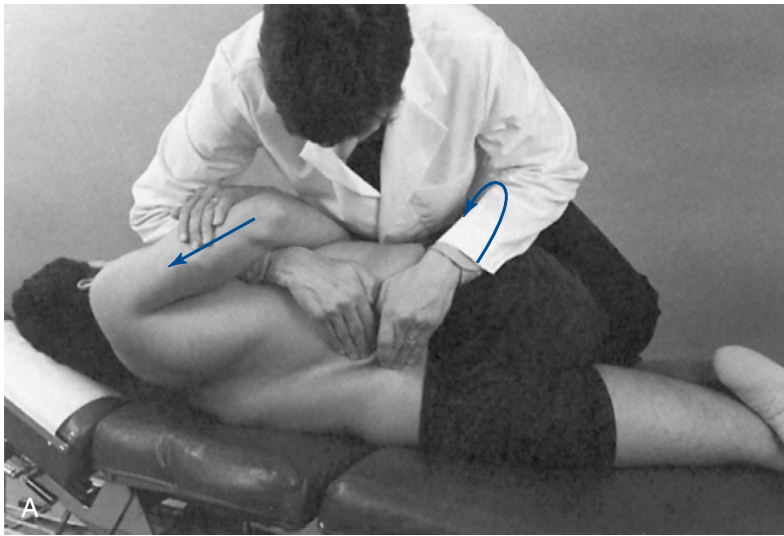
Figure 5-238 Assisted position, with a hypothenar contact applied to the right L3 spinous process to induce right rotation and gapping of the right L3–4 articulation.

the superior spinous process on the side of rotational restriction (side of spinous rotation) or over the mammillary process on the side opposite the rotational restriction. Laterally flex the patient away from the side of desired facet gapping and rotate the torso in the direction of restriction (Figure 5-240).

Rotational restrictions may also be treated with assisted side-posture mammillary push adjustments or assisted sitting mammillary push adjustments. The contacts are applied to the superior vertebra of the involved motion segment on the side of posterior body rotation (side opposite the rotational restriction). These methods are directed at distracting the motion segment inferior to the point of contact. These procedures establish a contact on

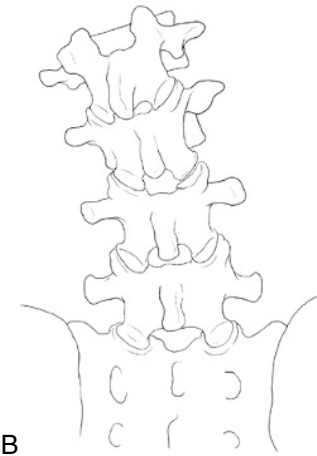
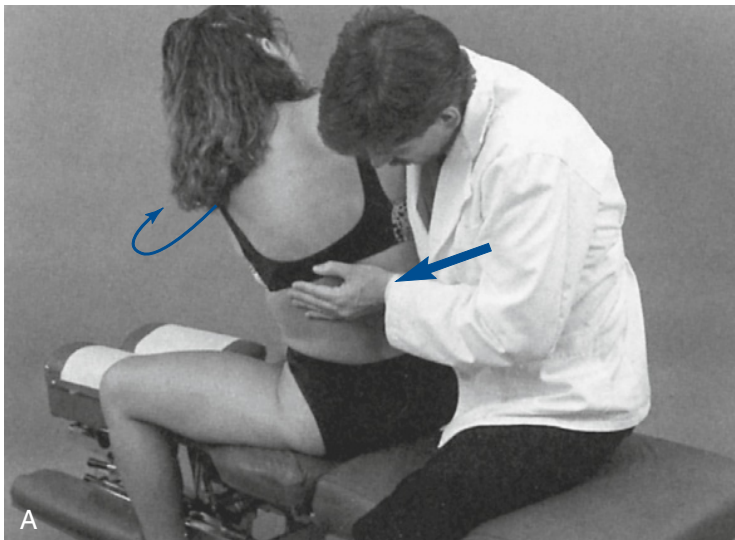
the side of facet approximation contralateral to the side of facet gapping. Therefore, they may be less effective at inducing joint gapping than the previous methods described, which establish contacts on the same side of the desired joint gapping.

In the side-posture method, take care to maintain the patient's relatively neutral shoulder position. Excessive posterior shoulder rotation opposes the direction of adjustive thrust and may place unwanted distractive tension at the joint above the desired level. In addition, consider incorporating a superior inclination to the adjustive thrust (Figure 5-241). This serves to direct the thrust along the plane of the lumbar sagittal facets and may minimize compression of the facet joints.



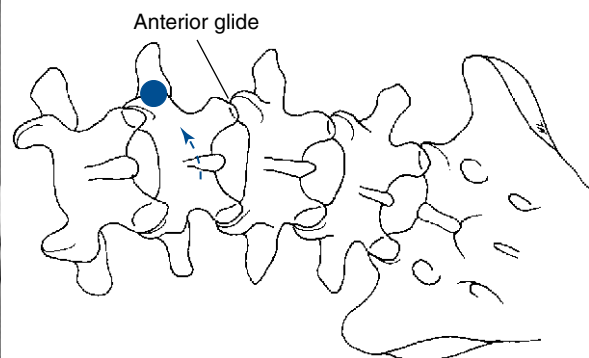
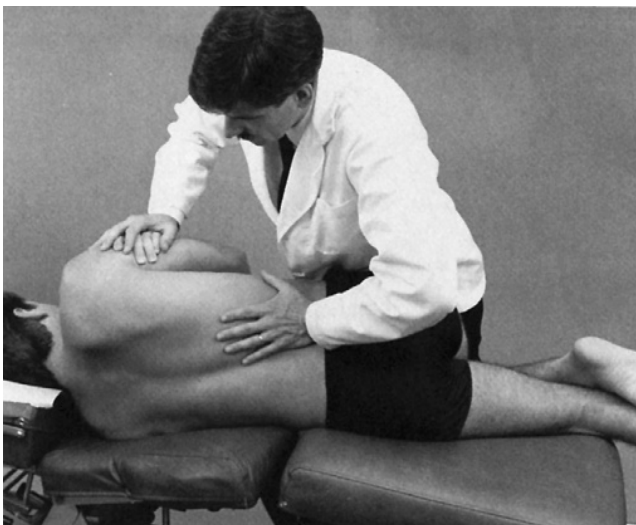
5-239

Figure 5-239 Digital contacts applied to the right lateral surface of the L3 spinous process and left lateral surface of the L4 spinous process to induce right rotation and gapping of the right L3–4 articulation.



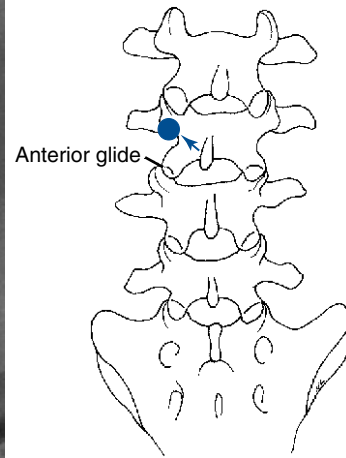
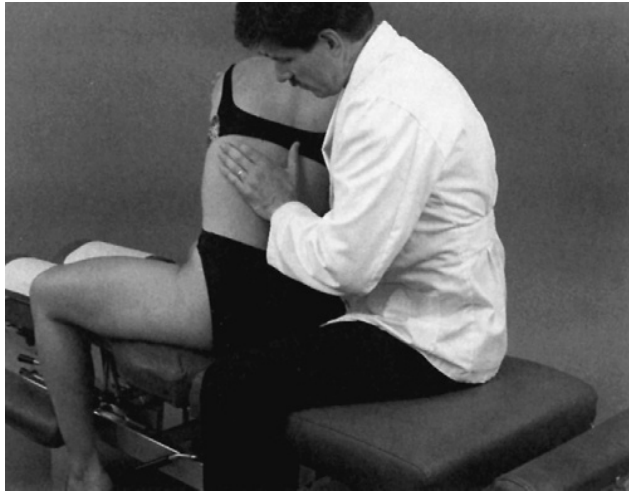
5-240

Figure 5-240 Assisted method, with a hypothenar contact applied to the right lateral surface of the L3 spinous process to induce right rotation and gapping of the right L3–4 articulation.



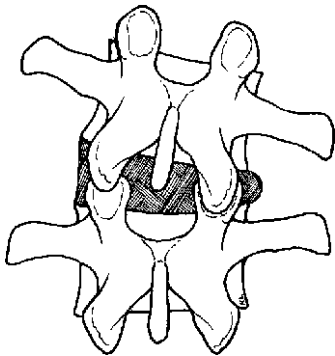
5-241

Figure 5-241 Assisted method, with a hypothenar contact applied to the right L3 mammillary process (dot) to induce left rotation.



5-242

Figure 5-242 Assisted method, with a hypothenar contact applied to the left L3 mammillary (*dot*) to induce right rotation or right lateral flexion.



5-243

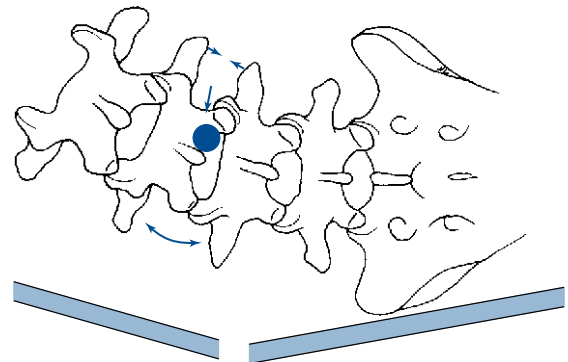
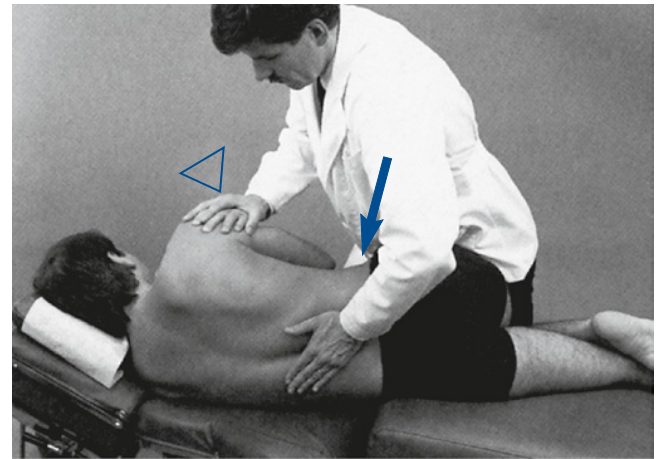
Figure 5-243 Illustration of right lateral flexion, demonstrating the right facet joints gliding together and the left facet joints gliding apart.

When using a sitting mammillary push adjustment, some lateral flexion of the patient away from the side of contact may assist in distraction the affected joint (Figure 5-242). If the patient is laterally flexed toward the side of contact and the adjustive thrust is directed anteriorly without superior distraction, there is a risk of unnecessary compression of the joint.

Lateral Flexion Adjustments

Lateral flexion dysfunction in the lumbar spine may result from a loss of disc closure and approximation of the facet joints on the side of lateral flexion dysfunction or contralateral loss of distraction of the facet and soft tissues on the side opposite the lateral flexion restriction (Figure 5-243).

Lateral flexion dysfunction is commonly treated with side-posture adjustments, but may also be treated in sitting positions. In side-posture positions, the adjustive contacts are established on the spinous process or the mammillary processes. Assisted patient positions are used with spinous process contacts with pillows or positioning of the lumbar and pelvic sections to enhance the lateral flexion position. The patient is placed on the side opposite the lateral flexion restriction and the patient is laterally flexed away from the table (laterally flexed toward the doctor) (Figure 5-244). After tension is developed, the doctor thrusts



5-244

Figure 5-244 Assisted method, with a hypothenar contact applied to the right lateral surface of the L3 spinous process (*dot*) to induce right lateral flexion of the L3–4 motion segment.

posteriorly to anteriorly and laterally to medially to induce segmental lateral flexion and closure of the up-side facet joint and disc and separation of the down-side facets and disc.

When using mammillary contacts, the patient is placed with the side of lateral flexion restriction down. The patient is laterally flexed toward the table to distract the up-side joint. A roll

may be placed under the patient to assist in the development of distraction. The contacts are established on either the upper or lower vertebra. With a contact on the superior vertebra, the thrust is delivered anteriorly and superiorly in the direction of trunk bending to distract the joints below the contact (Figure 5-245). With an inferior vertebra contact, the thrust is directed anteriorly and inferiorly to distract the joints above the contact. Upper vertebra contacts are much more commonly applied and easier on the doctor's wrist and shoulder.

When mammillary contacts are applied to accentuate disc closure, the patient is placed with the side of lateral flexion restriction up. The patient is laterally flexed away from the table, and the doctor thrusts posteriorly to anteriorly, laterally to medially, and superiorly to inferiorly to induce approximation of the facets and disc closure (Figure 5-246). This procedure is not commonly used unless lateral flexion restrictions are also coupled with opposite-side rotation restrictions (posterior right inferior [PRI] or posterior left inferior [PLI] listings).

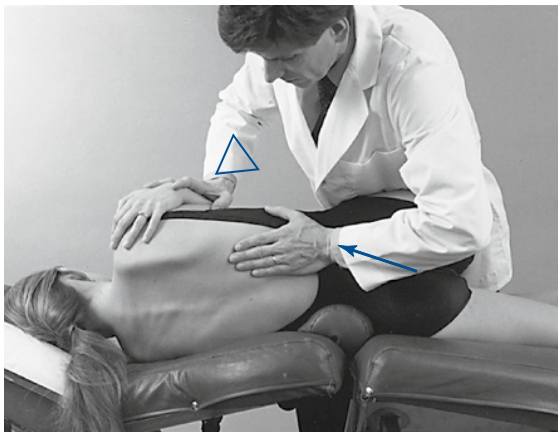
It is unlikely that adjustive VECs directed up or down the spine in a patient positioned in a neutral position will induce lateral flexion.³² The production of lateral flexion is more likely if

the patient is prestressed and allowed to move in the direction of lateral flexion restriction (Figure 5-246). The use of rolls, wedges, and articulating tables can assist in this capacity.

Flexion and Extension Adjustments

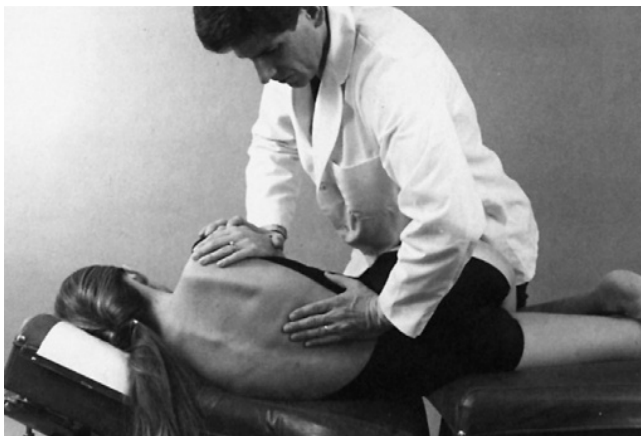
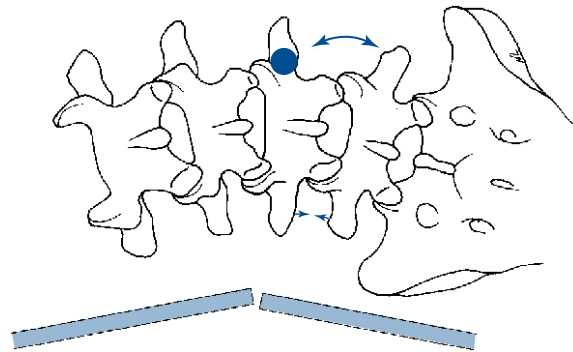
Flexion dysfunction is commonly treated in the side-posture position, with contacts established over the spinous processes. The spine is flexed at the level of dysfunction, and contacts are applied to the superior or inferior vertebra of the involved motion segment to induce separation of the interspinous space and posterior joints. With assisted methods, the contact is established on the superior vertebra, and the thrust is directed anteriorly and superiorly (Figure 5-247). With resisted methods, the contact is applied to the inferior vertebra and the thrust is directed anterior and inferiorly. Resisted methods are not commonly applied, except at the lumbosacral junction, where the contacts are established on the sacral apex.

Extension dysfunction is also treated in side-posture position with spinous contacts. In this case, the involved motion segment is allowed to extend, and the doctor establishes contacts over the superior or inferior spinous process and directs the adjustive thrust to induce approximation of the posterior joints and disc and



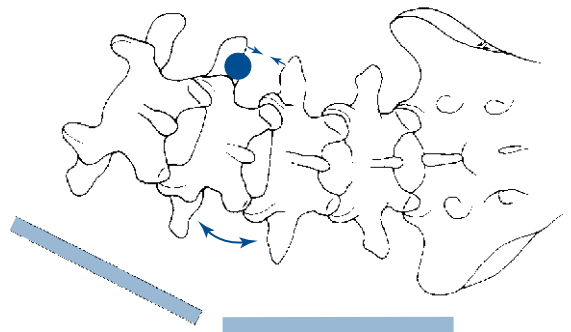
5-245

Figure 5-245 Assisted method, with a hypothenar contact applied to the right L4 mammillary process (*dot*) to induce left lateral flexion of the right L4-5 motion segment.



5-246

Figure 5-246 Assisted method, with a hypothenar contact applied to the right L3 mammillary (*dot*) to induce right lateral flexion of the L3-4 motion segment.



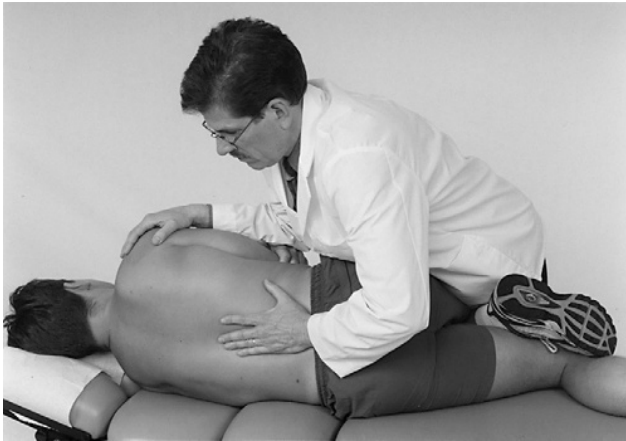


Figure 5-247 Assisted method, with hypothenar contact applied over the L5 spinous process to induce distraction in the L5–S1 motion segment.

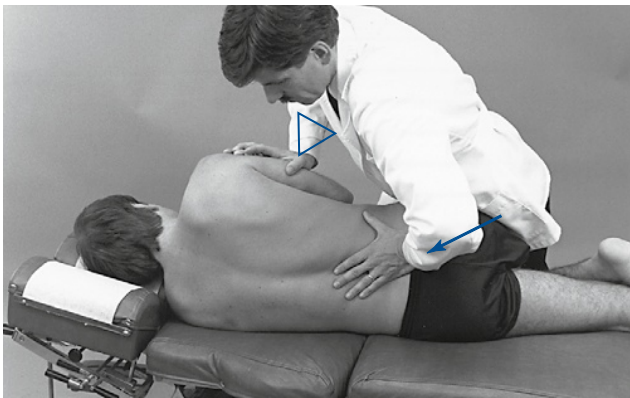
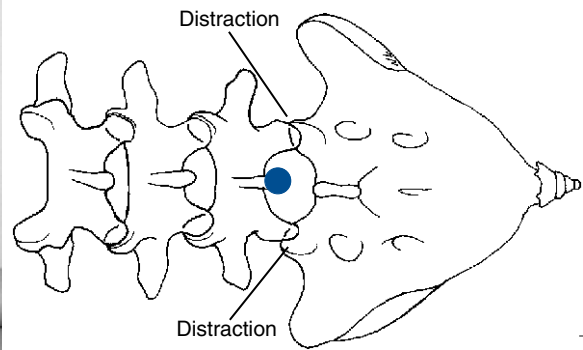
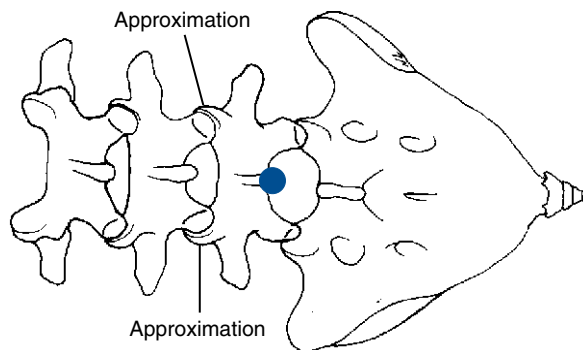


Figure 5-248 Resisted method, with a hypothenar contact applied to the L5 spinous process (*dot*) to induce extension in the L4–5 motion segment.



distraction of the anterior elements (Figure 5-248). The resisted method may be the procedure of choice because of the associated slight I-S VEC that minimizes stress to the doctor's shoulder.

It is unlikely that adjustive VECs directed up or down the spine in a patient positioned in a neutral position will induce flexion or extension.³² The production of flexion or extension is more likely if the patient is prestressed and allowed to move in the direction of restriction.

As mentioned previously, prone or knee-chest adjustments may be especially efficient in the delivery of lumbar extension adjustments. They are applied with unilateral or bilateral contacts, depending on whether you wish to induce extension with or without coupled rotation. The contacts may be established over the spinous processes but are more commonly applied over the mammillary processes.

LUMBAR ADJUSTMENTS (BOX 5-9)

Side Posture

Hypothenar/Mammillary Push (Figure 5-249)

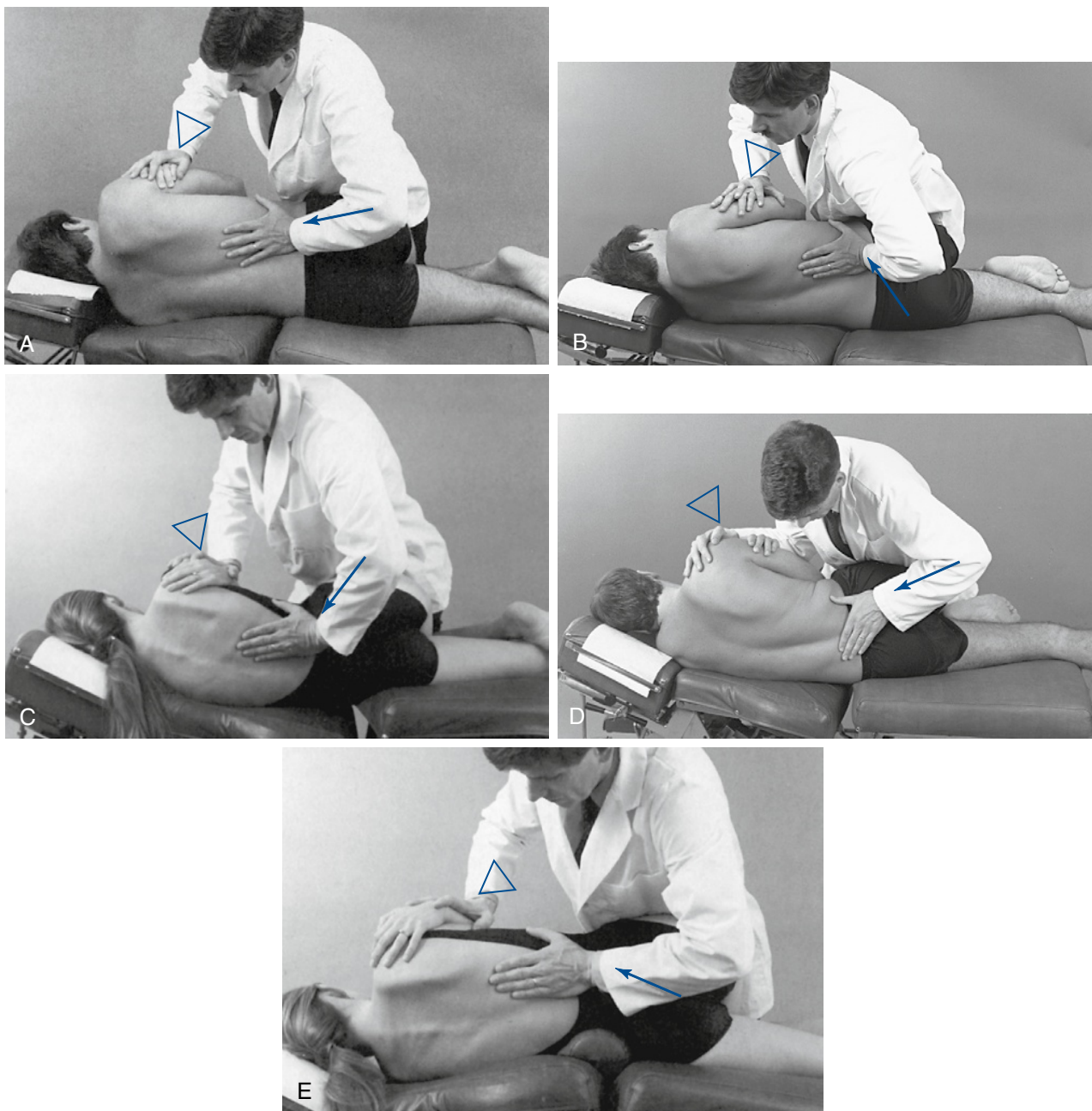
IND: Restricted rotation or lateral flexion, L1–L5. Rotation or lateral flexion malpositions, L1–L5.

PP: The patient lies in side posture, with the head supported on the elevated cervical section or pillow. The patient's down-side arm is crossed over the chest, with the hand resting on the

BOX 5-9 Lumbar Adjustments

- Side posture
 - Hypothenar/mammillary push (Figure 5-249)
 - Hypothenar/spinous push (Figure 5-250)
 - Hypothenar/spinous pull (Figure 5-251)
 - Digit/spinous push-pull (Figure 5-252)
- Prone
 - Bilateral thenar/mammillary push (Figure 5-253)
 - Hypothenar/mammillary push (Figure 5-254)
 - Hypothenar/spinous push (Figure 5-255)
- Knee-chest
 - Hypothenar/spinous push (Figure 5-256)
 - Bilateral thenar/mammillary push (Figure 5-257)
 - Hypothenar/mammillary push (Figure 5-258)
- Sitting
 - Hypothenar/mammillary or spinous push (Figure 5-259)

opposite shoulder or lateral rib cage. The patient's down-side leg is extended along the length of the table, and the upper leg and thigh are flexed. The patient's foot is placed over the popliteal space of the down-side leg. Optional arm and leg positions are covered in Figures 5-231 through 5-233.



5-249A, B, C, E

Figure 5-249 **A**, Assisted method, with a hypothenar contact applied to the right L4 mammillary process to induce left rotation at the right L4–5 motion segment. **B**, Resisted method, with a hypothenar contact applied to the right L4 mammillary process to induce right rotation and gapping of the right L3–4 articulation. **C**, Assisted method, with a hypothenar contact applied to the right L4 mammillary process to induce right lateral flexion of the left L4–5 motion segment. **D**, Resisted method, with a hypothenar contact applied to the right sacral base to induce right lateral flexion in the L5–S1 motion segment. **E**, Assisted method, with a hypothenar contact applied to the right L3 mammillary process to induce left lateral flexion in the L3–4 motion segment.

DP: Stand in a fencer stance, angled approximately 45 degrees to the patient. Support the patient's pelvis by contacting the patient's thigh with your caudad thigh (see Figure 5-233, *A*) or by straddling the patient's flexed leg between your thighs (see Figure 5-233, *C* and *D*).

CP: Hypothenar (pisiform) of the caudal hand, with the fingers running parallel to the spine.

SCP: Mammillary process.

IH: The patient's up-side shoulder and overlapping hand.

VEC: Descriptions of the adjustive VECs follow.

P: Ask the patient to lie on the appropriate side and to straighten the down leg. Position the patient's shoulders and flex the upper thigh to distract the interspinous space of the dysfunctional motion segment. Establish the vertebral and thigh contacts and develop preadjustive tension. At tension, generate an impulse thrust by dropping your body weight and thrusting through the shoulder.

Rotation restrictions: Assisted method: To induce rotation from a neutral position, place the patient on the side opposite the posterior body rotation (side of rotational restriction), and minimize rotation between the patient's shoulders and pelvis. Establish

the segmental contact by sliding laterally over the superior vertebra on the side of posterior body rotation. For example, if the L4 body is rotated posteriorly on the right (left rotation restriction), the contact should be established over the right L4 mammillary (see Figure 5-249, *A*). At tension, deliver an impulse thrust anteriorly and superiorly, parallel to lumbar facet planes.

Resisted method: To effect rotation with a resisted method, place the patient on the side opposite the rotational restriction and contact the mammillary of the inferior vertebra on the side of rotational restriction. For example, when treating an L3–4 right rotation restriction (L3 left posterior body rotation), place the patient on the left side and establish a contact over the right L4 mammillary (see Figure 5-249, *B*).

To develop preadjustive tension, rotate the patient's shoulders posteriorly, laterally flex the trunk toward the adjusting bench, and counter-rotate the patient's pelvis anteriorly. This should induce gapping distraction in the motion segment ipsilateral superior to the point of contact.

Shoulder rotation and lateral flexion are induced by pulling the patient's down-side arm anteriorly and inferiorly (see Figure 5-231, *B* and *C*). Lateral flexion of the patient may be assisted by elevating the thoracolumbar section of an articulating adjustive bench or by placing a roll under the patient's lumbar spine. Forward rotation of the patient's pelvis is aided by downward traction along the patient's flexed thigh and hip by the doctor's anterolateral thigh (see Figure 5-223, *A*) or lower abdomen (see Figure 5-233, *C* and *D*).

The degree of shoulder rotation depends on the area being treated. It is greater in the upper lumbar spine as compared with the lower lumbar spine. Excessive counter-rotation through the shoulders may be unnecessarily uncomfortable to the patient and may be stressful to his or her intercostal muscles.

At tension, deliver an impulse thrust through the body and contact arm. The adjustive VEC follows the movement of the pelvis and should not be directed straight anteriorly. Thrusts directed anteriorly have a tendency to induce segmental extension instead of rotation and joint gapping. The contact must remain light, and the VEC must reinforce the pelvic rotation by incorporating a strong M-L component (see Figure 5-249, *B*).

Resisted adjustive methods may also be used to treat rotational restriction coupled with opposite-side lateral flexion restrictions ([PRI], [PLI], left posterior superior [LPS], right posterior superior [RPS] listings).

Lateral flexion restrictions: Mammillary push adjustments for lateral flexion dysfunction are commonly treated with assisted patient positions. The patient is laterally flexed in the direction of restriction, with the contact established on the superior or inferior vertebra.

To maximize forces of approximation, contact the side of lateral flexion restriction (see Figure 5-249, *C* and *D*). To maximize forces of distraction, contact the side opposite the lateral flexion restriction (see Figure 5-249, *E*).

When contacting the open-wedge side, place the patient on the side opposite the lateral flexion restriction and laterally flex the patient away from the table. Lateral flexion is induced by elevating the patient's shoulders and pulling headward on the down-side arm (see Figure 5-232, *A*). The thoracolumbar section of an articulating adjustive bench may be lowered and released to aid in the production of lateral bending (see Figure 5-249, *C*

and *D*). Establish the contact on the superior vertebra or on the inferior vertebra. At tension, deliver an impulse thrust anteriorly, medially, and inferiorly with a superior vertebral contact (see Figure 5-249, *C*) and anteriorly, medially, and superiorly with an inferior vertebral contact (see Figure 5-249, *D*). Simultaneously gently pulling down on the patient's shoulder with IH contact effectively assists in the production of lateral flexion (see Figure 5-249, *D*). This superior vertebral contact method is commonly applied in the Gonstead technique to treat rotational restrictions coupled with opposite-side lateral flexion restrictions (PRI, PLI listings).

When contacting the closed-wedge side, place the patient on the side of lateral flexion restriction and laterally flex the patient toward the table. The thoracolumbar section of the adjustive bench may be elevated or a roll may be placed under the patient's lumbar spine to assist in the development of lateral flexion (see Figure 5-249, *E*). Deliver the adjustive thrust superiorly and posterior to anterior.

Hypothenar/Spinous Push (Figure 5-250)

IND: Restricted flexion, extension, rotation, and lateral flexion, L1–S1. Rotation, flexion, extension, and lateral flexion malpositions, L1–S1.

PP: The patient lies in the basic side-posture position.

DP: Stand in a fencer stance, angled approximately 45 degrees to the patient. Support the patient's pelvis by contacting the patient's thigh with your caudad thigh.

CP: Hypothenar of inferior hand, with fingers angled across the spine.

SCP: Lateral margin of the superior spinous process.

IH: The patient's up-side shoulder and overlapping hand.

VEC: L-M and P-A.

P: Ask the patient to lie on the appropriate side and to straighten the down-side leg. Position the patient's shoulders and flex the upper thigh to distract the interspinous space of the dysfunctional motion segment. Establish the vertebral and thigh contacts and develop preadjustive tension. Generate the spinous push adjustive thrust by dropping your body weight while thrusting through the shoulder. This adjustment is typically delivered with a nonpause thrust. Spinous push adjustments use assisted patient positions for rotation and lateral flexion restrictions and assisted or resisted positions for flexion or extension restrictions.

Rotation restrictions: To effect rotation, place the patient on the side opposite the rotation restriction (side opposite spinous rotation). Rotate the shoulder posteriorly in the direction of restriction and laterally flex the trunk toward the adjusting bench. Induce shoulder rotation and lateral flexion by pulling the patient's down-side arm anteroinferiorly (see Figure 5-250, *A*). Establish the segmental contact and remove superficial tissue slack by sliding medially onto the lateral surface of the superior spinous process (side of spinous rotation). The contact must remain light and well padded or it becomes painful.

Develop preadjustive tension by rotating the patient's shoulder and contacted vertebra posteriorly while the patient's pelvis and the segments below are rotated anteriorly. The forward rotation of the patient's pelvis is aided by downward traction along the patient's flexed thigh and hip by your anterolateral thigh. At tension, deliver an impulse thrust through the body and contact arm.

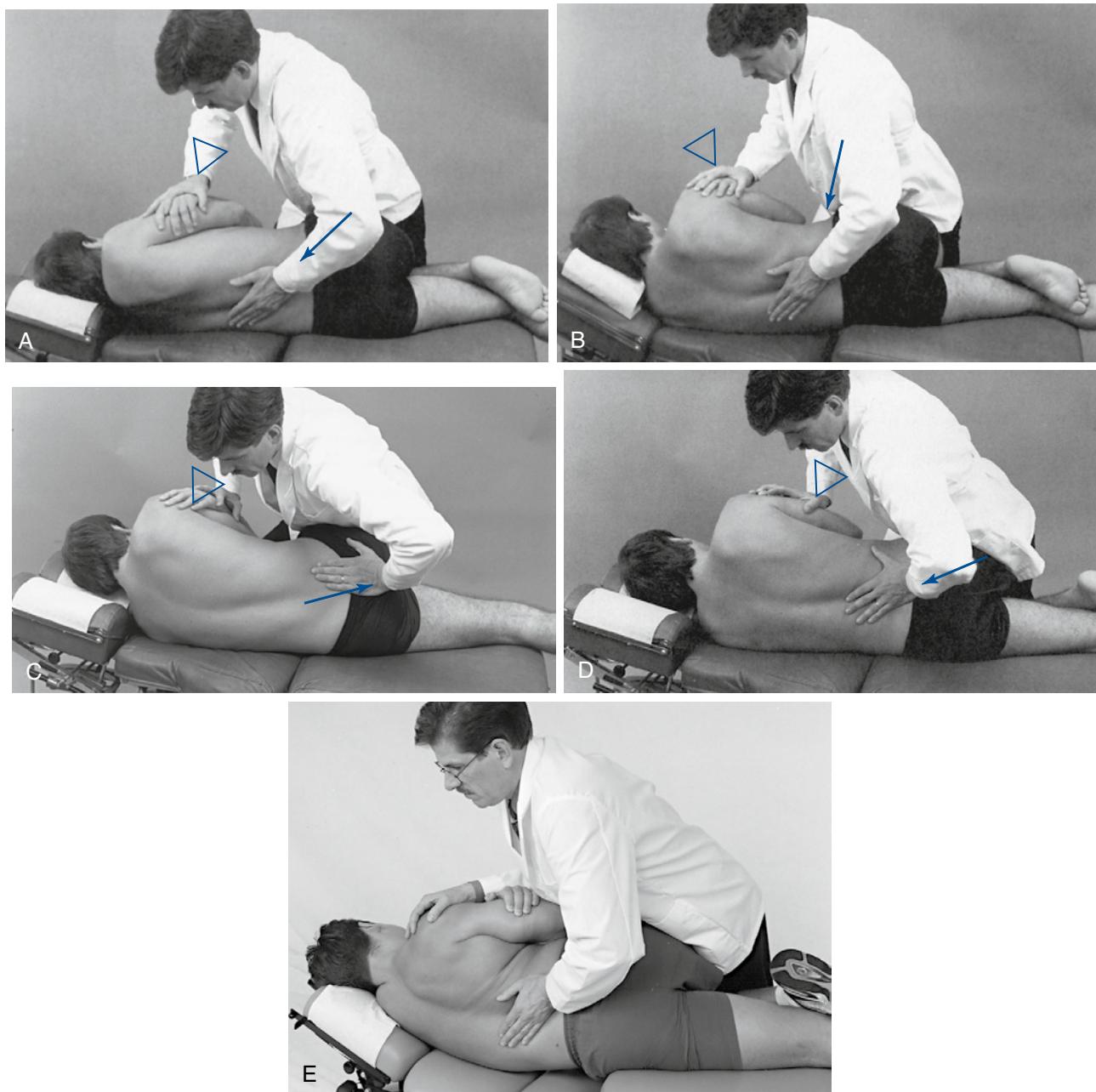


Figure 5-250 **A**, Assisted method, with a hypothenar contact applied to the right lateral surface of the L3 spinous process to induce right rotation in L3- motion segment. **B**, Assisted method, with a hypothenar contact applied to the right lateral surface of the L3 spinous process to induce right lateral flexion in the L3–4 motion segment. **C**, Resisted method, with a hypothenar contact applied to the sacral apex to induce flexion in the L5–S1 motion segment. **D**, Resisted method, with a hypothenar contact applied to the spinous process of L5 to induce extension in the L4–5 motion segment. **E**, Assisted spinous push adjustment, with contact established over the right lamina of the L4 spinous process to induce L4–5 right rotation and right lateral flexion.

Lateral flexion restrictions: When treating pure lateral flexion dysfunction, place the patient on the side opposite the lateral flexion restriction and laterally flex the patient away from the table. Induce lateral flexion by pulling headward on the patient's down-side shoulder. Placing a triangular pillow under the patient's head and shoulder or placing a patient on an articulating bench and lowering and releasing the thoracolumbar section may aid lateral flexion positioning (Figure 5-250, *B*).

Establish the adjustive contact and remove superficial tissue slack by sliding medially onto the lateral surface of the superior

spinous process. Develop preadjustive tension by transferring additional weight through your trunk into the spinous contact. At tension, deliver an impulse thrust through the body and contact arm. Simultaneously gently pulling down on the patient's shoulder with IH contact effectively assists in the production of lateral flexion (Figure 5-250, *E*).

Flexion restrictions: To treat flexion dysfunction, place the patient on either side and induce flexion in the lumbar spine. Establish the segmental contact with the proximal midline calconal or hypothenar contact on your caudal hand. When using an

assisted method, slide superiorly to contact the inferior tip of the superior spinous process and thrust superiorly and anteriorly.

When using a resisted method, slide inferiorly to contact the inferior vertebra and thrust anteroinferiorly. The resisted approach is commonly applied only when treating lumbosacral flexion restriction with a sacral contact (see Figure 5-250, C).

Extension restrictions: To treat extension dysfunction, use the same contacts as described for flexion dysfunction, but allow the lumbar spine to move into extension. With an assisted approach, contact the superior vertebra and thrust anteroinferiorly. With a resisted approach, contact the inferior vertebra and thrust anteriorly and superiorly.

To induce extension at the lumbosacral junction with an inferior vertebral contact, contact the sacral base at the level of S1 and thrust anteriorly and slightly superiorly (see Figure 5-250, D).

Combined same-side rotation and lateral flexion restriction: Assisted spinous push adjustments are commonly used for treating restrictions in rotation and same-side lateral flexion (PRS, PLS listings). The PP and thrust VEC are identical to those used in treating lateral flexion restrictions, with the exception that the patient's shoulder is rotated in the direction of restriction (see Figure 5-250, E).

Hypothenar/Spinous Pull (Figure 5-251)

IND: Restricted rotation or combined restrictions in rotation and opposite-side lateral flexion, L1–L5. Rotation or combined rotation and ipsilateral flexion malpositions, L1–L5.

PP: The patient lies in the basic side-posture position, with the foot of the patient's flexed leg hooked behind the popliteal space of the down-side leg.

DP: Square stance: Stand facing the patient and contact the patient's knee with either your knee or the distal surface of your leg (Figure 5-251, A).

Fencer stance: Stand facing the patient at a 45-degree angle. Straddle the patient's flexed knee and contact his or her mid-shin with your thigh (see Figure 5-251, B).

CP: Fingertips (digital contacts) of first three fingers of your inferior hand, with the forearm resting along the patient's posterolateral buttock and hip.

SCP: Lateral surface of the spinous process.

IH: Your IH contacts the patient's up-side shoulder and overlapping hand.

VEC: L-M pulling movement to induce axial rotation.

P: Ask the patient to lie on the appropriate side and to straighten the down-side leg. Flex the patient's upper thigh to distract the interspinous space of the dysfunctional motion segment. Then establish contacts on the spinous process, lateral hip and the patient's flexed leg. The spinous contacts are established by hooking the down side of the spinous process with the second, third, and fourth fingers while the forearm rests against the patient's posterolateral buttock and hip.

When using a square stance, contacts on the patient's leg are established with the doctor's knee or distal shin (Figure 5-251, A). When using a fencer stance, the contacts are established on the patient's leg with the doctor's mid-thigh (Figure 5-251, B).

At tension, a pulling impulse is generated by extending your contact shoulder while simultaneously inducing anterior pelvic rotation by accelerating through the patient's leg contact. To generate anterior pelvic rotation with a square stance and distal shin-to-patient knee contact, quickly extend the contact knee (Figure 5-251, A). With a knee-to-knee contact, extend your hip and drop your body weight through the patient's knee and forearm contact on the patient's lateral hip. With a fencer stance and thigh-to-patient's shin contact, generate rotation by accelerating your body weight through the contacts on the patient's lateral hip and shin (Figure 5-251, B). The digit contacts on the spinous are primarily used for sensing preadjustive tension. The primary adjustive force is generated through the leveraged contacts established on the patient's lateral thigh and lower extremity.

Rotation restriction: This adjustment may be delivered with an assisted or resisted method. Resisted methods that induce counter-rotation are likely more effective in developing rotational tension and gapping.^{65,66}

Assisted method: When using the neutral patient position, place the patient on the side of rotational restriction and establish a contact on the down-side of the superior spinous process (side of spinous rotation) (see Figure 5-251, A). Develop preadjustive tension by anteriorly rotating the patient's pelvis with your leg and forearm while applying cephalic traction to the patient's shoulder.

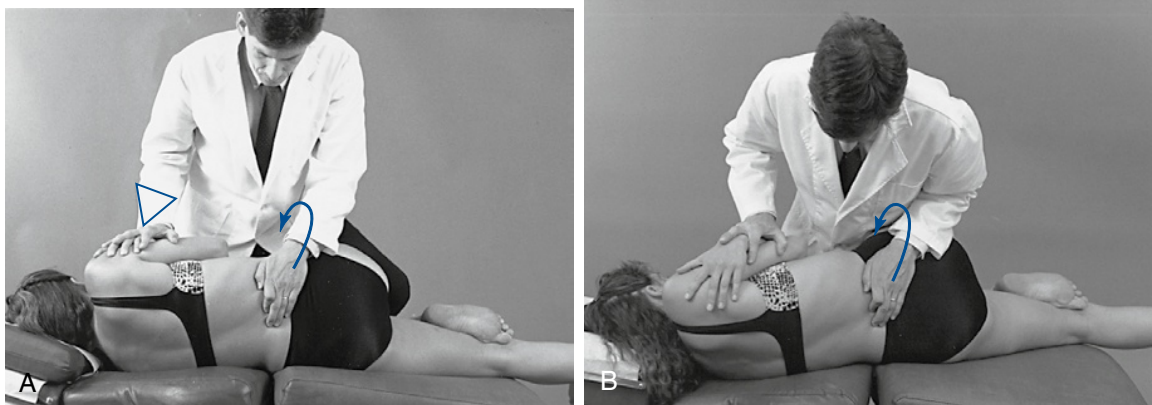


Figure 5-251 A, Assisted method, illustrating a digital contact applied to the left lateral surface of the L4 spinous process to induce left rotation in the L4–5 motion segment. B, Resisted method, illustrating a digital contact applied to the left lateral surface of the L5 spinous process to induce right rotation in the L4–5 motion segment and gapping of the right L4–5 articulation.

Resisted method: With a resisted method, place the patient on the side opposite the restriction (side opposite spinous rotation) and establish the segmental contact on the down-side of the inferior spinous process. For example, when treating a right rotation restriction (left posterior body rotation) at the L3–4 motion segment, place the patient on the left side and contact the left side of the L4 spinous process (see Figure 5-251, B).

Develop preadjustive tension by rotating the patient's shoulders posteriorly in the direction of segmental restriction as you rotate the patient's pelvis anteriorly with your leg and forearm. Induce posterior shoulder rotation and lateral flexion of the patient's trunk toward the table by pulling the patient's down-arm anteroinferiorly.

Resisted spinous pull adjustments may also be applied for combined rotational and opposite-side lateral flexion restrictions (PRI, PLI listings). The contacts and positioning are the same, but lateral flexion toward the adjusting bench is maximized. This may be accomplished by raising the thoracolumbar section of an articulating adjusting bench or by placing a roll under the patient's lumbar spine.

Digit/Spinous Push-Pull (Figure 5-252)

IND: Restricted rotation or combined restrictions in rotation and opposite-side lateral flexion, L1–L5. Rotation or combined rotation and ipsilateral lateral flexion malpositions, L1–L5.

PP: The patient lies in the basic side-posture position, with the foot of the patient's flexed leg hooked behind the popliteal space of the down-side leg.

DP: Square stance: Stand, facing the patient and contact the patient's knee with either your knee or the distal surface of your leg (see Figure 5-252, B).

Fencer stance: Stand, facing the patient at a 45-degree angle. Straddle the patient's flexed knee and contact his or her mid-shin with your thigh (see Figure 5-252, A).

CP: The fingertips (digital) of the cephalic hand reach under the patient's up-side arm to contact the lateral surface of the superior spinous process. The fingertips of the caudal hand hook

the inferior spinous process while the forearm contacts the patient's posterolateral buttock and thigh.

SCP: Adjacent spinous processes.

VEC: The superior hand thrusts (pushes) laterally to medially and inferiorly to superiorly. The inferior hand thrusts (pulls) laterally to medially in the opposing direction.

P: Place the patient in side posture. Flex the patient's upper thigh to distract the interspinous space of the dysfunctional motion segment. Rotate the patient's shoulder posteriorly in the direction of segmental restriction and flex the trunk laterally toward the adjusting bench.

The doctor establishes appropriate contacts on the adjacent spinous process and develops local joint tension by counter-rotating the pelvis, shoulders, and segmental contacts. As the shoulders are rotated posteriorly, the patient's pelvis and contacted vertebra are counter-rotated anteriorly. This should induce distraction in the motion segment between the established contacts. Posterior shoulder rotation is greater when treating upper lumbar dysfunction as compared with lower lumbar dysfunction.

At tension, deliver a high-velocity countertorquing thrust through both contact arms, reinforced by a shallow thrust through the contacts established on the patient's shoulder, leg, or shin. Take care not to apply undue pressure to the patient's lateral rib cage with the superior forearm contact. (The digit contacts on the spinous are primarily used for sensing preadjustive tension. The primary adjustive force is generated through the leveraged contacts established on the patient's lateral thigh, proximal shoulder and lower extremity.)

Prone

Bilateral Thenar/Mamillary Push (Figure 5-253)

IND: Restricted extension, L1–L5. Flexion malpositions, L1–L5.

PP: The patient lies prone.

DP: Stand in a fencer stance on either side of the patient.

CP: Bilateral thenar contacts parallel to the spine, with the fingers fanned and running medially to laterally.

SCP: Mamillary processes.

VEC: P-A.



Figure 5-252 Digital contacts applied to the right lateral surface of the L2 spinous process and left lateral surface of the L3 spinous process to induce right rotation and gapping of the right L2–3 articulation. **A**, Illustrates contact between the doctor's thigh and patient's leg. **B**, Illustrates contact between the patient's knee and the doctor's knee.

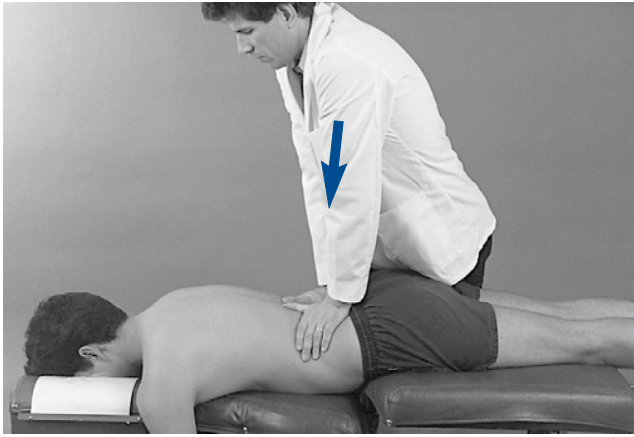


Figure 5-253 Bilateral thenar contacts applied to the mammillary processes of L3 to induce extension at the L3–4 motion segment.

P: The patient lies prone. Establish bilateral thenar contacts and develop joint tension by transferring additional body weight into the contacts. At tension, deliver a combined impulse thrust through the arms, trunk, and body. Releasing and lowering the thoracic section of an articulating table may assist in the development of segmental extension (see Figure 5-253). The thrust may be applied with a lumbar drop section. The thrust should be shallow to avoid hyperextension of the back.

Hypothenar/Mammillary Push (Figure 5-254)

IND: Restricted lateral flexion or rotation coupled with restricted extension, L1–L5. Rotation or lateral flexion malpositions coupled with flexion malpositions, L1–L5.

PP: The patient lies prone.

DP: Stand in a fencer stance or square stance on the side of adjustive contact.

CP: Hypothenar (pisiform), with arched hand and fingers running parallel to the spine.

SCP: Mammillary process.

IH: Your IH reinforces the contact or reaches around to grasp the anterior ilium or rib cage on the side of adjustive contact.

VEC: P-A and S-I or I-S, depending on the restriction being treated.

P: The patient lies prone, with the thoracic section released and lowered to assist in the development of segmental extension. Remove superficial tissue slack and establish adjustive contact. Develop preadjustive tension by transferring additional body weight into the contact. Deliver an impulse thrust through the arms, trunk, and body. The thrust may be applied with a lumbar drop section.

The contact may be reinforced with the IH, or the IH may contact the ipsilateral anterior ilium. When applying a reinforcing contact, stand on the side of adjustive contact (see Figure 5-254, A). If the IH contacts the ilium, stand on the side opposite the adjustive contact (see Figure 5-254, B).

When using an ilial contact, develop preadjustive tension by lifting and tractioning inferiorly against the ilium as the weight of your trunk is transferred anteriorly and superiorly against the contact. The counterdistraction tension through the ilium should be limited, and the patient's pelvis should not be rotated off the table more than 1 to 2 inches. Deliver the thrust with an impulse and body-drop thrust through the contact hand. It is unlikely that lateral flexion can be effectively induced in prone neutral positions. Prestressing the patient into lateral flexion and inducing lateral flexion during the delivery of the adjustment may assist in the production of lateral flexion.

Hypothenar/Spinous Push (Figure 5-255)

IND: Restricted extension coupled with rotation or lateral flexion restrictions, L1–L5. Flexion malpositions coupled with rotation or lateral flexion malpositions, L1–L5.

PP: The patient lies prone.

DP: Stand in a modified fencer stance or square stance on the side of adjustive contact.



Figure 5-254 A, Hypothenar contact applied to the right L1 mammillary process to induce extension and left rotation at the L1–2 motion segment. B, Hypothenar contact applied to the right L1 mammillary process, with countertraction applied through the anterior pelvis to induce left rotation at the right L1–2 motion segment.



5-255

Figure 5-255 Hypothenar contact applied to the left L2 spinous process to induce extension, left lateral flexion, and left rotation of the L2–3 motion segment.

CP: Mid-hypothenar.

SCP: Lateral proximal surface of the spinous process of the superior vertebra.

IH: Your IH supports the contact hand on the dorsal surface, with the fingers wrapped around the wrist.

VEC: P-A, L-M, and S-I.

P: The patient lies prone, with the thoracolumbar section of the table released and lowered to assist in the development of segmental extension. Establish a fleshy hypothenar contact against the spinous process on the side of rotation restriction (side of spinous rotation). The contact is developed by sliding medially onto the spinous process while inducing a slight clockwise or counterclockwise torquing movement, depending on the side of contact. The torquing movement is applied to assist in the development of a firm contact.

Develop preadjustive tension by transferring additional body weight into the contacts. At tension, deliver an impulse thrust through the arms, trunk, and body. The thrust should be shallow to avoid hyperextension of the patient's back. The thrust may be applied with a lumbar drop section. This adjustment is commonly applied in the treatment of coupled restrictions in rotation and same-side lateral flexion (PRS, PLS listings)

Knee-Chest

Hypothenar/Spinous Push (Figure 5-256)

IND: Restricted extension, coupled with lateral flexion, or rotation restrictions, L1–L5. Flexion, rotation, or lateral flexion malpositions, T4–T12.

PP: Position the patient in the knee-chest position, with the chest support placed so that the patient's pelvis is even with or slightly lower than the thoracic spine. The patient's femurs should be angled between 95 and 110 degrees.

DP: Stand at the side of the table in a square stance, typically on the side of the contact. You may also stand in a fencer stance, facing caudad.

CP: Mid-hypothenar.

SCP: Lateral surface of the spinous process.

IH: Your IH supports the contact hand on the dorsal surface, with the fingers wrapped around the wrist.

VEC: P-A, L-M, and S-I.

P: The IH first raises the patient's abdomen to make the spinous processes more prominent and available for establishing the contacts (see Figure 5-256, A). The patient is then instructed to allow the abdomen to drop and, at tension, an impulse thrust is delivered (see Figure 5-256, B). The patient is vulnerable to hyperextension in this adjustment, and the thrust must be shallow and nonrecoiling. Prestressing the patient into lateral flexion and inducing lateral flexion during the delivery of the adjustment may assist in the production of lateral flexion.

Bilateral Thenar/Mamillary Push (Figure 5-257)

IND: Restricted extension, L1–L5. Flexion malpositions, L1–L5.

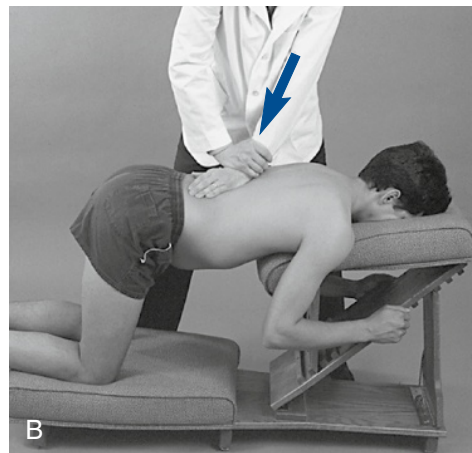
PP: Position the patient in the knee-chest position, with the chest support placed so that the patient's pelvis is even with or slightly lower than the thoracic spine. The patient's femurs should be angled between 95 and 110 degrees.

DP: Stand in a fencer stance on either side of the patient.

CP: Bilateral thenar contacts parallel to the spine, with the fingers fanned and running medially to laterally.

SCP: Mamillary processes.

VEC: P-A.



5-256

Figure 5-256 Knee-chest adjustment. **A**, Development of the adjustive contact and use of an adjusting bench in circumstances in which a knee-chest table is not available. **B**, Hypothenar contact applied to the left L1 spinous process to induce extension, left lateral flexion, and left rotation of the L1–2 motion segment.

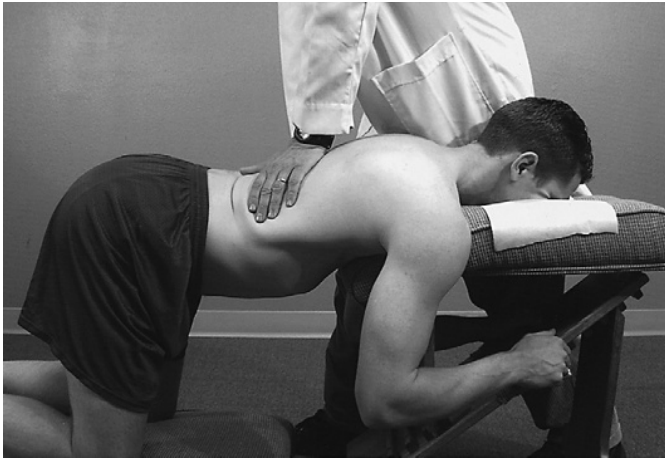


Figure 5-257 Bilateral thenar contacts established over the mammillary processes of L2 to induce extension at the L2–3 motion segment.

P: Establish the contacts on the superior or inferior vertebra of the involved motion segment. When contacting the superior vertebra, the adjustive thrust is delivered anteriorly and slightly inferiorly. When contacting the inferior vertebra, the thrust is delivered anteriorly and slightly superiorly.

The patient is instructed to allow the abdomen to drop and, at tension, an impulse thrust is delivered. The patient is vulnerable to hyperextension in this adjustment, so the thrust must be shallow and nonrecoiling.

Hypothenar/Maximillary (Figure 5-258)

IND: Restricted extension coupled with rotation or lateral flexion restrictions, T4–T12. Flexion malpositions coupled with rotation or lateral flexion malpositions, T4–T12.

PP: Position the patient in the knee-chest position, with the chest support placed so that the patient's pelvis is slightly lower than the thoracic spine. The patient's femurs should be angled between 95 and 110 degrees.

DP: Stand at the side of the table in a square stance, typically on the side of the contact. You may also stand in a fencer stance, facing caudally.

CP: Hypothenar (pisiform).

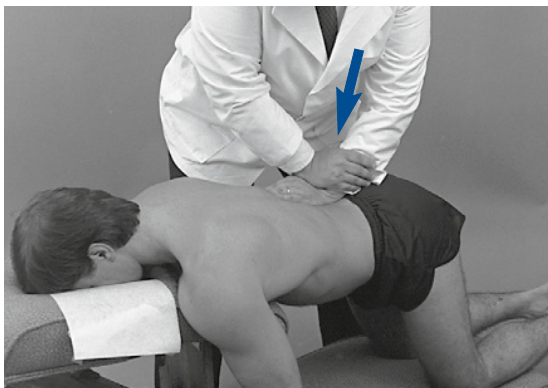


Figure 5-258 Hypothenar contact applied to the right L4 mammillary process to induce extension and left rotation.

SCP: Mammillary process.

IH: Your IH supports the contact hand on the dorsal surface, with the fingers wrapped around the wrist. The IH may be placed on the opposite side to stabilize or impart an assisting impulse.

VEC: P-A.

P: The IH first raises the patient's abdomen. Establish a contact on the mammillary process. Then instruct the patient to allow the abdomen to drop and, at tension, deliver an impulse thrust. The patient is vulnerable to hyperextension in this adjustment, so the thrust must be shallow and nonrecoiling. Prestressing the patient into lateral flexion and inducing lateral flexion during the delivery of the adjustment may assist in the production of lateral flexion.

Sitting

Hypothenar/Mammillary or Spinous Push (Figure 5-259)

IND: Restricted rotation or rotation with lateral flexion, T12–L5, which may be coupled with restricted extension or flexion. Rotation or lateral flexion malpositions, T12–L5, which may be coupled with flexion or extension malpositions.

PP: The patient sits, with legs straddling the adjusting bench and with knees locked against each side. The arms are folded across the chest, with the hands grasping the shoulders.

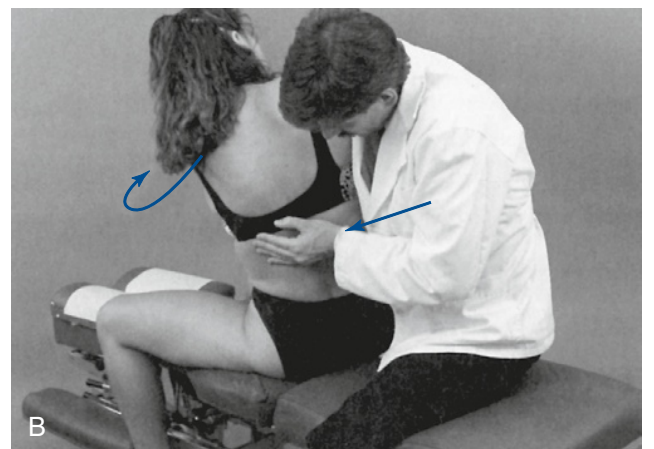
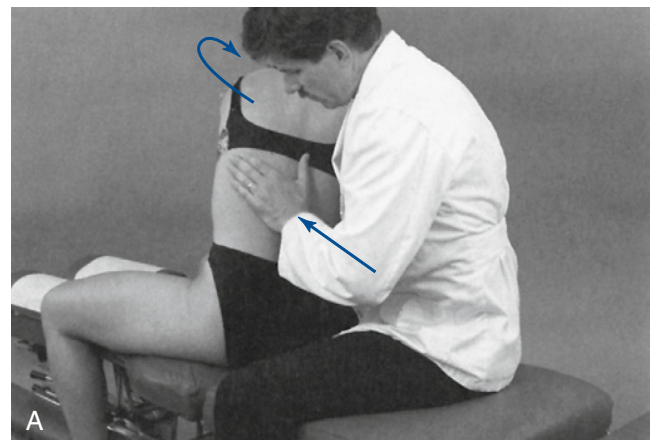


Figure 5-259 **A**, Hypothenar contact applied to the left mammillary process of L2 to induce right rotation or right lateral flexion of the L2–3 motion segment. **B**, Hypothenar spinous contact applied to the right lateral surface of L2 to induce right rotation or left lateral flexion of the L2–3 motion segment.

DP: The doctor may sit behind the patient, straddle the bench, or stand at the caudal end of the bench. In the standing position, the doctor may rest his elbow against his or her anterior ilium.

CP: Hypothenar (pisiform) of contact hand.

SCP: Mammillary or spinous process of superior vertebra.

IH: Your IH reaches around the patient to clasp the patient's opposite arm.

VEC: Pulling rotatory force generated by doctor's anterior arm contact and torso. P-A and L-M through the contact hand to assist in the production of rotation

P: Ask the patient to sit with crossed arms. Preadjustive tension is typically developed by flexing, laterally flexing, and rotating the patient in the direction of joint restriction (assisted method). Once tension is established, an impulse thrust is delivered by inducing a twisting thrust generated through the doctor's indifferent arm, trunk and contact. The direction of induced lateral flexion and the point of adjustive contact depend on the restriction being treated. Although this adjustment may be applied in all lumbar regions, it is probably most effectively applied in the lower thoracic spine and upper lumbar spine.

Rotation restrictions: When treating rotational dysfunction, the doctor may establish contacts on the spinous process or mammillary process. When using a mammillary contact, establish the contact on the superior vertebra on the side opposite the rotation restriction (side of posterior body rotation). Develop preadjustive tension by flexing, rotating, and laterally flexing the patient away from the side of contact (Figure 5-259, *A*). At tension, deliver a thrust to induce rotation. This method is applied to induce maximal distraction in the facet joint ipsilaterally inferior to the point of contact. It is also applied to treat restrictions in rotation and same-side lateral flexion (e.g., right or left rotation restriction coupled with the corresponding right or left lateral flexion restriction).

To use a spinous process contact, slide medially and establish a fleshy mid-hypothenar contact on the lateral surface of the spinous process on the side of rotation restriction (side of spinous rotation). Develop preadjustive tension by flexing, rotating, and laterally flexing the patient away from the side of contact (Figure 5-259, *B*). At tension, deliver a thrust to induce rotation. This contact should induce maximal distraction in the facet joint ipsilateral inferior to the side of spinous contact. This method is also commonly applied when treating combined restrictions in rotation and opposite-side lateral flexion (e.g., right or left rotation restriction coupled with the opposing right or left lateral flexion restriction).

Lateral flexion restrictions: Lateral flexion dysfunction is treated by contacting the mammillary process of the superior vertebra on the side opposite the lateral flexion restriction (Figure 5-259, *A*). Develop preadjustive tension by flexing, laterally flexing, and rotating the patient away from the side of contact. At tension, deliver a thrust to induce rotation.

Sitting lumbar adjustments produce rotational tension in the patient's spine, regardless of the dysfunction being treated. If the patient cannot tolerate rotation of the spine, he or she is not a good candidate for sitting lumbar adjustments.

PELVIC JOINTS

Probably the least understood and most controversial function of any area in the musculoskeletal system is that of the bones and joints that compose the pelvic mechanism. The two sacroiliac joints posteriorly, together with the pubic symphysis anteriorly, form a three-joint complex, with much the same function as the typical vertebral functional unit. Although originally viewed as an immobile joint, there is now no doubt that the sacroiliac joints are mobile diarthrodial joints, important to the statics and dynamics of posture and gait. They must provide support for the trunk while functioning to guide movement and helping to absorb the compressive force associated with locomotion and weight-bearing.

Grieve⁶⁷ believes that this articulation, together with the craniovertebral region and other transitional areas, is of prime importance in understanding the conservative treatment of vertebral joint problems. Moreover, dysfunction of the sacroiliac joint is often ignored by other health care practitioners as an insignificant feature of musculoskeletal problems. The sacroiliac dysfunctional syndrome is a legitimate clinical entity that is separate from other painful low back conditions.⁶⁷⁻⁷¹

FUNCTIONAL ANATOMY OF THE SACROILIAC JOINTS

The pelvic complex comprises the two innominate bones, with the sacrum between. The ilium, ischium, and pubic bone fuse at the acetabulum to form each innominate (Figure 5-260). The sacrum is a fusion of the five sacral segments and is roughly triangular in shape, giving it the appearance of a wedge inserted between the two innominate bones (Figure 5-261). The sacral base has two superior facets that articulate with L5 and the sacral apex, which points downward to articulate with the coccyx. The apex of the sacrum is oval and articulates with the coccyx by means of a disc. By about 30 years of age, the disc disappears, and the

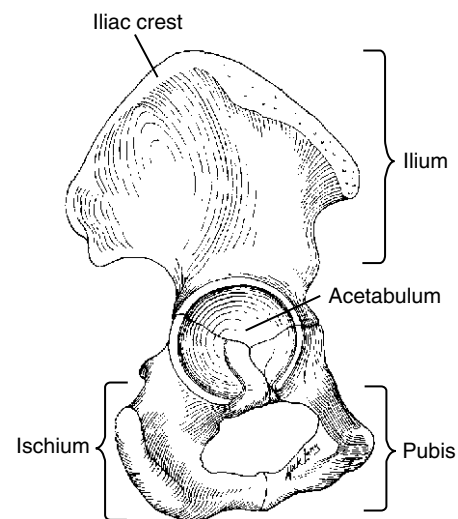


Figure 5-260 Lateral view of the right innominate, showing the ilium, ischium, and pubic contributions to the acetabulum.

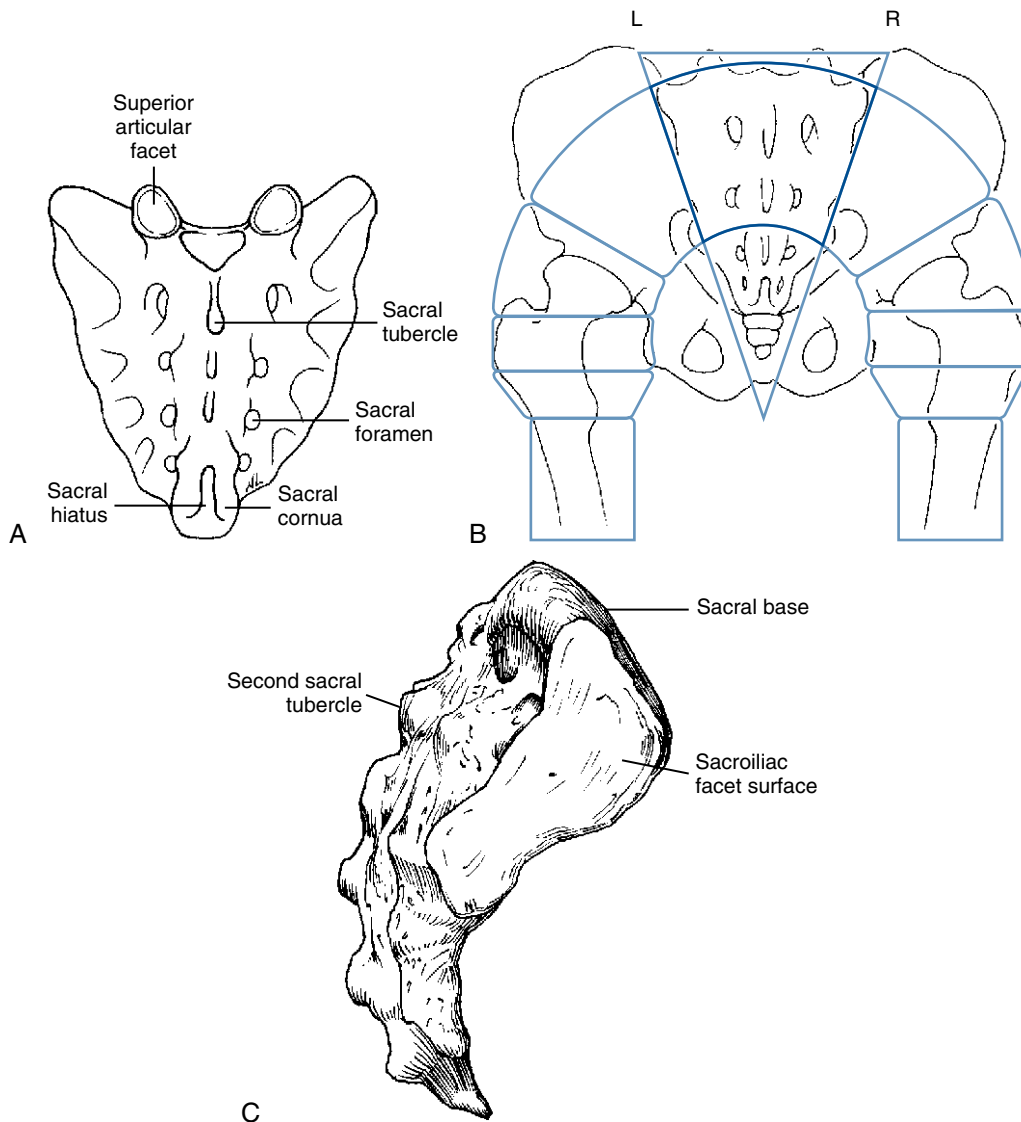


Figure 5-261 A, Sacrum viewed from the posterior is triangular and serves as a keystone (B) in the arch between the two columns formed by the lower extremities. C, Lateral view of the sacrum.

two structures are essentially fused together. The sacral tubercles, located in the midline, correlate with the spinous processes of the fused vertebra. The tubercles on the posterolateral aspect correlate with transverse processes.

The posterior sacroiliac joints are true synovial joints. They have a joint cavity containing synovial fluid and are enclosed by a joint capsule. The shape and configuration of the posterior joints are unique and important to their function. The articular surface is described as auricular (ear-shaped), a letter C, or a letter L lying on its side (Figure 5-262). The articular surfaces have different contours that develop into interlocking elevations and depressions. This bony configuration produces what has been termed a *key-stone effect* of the sacrum, effectively distributing axial compressive forces through the pelvic mechanism (see Figure 5-261, B). Forces from the lower extremities divide, heading upward toward the spine and anteriorly toward the pubic symphysis, and downward forces of gravity on the spine split to both sides (Figure 5-263).

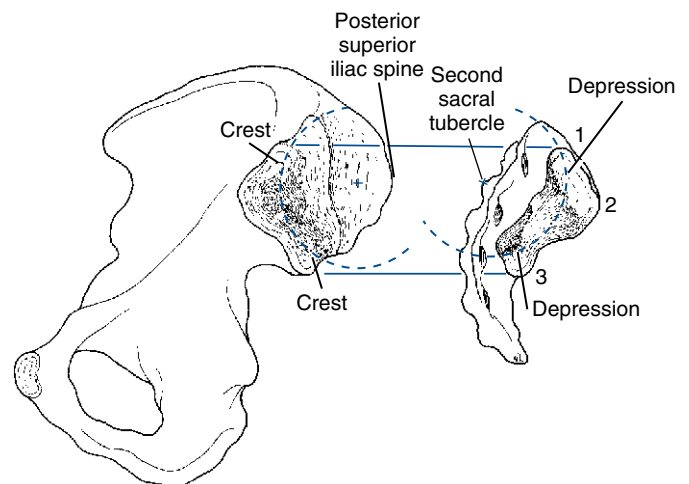


Figure 5-262 Auricular-shaped surfaces of the posterior joints of the sacroiliac articulation.

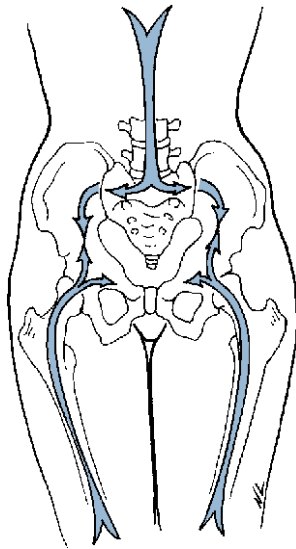


Figure 5-263 Forces from gravity above meet with forces from the lower extremities at the sacroiliac and hip articulations.

The morphologic configuration of the sacroiliac joints is not static and is extremely variable from individual to individual.^{72,73} At birth, the joints are undeveloped, smooth, and flat. Only after an individual becomes ambulatory do the joints begin to take on their adult characteristics. In the teenage years, the joint surfaces begin to roughen and develop their characteristic grooves and ridges. In the third to fourth decades, this process is well established, and by the fifth and sixth decades, the joint surfaces may be very eroded. In later years, a high percentage of male patients will have developed interarticular adhesions across the sacroiliac joints and will have lost sacroiliac joint motion.^{72,73}

A number of strong ligaments aid in stabilizing the pelvic mechanism (Figure 5-264). The posterior sacroiliac ligaments run from the sacrum to the iliac tuberosity and posterosuperior iliac spine (PSIS). They continue laterally with the sacrotuberous ligament and medially with the thoracolumbar fascia. The sacrotuberous ligament extends from the lower portion of the sacrum obliquely downward to the ischial tuberosity. It continues caudally with the tendon of the long head of the biceps femoris. The anterior sacroiliac ligament consists of numerous bands attaching from the lateral edge of the sacrum to the auricular surface of the ilium. The sacrospinous ligament is triangular and extends from the lower lateral edge of the sacrum and the upper edge of the coccyx to the ischial spine.

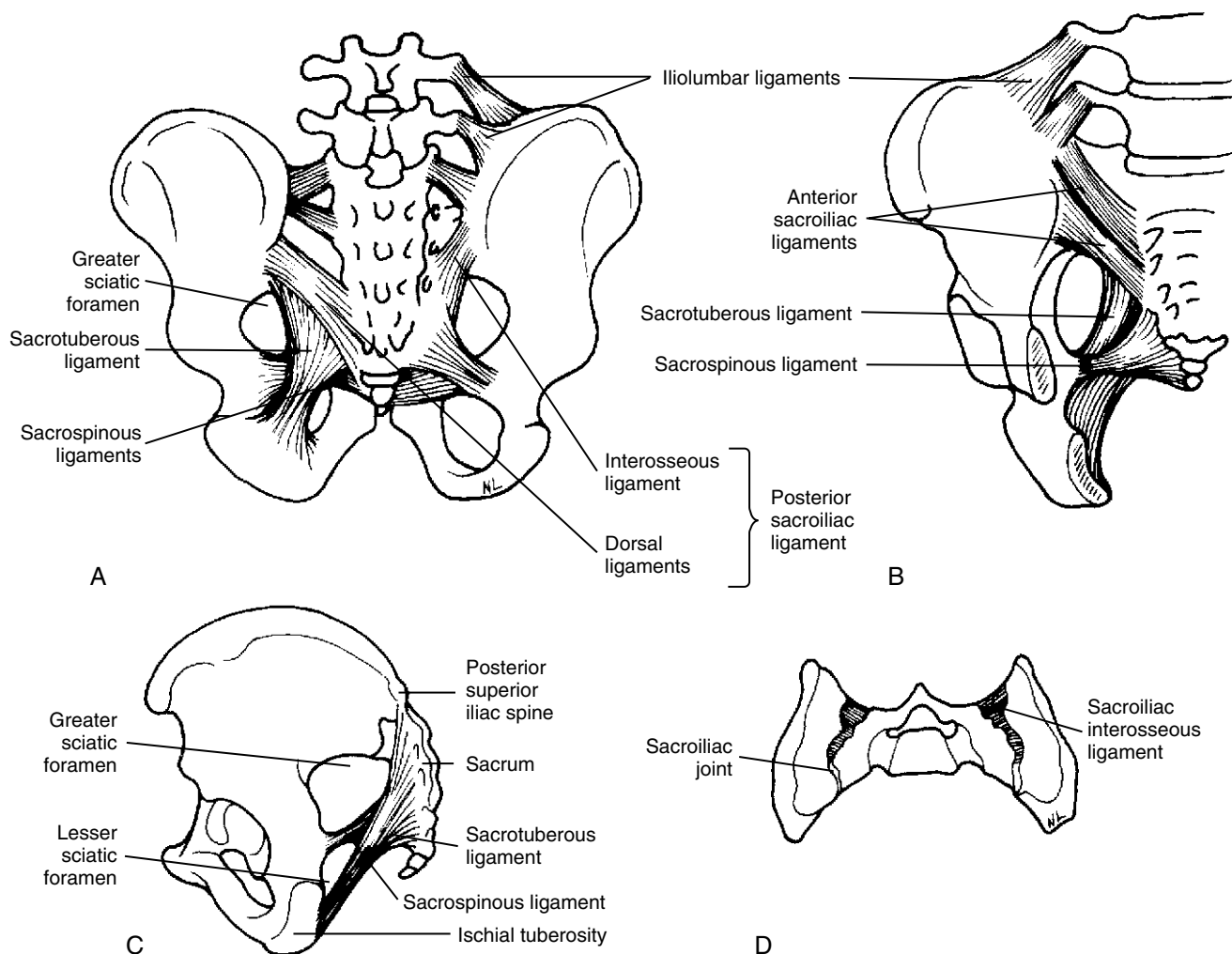


Figure 5-264 Ligaments of the posterior sacroiliac articulations. A, Posterior view. B, Anterior view. C, Lateral view. D, Transverse section.

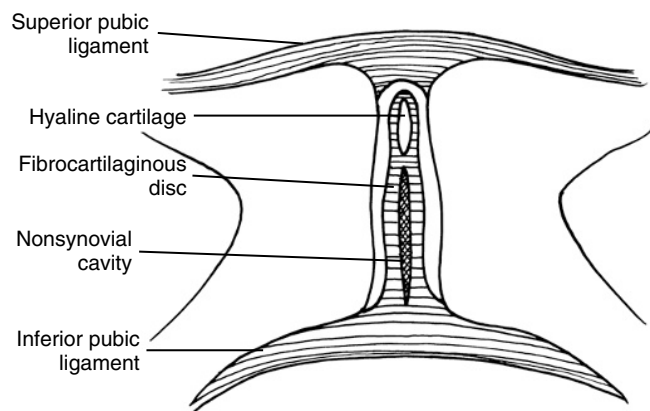


Figure 5-265 Pubic symphysis: anterior joint of the sacroiliac three-joint complex.

The sacrotuberous and sacrospinal ligaments limit posterior movement of the sacral apex; the posterior sacroiliac ligament limits anterior movement of the sacral base. Anteriorly, the pubic bones are joined by the symphysis pubis, a cartilaginous joint containing a fibrocartilaginous interpubic disc (Figure 5-265). The superior pubic ligament connects the pubic bones superiorly; the inferior pubic ligament connects the lower borders of the symphysis pubis, forming the upper boundary of the pubic arch. Anteriorly, there is evidence of connective tissue layers passing from one bone to another. They are interlaced with fibers of the external oblique aponeuroses and the medial tendons of the rectus abdominus muscles. Posteriorly, there is also some fibrous tissue that is continuous with the periosteum of both pubic bones. The inguinal ligament, the lower reflected aponeurotic margin of the external oblique muscle, extends from the ASIS to the pubic tubercle.

Although some of the strongest muscles of the body surround the sacroiliac joint, none are intrinsic to it or act upon it directly.⁶⁷ However, the surrounding muscle mass may influence the mechanical behavior of the joint or respond to the stresses applied to it.

SACROILIAC MOTIONS

Although it has become accepted that the sacroiliac is a truly movable joint, there is still controversy as to exactly how it moves, how much it moves, and where axes of motion might be located. A number of different hypotheses and models of pelvic mechanics have been proposed.^{71,74-80} Recent models reinforce the reality of sacroiliac motion but also stress the sacroiliac joint's important role in maintaining stability during the transverse of forces between the lower extremity and the spine.⁸¹

The sacroiliac joint is most active during locomotion, with movement occurring primarily in the oblique sagittal plane. During locomotion the sacroiliac joints flex and extend in unison with the corresponding hip joint. In the process of ambulating, each sacroiliac joint goes through two full cycles of alternating flexion and extension. Movements of flexion or extension in one joint are mirrored by the opposite movement at the other joint.

Illi's model of sacroiliac motion⁸² proposes that compensatory movements at the sacrum and lumbosacral junction occur to help absorb the pelvic torsion induced by these opposing movements of

flexion and extension. He suggests that as one innominate flexes (the PSIS moves posteriorly and inferiorly), the ipsilateral sacral base moves anteriorly and inferiorly, and as the other innominate extends (moves anteriorly and superiorly), the sacral base on that side moves posteriorly and superiorly (Figure 5-266). If the described actions of the sacrum are envisioned as one continuous motion, a picture of an oblique and horizontal, figure-eight-shaped rocking movement of the sacrum becomes apparent (Figure 5-267).

Illi⁸² further postulates that alternating movements of flexion act through the iliolumbar ligament to dampen motion at L5 and hence the whole spine.⁷⁸ As the ilium moves posteriorly, L5 is pulled posteriorly and inferiorly through tension in the iliolumbar ligament, and the rest of the lumbar spine undergoes coupled motion in slight rotation and lateral flexion (type I movement).

There has been a tendency in the chiropractic profession to refer to the complementary ipsilateral anterior and inferior and posterior and superior sacral movements as *extension* and *flexion*. This has led to the confusing situation in which the same restriction in sacroiliac movement may be referred to interchangeably

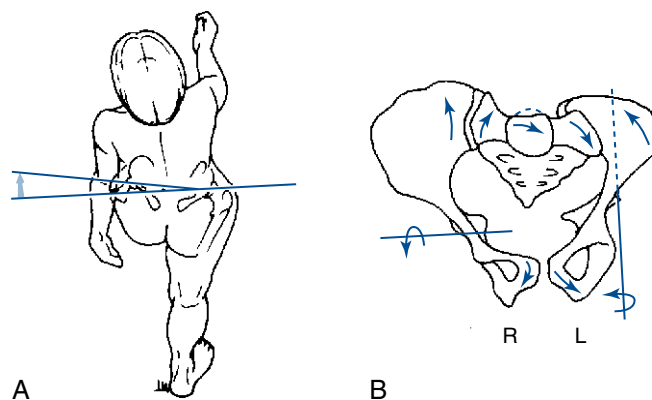


Figure 5-266 Movements of the pelvic joints during gait. **A**, Posterior view, illustrating left hip and sacroiliac (SI) joint flexion and right hip and SI joint extension. **B**, Anterior view, illustrating reciprocal movements of the innominate and sacrum during SI joint flexion and extension. As the left innominate moves posteriorly, a pivoting motion occurs at the pubic symphysis, and the left sacral base moves anteroinferiorly. As the right innominate moves anteriorly, the sacral base moves posteriosuperiorly.

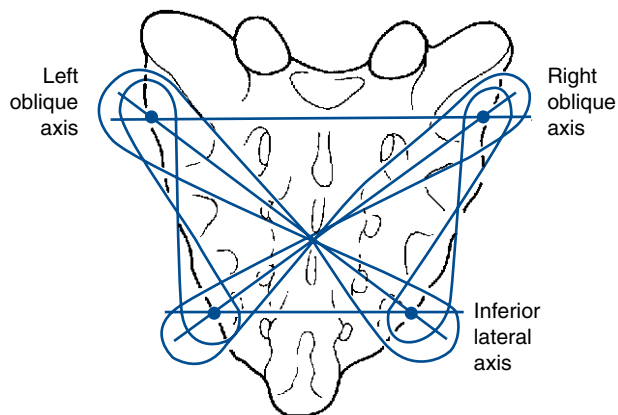


Figure 5-267 The proposed axes of motion in the sacroiliac articulation allow a "gyroscopic" figure-eight movement.

as *flexion* and *extension* restrictions. For example, during sacroiliac flexion, if the PSIS is the point of reference and perceived to be limited in its PI movement, the restriction is described as a *sacroiliac flexion restriction*. However, if the sacral base is the point of reference and a loss of complementary ipsilateral anterior inferior (AI) movement of the sacral base is perceived, then the same sacroiliac restriction is labeled as an *extension restriction*.

The obvious problem with this approach is that both of these movements occur during sacroiliac flexion, and it is not possible for a joint to both flex and extend at the same time. The basis of this misunderstanding probably relates to the fact that the sacrum articulates with both the sacroiliac joint and the lumbosacral joint. Lumbosacral extension does involve AI movement of the sacral base, but the same movement relative to the ilium occurs during sacroiliac flexion, not extension. Pure sagittal plane movements of the sacral base anteroinferiorly (nutation) and posteriorly and inferiorly (counternutation) do occur, but only during trunk flexion and extension and when changing from the upright, seated, and recumbent positions. These movements are appropriately labeled as *flexion* and *extension*, but they are defined relative to the lumbosacral articulation, not the sacroiliac joints (Figure 5-268).

To avoid confusion, we suggest applying the descriptions of *flexion* and *extension* to joint movement, not movement of the sacrum in space. If anterior glide of the sacral base is restricted at the lumbosacral articulation, it should be referred to as a *lumbosacral extension restriction*, but if the movement restriction is perceived to be across the sacroiliac joint, it should be referred to as a *sacroiliac flexion restriction*.

If the sacroiliac joints are observed from the posterior and the PSIS and posterior sacral base are used as reference points, sacroiliac flexion and extension movements may be described as follows:

- Flexion of the sacroiliac incorporates posterior and inferior movement of the PSIS and ipsilateral anterior and inferior movement of the sacral base.
- Extension of the sacroiliac incorporates anterior and superior movement of the PSIS and ipsilateral posterior and superior movement of the sacral base (see Figure 5-266).

It is also important to understand and appreciate that with flexion and extension movements at the posterior sacroiliac joints, motion of the pubic symphysis in rotation about a transverse axis must occur. Although this twisting type of motion is considered the only normal motion at the pubic symphysis, small amounts of translational movements forward, backward, up, and down may occur. Large translational movements of the pubic symphysis may also occur, but are considered abnormal and present only with an unstable pelvic complex (see Figure 5-268).

Abnormal movement and malpositions of the pubic symphysis have been described.⁸³ A significant vertical shear in which the pubic bone on one side is significantly higher than the other occurs and can be palpated, as well as seen on x-ray film.⁸⁴ Separation of the pubic symphysis, although less often encountered and occurring mainly in pregnancy, can also be observed on x-ray film. Palpable pain may occur in dysfunctional patterns of the pubic symphysis.

EVALUATION OF THE PELVIC COMPLEX

The examination of the pelvic complex should incorporate an assessment of alignment, tone, texture, and tenderness of the pelvic ring and its related soft tissues. Also, the sacroiliac joints should be examined for pain and mobility.

Observation

The alignment of the pelvic ring and sacroiliac joints should be assessed in standing, sitting, and prone positions. Standing postural evaluation has been previously described within the section on evaluation of the lumbar spine (see Figures 5-213 through 5-215). Sitting and prone evaluation of pelvic alignment incorporates an evaluation of the same pelvic landmarks and provides the doctor the opportunity to compare alignment in weight-bearing and non-weight-bearing positions. For example, a low iliac crest standing and sitting may identify a discrepancy in bony symmetry of the innominates versus a discrepancy in leg length. A low iliac crest in the standing position that appears high in the prone position may indicate the presence of sacroiliac dysfunction and a functional short leg versus an anatomic short leg.

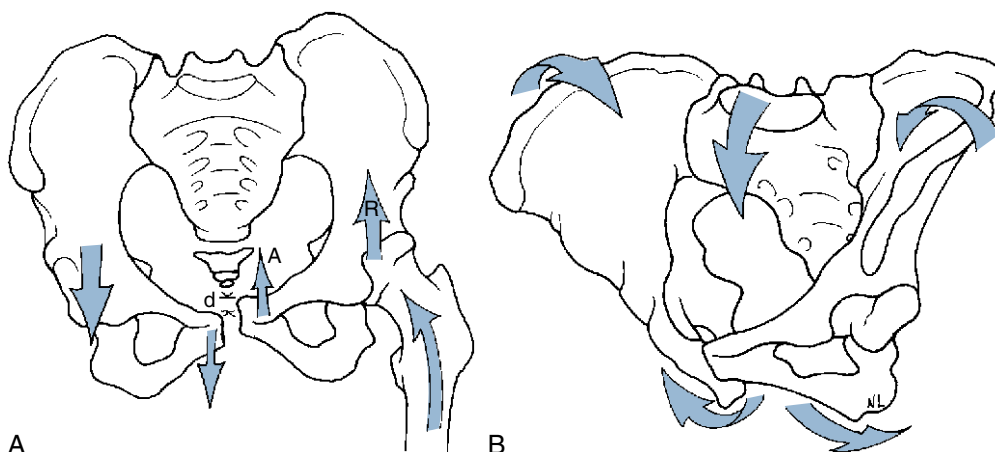


Figure 5-268 Although the usual movement of the pubic symphysis is rotation about a transverse axis, the mechanisms exist for shear (A) and separation or compression (B) of the pubis. B also demonstrates nutation of the sacral base and extension of the lumbosacral articulation.

Static Palpation

The palpatory assessment of pelvic bony and soft tissue structures is primarily conducted in the prone position. To evaluate the bony landmarks, establish bilateral contacts with the thumbs or fingertips and compare the contour and alignment of the iliac crests, PSISs, sacral base, and sacral apex (Figure 5-269). The palpatory depth of the sacral base just medial to the PSIS and the distance

between the second sacral tubercle and the PSIS should be evaluated on each side and compared (see Figure 5-269).

Flexion (PI) or extension (AS) malpositions of one innominate as compared with the other are possible indications of pelvic dysfunction. These positions of distortion may be identified with the aid of palpation and observation. Table 5-9 lists the clinical findings that have been empirically reported by the chiropractic



Figure 5-269 Prone palpation of the bony landmarks of the pelvis. **A**, Palpation of the alignment of the iliac crests. **B**, Palpation of the alignment of the posterosuperior iliac spine. **C**, Palpation of the alignment of the sacral apex. **D**, Palpation of the sacral sulcus. (Note the depth and alignment of the sacral base.)

TABLE 5-9 Clinical Findings Associated with Pelvic Distortion

Posterior Innominate Flexion Malposition

Prominent medioinferior PSIS
Low gluteal fold
Elevated ASIS
AI sacral base
Contralaterally deviated sacral apex
Ipsilateral posteroinferior rotated L5
Ipsilateral convexity of lumbar lateral curve
Functional leg length deficiency

Anterior Innominate Extension Malposition

Anterior, lateral and superior PSIS
High gluteal fold
Lowered ASIS
Posterosuperior sacral base
Ipsilaterally deviated sacral apex

profession to reflect each distortion. However, remember that identification of altered alignment is not confirmation of dysfunction. The evaluation of alignment, like all physical procedures, is prone to examiner error, and congenital asymmetries of the pelvis are not uncommon.

The soft tissues of the lumbosacral, sacroiliac, and gluteal regions are also palpated with the palmar surfaces of the fingers or thumbs. In the patient complaining of low back pain, it is important to carefully distinguish between several common sites and sources of pain. Pain arising in the sacroiliac joint, iliolumbar ligaments, lumbar motion segments, or adjacent gluteal soft tissues may all be responsible for the subjective complaint of generalized lumbosacral or sacroiliac pain.

The sacroiliac ligaments are palpated just medial to the PSIS, and the iliolumbar ligaments are palpated between the L5 transverse process and iliac crest. Pain arising from the myofascial origins of the gluteal and piriformis muscles is identified by exploring their attachments along the PSIS, iliac crest, and sacral ala (Figure 5-270).

Incorporated into the evaluation of the bony structures of the pelvis is an evaluation of comparative leg length. Anatomic

discrepancies in length may predispose the patient to pelvic dysfunction, and functional leg length inequality is considered a potential significant sign of sacroiliac subluxation and dysfunction. Leg length is traditionally evaluated in the prone position by observing the comparative length of the heels or medial malleoli (Figure 5-271, *A*). If discrepancies are noted, the legs should be elevated at 90 degrees of knee flexion to screen for a shortened tibia (Figure 5-271, *B*). The elevated feet must be maintained in neutral upright position. Internal or external rotation of the hip induces a false indication of tibial shortening.

In cases of suspected sacroiliac dysfunction and associated functional leg length inequality, the doctor should also evaluate and compare leg length in the supine and sitting positions. Functional leg length inequality that is secondary to sacroiliac subluxation and dysfunction may reverse from the supine to sitting position, whereas anatomic leg length inequality or functional inequality secondary to dysfunction at other sites likely will not (Figure 5-272).

A unique method incorporating prone leg length evaluation is also commonly applied in chiropractic. This method is referred to as the *Derfield pelvic leg check (DPLC)*. This procedure is purported



Figure 5-270 Palpation of the lumbosacral and gluteal soft tissues. **A**, Palpation for tenderness of the right iliolumbar ligament. **B**, Palpation of the origins of the gluteal muscles. **C**, Palpation of the origins of the piriformis muscles.

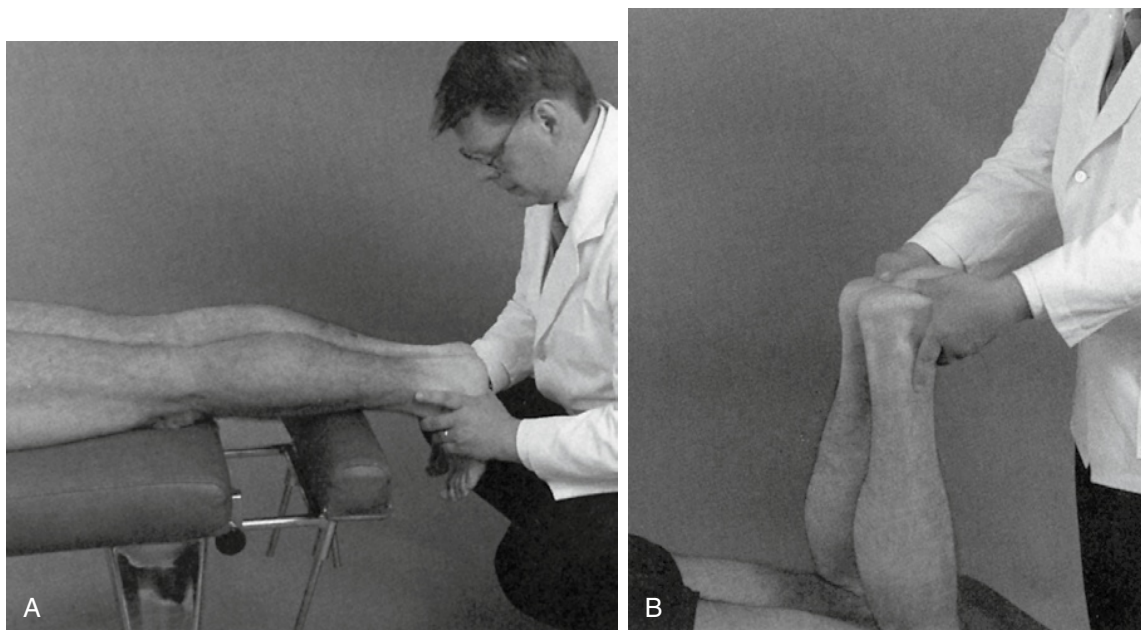


Figure 5-271 Prone evaluation of leg length. **A**, Knees extended. **B**, Knees flexed.

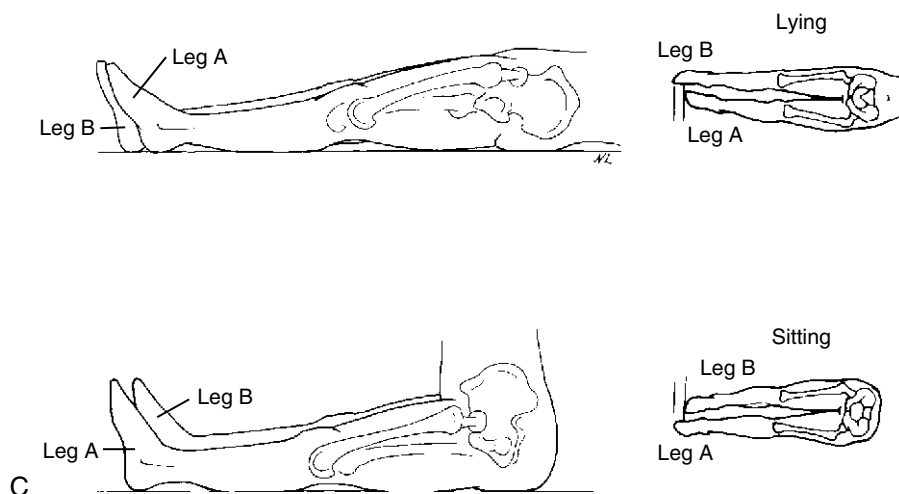
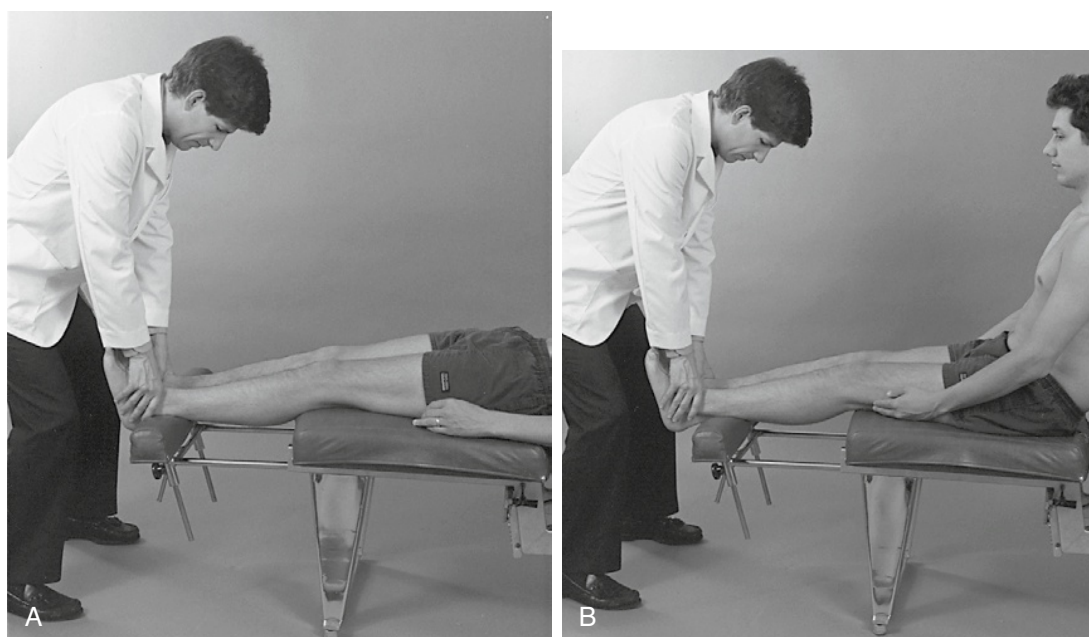


Figure 5-272 Supine evaluation of leg length. **A**, Lying supine. **B**, Sitting. **C**, Changes in leg length observed from the lying to the sitting position may indicate a sacroiliac dysfunction. On the side of the relative posterior innominate (*leg A*), the acetabulum is displaced anteriorly, creating a leg deficiency in the supine position, which lengthens on sitting. (Modified from Gatterman MI: *Chiropractic management of spine related disorders*, Baltimore, 1990, Williams & Wilkins.)

to detect sacroiliac dysfunction and determine the side and nature of the dysfunction.^{85,86} The reliability of DPLC as a separate diagnostic procedure has not been evaluated. The validity of the DPLC to ascertain pelvic dysfunction has not been evaluated.

The test is based on the premise that pelvic dysfunction is associated with misalignment of one ischium as compared with the other, and that pelvic misalignment will be reflected in functional unleveling of the legs. The relative short-leg side is envisioned to be associated with a PI malposition of the ischium. The relative long-leg side is associated with an AS malposition of the ischium.

The DPLC is performed in a prone position and incorporates the evaluation of leg length in a knee-extended position, followed by an evaluation in 90 degrees of knee flexion (see Figure 5-271). If leg length inequality is noted in the knee-extended position, the knees are flexed to 90 degrees to observe for a change in comparative length. A Derifield positive response (D+) is observed when the comparative short leg gets longer. A Derifield negative response (D-) is observed when the short leg remains short or appears even shorter. A D+ indicates that the sacroiliac dysfunction is on the side of the short leg and a D- response indicates that the dysfunction is on the side of the long leg.⁸⁵

The DPLC is theorized to detect pelvic dysfunction as a result of associated reactive hypertonicity in the anterior thigh muscles on the side of sacroiliac dysfunction. When the reactive anterior thigh muscles are stretched in the knee-flexed position, they contract, firming up the thigh and increasing its A-P diameter. The net effect is to elevate the leg of the table on the dysfunctional side. Therefore, on the D+ side, the short leg gets longer, decreasing or reversing the inequality. On the D- side, the long leg gets longer, increasing the inequality.⁸⁵

Joint Play

A variety of different joint provocation techniques have demonstrated their reliability and validity for identifying the sacroiliac joint as a source of LBP. Many of these procedures can be classified as JP procedures and include compression, distraction, torsion, and shear tests. Palpatory localization of pain to the sacroiliac joint, in conjunction with positive joint provocation testing, should provoke suspicion of the sacroiliac joint as one potential source of the patient's LBP.

Sitting Joint Play. To assess sitting JP of the sacroiliac joint, place the patient in the sitting position, with the arms relaxed on the lap. Sit behind the patient, shifted slightly to the contralateral side. Establish the palpation contact with the thumb along the medial aspect of the PSIS, with the supporting forearm placed across the patient's shoulders (Figure 5-273). Guide the patient in lateral flexion away from the side of contact until tension at the sacroiliac joint is felt. At tension, apply additional downward pressure through the indifferent arm, coupled with lateral pressure from the contact thumb. Slight give should be perceived during evaluation, and abnormal resistance or pain may be associated with sacroiliac dysfunction.

Prone Joint Play

Sacroiliac Flexion. To evaluate flexion JP, reach across with the caudal hand and cup the patient's anterior ilium. Then palpate the sacroiliac joint with the cephalic hand by locating the fingertips just medial to the PSIS over the dorsal aspect of the ipsilateral sacral ala (sulcus of the SI joint) (Figure 5-274). To execute the procedure, pull posteriorly with the anterior ilial contact and



Figure 5-273 Thumb contact applied to the right posterosuperior iliac spine to evaluate joint play of the right sacroiliac articulation.



Figure 5-274 Anterior-to-posterior pressure applied to the right ilium and digital contacts applied to the right sacroiliac joint assess for the presence of flexion joint play.

palpate for posterior glide of the ilium. Counterpressure asserted against the sacral base through the doctor's thenar or hypothenar may also be applied to induce more shear across the joint. Pain indicates that the sacroiliac joint may be dysfunctional and contributing to the patient's LBP.

Sacroiliac Extension. To assess extension, contact the sacral apex with the caudal hand and a contact on the ipsilateral PSIS with the hypothenar of the cephalic hand (Figure 5-275). Apply anterior and superior pressure against the ilium and anterior and inferior movement against the sacral apex. Normal movement is



Figure 5-275 Counterpressure applied through a hypothenar contact over the right posterosuperior iliac spine and a hypothenar contact over the sacral apex to assess extension joint play in the right sacroiliac joint.



Figure 5-277 Hypothenar contact applied to the right inferior margin of the sacrum exerts pressure superiorly against counterpressure applied by a calcaneal contact over the right iliac crest to assess superior sacral glide.

reflected by a sense of give and separation into extension. Pain indicates that the SI joint may be dysfunctional and contributing to the patient's LBP.

Inferior Sacral Glide. The patient is placed in the prone position, and the doctor reaches across the patient with the heel of the caudal hand to contact the patient's lower ischium. The ulnar side of the cephalic hand contacts the superior dorsal surface of the sacrum, also on the contralateral side (Figure 5-276). To execute the procedure, apply caudal pressure against the sacrum as counterstabilizing pressure is maintained against the ischium. Pain during the test may indicate sacroiliac dysfunction.

Superior Sacral Glide. The patient is placed in the prone position. The doctor reaches across the patient with the ulnar side of the caudal hand to contact the inferior and lateral margin of the sacrum. The cephalic hand stabilizes the iliac crest with a broad contact (Figure 5-277). Cephalic pressure is applied against the sacral contact as counterpressure is maintained against the ilium. Pain during the test may also indicate sacroiliac dysfunction.

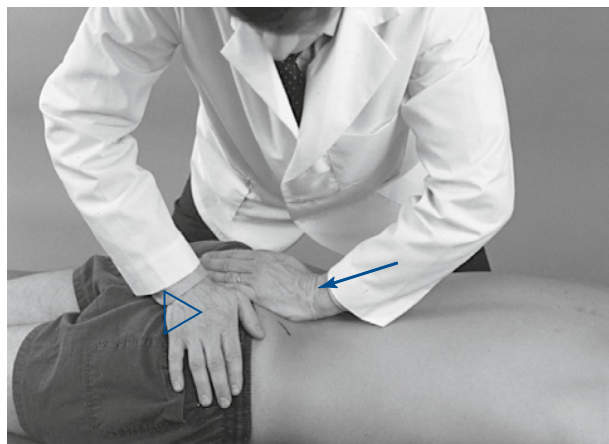


Figure 5-276 Hypothenar contact applied to the right sacral base exerts pressure inferiorly against counterpressure applied by a calcaneal contact over the right ischium to assess inferior sacral glide.

Motion Palpation

Sitting Mobility Test

Sacral Push. The patient is asked to sit with the arms relaxed on the thighs. The doctor sits behind the patient and establishes bilateral thumb contacts across the patient's sacroiliac joints and sacral ala (Figure 5-278). The patient is then asked to extend back and rotate around the doctor's thumbs. With proper sacroiliac and lumbosacral joint motion, the doctor's thumbs should move symmetrically forward with the patient's sacral base. Restricted anterior gliding of the sacral base may indicate sacroiliac dysfunction.



Figure 5-278 Thumb contacts push posteriorly to anteriorly over the sacral base on both sides to assess anterior glide of the sacrum.

Standing Sacroiliac Tests

Upper Sacroiliac Mobility. The patient is asked to stand, supporting himself or herself by reaching out to contact the wall or a chair. The doctor stands or sits behind the patient and establishes thumb contacts on the patient's PSIS and second sacral tubercle or ipsilateral sacral base (Figure 5-279). The patient is then instructed to flex his or her ipsilateral hip. This induces flexion of the hip and sacroiliac joint. This procedure may be done with the patient's knee either flexed or straight. If the patient is instructed to keep his or her knee bent during hip flexion, instruct the patient to raise the hip to approximately 90 degrees. If the patient is instructed to keep his or her leg straight, instruct the patient to raise his or her leg to approximately 45 degrees.

With normal movement, the doctor's thumbs approximate as the PSIS moves posteriorly and inferiorly toward the relatively stationary second sacral tubercle (see Figure 5-279, *A*). Sacroiliac flexion restrictions should be suspected when the thumbs do not approximate and the pelvis rotates obliquely around the opposite hip.

After flexion has been evaluated, the patient is instructed to raise the contralateral leg to a level above 90 degrees. This induces posterior nodding of the sacral base and sacroiliac extension on the side of palpation. With normal movement, the doctor's thumbs move apart as the PSIS moves anteriorly and superiorly away from the second sacral tubercle (Figure 5-279, *B*).

Lower Sacroiliac Mobility. To assess mobility of the lower sacroiliac joint, the palpation contacts are moved down to the sacral apex and adjacent ischium (Figure 5-280). The patient is again instructed to raise the ipsilateral leg to approximately 90 degrees. Flexion in the lower joint is assessed, and the doctor's thumbs should separate as the ischium moves anteriorly and superiorly,

relative to the sacral apex (see Figure 5-280, *A*). Extension of the lower sacroiliac joint is evaluated by elevating the opposite leg to a level above 90 degrees. Normal extension of the lower joint is demonstrated by separation of the sacral and ischial contacts (Figure 5-280, *B*).

The standing sacroiliac mobility tests (Gillet tests) have demonstrated poor to mixed reliability and have not been subjected to significant validity testing. They may assist the doctor in refining his or her adjustive approach, but should not be used as a stand-alone procedure for determining whether a patient has pelvic dysfunction.

Sacroiliac Joint Stability Testing

The sacroiliac joint is postulated to have a self-bracing mechanism that is clinically important to proper lumbopelvic function. "The self bracing mechanism is induced through pre-activation of the local muscle system of the lumbopelvic region prior to movement, with subsequent tensioning of the pelvic ligaments, thoracolumbar fascia and compression of the joint surfaces."⁸¹ This mechanism induces a close-packed position of the sacroiliac joint (nutation of sacrum and relative posterior rotation of the innominate) to assist in load transverse.

Clinical assessment of sacroiliac stability has been developed (the Stork test). The Stork test is a modification of the standing SI motion test (the Gillet test). During the single-leg standing support phase, the doctor palpates the PSIS and the second sacral tubercle. In a stable sacroiliac joint, the self-bracing mechanism moves the PSIS in a posteroinferior direction or maintains the PSIS in a neutral position. An unstable joint is indicated by anterosuperior movement of the PSIS (positive test). The Stork test has demonstrated good reliability in one study, but has not been tested for validity.⁸¹

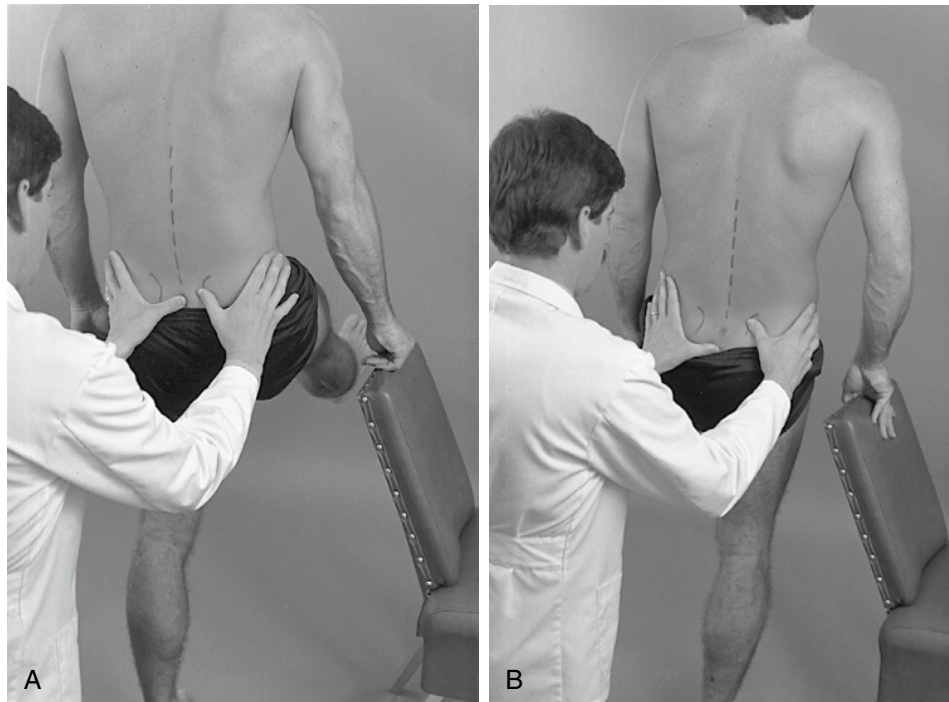
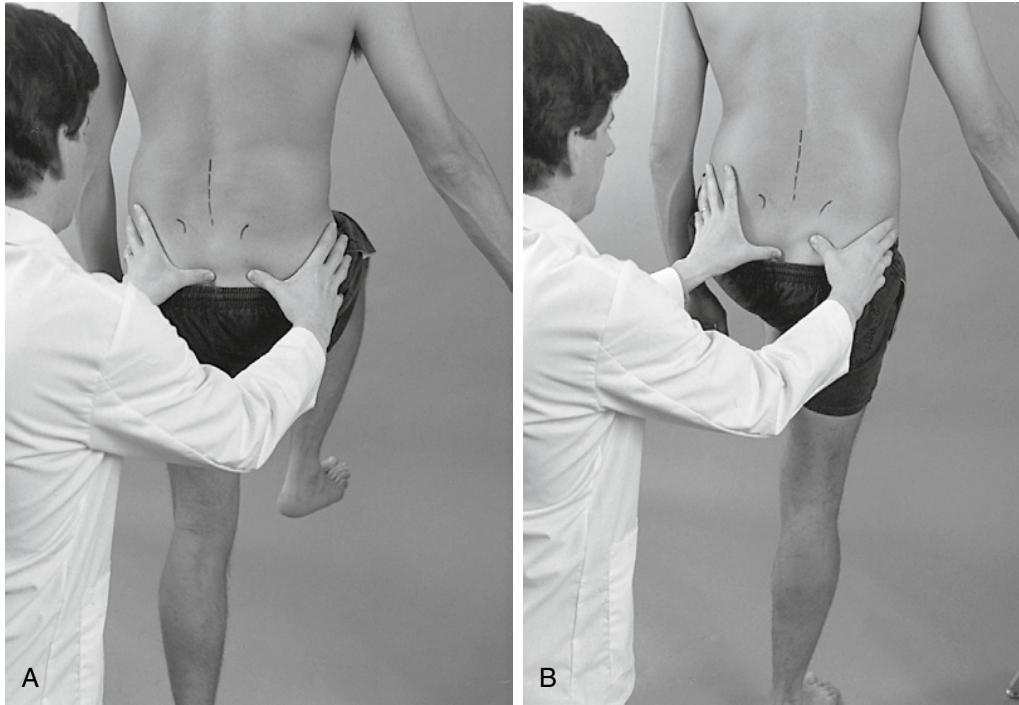


Figure 5-279 Standing sacroiliac joint evaluation for right upper joint movement. After thumb contacts are established over the right posterosuperior iliac spine and sacral tubercle, the patient flexes the ipsilateral hip to assess sacroiliac (SI) joint flexion (approximation of the thumbs) (*A*) and the contralateral hip to assess SI joint extension (separation of thumbs) (*B*).



5-280

Figure 5-280 Standing sacroiliac joint evaluation for right lower joint movement. After thumb contacts are established over the right sacral apex and the soft tissue lateral to the sacral apex in line with the posterosuperior iliac spine, the patient flexes the ipsilateral hip to assess SI joint flexion (separation of thumbs) (A) and the contralateral hip to assess SI joint extension (separation of thumbs) (B).

Pubic Symphysis Dysfunction

The symphysis is not a synovial joint and, as such, does not demonstrate significant movement. However, some degree of shifting and shearing movement at the symphysis is probably present with locomotion and occurs by virtue of its fibrocartilaginous structure. As a result, dysfunction at this joint may contribute to pelvic dysfunction and pain. The question then remains: If the symphysis is capable of contributing to sacroiliac dysfunction, how does a doctor determine if the symphysis should be manipulated?

Although methods for assessing glide are presented, it is doubtful that mobility at the symphysis is palpable. Therefore, pain on stress testing may be more indicative of dysfunction than misalignment and mobility testing. The final decision as to whether pubic symphysis dysfunction is present is a clinical judgment based on the physical findings and the patient's prior response to treatment. In cases that are not responding as expected, it may be appropriate to institute a course of trial manipulation of the symphysis pubis after contraindications have been ruled out.

Evaluation of the Pubic Symphysis

Palpation of the pubic articulation must be approached cautiously because of its proximity to the genitalia and its sensitivity to pressure. Palpation of the pubic bone and symphysis can occur through light clothing or a gown. The pubic symphysis should be accessed from the superior direction and its location can be aided by having the patient locate the pubic bone before palpation.

To palpate the pubic symphysis, place the patient in the supine position. Begin by accessing the ASISs of the ilium with the fingertips. Meet the thumbs in the midline superior to the pubic symphysis and proceed inferiorly until the symphysis is contacted.

Once the superior margins of the pubic bone are located, the doctor may palpate the anterior aspect of each pubic bone with the thumbs or index fingers of both hands (Figure 5-281).

After locating the pubic bone, the doctor should proceed to evaluate alignment and the joints' response to gentle provocation. Alignment should be assessed for changes in A-P and S-I orientation from one side to the other. Joint provocation is performed by applying gentle A-P and S-I springing movements across the joint. Finally, ask the patient to actively tilt the pelvis sideways or, alternately, elongate and shorten the legs as you palpate for subtle shifting between the two pubic bones. Any direction that causes pain, as well as any movement abnormality, may indicate disease or dysfunction in the pubic articulation.

Evaluation of the Coccyx

External evaluation of the coccyx and its articulation is performed with the patient in a prone position. Begin by palpating the sacrococcygeal joint space for abnormal alignment and tenderness. Apply P-A stress to the sacrococcygeal joint with double thumbs to induce JP movement.

Internal palpation of the coccyx for misalignment or tenderness may be performed in the prone or side-lying position. When performing this evaluation, use the same standards of patient draping and technique that would be used in a digital rectal examination. When patients of the opposite gender are evaluated, it is standard of care that an assistant be present.

With internal palpation, the coccyx can be grasped between the index finger and thumb of the doctor and evaluated for A-P glide and tenderness. Internal coccyx evaluation is most helpful in identifying anterior deviation of the coccyx, posterior glide



Figure 5-281 Palpation of the pubic symphysis for pain and displacement. **A**, Anterior-to-posterior pressure is applied to assess anterior displacement and posterior glide. **B**, Superior-to-inferior and inferior-to-superior pressure is applied to assess for superior or inferior displacement and glide.

restrictions, or tenderness of myofascial attachments to its anterior and lateral surfaces. Misalignment with tenderness or loss of JP is characteristic of dysfunction.

OVERVIEW OF PELVIC ADJUSTMENTS

Movement at the sacroiliac joint occurs primarily in the semisagittal plane along the angle of the joint's articular surface.^{76,77,79,84} These movements occur primarily during locomotion and when changing from supine to sitting positions or from sitting to standing positions. They involve movements of the ilium and sacrum in reciprocally opposing directions⁸² and have been referred to previously as movements of flexion and extension.^{78,79}

To treat sacroiliac flexion or extension dysfunction, adjustive contacts may be applied on the ilium or the sacrum. Flexion dysfunction is treated by contacting the ilium and inducing posteroinferior movement of the ilial articular surface relative to the ipsilateral sacral articular surface or by contacting the sacrum and inducing anterior and inferior movement of the sacral articular surface relative to the ipsilateral ilial articular surface.

Extension dysfunction is induced by contacting the ilium and inducing anterosuperior movement of the ilial articular surface relative to the ipsilateral sacral articular surface or by contacting the sacrum and inducing posterior and superior movement of the sacral articular surface relative to the ilial articular surface. These adjustments may be applied in a variety of patient positions and may incorporate methods that establish adjustive contacts on both sides of the articular surfaces.

Side-Posture Pelvic Adjustments

Side-posture sacroiliac adjustments are the most common manipulative methods used for treating sacroiliac joint dysfunction. Like lumbar side-posture adjustments, they offer flexibility in patient position and added leverage. They can, however, produce unwanted rotational tension in the lumbar spine. This can be minimized by limiting counter-rotation of the patient's shoulders and by emphasizing traction and tension on the sacroiliac joint through the contact hand.

Prone Pelvic Adjustments

Prone pelvic adjustments can be appropriate alternatives to side-posture pelvic adjustments. They do not produce the preadjustive joint distraction of side-posture adjustments, but they may be more appropriate for situations in which a side-posture position would unduly stress the lumbar spine.

Symphysis Adjustments

The adjustive procedures that are applied to the pubic symphysis often use an initial patient contraction of specific muscles against resistance supplied by the doctor. The muscle contraction first pulls on the symphysis in the desired direction and when, or if, tolerated by the patient, an impulse thrust is applied. This is not a synovial joint, and cavitation is not likely. Sometimes a noise that indicates a soft tissue release or tendinous snap will be heard or felt.

Coccyx Adjustments

Coccygeal pain may occur after a fall or childbirth and can be very persistent. The benefits of coccygeal adjustment will most likely be realized either very quickly or not at all. The external contact uses a tissue-pull procedure and is probably less effective but more comfortable for the patient. The internal contact has the doctor placing a finger intrarectally to establish a contact on the anterior aspect of the coccyx. Use of latex gloves is necessary to provide a sanitary barrier. Appropriate patient gowning and draping as well as a thorough explanation of the procedure is important to maintain professional boundaries. When this procedure is performed on patients of the opposite sex, it is standard of care that an assistant be present.

PELVIC ADJUSTMENTS (BOX 5-10)

Side-Posture

Hypothenar/Ilium Push (PI Ilium) (Figure 5-282)

IND: Restricted sacroiliac extension. Flexion malposition of the ilium (PI).

PP: The patient lies in the basic side-posture position.

BOX 5-10 Pelvic Adjustments

- Side posture
 - Hypothenar/ilium push (PI ilium) (Figure 5-282)
 - Hypothenar/sacral base push (PS sacrum) (Figure 5-283)
 - Hypothenar/ischium push (AS ilium) (Figure 5-284)
 - Hypothenar/sacral apex push (AI sacrum)
- Prone
 - Hypothenar/ilium sacral apex push (PI ilium or AI sacrum) (Figure 5-285)
 - Hypothenar/ischium sacral base push (AS ilium or PS sacrum) (Figure 5-286)
 - Hypothenar/ilium push with hip extension (PI ilium) (Figure 5-287)

AI, Anteroinferior; AS, anterosuperior; PI, posteroinferior; PS, posterosuperior.

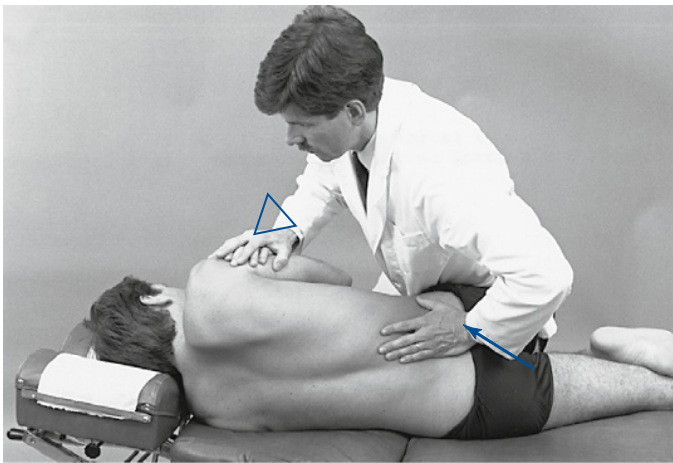
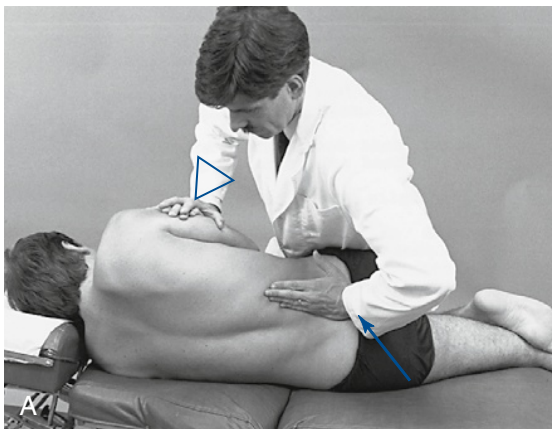


Figure 5-282 Hypothenar contact established over the right posterosuperior iliac spine to induce extension of the right sacroiliac joint (posteroinferior ilium).

DP: Stand in a fencer stance, angled approximately 45 degrees to the patient. Support the patient's pelvis by contacting the patient's thigh with your thigh or by straddling the patient's bent upper leg between your thighs.



CP: Hypothenar of caudal hand.

SCP: Medial margin of the PSIS.

IH: Your IH contacts the patient's up-side shoulder and overlapping hand.

VEC: P-A, M-L, and I-S.

P: Place the patient in side posture, with the involved side up. Flex the upper thigh to between 60 and 80 degrees. Establish the ilial and leg contacts and develop preadjustive tension by distracting and extending the involved sacroiliac joint.

Produce joint distraction by lowering your body weight on to the patient's lateral thigh. Produce joint extension by leaning your torso P-A, I-S into the contact. The IH stabilizes the patient's shoulder and applies gentle traction cephalically and posteriorly. Take care to avoid excessive posterior rotation of the patient's upper torso. At tension, generate a thrust by dropping upper body weight into the contact. Medial to lateral pressure against the PSIS is an important component of this procedure as it assists in distracting the sacroiliac joint.

Hypothenar/Sacral Base Push (PS Sacrum) (Figure 5-283)

IND: Restricted sacroiliac flexion; unilateral PS malposition of the sacrum.

PP: The patient lies in the basic side-posture position.

DP: Stand in a fencer stance, angled approximately 45 degrees to the patient. Support the patient's pelvis by contacting the patient's thigh with your inferior thigh or by straddling the patient's bent upper leg between your thighs.

CP: Hypothenar of the caudal hand.

SCP: Superior sacral base, just medial to the PSIS on the side of sacroiliac dysfunction.

IH: Your IH contacts the patient's up-side shoulder and overlapping hand.

VEC: P-A and slightly I-S.

P: Place the patient in side posture, with the dysfunctional sacroiliac joint against or away from the table. Flex the upper thigh to between 60 and 80 degrees. Establish the ilial and leg contacts and develop preadjustive tension. The IH stabilizes the patient's shoulder and applies gentle traction cephalically and posteriorly. Take care to avoid excessive posterior rotation of the patient's upper torso.

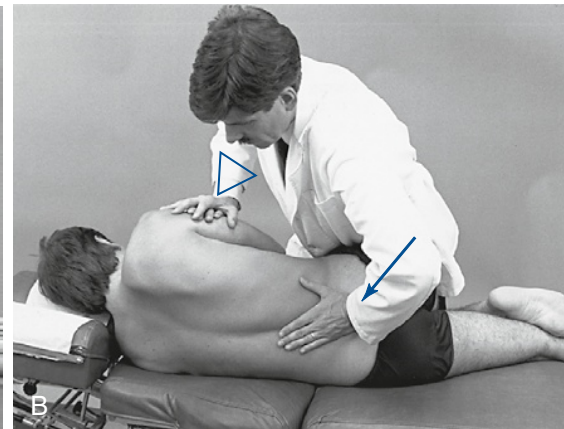


Figure 5-283 Hypothenar contact established over the right (A) and left (B) side of the sacral base to induce sacroiliac flexion by inducing posterior-to-anterior movement of the sacral base on the side of contact.

Dysfunctional side up: The patient lies on the side opposite the dysfunctional sacroiliac joint. Induce sacroiliac flexion by pushing the sacral base forward. Produce sacroiliac joint distraction by lowering your body weight through the thigh contact (Figure 5-283, *A*). At tension, generate a thrust by dropping your body weight into the sacral contact (thenar contact may be substituted for the hypothenar contact).

Dysfunctional side down: The patient lies on the side of sacroiliac joint dysfunction. Establish the adjustive contact on the sacral base on the down-side and rotate the patient slightly farther toward you. At tension, generate an anteriorly directed thrust body drop, along the plane of the sacroiliac articulation (see Figure 5-283, *B*). This method may be preferable to dysfunctional-side-up adjustments because of the stability and resistance provided by placing the dysfunctional joint against the table and the more effective positioning of the doctor's body weight over the joint.

Hypothenar/Ischium Push (AS Ilium) (Figure 5-284)

IND: Restricted sacroiliac flexion. Extension malposition of the ilium (AS).

PP: The patient lies in the basic side-posture position.

DP: Stand in a low fencer stance, straddling the patient's flexed upper knee. Support the patient's flexed leg against the proximal anterior thigh of your caudal leg.

CP: Soft broad hypothenar contact of the caudad hand, with the fingers spread and pointing cephalad (see Figure 5-284, *A*).

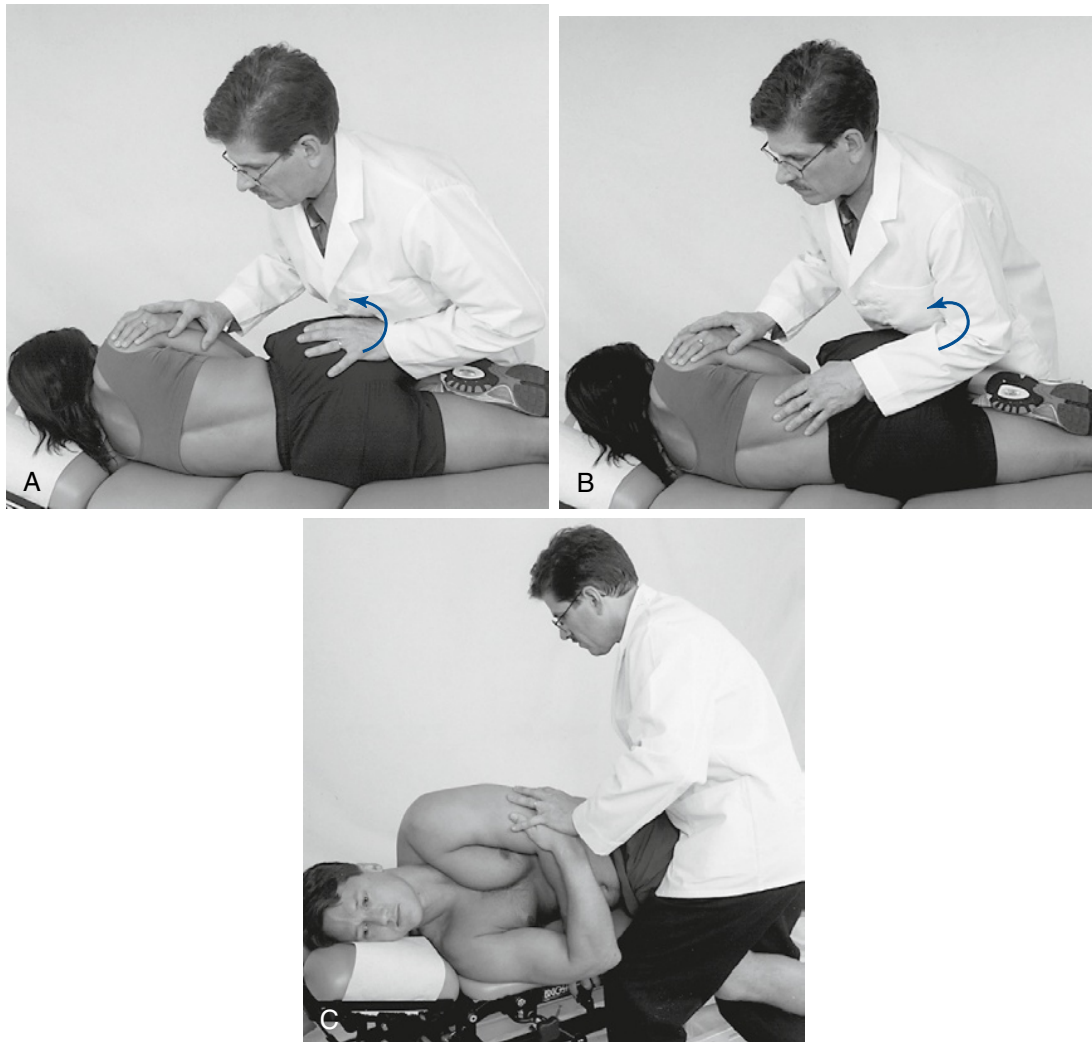
SCP: Medial inferior ischium.

IH: The cephalad hand stabilizes the patient's upper shoulder, maintaining spinal flexion.

VEC: P-A.

P: Place the patient in side posture, with the involved side up. Slightly flex the patient's trunk and flex the upper thigh to 90 degrees or more. Establish a broad soft contact along the inferior and medial margin of the ischium.

Preadjustive tension is produced by inducing sacroiliac distraction and flexion. Sacroiliac distraction is induced by dropping the patient's flexed thigh toward the floor and by lowering the doctor's body weight against the patient's flexed hip. Sacroiliac flexion is induced by maintaining inferior directed pressure with the contact hand as the doctor directs cephalic pressure against the patient's flexed leg with his caudal leg.



5-284A, B

Figure 5-284 To induce flexion (anterosuperior ilium) of the right sacroiliac joint, a hypothenar contact (**A**) or a forearm contact (**B**) is established over the posterior aspect of the ischial tuberosity (**C**). The doctor's leg contacts are modified in this adjustment to a straddle-leg position.

At tension, a body-centered thrust is delivered anteriorly along the shaft of the femur through the trunk and lower extremities. Your lower abdomen may impact the patient's posterior lateral buttock and hip. A slight variation of this procedure involves using a forearm contact instead of a hypothenar contact (Figure 5-284, *B*). Optional doctor stances and leg contacts may also be used in this adjustment (Figure 5-284, *C*).

Hypothenar/Sacral Apex Push (AI Sacrum) (Figure 5-285)

IND: Restricted sacroiliac extension or unilateral anteroinferior malposition of the sacrum.

PP: The patient lies in the basic side-posture position.

DP: Stand in a low fencer stance, straddling the patient's flexed upper knee. Support the patient's flexed leg against the proximal anterior thigh of your caudal leg.

CP: Hypothenar of the caudal hand, with the fingers pointing cephalad (Figure 5-285, *A*).

SCP: Apex of the sacrum.

IH: Your superior hand stabilizes the patient's upper shoulder, maintaining spinal flexion.

VEC: P-A.

P: Place the patient in side posture, with the involved side down. Slightly flex the patient's trunk and flex the upper thigh above 90 degrees. Establish a broad soft contact along the up-side of the sacral apex.

Develop preadjustive tension by inducing lumbosacral flexion and forward rotation of the pelvis. This produces posterior

nodding and rotation of the sacral base on the down-side and extension of the down-side sacroiliac joint. Induce anterior pelvic rotation by dropping the patient's flexed thigh toward the floor and by lowering your body weight against the patient's flexed hip. Induce posterior nodding of the sacral base by pulling inferiorly with the contact hand against the sacral apex as you push cephalically against the patient's flexed leg with your caudal leg.

At tension, deliver a body-centered thrust anteriorly through the trunk, lower extremities, and shoulder. Your lower abdomen may impact the patient's posterolateral buttock and hip. A slight variation of this procedure involves using a forearm contact instead of a hypothenar contact (Figure 5-285, *B*).

Prone

Hypothenar/Ilium Sacral Apex Push (PI Ilium or AI Sacrum) (Figure 5-286)

IND: Restricted sacroiliac extension. Posteroinferior malposition of the ilium or unilateral anteroinferior malposition of the sacrum.

PP: The patient lies prone.

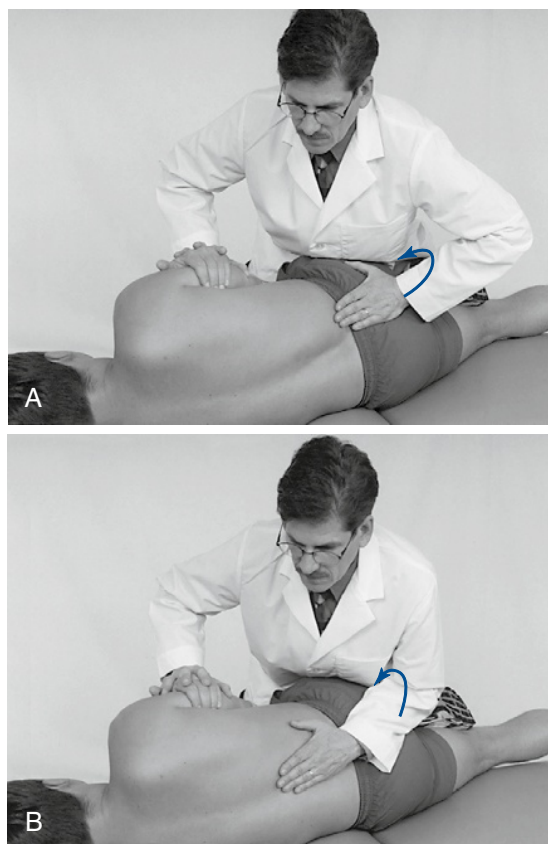


Figure 5-285 Hypothenar contact (**A**) or a forearm contact (**B**) is established over the sacral apex to induce extension of the left sacroiliac joint.



Figure 5-286 Adjustment for restricted extension in the left sacroiliac joint using bilateral contacts over the left posterosuperior iliac spine (PSIS) and sacral apex (**A**) and a unilateral contact over the PSIS (**B**).

DP: Stand in a modified fencer stance on the side opposite the dysfunction.

CP: Hypothenar contacts of both hands.

SCP: Medial superior margin of the PSIS and sacral apex (see Figure 5-286, *A*).

VEC: P-A, I-S, and M-L with the PSIS contact. P-A, S-I, and M-L with the sacral apex contact.

P: Position the patient in the prone position. Reach across the patient with the caudal hand and establish a hypothenar contact on the contralateral PSIS. With the cephalic hand, reach inferiorly to establish a contact on the sacral apex (Figure 5-286, *A*).

Develop preadjustive tension by leaning anteriorly and superiorly with the iliac contact and anteroinferiorly with the sacral contact. A pelvic block may be positioned under the greater trochanter to assist in the development of sacroiliac extension. At tension, deliver a high-velocity thrust combined through the arms, trunk, and body.

When using this adjustment with a drop table, place the patient's ASISs in the break between the adjusting table's pelvic and lumbar sections. The adjustive thrust should not be delivered until appropriate drop-piece tension has been established.

If desired, a PI ilium or sacroiliac extension restriction may be treated with a unilateral contact on the PSIS. With this method, you may stand on either side of the patient (Figure 5-286, *B*).

Hypothenar/Ischium Sacral Base Push (AS Ilium or PS Sacrum) (Figure 5-287)

IND: Restricted sacroiliac flexion. AS malposition of the ilium or unilateral PS malposition of the sacrum.

PP: The patient lies prone.

DP: Stand in a modified fencer stance on the side opposite the dysfunction.

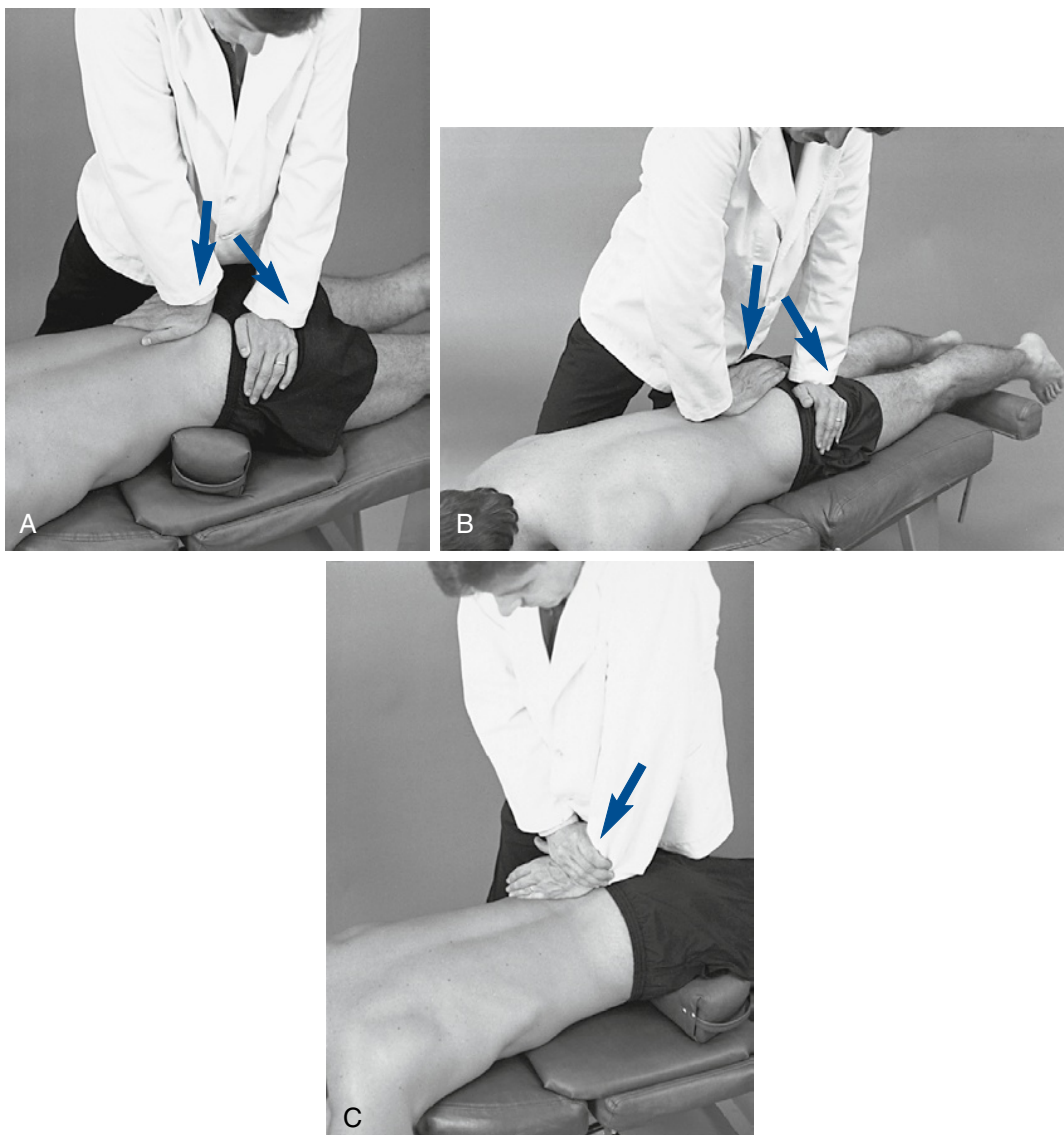


Figure 5-287 Adjustment for restricted flexion of the left sacroiliac joint, using contacts over the left ischium and sacral base with blocks (*A*) or without blocks (*B*). *C*, Unilateral sacral base contact.

CP: Proximal palmar surface of the caudal hand for ischial contact and thenar or hypothenar eminence of cephalic hand for sacral base contact (Figure 5-287, *A* and *B*).

SCP: Inferior ischium or anterior ilium and proximal sacral base.

VEC: P-A and S-I with ischial contact. P-A and I-S with sacral contact.

P: Position the patient in the prone position. Reach across the patient with the caudal hand and establish a palmar contact on the inferior ischium. With the thenar or hypothenar of the cephalic hand, contact the superior margin of sacral base just medial to the PSIS on the same side.

Develop preadjustive tension by leaning anteroinferiorly with the ischial contact and anteriorly and superiorly with the sacral contact. A pelvic block may be positioned under the ASIS to assist in the development of sacroiliac flexion (see Figure 5-287, *A*).

At tension, deliver a high-velocity combined thrust through the arms, trunk, and body.

When using this adjustment with a drop table, place the patient's ASISs at the superior end of the table's pelvic section. The adjustive thrust should not be delivered until appropriate drop-piece tension has been established.

If desired, a posterosuperior sacral base or sacroiliac flexion restriction may be treated with a unilateral contact on the involved PS sacral base. With this method, you may stand on either side of the patient (see Figure 5-287, *C*).

Hypothenar/Ilium Push with Hip Extension (PI Ilium) (Figure 5-288)

IND: Restricted sacroiliac extension. PI malposition of the ilium.

PP: The patient lies prone.

DP: Stand in a fencer stance on the side opposite the dysfunction.

CP: Hypothenar of cephalic hand.

SCP: MS margin of the PSIS.

IH: The caudal hand grasps the patient's distal thigh on the same side as the adjustive contact.

VEC: P-A, I-S, and M-L.

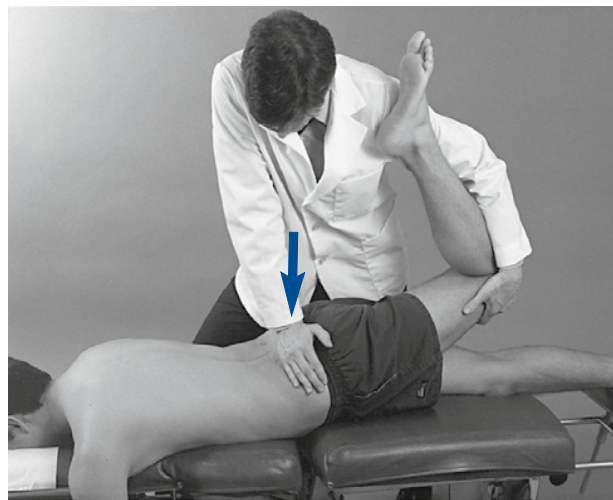


Figure 5-288 Contact over the left posterosuperior iliac spine while extending the hip to induce extension of the left sacroiliac joint.

P: Position the patient in the prone position. Reach across the patient with the cephalic hand and establish a hypothenar contact on the contralateral PSIS. With the caudal hand, reach across the patient and grip the patient's contralateral thigh just proximal to the knee.

Develop joint tension by leaning into the contact and gently extending the patient's thigh. The patient's knee on the elevated side may be flexed or remain extended. Accompanying hip extension is minimal and should not elevate the patient's pelvis off the table.

At tension, deliver an impulse thrust through the ilial contact, reinforced by a shallow lift with the thigh contact. The thrust through the hip must be shallow. It is possible to overextend the hip, damaging the capsule and its adjacent soft tissues. This adjustment is contraindicated in patients with hip pathology or meralgia paresthetica.

When using this adjustment with a drop table, place the patient's ASISs in the break between the adjusting table's pelvic and lumbar sections. The adjustive thrust should not be delivered until appropriate drop-piece tension has been established.

Prone Pelvic Blocking (Figure 5-289)

IND: AS or PI malposition of the ilium.

PP: The patient lies prone with a firm surface (padded board) under the pelvic area.

DP: The doctor has a passive role. Place padded wedges (pelvic blocks) under both sides of the pelvis.

CP: Padded wedge (pelvic block).

SCP: ASIS and anterior aspect of hip.

P: Position the patient in the prone position on a padded board. Place a padded wedge under the ASIS on the side of the anterior innominate and another under the anterior aspect of the



Figure 5-289 Prone pelvic blocking for a left anterosuperior and right posteroinferior distortion pattern, using gravity over time to induce movement at the sacroiliac joints. There is no thrust.

hip on the side of the posterior innominate to develop sacroiliac flexion.

No thrust is given; gravity provides the force applied over time. This procedure does not fit the definition of an HVLA adjustment because of the lack of a high-velocity thrust.

PUBIC SYMPHYSIS ADJUSTMENTS (BOX 5-11)

Box 5-11 Pubic Symphysis Adjustments

- Supine
 - Hypothenar/Thigh (Figure 5-290)
 - Hypothenar/Pubis (Figure 5-291)
 - Hypothenar/Ilium, Palmar/Ischium (Figure 5-292)
 - Pubic Distraction (Figure 5-293)

Supine

Hypothenar/Thigh (Superior Pubis) (Figure 5-290)

IND: Restricted inferior glide and superior malposition of the pubis.

PP: The patient lies supine, with the side of involvement at the edge of the table. The corresponding leg hangs off the table. The PSIS is just on the table.

DP: Stand on the involved side, facing caudad in a fencer's stance.

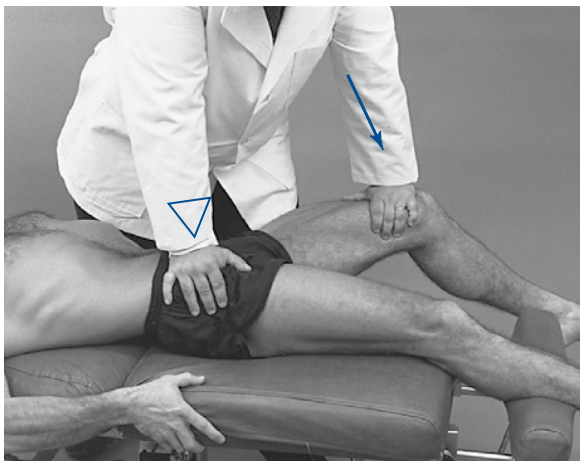
CP: A palmar contact of the caudal hand.

SCP: Distal femur of the leg on the involved side.

IH: The cephalad hand establishes a palmar contact over the ASIS on the uninvolved side.


VEC: S-I.

P: While stabilizing the pelvis with the IH, the contact hand applies an A-P stress on the patient's thigh. Ask the patient to attempt to raise the thigh against the resistance and after 4 to 5 seconds, deliver a slight and shallow impulse thrust downward to the distal thigh. After the adjustment is applied, the patient should relax, maintaining the adjustive position for approximately 1 to 2 minutes.



 5-290 **Figure 5-290** Left superior pubis adjustment.



 5-291 **Figure 5-291** Right anterior pubis adjustment; also an alternative for a right superior pubis adjustment.

Hypothenar/Pubis (Anterior Pubis) (Figure 5-291)

IND: Restricted posterior glide and anterior malposition of the pubis (alternate procedure for superior pubis).

PP: The patient lies supine, with the uninvolved knee and hip flexed and the foot flat on the table.

DP: Stand on the uninvolved side, facing obliquely caudad.

CP: A hypothenar knife-edge contact of the cephalad hand.

SCP: Anterior aspect of the involved pubic ramus (superior aspect for superior pubis).

IH: The caudal hand either reinforces the contact hand with the fingers wrapped around the wrist or establishes a palmar contact over the distal thigh of the uninvolved leg and applies additional flexion stress.

VEC: A-P.

P: At tension, deliver a quick and shallow impulse thrust A-P to the involved pubis. This technique is best done with the use of a pelvic drop section.

Hypothenar/Ilium, Palmar/Ischium (Inferior Pubis) (Figure 5-292)

IND: Restricted superior glide of the pubis. Inferior malposition of the pubis.



 5-292 **Figure 5-292** Right inferior pubis adjustment.

PP: The patient lies supine, with the knee and hip on the involved side fully flexed.

DP: Stand on the side of the table opposite the side of involvement, leaning over the patient, with your upper body contacting the anterior aspect of the patient's tibia.

CP: The caudal hand grasps the patient's lower ischium while the cephalad hand reaches over the patient and contacts the ASIS on the side of involvement.

VEC: P-A and I-S with the caudal hand. A-P with the cephalic hand.

P: Develop preadjustive tension by applying body weight to the patient's flexed leg. At tension, deliver a thrust with the caudal hand while the cephalic hand thrusts posteriorly through your torso while generating a pulling thrust.

Pubic Distraction (Figure 5-293)

IND: Pubic dysfunction, pubic symphysis pain with palpation, and joint provocation.

PP: The patient lies supine, flexing both knees and hips and resting the feet flat on the table, close to one another.

DP: Stand at or kneeling on the foot end of the table, facing the patient.

CP: Palmar contacts of both hands.

SCP: Medial aspects of both knees.

VEC: M-L.

P: Separate the patient's knees and grasp the medial aspects of the patient's knees. With crossed forearms between the knees, ask the patient to squeeze the knees together for several seconds or until sufficient adductor muscle fatigue occurs. Then deliver a shallow impulse thrust to both knees.



5-293

Figure 5-293 Pubic distraction.

COCCYX ADJUSTMENTS (BOX 5-12)

Box 5-12 Coccyx Adjustments

- Prone
 - Thumb/External Coccyx Push (Figure 5-294)
 - Index/Internal Coccyx Pull (Figure 5-295)

Prone

Thumb/External Coccyx Push (Figure 5-294)

IND: Restricted coccyx movement, malposition of the coccyx, and coccygodynia.

PP: The patient lies in the prone position, with the thoracic and pelvic pieces raised or a Dutchman roll placed under the ASIS. The buttocks should be appropriately draped.

DP: Stand at the side of the table, assuming a fencer's stance and facing cephaladly.

CP: The thumb contact of the cephalad hand.

SCP: The base of the coccyx (skin to skin).

IH: The pisiform-hypothenar contact of the caudad hand is established over the thumbnail of the contact hand, with the fingers lying loosely over the dorsum of the contact hand.

VEC: I-S.

P: Draw tissue slack out in a cephalad direction with both hands. At tension, deliver a cephalad and slightly P-A impulse thrust over the coccygeal base, producing a mixed tissue pull and osseous adjustive technique.

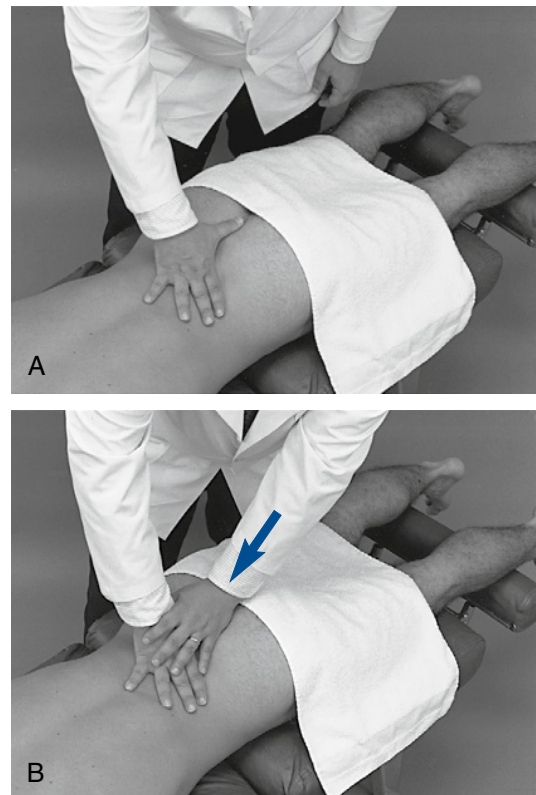


Figure 5-294 External coccyx adjustment. **A,** Tissue pull, with thumb contact taken. **B,** Reinforced contact, with thrust headward.

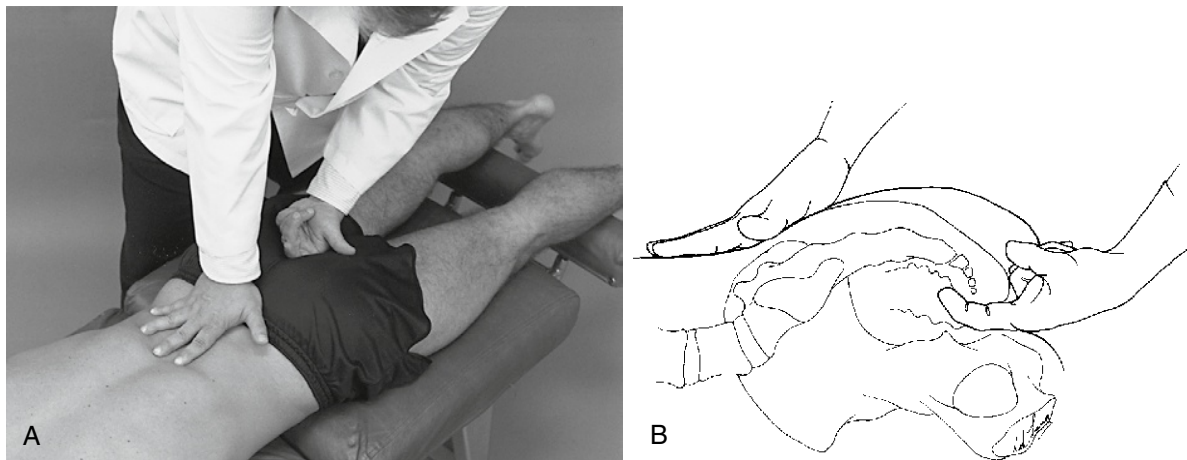


Figure 5-295 A, Simulation of an internal coccyx adjustment. B, Illustration of contacts.

Index/Internal Coccyx Pull (Figure 5-295)

IND: Restricted coccyx movement, malposition of the coccyx, and coccygodynia.

PP: The patient lies in the prone position, with the thoracic and pelvic pieces raised or a Dutchman roll placed under the ASIS.

DP: Stand at the side of the table, assuming a fencer's stance and facing cephaladly.

CP: A digital contact, with a gloved and lubricated middle finger of the caudad hand. A palmar-calcaneal contact of the cephalad hand over the upper half of the sacrum.

SCP: The anterior surface of coccyx, intrarectally.

IH: A palmar-calcaneal contact of the cephalad hand over the upper half of the sacrum.

VEC: S-I.

P: The intrarectal contact applies tension to the coccyx inferiorly and slightly posteriorly. Deliver a gentle impulse thrust through the contact on the sacrum while simultaneously delivering a very gentle and shallow posterior thrust against the coccyx.

OUTLINE

ROLE OF THE PERIPHERAL

JOINTS

283

TEMPOROMANDIBULAR JOINT

283

Functional Anatomy

284

Biomechanics

286

Evaluation

288

Adjustive Procedures

291

SHOULDER

294

Functional Anatomy

294

Biomechanics

298

Evaluation

298

Adjustive Procedures

302

ELBOW

Functional Anatomy

315

Biomechanics

316

Evaluation

317

Adjustive Procedures

319

WRIST AND HAND

322

Functional Anatomy

326

Biomechanics

326

Evaluation

327

Adjustive Procedures

329

HIP

333

Functional Anatomy

337

Biomechanics

337

Evaluation

344

Adjustive Procedures

346

KNEE

349

Functional Anatomy

350

Biomechanics

353

Evaluation

354

Adjustive Procedures

358

ANKLE AND FOOT

364

Functional Anatomy

365

Biomechanics

367

Evaluation

368

Adjustive Procedures

371

ROLE OF THE PERIPHERAL JOINTS

From the beginnings of chiropractic, practitioners have treated the nonspinal joints. D.D. Palmer¹ described his treatment of the feet in 1910. Applying manual procedures to the peripheral joints is a skill that is taught in all accredited chiropractic programs.

The extremity joints are lever-hinge complexes that translate forces into motion, but in so doing, can also amplify forces negatively to the neuromusculoskeletal (NMS) system. The mechanical principles that determine what functions the body can perform are the same, regardless of the activity, whether it's an athletic, recreational, occupational, or everyday task.

Understanding biomechanics (the application of mechanical laws to living structures) is paramount when confronted with a clinical dysfunctional process affecting the musculoskeletal system and, specifically, a peripheral joint. The joint, its ligamentous structures, and its capsule form the hinge for the bony lever to move about when a force from the muscles is provided. Proper joint function depends on the integrity of the soft tissues and the alignment of the bony joint surfaces.

By incorporating biomechanical principles in clinical practice, the chiropractor will better understand the nature and extent of the manipulable lesion, as well as the way the patient's NMS system is influenced locally and globally. Moreover, mechanisms for the dysfunctional changes will become apparent, as will the extent and effect of the patient's adaptational process.

Impairments involving the muscles, ligaments, and joints of the extremities can significantly reduce the quality or the ease of performing many important activities related to personal care, livelihood, and recreation. The rationale for treating peripheral joints includes correction of biomechanical problems and reflex-triggered functional syndromes.² Biomechanical problems are characterized primarily by joint pain, but swelling and paresthesia may be present. These problems may be the result of trauma (sprains, strains, athletic injuries, or work injuries) or repetitive activities and postures (carpal tunnel syndrome, plantar fasciitis, or foot pronation).

Reflex-triggered functional syndromes occur through a series of distortions affecting the kinetic chain. They are a result of mechanical deficiencies (short leg) or muscular insufficiencies or deformities (valgus or varus). These problems present as clinical findings associated with specific areas of the body, but the major dysfunctions are located somewhere else. For example, a patient may have acute or recurrent low back pain that appears to be localized to the sacroiliac joints, but treatment to the sacroiliac joints is ineffective. On further examination, a dysfunctional metatarsal joint is found that disturbs normal proprioceptive function, leading to sacroiliac changes.

As is the case with manual procedures applied to the spine, a detailed examination must be conducted to determine the necessity for care and the type of procedures to be applied to the peripheral joints. In addition to joint manipulation and adjustment to the peripheral joints, consideration should be given to the use of soft tissue procedures, hot and cold modalities, stretching exercises, rehabilitation, taping and supports, and orthotics.

It is necessary to assign a clear and specific name to each technique procedure for teaching and testing purposes. The adjustive techniques in this chapter have been given names that are based on the involved joint or region, patient position, contact used by the clinician, body part contacted, and any necessary additional information (e.g., push, pull, with distraction, etc.), as well as the induced joint movement. These names follow the patterns used by the U.S. National Board of Chiropractic Examiners and are designed to be helpful in the teaching and testing for competence of the procedures.

TEMPOROMANDIBULAR JOINT

The weight of the head must be balanced and stabilized atop the spine. A biomechanical relationship exists between the forces developed in stabilization of the cervical spinal segments, tension in the deep cervical fascia, movements of the temporomandibular joints (TMJs), and activity of the hyoid bone muscles, as well as the structures of the shoulder girdle (Figure 6-1). More importantly, postural stresses, muscle tone, malocclusion of the teeth, and joint dysfunction

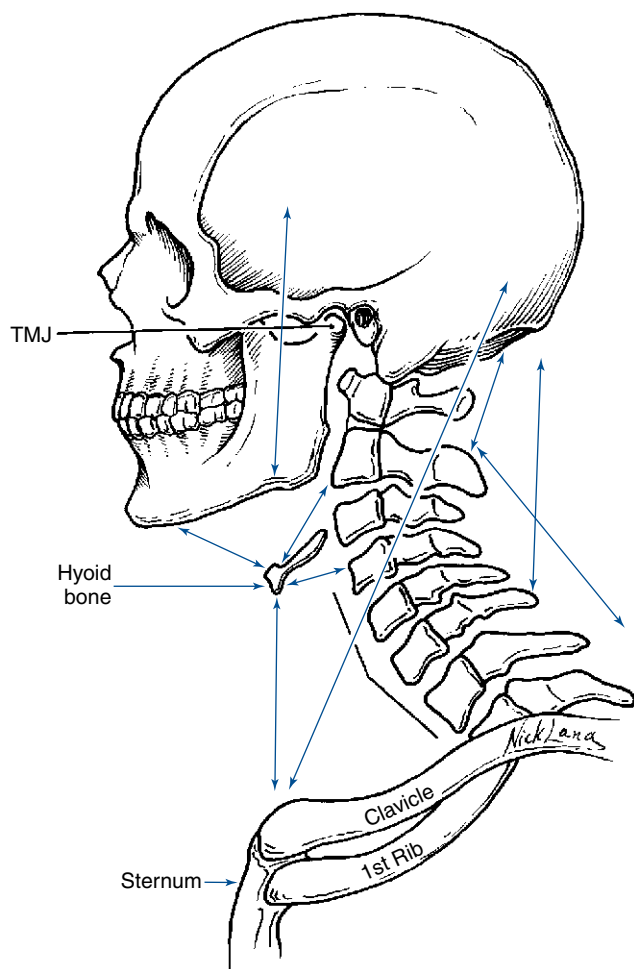


Figure 6-1 Biomechanical relationship necessary to stabilize the head and the cervical spine segments. Arrows indicate direction of muscle pull. (Modified from Grieve G: *Common vertebral joint problems*, ed 2, Edinburgh, 1988, Churchill Livingstone.)

have clinical relationships with neck pain, headache, orofacial pain, and abnormalities of chewing and swallowing. An association is formed between two of the body's most complicated joint systems—the TMJ and the atlanto-occipital joint. Both of these joint systems should be evaluated in patients complaining of head and neck pain.

The craniomandibular complex is composed of the TMJ, the teeth, muscles of mastication, and the hyoid bone. The TMJ is one of the most active joints in the body, moving more than 2000 times per day in its functions of mastication, swallowing, respiration, and speech. The head is tethered to the body by the muscles that move the TMJ and the atlanto-occipital joint. Head posture depends on the tone of these muscles, thus developing an intimate association between movements of the head and mandible. Because of this relationship, a change in either cervical spine posture, head posture, or mandibular rest position can create a change in the others.

FUNCTIONAL ANATOMY

Osseous Structures

The mandible, the largest and strongest bone of the face, articulates with the temporal bones while accommodating the lower teeth (Figure 6-2). The body of the mandible runs horizontally and has two posterior rami. The rami are perpendicular to the body and

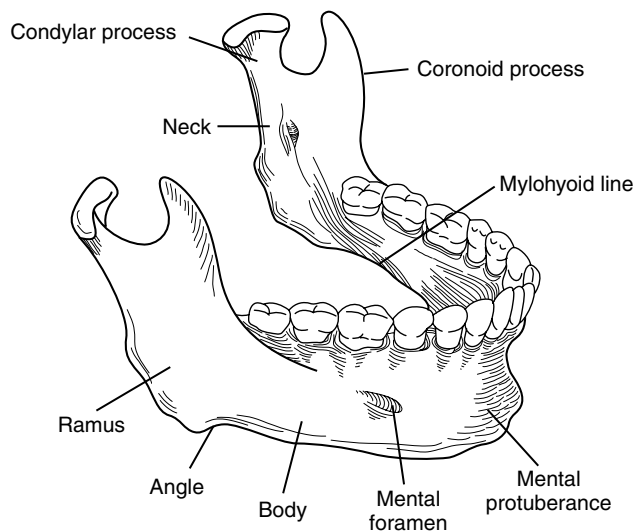


Figure 6-2 The osseous components of the mandible. (Modified from Hertling D, Kessler RM: *Management of common musculoskeletal disorders: Physical therapy principles and methods*, ed 2, Philadelphia, 1990, JB Lippincott.)

form an inferior palpable angle. Each ramus has two processes: the coronoid process, serving as a point of attachment for muscles, and the condylar process, for articulation with the temporal bone via the intra-articular disc. Lines drawn through the axis of each condyle intersect just anterior to the foramen magnum, the significance of which is in visualizing a line of correction for manipulative procedures (Figure 6-3). The temporal bone has a concave mandibular fossa, with the convex articular eminence just anterior to it.

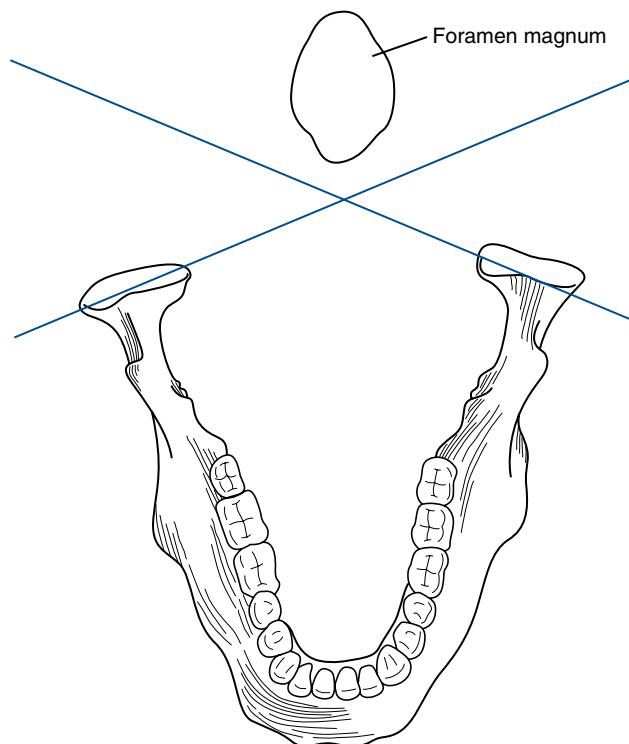


Figure 6-3 Line drawn through the axes of the mandibular condyles will intersect just anterior to the foramen magnum. (Modified from Hertling D, Kessler RM: *Management of common musculoskeletal disorders: Physical therapy principles and methods*, ed 2, Philadelphia, 1990, JB Lippincott.)

Functionally, the mandibular fossa serves as a receptacle for the condyles when the joint is in a closed-packed position (teeth approximated). During opening, closing, protrusion, and retrusion, the convex surface of the condyle must move over the convex surface of the articular eminence (Figure 6-4). The existence of the intraarticular disc compensates functionally for the incongruity of the two opposing convex surfaces.³ The disc also separates the joint into an upper and lower portion or compartment, each with synovial linings. The outer edges of the disc are connected to the joint capsule.

Ligamentous Structures

Four ligaments serve as secondary stabilizers for the joint. They are the articular capsule, temporomandibular ligament, stylomandibular ligament, and sphenomandibular ligament (Figure 6-5). The primary function of the joint capsule is to enclose the joint, but because the disc is tethered to it, the joint capsule also causes the disc to move forward when the condyle moves forward. The temporomandibular ligament is the main suspensory ligament of the

mandible during opening movements of the jaw. It also prevents excessive forward, backward, and lateral movements. The stylo-mandibular ligament prevents excessive anterior movement of the mandible and, as such, serves as a stop for the mandible in extreme opening. The sphenomandibular ligament functions as a suspensory ligament for the mandible during wide opening of the joint.

A mandibular-malleolar ligament connecting the neck and anterior process of the malleus to the medioposterior aspect of the joint capsule has been reported.^{4,5} The clinical significance of this structure lies in making an anatomic connection between the TMJ and the middle ear. The mandibular malleolar ligament passes through the petrotympanic fissure to connect the malleolus to the meniscus and the capsular ligament of the TMJ. The anterior tympanic artery, which is responsible for supplying blood to the tissue around the tympanic membrane, and the chorda tympani nerve, which gives pain sensation to the tongue, also travel through this fissure. Irritation to these structures can cause symptoms such as ear pain, tinnitus, vertigo, subjective hearing loss, hyperacusis, tongue pain, and muscle pain.

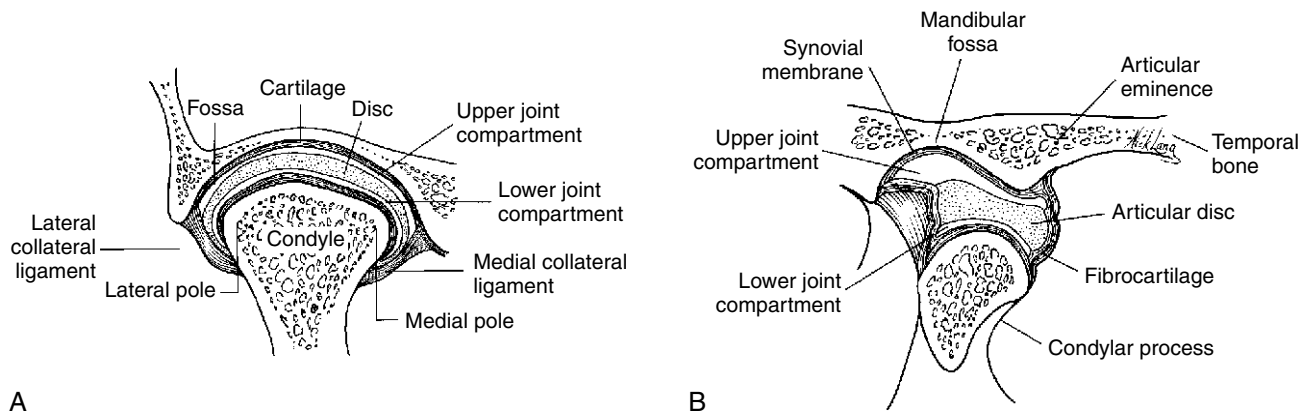


Figure 6-4 A, Coronal (frontal) section through the temporomandibular joint (TMJ) in the closed position. B, Sagittal section through the TMJ in the open position.

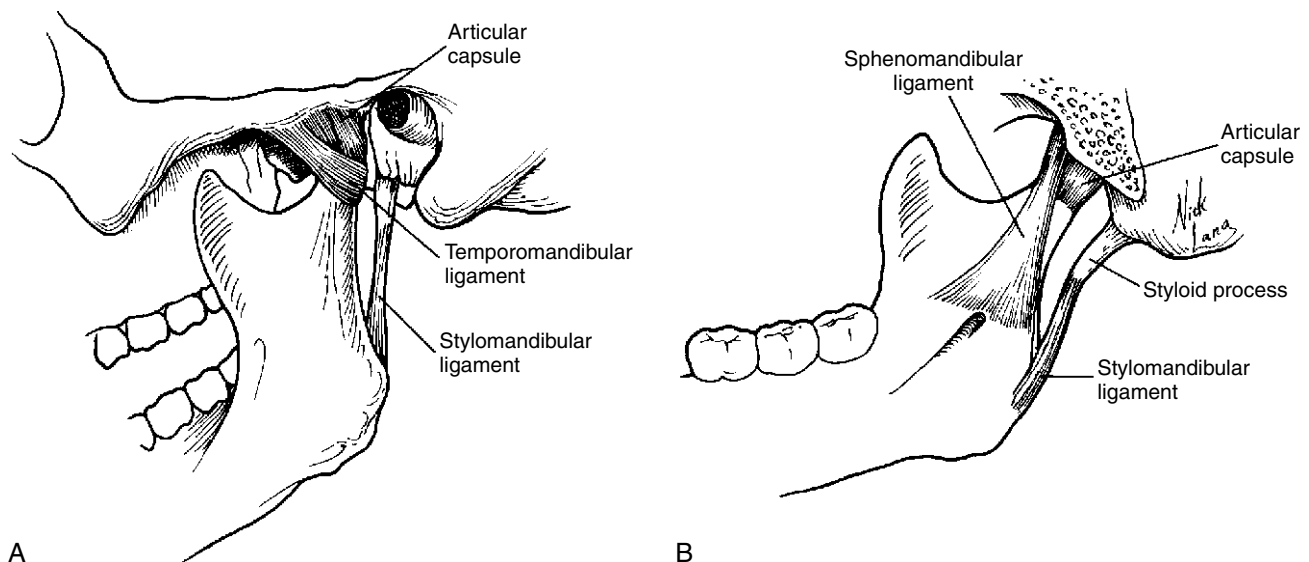


Figure 6-5 Lateral view of the left temporomandibular joint (TMJ) (A) and medial view of the right TMJ (B), showing the ligamentous structures.

Musculature

The primary movers of the mandible in elevation are the temporalis, masseter, and medial pterygoid muscles (Table 6-1 and Figure 6-6). The posterior fibers of the temporalis also retract the mandible while maintaining the condyles posteriorly. The superficial fibers of the masseter protrude the jaw, and the deep fibers act as a retractor. The deep fibers also attach to the lateral aspect of the joint capsule. The medial pterygoid can protrude the mandible, as well as deviate the jaw laterally (Figure 6-7).

The major depressors of the mandible are the lateral pterygoid, suprahyoid, and infrahyoid muscles. The lateral pterygoid also

attaches to the mandibular condyle and the intraarticular disc, thereby serving as a significant stabilizer of the joint. It is the primary muscle used in opening the mouth, but can also assist in lateral movements and protrusion. Of clinical importance is its frequency of involvement in cases of TMJ dysfunction.

The suprahyoid muscle group is composed of the digastric, stylohyoid, geniohyoid, and the mylohyoid muscles (Figure 6-8). The digastric muscle pulls the mandible downward and in a posterior direction. The stylohyoid muscle initiates and assists jaw opening, but also draws the hyoid bone upward and backward when the mandible is fixed. The geniohyoid muscle pulls the mandible downward and backward. The mylohyoid muscle elevates the floor of the mouth and assists in depressing the mandible when the hyoid is fixed or in elevating the hyoid when the mandible is fixed.

The prime function of the infrahyoid muscle group, composed of the sternohyoid, thyrohyoid, and omohyoid muscles, is to stabilize or depress the hyoid bone. This action then allows the suprahyoid muscles to act on the mandible.

TABLE 6-1 Actions of the Muscles of the Temporomandibular Joint

Action	Muscles
Mandibular elevation (closing)	Temporalis, masseter, and medial pterygoid
Mandibular depression (opening)	Lateral pterygoid, suprahyoid, and infrahyoid
Protrusion (anterior glide)	Superficial fibers of the masseter, medial pterygoid, and lateral pterygoid
Retrusion (posterior glide)	Temporalis and deep fibers of the masseter
Lateral glide	Medial pterygoid and lateral pterygoid
Hyoid elevation	Stylohyoid, mylohyoid, and digastrics
Hyoid depression	Infrahyoid

BIOMECHANICS

Posture and Alignment

In the normal relaxed and inactive state of the TMJ, referred to as the *mandibular postural rest position*, the teeth should not touch. Instead, there should be an intraocclusal space, termed the *freeway space*, of approximately 3 to 5 mm. This position is the result of the equilibrium between the muscle tone of the mandibular elevators and the force of gravity. Moreover, it is influenced by the state of the anterior and posterior neck muscles, head posture, and inherent elasticity of the mandibular muscles. This resting posture is decreased in people who brux, or clench, their teeth and is increased in mouth breathers. Disturbances in the resting position may affect the remodeling or reparative processes, leading to an unhealthy TMJ system.

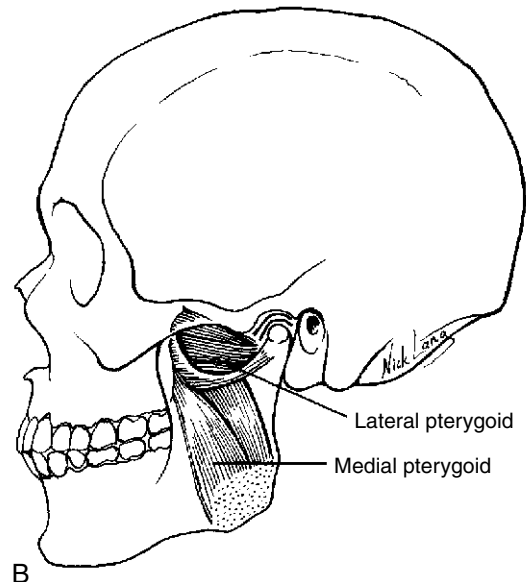
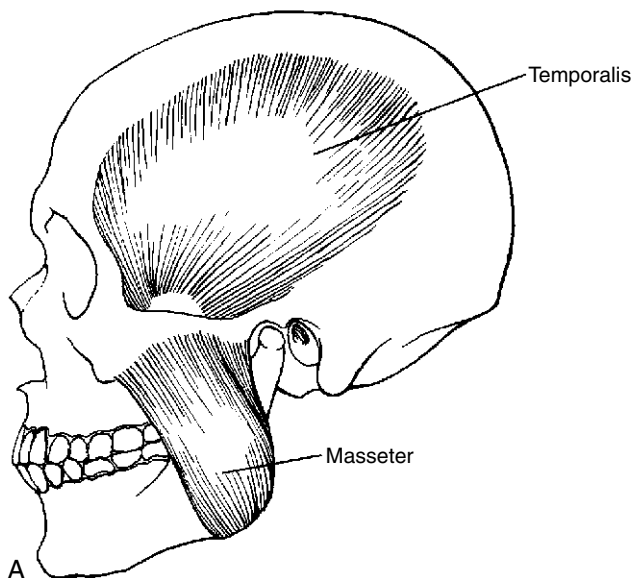


Figure 6-6 Intrinsic musculature of the mandible. **A**, Left temporalis and masseter, externally. **B**, Left medial and lateral pterygoid, with proximal mandible removed.

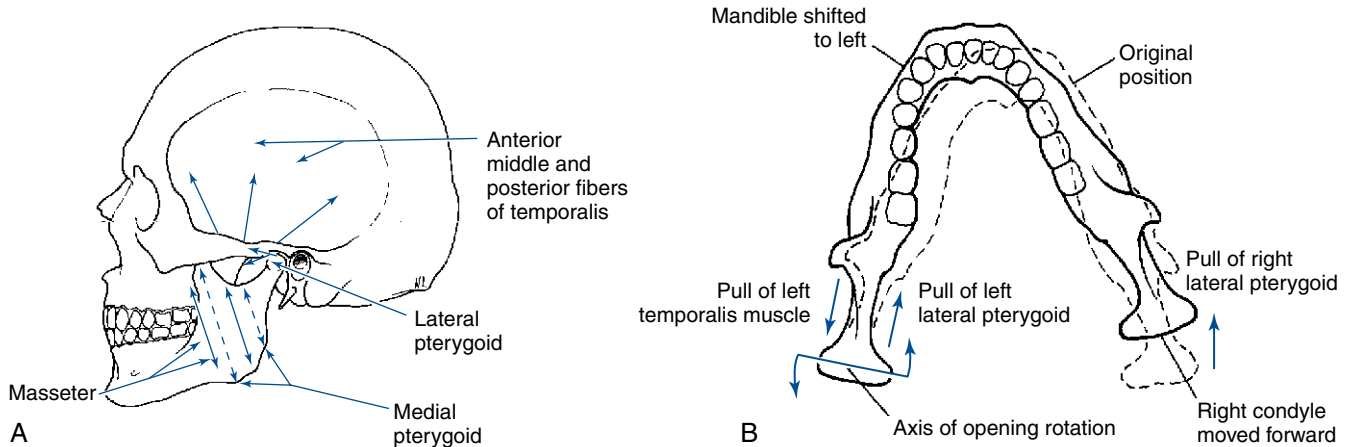


Figure 6-7 The directions of muscular pull in the temporomandibular joint. **A**, Opening, closing, and anterior glide. **B**, Left lateral deviation.

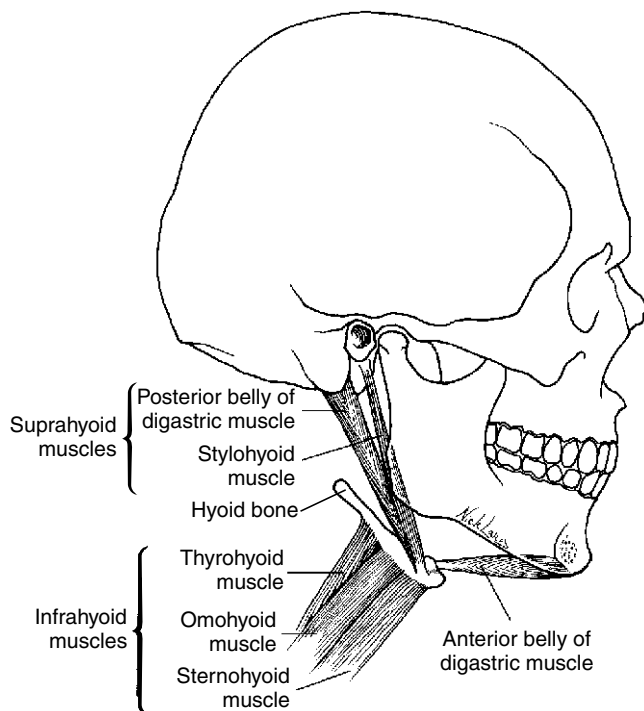


Figure 6-8 The suprahyoid and infrahyoid muscle groups of the mandible.

The position of the mandible when the teeth are fully occluded is termed the *intercuspal position* and is directly influenced by the state of the dentition. In this position, the condylar location is also affected by the dentition, which may differ from that imposed by muscle action. Furthermore, the intercuspal position may have an effect on the mandibular resting posture by disturbing the balance of the musculature, which in turn can affect the head posture and cervical spine function. Therefore, the state of the dentition should not be ignored in patients who have chronic neck pain.

The TMJ undergoes the coupled motions of rotation and translation. Rotational movement of the condyle occurs about a transverse axis between the condyle and the intraarticular disc. This movement causes the first 12 to 15 mm of mandibular opening and closing. Translational movement of the condyle consists of a downward and forward gliding movement of the disc-condyle complex and depends on the synchronous movement of disc and condyle (Figure 6-9). Rotational movements occur mainly in the inferior (subdiscal) joint space, and translational movements occur mainly in the superior (supradiscal) joint space.

Mandibular movement is a dynamic combination of opening, closing, anterior glide (protrusion), posterior glide (retrusion), and lateral glide. Mandibular opening involves the contraction of the lateral pterygoids and digastrics, with assistance from the suprahyoid and infrahyoid muscles. Indirectly, the posterior

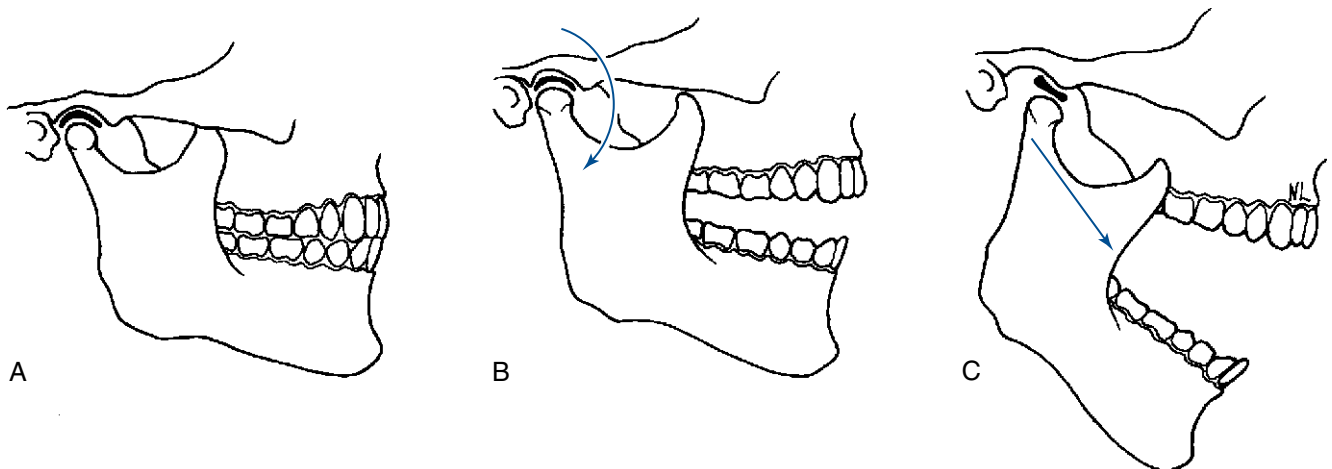


Figure 6-9 Mandibular movement is a complex relationship of rotational and translational movements. **A**, Closed position. **B**, Initial opening is rotational. **C**, Full opening requires forward translation with continued rotation.

TABLE 6-2 Arthrokinematic and Osteokinematic Movements of the Temporomandibular Joint

Osteokinematic Movements		Arthrokinematic Movements
Mandibular elevation and depression	40 to 60 mm of incisor separation	Combination of spin and glide occurring in the upper and lower joint compartments
Mandibular retraction and protrusion	5 to 10 mm of incisor separation	Glide movement occurring in the upper joint compartment
Mandibular lateral deviation	5 to 10 mm of incisor separation	Glide, spin, and angulation occurring mostly in the upper compartment but also in the lower joint space

cervical muscles must contract to prevent neck flexion and permit the mandible to drop away from the cranium. The first few degrees of opening are rotational movements about a transverse axis. Then the condyle and disc complex must translate forward over the articular eminence, allowing condylar rotation to continue. The closing sequence is the reverse. The condyle and disc complex translate posteriorly with condylar rotation, which brings the joint to its resting position, or intercuspal position. The masseter, medial pterygoid, and temporalis muscles are responsible for this action. Table 6-2 lists the osteokinematic and arthrokinematic movements of the TMJ.

Lateral glide of the mandible involves a pivoting rotation of the condyle on the side to which the mandible is moving and a translation of the other condyle. For lateral glide to the right, the left lateral pterygoid and the anterior bellies of both the digastrics contract, causing the left condyle to move downward, forward, and medially. Meanwhile, contraction of the right temporalis and the right lateral pterygoid rotates the right condyle in the fossa and displaces the mandible to the right. Box 6-1 identifies the close-packed and loose-packed positions for the TMJ.

EVALUATION

Problems affecting the TMJ can be broadly classified into developmental abnormalities, intracapsular diseases, and dysfunctional conditions (Box 6-2). Developmental abnormalities include hypoplasia, hyperplasia, impingements of the coronoid process, chondromas, and ossification of ligaments, such as the stylohyoid ligament (Eagle syndrome). The intracapsular diseases include degenerative arthritis, osteochondritis, rheumatoid arthritis, psoriatic arthritis, synovial chondromatosis, infections, steroid necrosis, gout, and metastatic tumors.⁶ Developmental abnormalities and intracapsular diseases must be ruled out by using appropriate evaluative procedures, including diagnostic imaging and clinical laboratory studies. The focus here is on the dysfunctional conditions affecting the TMJ, which are categorized as extracapsular (myofascial pain syndromes and muscular imbalance); capsular (sprain, hypomobility and hypermobility, and synovial folds); and intracapsular (disc displacement and disc adhesions).

Inspect the face and jaw, looking for bony or soft tissue asymmetry, misalignment of the teeth, and evidence of swelling (Figure 6-10). Observe the jaw during opening and closing to identify excursions deviations and the presence of clicking⁷⁻⁹ (Figure 6-11). Ask the patient if the movements are painful. Normal opening should accommodate three of the patient's

BOX 6-1 Close-Packed and Loose-Packed (Rest) Positions for the Temporomandibular Joint

CLOSE-PACKED POSITION

The maximal intercuspal position, in which all teeth make full contact with one another (clenched)

LOOSE-PACKED POSITION

The “freeway space” position of slight mandibular opening, which occurs because of equilibrium between jaw-opening and jaw-closing muscles

BOX 6-2 Classification of Problems Affecting the Temporomandibular Joint

DEVELOPMENTAL ABNORMALITIES

Hypoplasia
Hyperplasia
Impingement of the coronoid process
Chondromas
Ossification of ligaments (e.g., Eagle syndrome)

INTRACAPSULAR DISEASES

Degenerative arthritis
Osteochondritis
Inflammatory arthritis (rheumatoid and psoriatic arthritis and gout)
Synovial chondromatosis
Infections
Metastatic tumors

DYSFUNCTIONAL CONDITIONS

Extracapsular Conditions

Myofascial pain syndrome
Muscular imbalance (strain or spasm)

Capsular conditions

Sprain, capsulitis, synovitis
Hypomobility
Hypermobility
Synovial folds

Intracapsular conditions

Disc displacement
Disc adhesions

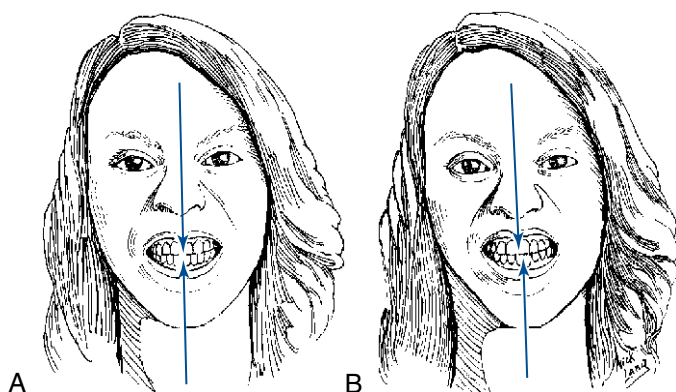


Figure 6-10 Intercuspal alignment. **A**, Normal alignment. **B**, Left lateral deviation.

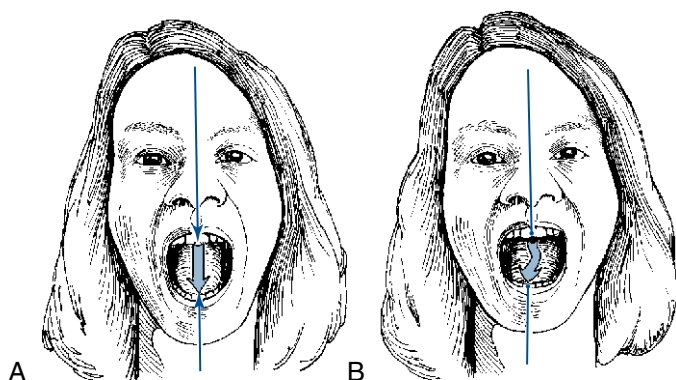


Figure 6-11 Mandibular gait pattern. **A**, Normal pattern. **B**, C-shaped deviation.

fingers inserted between the incisors. If not, hypomobility resulting from joint dysfunction or a closed lock as a result of disc displacement should be suspected. If opening beyond three fingers occurs, hypermobility from capsular attenuation is likely (Figure 6-12).

Palpate the joint by placing the fifth digit into the patient's external auditory meatus with the palmar surface forward (Figure 6-13). Before placing the finger into the ear, push on the tragus to determine if the external ear canal is painful. The position of the condyles and intra-articular clicking can be felt from within the external canal. The lateral aspect of the joint and capsule can be palpated externally for position, pain, and movement disorders (Figure 6-14). Palpate the muscles to determine changes in tone, texture, and tenderness. The lateral pterygoid can be palpated intraorally by following the buccal mucosa to the medial aspect of the TMJ just proximal to the tonsils (Figures 6-15 and 6-16). According to investigations, the lateral pterygoid muscle is practically inaccessible for intraoral palpation because of topographic and anatomic reasons.¹⁰⁻¹³ Palpation of the lateral pterygoid muscle is also characterized by poor interexaminer agreement.¹⁰ In contrast to these reports, Stelzenmüller and associates¹⁴ reliably confirmed the palpation of the lateral pterygoid muscle, which was controlled by two imaging procedures and electromyography. All three of the procedures confirmed palpation. The difficulty in reliably identifying the muscle seems to be due to



Figure 6-12 Generally, and only as a screen, three of the patient's fingers should fit between the incisors when fully opened.

BOX 6-3

Accessory Joint Movements of the Temporomandibular Joint

Long-axis distraction
Lateral glide
Anterior glide
Posterior glide

the fact that the medial pterygoid muscle must be passed before palpating the lateral pterygoid muscle.¹⁴ Note also the position and movement of the hyoid bone, as well as the state of the associated soft tissues.

Perform accessory motions of the TMJ by placing the gloved thumbs intraorally over the lower teeth and wrapping the fingers around the mandible externally (Figure 6-17). Apply passive stress in long-axis distraction, lateral glide, and anterior-to-posterior (A-P) glide (Box 6-3). Normally, a springing end feel is perceived (Figure 6-18).

A general survey of the oral cavity should be done to rule out dental or oral lesions. Ask the patient to close the teeth together quickly and sharply. Normally, this is not painful, and a broad clicking of the dental surface should be heard. If local pain is produced or only a faint, single striking sound is heard, a dental source (acute malocclusion, tooth abscess, or periodontal disease) is indicated, and a referral to a dentist is advised. In addition, it is important to remember the intimate neurologic relation of the TMJ with the atlanto-occipital articulation and the need to evaluate both joints when either is symptomatic.

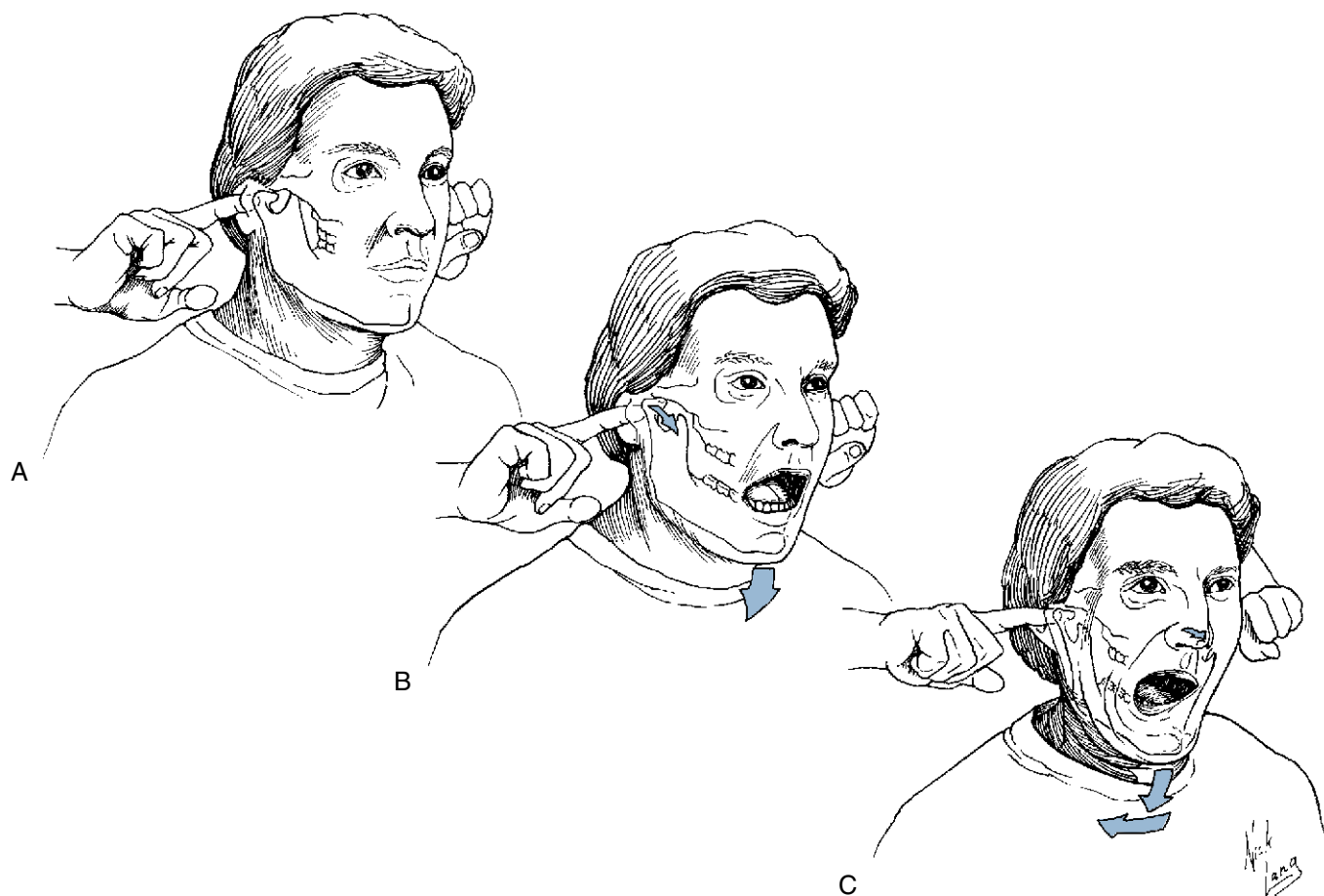


Figure 6-13 Intra-auricular palpation of the temporomandibular joint. **A**, Finger is placed in external auditory meatus. **B**, Movement of condyle is palpated on opening and closing. **C**, Asymmetric joint movement.

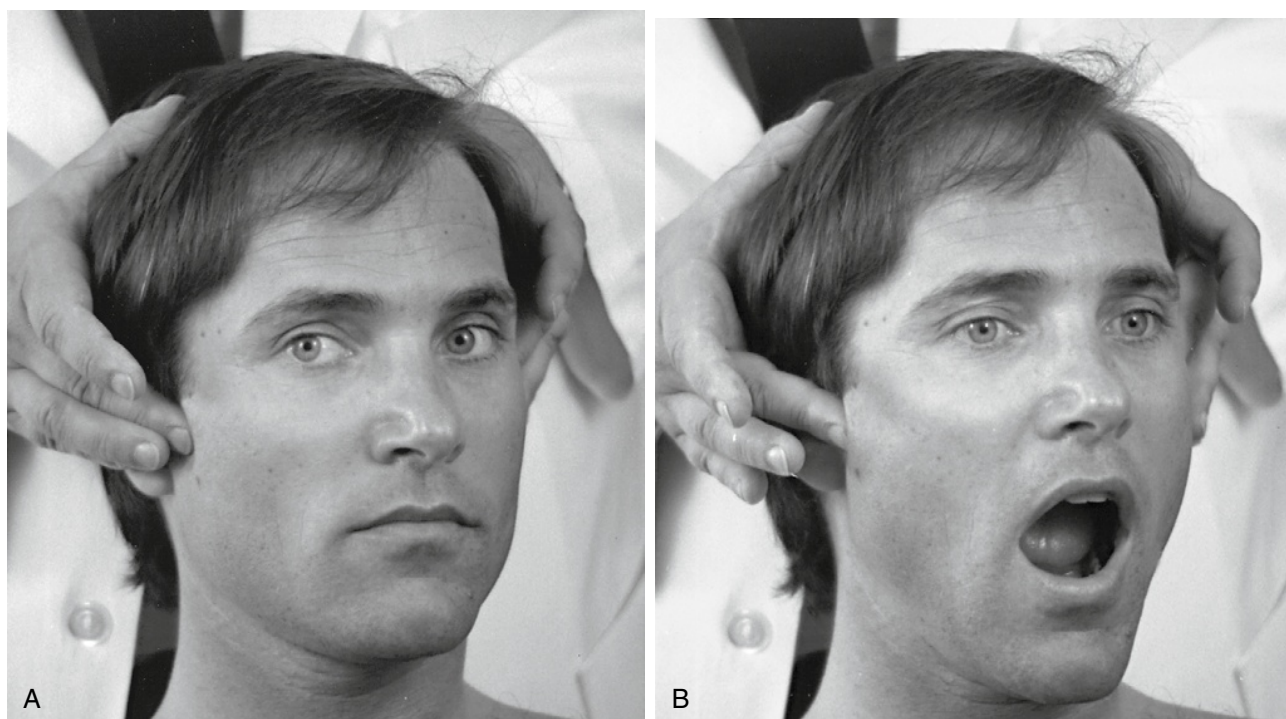


Figure 6-14 External palpation of the temporomandibular joint space. **A**, Jaw closed. **B**, Jaw open.

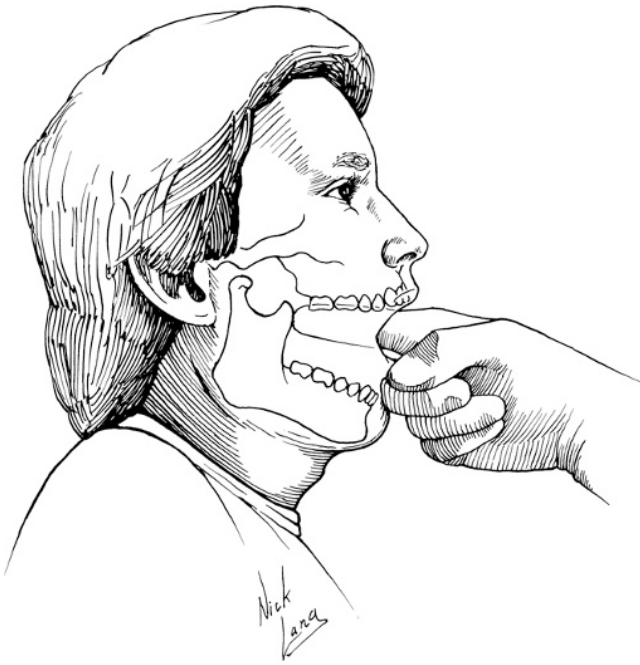


Figure 6-15 Palpation of pterygoid muscles with intraoral contact.



Figure 6-16 Intraoral palpation of the pterygoid muscles.

ADJUSTIVE PROCEDURES

The manipulative techniques used to treat TMJ disorders aim to restore normal joint mechanics, which will then ideally allow full pain-free functioning of the mandible. Two basic types of adjustive procedures are used for the different forms of TMJ dysfunction: distraction and translation techniques (Box 6-4). See Box 5-3 for abbreviations used in illustrating techniques.

Distraction Techniques

Distraction procedures create a slow and controlled joint gaping or separation of the joint surfaces. Typically, they require an intraoral contact, making the use of rubber gloves necessary.

TMJ Supine: Bilateral Thumb/Mandible; Long Axis Distraction^{3,7,8} (Figure 6-19)

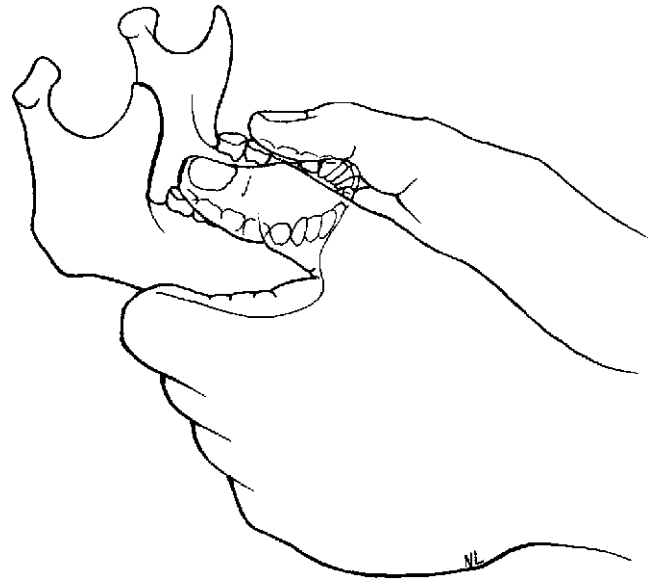


Figure 6-17 Double-thumb intraoral contact on mandible.

BOX 6-4

Temporomandibular Adjustive Techniques

Supine bilateral thumb/mandible distraction; long-axis distraction (Figure 6-19)
 Seated or supine bimanual thumb/lower molars; plica entrapment reduction (Figure 6-21)
 Seated reinforced palmar/distal mandible; anterior-to-posterior glide (Figure 6-22)
 Supine reinforced thumb/proximal mandible; lateral-to-medial glide (Figure 6-24)
 Seated thenar/proximal mandible; lateral-to-medial glide (Figure 6-25)

IND: Reduce an acutely dislocated disc, treat loss of accessory joint movements, and influence disc nutrition.

PP: The patient is supine, with the mouth slightly open. A head belt or an assistant may be used to stabilize the patient's head.

DP: With gloved hands, stand at the side of the table, facing the patient on the side of the joint dysfunction.

SCP: Lower teeth on the involved side.

CP: Use your cephalic hand, with a thumb contact on the lower teeth of the affected side. Wrap your fingers around the mandible externally, with the index fingers along the body of the mandible.

IH: Use your caudal hand, if jaw excursion allows, to reinforce the CP with a thumb contact on top of the CP. If this is not possible, place the thumb on the lower teeth of the other side.

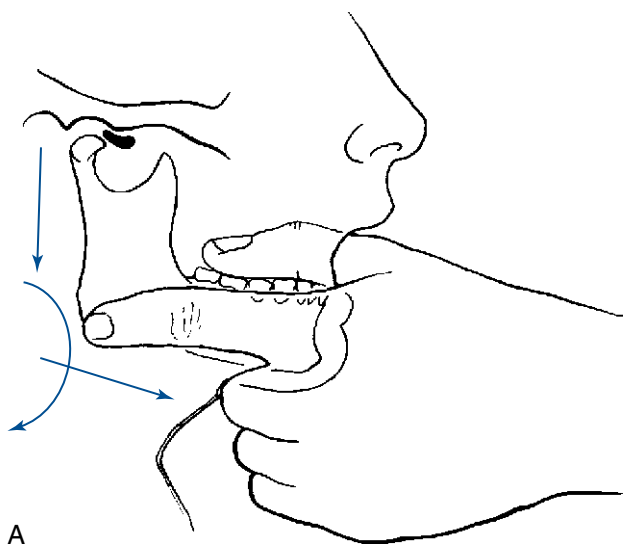
VEC: Long-axis distraction.

P: Ask the patient to swallow, and then apply distraction to the caudal joint surfaces. For an acute anterior dislocation of the disc (Figure 6-20), tip the condyle anteriorly to position it under the disc and add a posterior-to-anterior (P-A) force. For an acute posterior dislocation of the disc, tip the condyle



Figure 6-18 Accessory joint movement evaluation for the temporomandibular joint.

posteriorly to position it under the disc and add an A-P force. For loss of specific accessory joint movements and for stimulation of mechanoreceptors, this procedure can be done



A



Figure 6-19 Long-axis distraction manipulation of the temporomandibular joint.

with the addition of lateral glide movements, dorsal-ventral glide movements, or both.

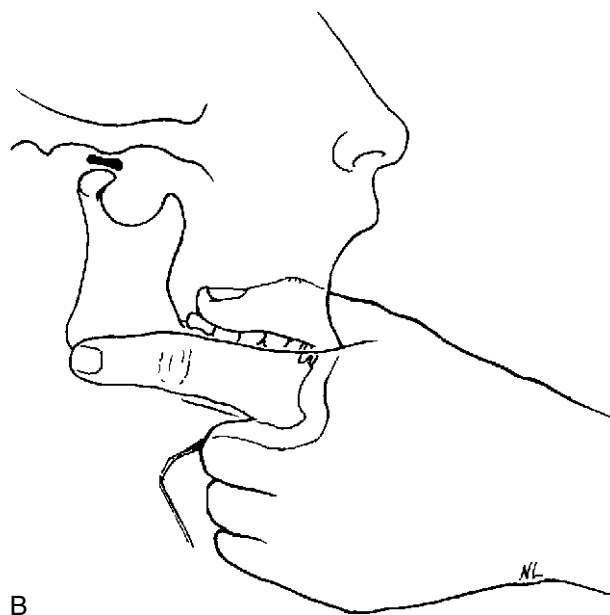
The TMJ is prone to plica formation (synovial fold) especially with sustained, extreme opening. When a synovial fold becomes entrapped, pain is produced on occlusion and when the disc moves medially or the condyle moves laterally. To reduce the entrapped fold of synovial tissue, the distraction technique must be combined with contraction of the patient's ipsilateral masseter muscle.

TMJ Seated or Supine: Bilateral Thumb/Lower Molars; Plica Entrapment Reduction⁷ (Figure 6-21)

IND: Plica entrapment (synovial fold).

PP: The patient is seated or supine, with the head stabilized and the mandible deviated away from the involved side.

DP: Stand at the side of the patient.



B

Figure 6-20 Distraction technique for an anteriorly dislocated disc for the temporomandibular joint. **A,** Starting position. **B,** Distraction of the condyle with tilt and anterior translation under the disc.



6-21

Figure 6-21 Manipulation for plica entrapment of the left temporomandibular joint.

SCP: Lower teeth on the involved side.

CP: Establish the thumb contact over the lower teeth, with the fingers grasping the mandible.

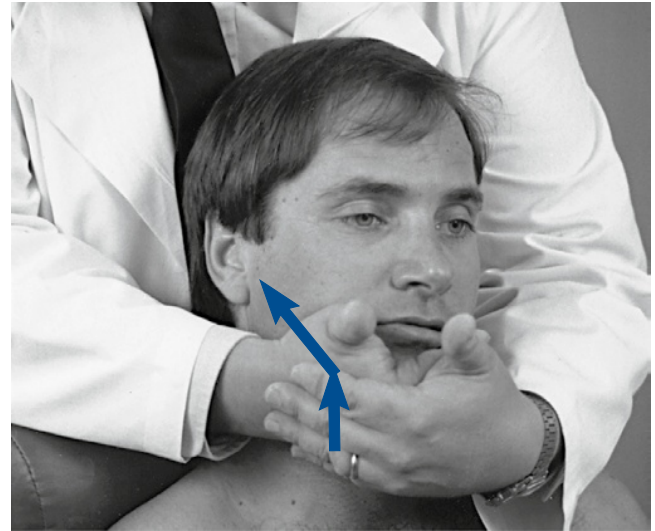
VEC: Long-axis distraction.

P: Apply a long-axis distraction force while maintaining the mandible in lateral deviation away from the problem. Then ask the patient to move the mandible (contracting the deep fibers of the masseter) toward the affected side, against the applied resistance.

Translation Techniques

Translational adjustive techniques use a line of correction parallel to the plane of the joint surfaces. In addition, a compressive loading force may be applied. The primary effect of this procedure is to move the disc posteriorly, which is specifically indicated for releasing minor disc adhesions (intracapsular adhesions). Translational techniques use extraoral contacts, and gloves are not necessary. Caution must be the rule when using translational techniques, because they carry a higher risk of complication than distraction techniques. If a translational technique is applied to a joint in which the disc is displaced anterior to the condyle, it is possible to injure the retrodiscal tissue or produce a hemarthrosis (blood in the joint) resulting in a medical emergency.

TMJ Seated: Reinforced Palmar/Distal Mandible; Anterior-to-Posterior Glide^{7,9} (Figure 6-22)



6-22

Figure 6-22 Anterior-to-posterior translational manipulation for the right temporomandibular joint.

IND: Release intracapsular discal adhesions, restricted anterior to posterior glide accessory joint movements, anterior misalignment of the mandibular condyle.

PP: The patient is seated.

DP: Stand behind the patient, with a rolled towel or pillow placed between you and the patient to support the cervical spine.

SCP: Ramus of the mandible.

CP: Your ipsilateral hand takes a knife-edge palmar contact over the ramus of the mandible while the fingers cradle the chin.

IH: Your contralateral hand reinforces over the CP.

VEC: A-P, along the line of the articular eminence (Figure 6-23).

P: First apply a compressive loading force (inferior-to-superior [I-S]), and then deliver the thrust as an impulse anteriorly to

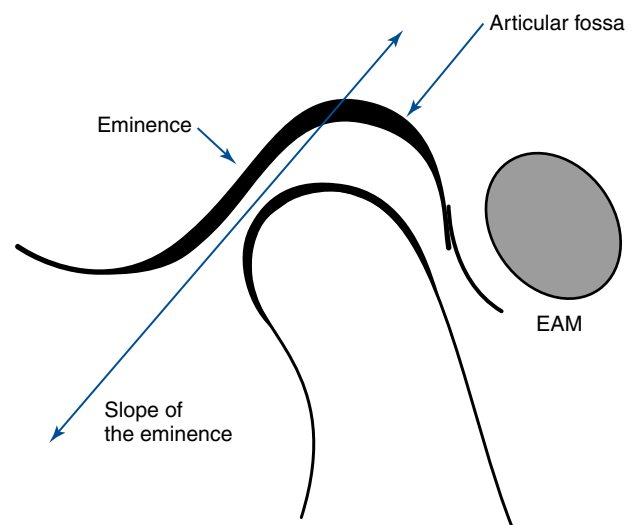


Figure 6-23 The slope of the articular eminence must first be established because this will determine or influence the direction of a translational thrust. External palpation of the eminence is used to determine the slope. (Modified from Curl D: Acute closed lock of the temporomandibular joint: Manipulative paradigm and protocol, *J Chiro Tech* 3[1]:13, 1991.)



Figure 6-24 Supine lateral-to-medial translation manipulation of the right temporomandibular joint.

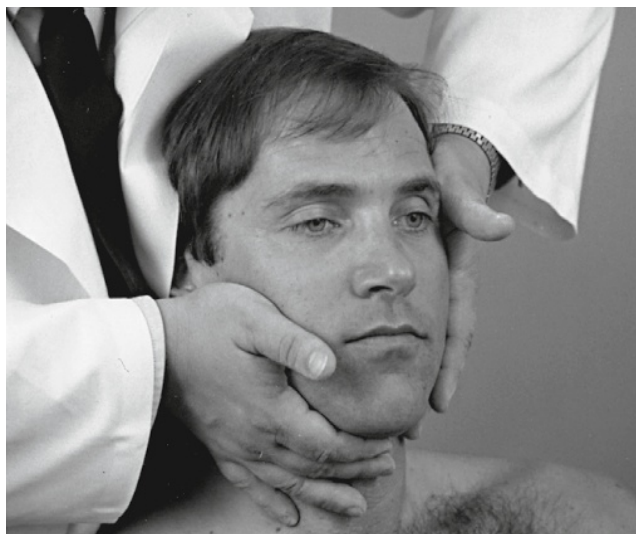


Figure 6-25 Seated lateral-to-medial translation manipulation of the right temporomandibular joint.

posteriorly and along the line of the articular eminence. To distract the patient's attention and to prevent jamming of the teeth, have the patient open his or her mouth and slowly close it; make the adjustment when he or she is closing the mouth.

TMJ Supine; Reinforced Thumb/Proximal Mandible: Lateral-to-Medial Glide^{3,7} (Figure 6-24)

IND: Restricted lateral to medial glide accessory joint movements, lateral misalignment of the mandibular condyle.

PP: The patient is supine, with the head turned slightly and the affected side up.

DP: Stand at the side of the table toward the side of contact.

SCP: Neck of the mandible.

CP: Establish a reinforced double-thumb contact over the neck of the mandible.

VEC: Lateral-to-medial (L-M).

P: Deliver an impulse thrust in an L-M direction.

TMJ Seated: Thenar/Proximal Mandible; Lateral to Medial Glide (Figure 6-25)

IND: Restricted lateral to medial glide accessory joint movements, lateral misalignment of the mandibular condyle.

PP: The patient is seated.

DP: Stand behind the patient and slightly to the side of involvement.

SCP: Proximal mandible.

CP: With your ipsilateral hand, establish a thenar contact over the proximal aspect of the mandible, just distal to the joint space.

IH: Use the contralateral hand to apply a broad palmar contact on the uninvolved side of the face and head.

P: With the patient's jaw relaxed (teeth not clenched), deliver an impulse thrust in an L-M direction.

One of the greatest difficulties encountered when adjusting the TMJ is the patient's inability to adequately relax the jaw muscles. Furthermore, it is detrimental to the joint to overstretch the upper

joint compartment; therefore, care must be taken when using intraoral contacts.

SHOULDER

The primary role of the shoulder is to place the hand in a functional position. To achieve this function, a great deal of joint mobility is necessary, which requires complex anatomy and biomechanics. The shoulder is not one joint, but rather is a relationship of anatomic and physiologic joints, forming a four-joint complex. The glenohumeral joint is a true anatomic joint and forms the shoulder proper. The sternoclavicular and acromioclavicular joints are also true anatomic joints formed between the manubrium of the sternum, the clavicle, and the acromial process of the scapula, respectively. The scapulocostal joint lacks a joint capsule and is therefore considered a physiologic joint, necessary to allow the smooth gliding of the scapula over the ribs (Figure 6-26).

These joints provide for an extensive range of active movement for the upper extremity. Many muscles work together synergistically to produce coordinated actions across multiple joints. Dysfunction from trauma or pathologic processes to any of the articulations can cause a significant reduction in the effectiveness of the entire upper limb.

FUNCTIONAL ANATOMY

Osseous Structures

The convex articular surface of the proximal humerus is directed slightly posteriorly, medially, and superiorly, and is met by the articular surface of the glenoid fossa of the scapula (Figure 6-27). A 45-degree angle is formed between the articular surface and the shaft of the humerus. The glenoid fossa is not a deep impression into the bone of the scapula, and by itself it is incongruous with

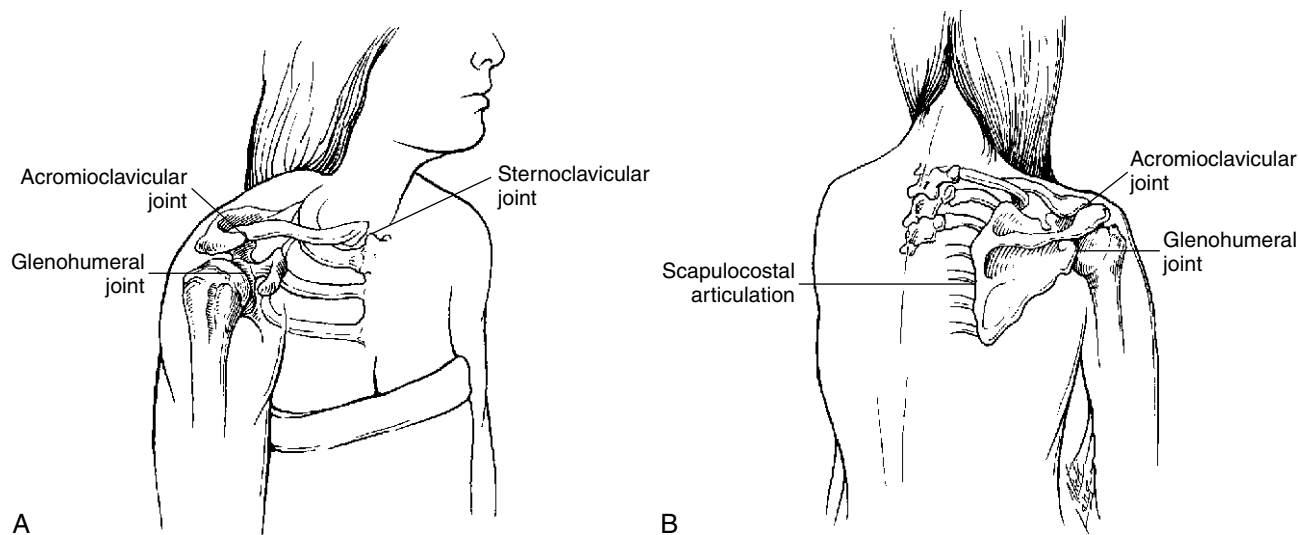


Figure 6-26 The four joints that make up the shoulder complex. **A**, Anterior view. **B**, Posterior view.

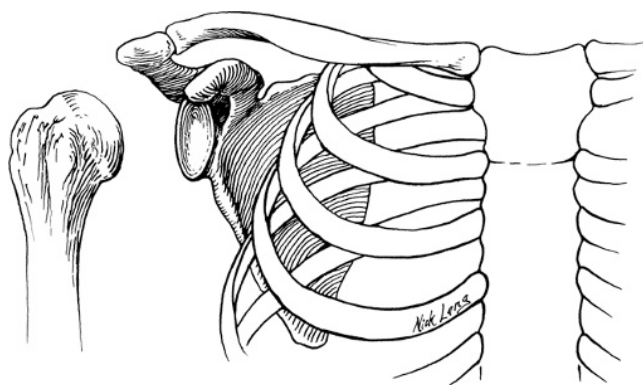


Figure 6-27 The proximal humerus articulates with the shallow glenoid fossa of the scapula.

the humeral head. The glenoid labrum is a fibrocartilaginous rim that encircles the fossa and provides a greater surface area of contact for the humerus, which helps provide some stability (Figure 6-28).

The S-shaped clavicle provides for more movement during elevation of the arm. The distal third of the clavicle is concave anteriorly, which directs the distal articular surface anteriorly and somewhat superiorly. The proximal end of the clavicle articulates with the upper and lateral edge of the manubrium and the superior surface of the first rib costocartilage (Figure 6-29). An intra-articular disc lies between the clavicle and manubrium joint surfaces and is important in preventing medial dislocations of the clavicle.

The scapula lies at a 30-degree angle away from the coronal plane and forms a 60-degree angle with the clavicle¹⁵ (Figure 6-30). It has the coracoid process for muscular attachment, projecting anteriorly and lying just medial to the glenoid fossa. The spine of the scapula arises from the medial border at about the T3 level and courses laterally and superiorly, ending as the acromion process.

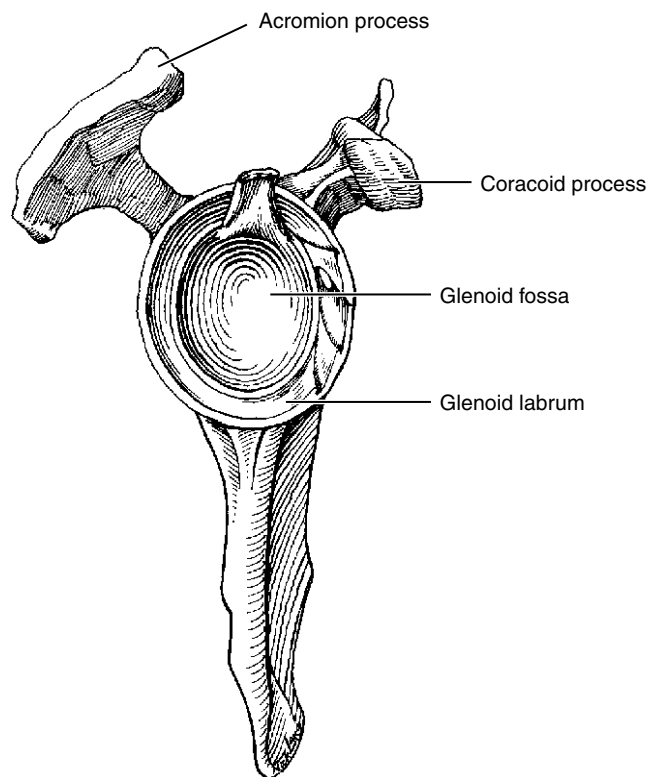


Figure 6-28 The glenoid fossa of the right scapula.

Ligamentous Structures

Many ligaments are associated with the shoulder, connecting one bone to another and providing a secondary source of joint stability (Figure 6-31). With the varied movements the shoulder can perform, these ligaments will become either slack or taut. Remember that a ligament is painlessly palpated unless it is injured or stretched.

The glenohumeral joint capsule is thin, lax, and redundant, with anterior folds when the arm is at rest. The glenohumeral ligaments

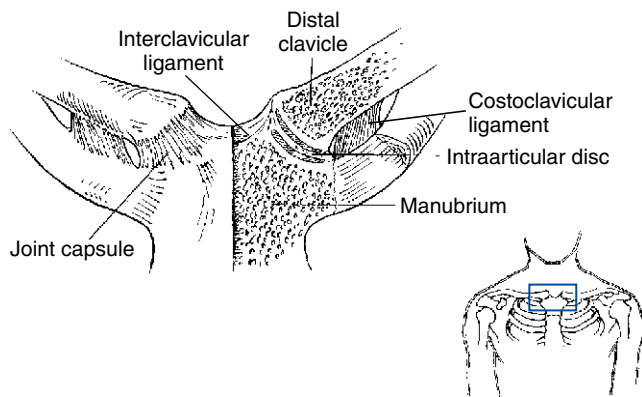


Figure 6-29 A coronal section through the sternoclavicular joints.

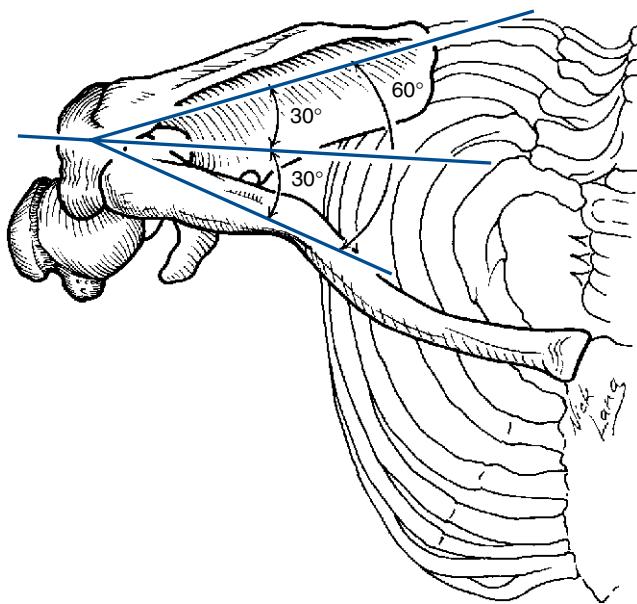


Figure 6-30 Apical view of the right shoulder complex showing the relationship between the scapula and clavicle.

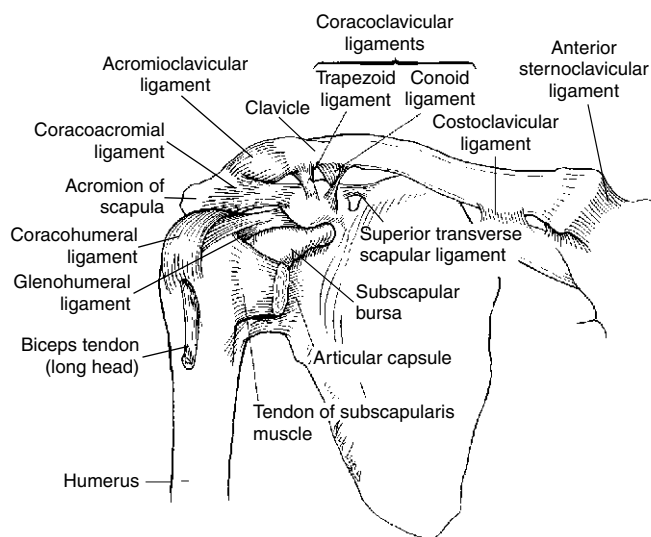


Figure 6-31 Ligaments of the shoulder complex.

provide some reinforcement to the joint capsule anteriorly while helping to check external rotation and possibly abduction. The coracohumeral ligament runs from the coracoid process to the greater tubercle, reinforces the superior aspect of the capsule, and checks external rotation and possible extension. The transverse humeral ligament attaches across the greater and lesser tubercles and serves to contain the tendon of the long head of the biceps muscle.

The acromioclavicular ligament strengthens the superior aspect of the joint capsule. It is intrinsically weak, however, and gives way when a force is applied to the acromion process or glenohumeral joint from above (Figure 6-32). The major stabilizing ligaments of the acromioclavicular joint are the coracoclavicular ligaments, which include the conoid and trapezoid ligaments. The conoid ligament twists on itself as it connects between the coracoid process and clavicle. It prevents excessive superior movement of the clavicle on the acromion, as well as retraction of the scapula, by not allowing the scapuloclavicular angle to widen. Furthermore, the conoid ligament tightens on humeral abduction, causing axial rotation of the clavicle that is necessary for full elevation of the arm. The trapezoid ligament also connects between the coracoid process and the clavicle, but lies distal to the conoid ligament. Its role is to check lateral movement of the clavicle, thereby preventing overriding of the clavicle on the acromion process. The trapezoid ligament also prevents excessive scapular protraction by not allowing the scapuloclavicular angle to narrow.

The sternoclavicular joint capsule is reinforced anteriorly and posteriorly by the anterior and posterior sternoclavicular ligaments, respectively (see Figure 6-32). The interclavicular ligaments reinforce the capsule superiorly. Lying just lateral to the joint, the costoclavicular ligament attaches between the clavicle and first rib and serves to check elevation of the clavicle. Its posterior fibers prevent medial movement, and the anterior fibers prevent lateral movement of the clavicle.

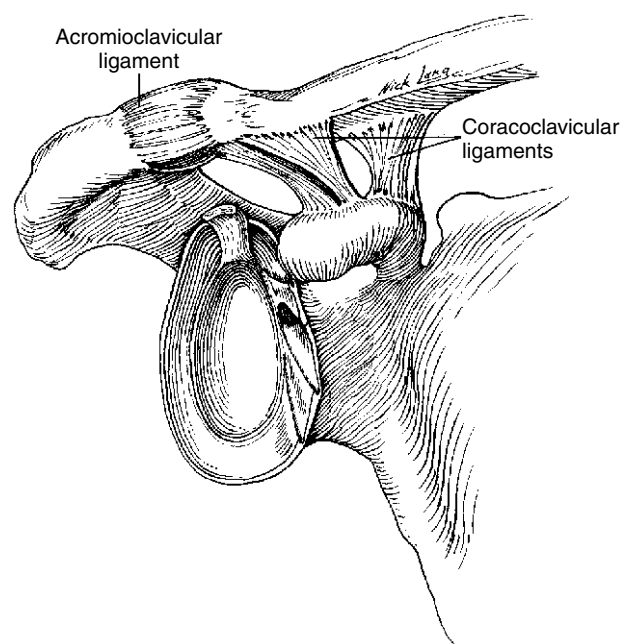


Figure 6-32 Ligaments of the acromioclavicular joint.

Musculature

Because of the high degree of mobility and the numerous muscles necessary to provide stability to the joint, eight or nine bursae are found about the shoulder joint to reduce friction between the moving parts. Irritation to the bursae, leading to an inflammatory response, is a common clinical occurrence. Of specific clinical significance are the subscapular bursae and the subacromial or subdeltoid bursae (see Figure 6-31). The subscapular bursa lies beneath the subscapularis muscle and overlies as well as communicates with the anterior joint capsule. Distension of this bursa occurs with articular effusion. The subacromial or subdeltoid bursa extends over the supraspinatus tendon and under the acromion process and deltoid muscle (Figure 6-33). It is susceptible to impingement beneath the acromial arch, and inflammation often follows supraspinatus tendinitis.

A review of the location and function of the numerous muscles that stabilize and supply the force for performing the varied movements of the shoulder is necessary to understand the dysfunctional conditions that affect the shoulder joint complex (Table 6-3). Although muscle tendons tend to lend stability to the joint, they do not prevent downward dislocation. The rotator cuff is composed of the supraspinatus, infraspinatus, teres minor, and subscapularis muscles. It is most notably the horizontal running fibers that prevent dislocation as they check the lateral excursion of the glenoid cavity, which in turn allows downward movement of the humerus. The factors preventing downward dislocation include the slope of the glenoid fossa, which forces the humerus laterally as it is pulled down; tightening of the upper part of the capsule and of the coracohumeral ligament; and the activity of the supraspinatus muscle working with the posterior fibers of the deltoid.

TABLE 6-3 Actions of the Muscles of the Shoulder Joint Complex

Action	Muscles
Flexion	Anterior deltoid, coracobrachialis, and pectoralis major (clavicular)
Extension	Latissimus dorsi, teres major, and posterior deltoid
Abduction	Middle deltoid, supraspinatus, and serratus anterior (scapular stability)
Adduction	Pectoralis major and latissimus dorsi
External rotation	Infraspinatus, teres minor, and posterior deltoid
Internal rotation	Subscapularis, pectoralis major, latissimus dorsi, teres major, and anterior deltoid
Scapular stabilization	Trapezius, serratus anterior, and rhomboids
Scapular retraction (medial glide)	Rhomboid major and minor
Scapular elevation	Trapezius and levator scapulae

The tendon of the long head of the biceps is unique in its relationship to the joint in that it originates from within the joint capsule. It arises from the upper margin of the glenoid fossa as a continuation of the glenoid labrum. It penetrates the capsule and passes through the intertubercular groove, which has been

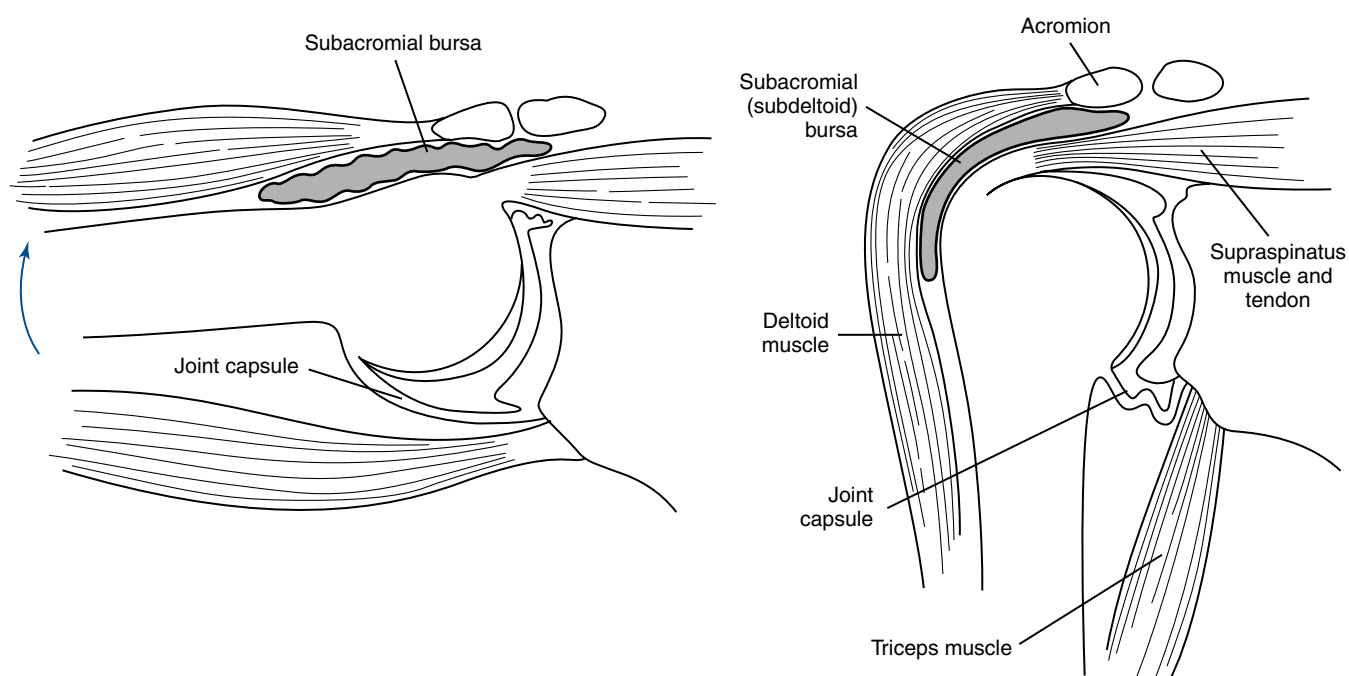


Figure 6-33 Subacromial bursa. **A**, With humerus pendulous. **B**, With humerus abducted. (Modified from Hertling D, Kessler RM: *Management of common musculoskeletal disorders: Physical therapy principles and methods*, ed 2, Philadelphia, 1990, JB Lippincott.)

converted to a tunnel by the transverse humeral ligament. The passage through the capsule, over the humeral head and between the two tubercles, is facilitated by a tubular sheath of synovial membrane.

Shoulder abduction can be artificially divided into two phases of movement, each involving different muscles to a greater or lesser degree. Shoulder movement is fluid, however, and the various muscular actions and motions run into one another. The first phase of movement, from 0 to 90 degrees of abduction, involves a coupling of the deltoid and supraspinatus muscles that draws the humerus up. To go farther, the scapula and shoulder girdle must also move. In the second phase, the serratus anterior, along with the upper and lower trapezius, tip the scapula so that the inferior angle moves laterally and the acromion process is elevated. This allows movement between 90 and 180 degrees.

Forward flexion of the shoulder begins with the contraction of the anterior fibers of the deltoid, coracobrachialis, and clavicular division of the pectoralis major muscles. Up to 60 degrees of flexion can occur before the scapula and shoulder girdle must move. Again, the serratus anterior and the upper and middle trapezius contract, tipping the scapula, raising the acromion process, and causing axial rotation at the acromioclavicular and sternoclavicular joints.

Internal (medial) rotation is accomplished by contraction of subscapularis, teres major, pectoralis major, latissimus dorsi, and the anterior deltoid muscles. For extreme ranges of internal rotation, the scapula will abduct from the pull of the serratus anterior and the pectoralis minor muscles.

The external (lateral) rotator muscles, in comparison with the internal rotators, are quite weak but are still very important to normal function of the upper limb. Clinically, they are easily strained. The infraspinatus, teres minor, and to a lesser degree, posterior deltoid muscles are responsible for external rotation of the humerus, with the rhomboids and trapezius muscles adducting the scapula for extreme movement.

Shoulder extension is accomplished by contraction of the latissimus dorsi, teres major, and posterior deltoid muscles, with some adduction of the scapula from contraction of the middle trapezius and rhomboid muscles. Adduction of the humerus is accomplished by contraction of the latissimus dorsi, teres major, and pectoralis major muscles, with the rhomboids adducting the scapula.

BIOMECHANICS

Scapulohumeral Rhythm

For the arm to be abducted from the side to overhead, motion of the scapula must be simultaneous and synchronous. During the first 30 degrees of abduction, the scapula seeks a stable position on the rib cage through contraction of the trapezius, serratus anterior, and rhomboid muscles (Figure 6-34). Beyond 30 degrees, 2 degrees of glenohumeral movement occur for every 1 degree of scapulocostal movement. Thus, 15 degrees of arm abduction results from 10 degrees of glenohumeral joint movement and 5 degrees of scapulocostal joint movement. Rotation of the scapula during abduction also enhances mechanical stability by bringing the glenoid fossa directly under the humeral head.

Acromioclavicular Joint

Arm abduction also requires axial rotation of the clavicle. For every 10 degrees of arm abduction, the clavicle must elevate 4 degrees. After 90 degrees of arm abduction (60 degrees of humeral abduction and 30 degrees of scapular rotation), the clavicle rotates to accommodate the scapula through its full 60 degrees of motion. This is achieved through the S-shape of the clavicle.¹⁶ The acromioclavicular joint usually has a meniscus that functionally divides the joint. Rotational movements occur through the conoid ligament and between the acromion and the meniscus. Hinging movements occur between the meniscus and the clavicle.¹⁷

Glenohumeral Joint

The rotator cuff muscles pull the humerus inferiorly (depression), and the glenohumeral joint capsule creates external rotation during abduction to permit the greater tuberosity to pass under the acromion and coracoacromial ligament (Figure 6-35). Carefully evaluate patients complaining of loss of full abduction with or without pain to determine whether the humerus is displaced superiorly or has lost inferior glide movements because of capsular ligament fibroadhesions or improper functioning of the rotator cuff muscles.

During shoulder movements away from the body (flexion, abduction, and extension), the superior aspect of the glenohumeral joint capsule becomes lax, so that it can no longer maintain joint integrity against external forces. The rotator cuff muscles carry the responsibility for keeping the humerus oriented to the glenoid fossa. Table 6-4 identifies the normal ranges of motion (ROMs) for the shoulder joint complex, and Table 6-5 describes the close-packed and loose-packed positions of the shoulder joints.

The minimally constrained ball-and-socket glenohumeral joint allows for a significant amount of movement, but is also susceptible to injury and instability. Rotational movements are the most frequent surface motion, although combinations of gliding and rolling also occur.¹⁷ For other movements, the humeral ball must displace with respect to the glenoid fossa.

Sternoclavicular Joint

Considerable gliding movements occur at the sternoclavicular joint, with the costoclavicular ligament serving as a fulcrum during shoulder motion. Similar to the acromioclavicular joint, in the sternoclavicular joint, the meniscus divides the joint into two functional units. A-P gliding occurs between the sternum and the meniscus, and superior-to-inferior (S-I) gliding occurs between the clavicle and the meniscus.¹² Rotation of the clavicle about its long axis is possible. A reciprocal motion occurs between the sternoclavicular joint and acromioclavicular joint during glide motions but not during rotational motions.

EVALUATION

The acromioclavicular joint is relatively weak and inflexible for the constant burden and repeated stresses it bears. A force applied to the acromion process or the glenohumeral joint from above causes the scapula to rotate around an axis located at the coracoid process. The acromioclavicular ligament, being intrinsically weak,

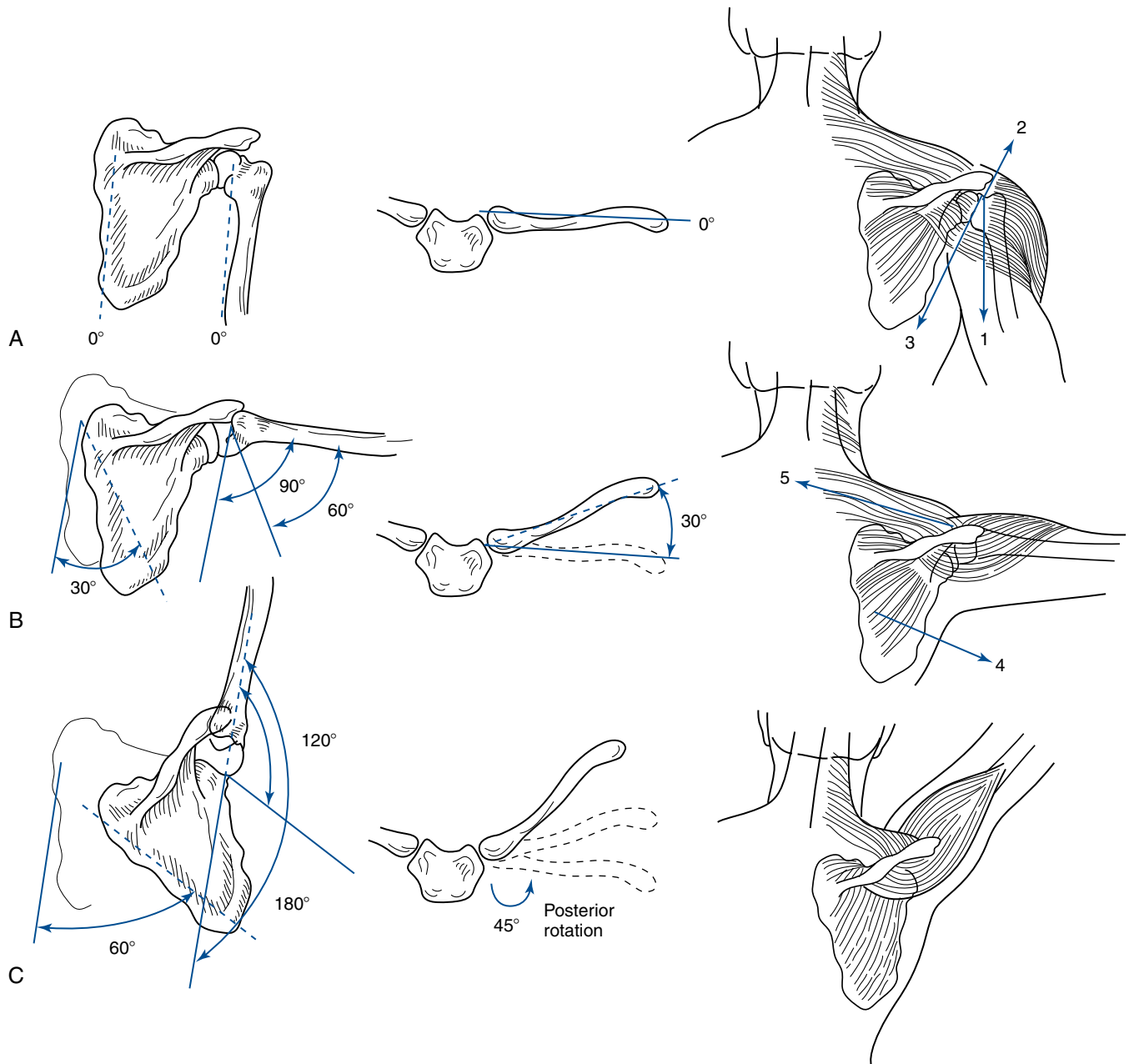


Figure 6-34 Scapulohumeral rhythm. **A**, Shoulder at 0-degree abduction, with the scapula, humerus, and clavicle in neutral positions. The weight of the extremity (1) is balanced by a force couple comprising an upward pull from the deltoid muscle (2) and a downward pull from rotator cuff muscles plus the pressure and friction of the humeral head (3). **B**, Shoulder at 90-degree abduction, with 30-degree scapular rotation and 60-degree humeral abduction; clavicle elevates 30 degrees; serratus anterior muscle (4) and upper trapezius muscle (5) force couple balance and rotate scapula. **C**, Shoulder at 180-degree abduction, with 60-degree scapular rotation and 120-degree humeral abduction; clavicle rotates 45 degrees to reach an additional 30-degree elevation. (Modified from Wadsworth CT: *Manual examination and treatment of the spine and extremities*, Baltimore, 1988, Williams & Wilkins.)

gives way, and the joint disrupts. A second mechanism of injury comes with a downward force of greater intensity, which lowers the clavicle on the first rib, with the rib becoming a fulcrum. Both the acromioclavicular and coracoclavicular ligaments tear, causing a complete acromioclavicular separation. This usually happens with a fall on the point of the shoulder or a fall on the hand of an outstretched arm. The sternoclavicular and costoclavicular ligaments also may be sprained during shoulder trauma.

Rotator cuff muscle strains are caused by falls on an outstretched arm, impingement against the coracoacromial arch, and minor or

repetitive stresses to a compromised tendon. Most injuries are to the supraspinatus tendon.

Because of the relatively poor blood supply near the insertion of the supraspinatus, nutrition to this area may not meet the demands of the tendon tissue. An inflammatory response arises in the tendon, creating a tendinitis that probably is a result of the release of enzymes and resultant dead tissue acting as a foreign body. The body may react by laying down scar tissue or even calcific deposits. This is then referred to as *calcific tendinitis*.

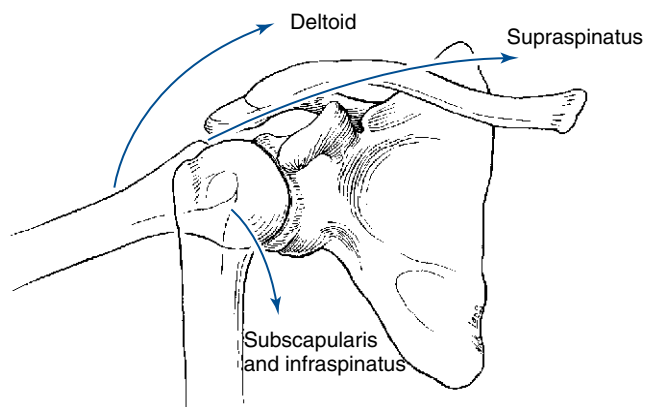


Figure 6-35 The actions of the rotator cuff muscles to depress the humeral head during shoulder abduction.

Bursitis, as a primary condition resulting from local trauma, is rare. As a secondary progression from tendinitis, however, it is very common. Acute bulging of the tendon compresses the bursa against the coracoacromial arch, resulting in inflammation and swelling of the bursa. This produces severe limitation of motion, as well as severe pain. The subacromial and subdeltoid bursae are most frequently involved.

Capsulitis (adhesive capsulitis or “frozen shoulder”) is a further progression of tendinitis-bursitis clinical phenomenon, with resultant adherence of the bursal walls, causing the supraspinatus and deltoid muscles to become “stuck together.” Continued immobility leads to capsular tightening and eventual capsular fibrosis. Degenerative joint disease, rheumatoid arthritis, immobilization, and reflex sympathetic dystrophy also cause capsular tightening that may lead to capsulitis.

The shoulder and arm are common sites for referred pain from the cervical spine, myocardium, gallbladder, liver, diaphragm, and breast. Usually the patient’s history suggests the origin of pain. Moreover, many of the muscles responsible for shoulder movement and proper function receive innervation from the C5 or C6 nerve root (NR), again signifying the importance of evaluating the cervical spine.

To begin the evaluation of the shoulder, observe the shoulder posture for the presence of asymmetry of shoulder heights, position of scapulae, and position of the humerus. Inspect the soft tissues for signs of atrophy and swelling. Identify osseous symmetry and pain production through static palpation of the sternoclavicular joint, clavicle, coracoid process, acromioclavicular joint, acromion process, greater tuberosity, bicipital groove, lesser tuberosity, spine of the scapula, and borders and angles of the scapula. Tone, texture, and tenderness changes should be identified through soft tissue palpation of the bursa, pectoralis major, biceps, deltoid, trapezius, rhomboids, levator, latissimus dorsi, serratus anterior, rotator cuff muscles, and teres major. Accessory joint motions for each of the four-component articulations should be evaluated when joint dysfunction is suspected (Table 6-6).

Assess long-axis distraction of the glenohumeral joint with the patient in the supine position and the involved arm at the side of body. Stand at the side of the table and use the inside hand to stabilize the scapula in the patient’s axilla. Use the other hand to grasp the humerus and stress it caudal, feeling for a springing motion (Figure 6-36).

Evaluate A-P glide with the patient supine and the involved arm slightly abducted. Standing between the patient’s arm and table, grasp the axillary, medial aspect of the proximal humerus with your inside hand and grasp the anterolateral aspect of the

TABLE 6-4 Arthrokinematic and Osteokinematic Movements of the Shoulder Joints

Osteokinematic Movements	Degrees	Arthrokinematic Movements
Glenohumeral flexion	120	Rotation and glide
Glenohumeral extension	55	Rotation and glide
Glenohumeral abduction	120	Roll and glide
Glenohumeral adduction	45	Roll and glide
Glenohumeral internal rotation	90	Rotation
Glenohumeral external rotation	90	Rotation
Clavicle internal and external rotation	10	Rotation
Clavicle elevation and depression	5	Roll and glide
Clavicle abduction and adduction	10	Roll and glide
Scapular internal and external rotation	25	Rotation and glide

TABLE 6-5 Close-Packed and Loose-Packed (Rest) Positions for the Shoulder Joints

Articulation	Close-Packed Position	Loose-Packed Position
Glenohumeral	Full abduction with external rotation	55 degrees of abduction with 30 degrees of horizontal adduction
Acromioclavicular	90 degrees of abduction	Physiologic rest position
Sternoclavicular	Full arm elevation	Physiologic rest position

TABLE 6-6 Accessory Joint Movements of the Shoulder Joint Complex

Joint	Movement
Glenohumeral	Long-axis distraction
	A-P glide
	P-A glide
	Internal rotation
	External rotation
	M-L glide
	Inferior glide in flexion
	Inferior glide in abduction
Sternoclavicular	I-S glide
	S-I glide
	A-P glide
	P-A glide
Acromioclavicular	I-S glide
	S-I glide
	A-P glide
	P-A glide
Scapulocostal	L-M glide
	M-L glide
	Clockwise rotation
	Counterclockwise rotation

A-P, Anterior-to-posterior; I-S, inferior-to-superior; L-M, lateral-to-medial; M-L, medial-to-lateral; P-A, posterior-to-anterior; S-I, superior-to-inferior.

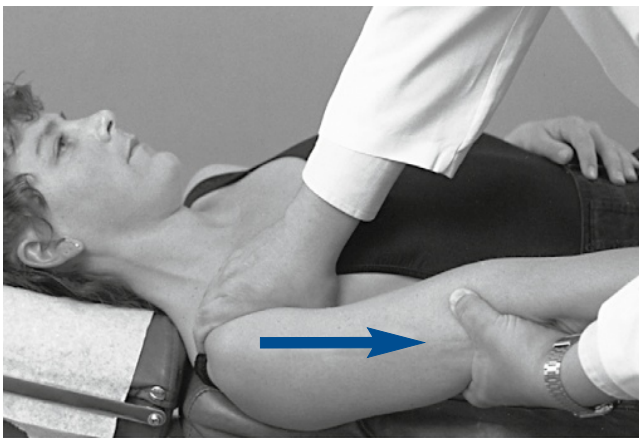


Figure 6-36 Assessment of long-axis distraction (inferior glide) of the right glenohumeral joint.

proximal humerus with your outside hand. Thumbs should be together. Stress the proximal humerus anteriorly to posteriorly by applying pressure with the thumbs. Conduct P-A glide with these same contacts, stressing the proximal humerus posteriorly to anteriorly with the fingers (Figure 6-37).

Medial-to-lateral (M-L) glide can be done with the patient in the supine position. Grasp the medial aspect of the proximal humerus with one hand while stabilizing the distal humerus at the elbow with the other hand. Using the elbow as a fulcrum, stress the proximal humerus medially to laterally (Figure 6-38).

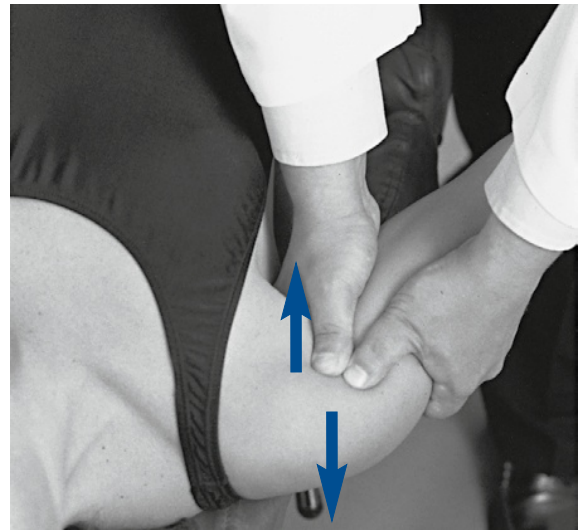


Figure 6-37 Assessment of anterior-to-posterior and posterior-to-anterior glide of the right glenohumeral joint.



Figure 6-38 Assessment of medial-to-lateral glide Long Axis Distraction (LAD) of the right glenohumeral joint.

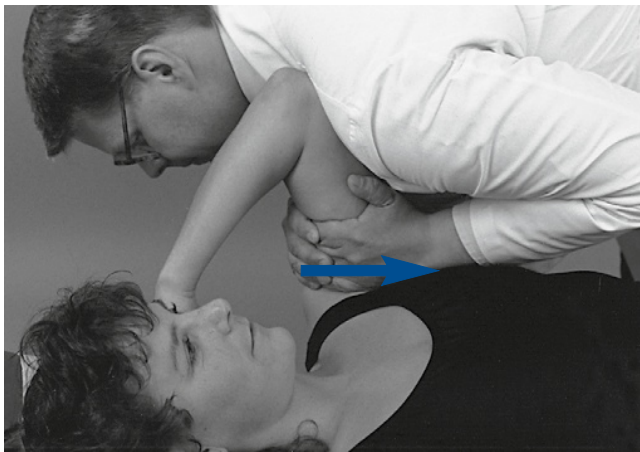
Assess internal and external rotation with the patient in the supine position, with the involved arm slightly abducted. Stand at the side of the table and use both hands to grasp the proximal humerus. Stress the humerus internally and externally (Figure 6-39).

Evaluate inferior glide in flexion with the patient in the supine position and the involved arm flexed to 90 degrees. Stand at the side of table, interlace the fingers of both hands around the proximal humerus, and rest the patient's elbow against your shoulder. Using the patient's elbow against your shoulder as a fulcrum, stress the patient's shoulder inferiorly (Figure 6-40).



▶ 6-39

Figure 6-39 Assessment of external rotation of the right glenohumeral joint.



▶ 6-40

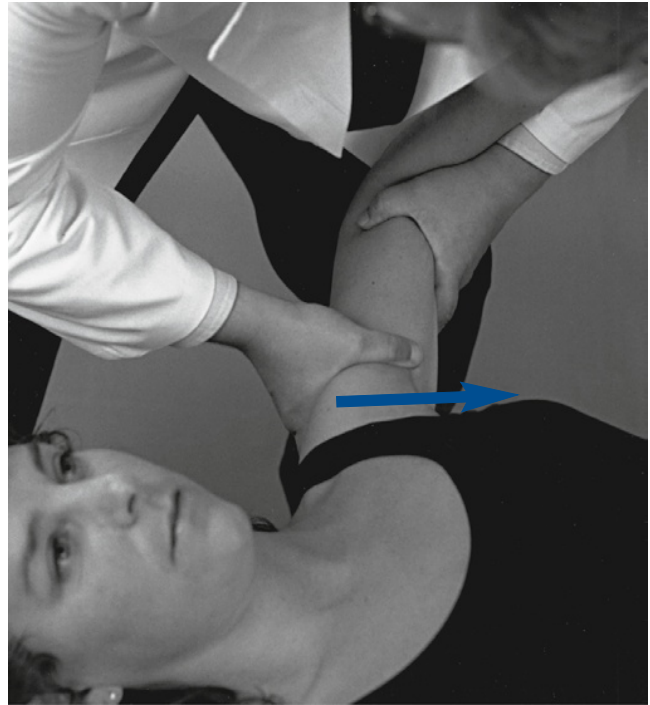
Figure 6-40 Assessment of inferior glide in flexion of the left glenohumeral joint.

Perform inferior glide in abduction with the patient in the supine position, with the involved arm abducted to 90 degrees. Stand at the head of the table and place the cephalic hand on the superior aspect of the proximal humerus while grasping the inferior aspect of the elbow with the other hand. Then stress the proximal humerus inferiorly (Figure 6-41).

The sternoclavicular joint has accessory movements in inferior glide, superior glide, posterior glide, and anterior glide. To evaluate each of these movements, stand behind the seated patient. Reach in front of the patient's neck and use a thumb contact on the proximal clavicle to stress it inferiorly, superiorly, posteriorly, and anteriorly (Figure 6-42).

Evaluate the acromioclavicular joint for accessory movements standing at the patient's side, with the patient seated. Grasp the distal clavicle with one hand and stabilize the scapula and shoulder with the other hand. Then stress the distal clavicle anteriorly, posteriorly, inferiorly, and superiorly (Figure 6-43).

The scapulocostal joint, although not an anatomic joint, also undergoes a stress evaluation. The findings are somewhat different in that a springing end feel or joint play movement is not expected. The stress evaluation is used to determine the integrity of the subscapular soft tissues and the supporting musculature. Decreased movement is still the positive finding.



▶ 6-41

Figure 6-41 Assessment of inferior glide in abduction of the left glenohumeral joint.

The movements assessed are medial glide, lateral glide, and rotation in both directions. Perform the evaluation standing at the side of the table, with the patient lying in the prone position. For medial glide, bring the patient's arm to rest along the side of the body and use both hands to contact the lateral aspect (axillary border) of the scapula. Stress the scapula medially (Figure 6-44). For rotational glide moving the inferior angle medially, place the patient's arm in the small of the back and use both hands to grasp the scapula so that the caudal thumb hooks the lateral aspect of the inferior angle. With both hands, twist the scapula so that the inferior angle is stressed medially (Figure 6-45). For rotational glide moving the inferior angle laterally, place the patient's hand behind his or her head and use both hands to grasp the scapula so that the cephalic thumb hooks the superior aspect of the axillary border and the caudal fingers hook the medial aspect of the inferior angle. With both hands, twist the scapula so that the inferior angle is stressed laterally (Figure 6-46). For M-L glide, the patient's arm hangs off the table; stand on the opposite side of the table and use both hands to contact the medial aspect (vertebral border) of the scapula. With both hands, stress the scapula laterally (Figure 6-47).

ADJUSTIVE PROCEDURES

The manipulative techniques used to treat shoulder disorders aim to restore normal joint mechanics, which will then ideally allow full pain-free functioning of the shoulder joints. All four joints should be evaluated for characteristics of dysfunction when there are shoulder symptoms present. Box 6-5 identifies the adjustive procedures for the joints of the shoulder.

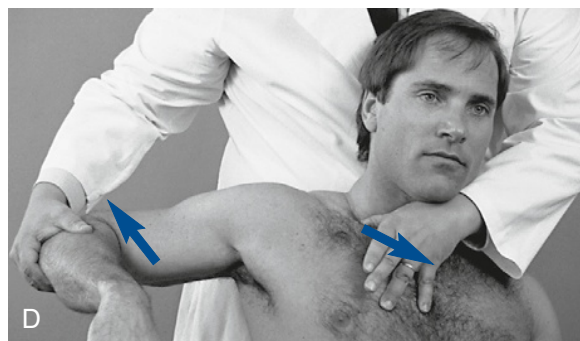
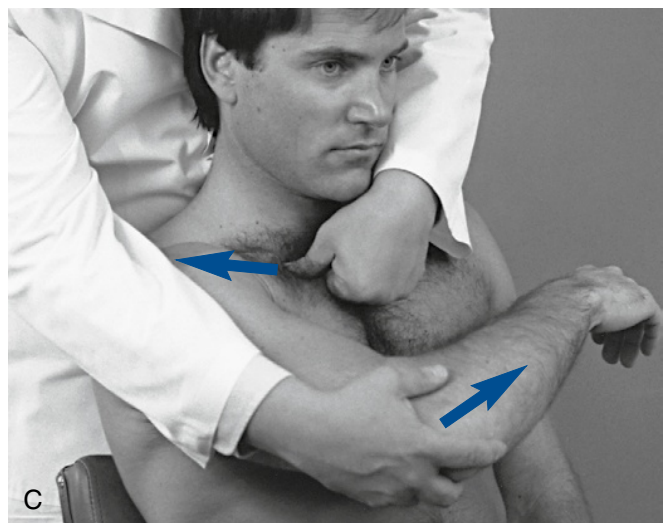
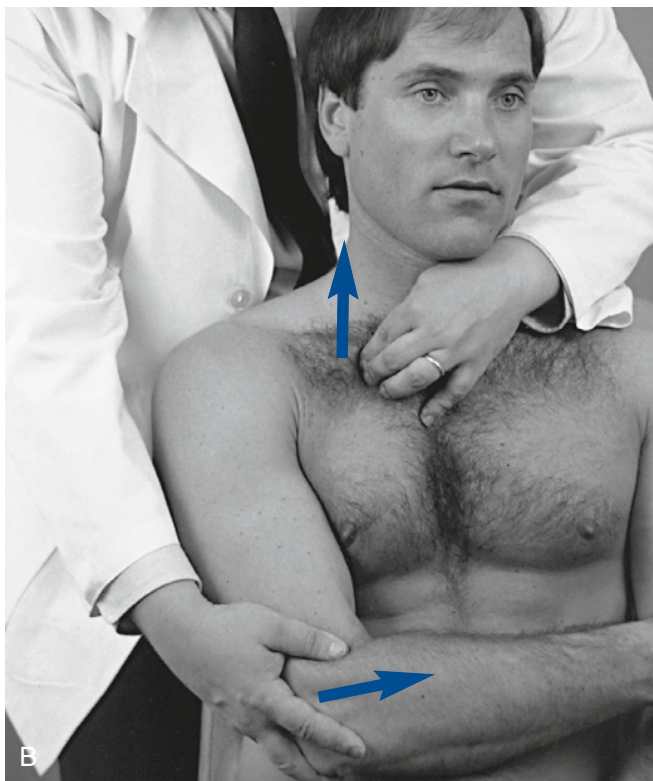
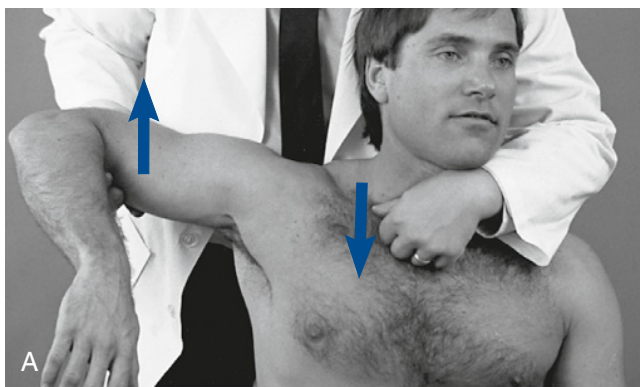


Figure 6-42 Assessment of the right sternoclavicular joint. **A**, Superior-to-inferior glide. **B**, Inferior-to-superior glide. **C**, Anterior-to-posterior glide. **D**, Posterior-to-anterior glide.

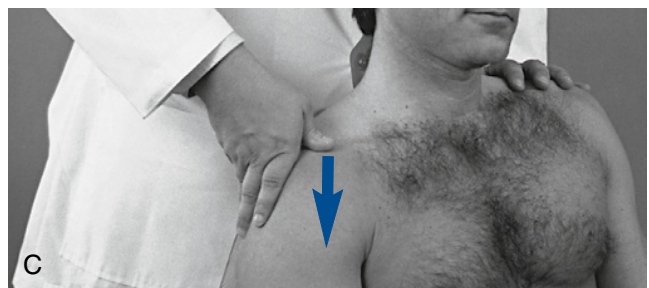


Figure 6-43 Assessment of the right acromioclavicular joint. **A**, Posterior-to-anterior glide. **B**, Anterior-to-posterior glide. **C**, Superior-to-inferior glide. **D**, Inferior-to-superior glide.



6-44

Figure 6-44 Assessment of the right scapulocostal articulation for lateral-to-medial glide.



6-45

Figure 6-45 Assessment of the right scapulocostal articulation for clockwise rotation, gliding the inferior angle medially.

Glenohumeral Joint

Supine:

Thumb Web/Axilla with Knee Extension: Long-Axis Distraction (Figure 6-48)

IND: Loss of long-axis accessory movement, superior misalignment of the humerus.

PP: The patient is supine, with the involved arm along the body.

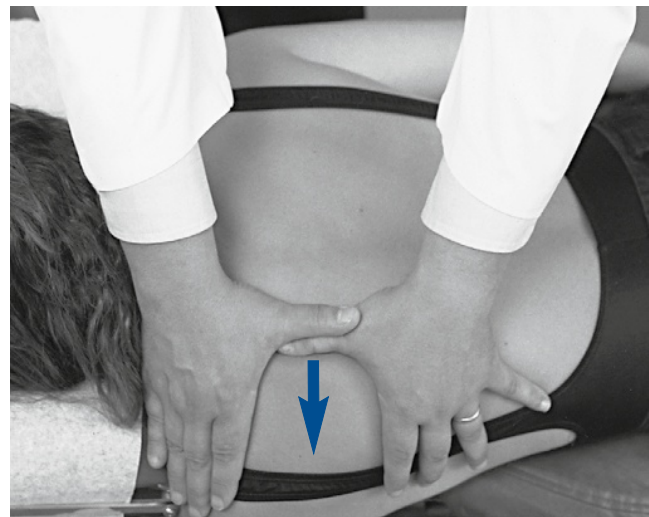
DP: Stand on the involved side, bring the patient's arm into slight abduction, and straddle the arm so that your slightly bent knees can grasp the patient's distal humerus just proximal to the epicondyles.

SCP: The patient's axilla.



6-46

Figure 6-46 Assessment of the right scapulocostal articulation for counterclockwise rotation, gliding the inferior angle laterally.



6-47

Figure 6-47 Assessment of the left scapulocostal articulation for medial-to-lateral glide.

CP: With your inside hand, establish a thumb web contact in the patient's axilla while applying downward pressure with your fingers on the shoulder girdle to stabilize it against the table.

IH: With your outside hand, use a digital contact over the lateral aspect of the joint to monitor for movement.

VEC: Long-axis distraction.

P: While maintaining the shoulder girdle against the table and applying slight superior pressure with the CP, make a quick "bunny hop" movement by extending both knees and drawing the humerus into long-axis distraction.

Bimanual Thumb Thenar Grasp/Proximal Humerus with Knee Extension; Anterior-to-Posterior Glide (Figure 6-49)

IND: Loss of A-P accessory movement, anterior misalignment of the humerus.

PP: The patient lies in the supine position, with the involved arm in slight abduction and the glenohumeral joint positioned off the edge of the table.

BOX 6-5 Shoulder Adjustive Techniques**GLENOHUMERAL SUPINE:**

Thumb web/axilla with knee extension; long-axis distraction (Figure 6-48)

Bimanual thumb thenar grasp/proximal humerus with knee extension; anterior-to-posterior glide (Figure 6-49)

Interlaced digital/proximal humerus; superior-to-inferior glide in flexion (Figure 6-50)

Index/proximal humerus; superior-to-inferior glide in abduction (Figure 6-51)

Bimanual thumb thenar grasp/proximal humerus with knee extension; internal or external rotation (Figures 6-52 and 6-39)

Bimanual thumb thenar grasp/proximal humerus; mobilization with distraction (Figure 6-53)

Bimanual grasp/hand; pendular abduction mobilization (Figure 6-54)

GLENOHUMERAL PRONE:

Bimanual thumb thenar grasp/proximal humerus with knee extension; posterior-to-anterior glide (Figure 6-55)

Bimanual thumb thenar grasp/proximal humerus; mobilization with distraction (Figure 6-56)

GLENOHUMERAL STANDING:

Interlaced digital/proximal humerus; superior-to-inferior glide in flexion (Figure 6-57)

Interlaced digital proximal humerus; superior-to-inferior glide in abduction (Figure 6-58)

GLENOHUMERAL SITTING:

Reinforced palmar/olecranon; anterior-to-posterior glide (Figure 6-59)

ACROMIOCLAVICULAR SUPINE:

Index/distal clavicle; superior-to-inferior glide (Figure 6-60)

Covered thumb/distal clavicle; inferior-to-superior glide (Figure 6-60)

Hypothenar/distal clavicle with distraction; anterior-to-posterior glide (Figure 6-62)

Digital/distal clavicle with distraction; posterior-to-anterior glide (Figure 6-63)

ACROMIOCLAVICULAR SITTING:

Web/distal clavicle; superior-to-inferior glide (Figure 6-64)

STERNOCLAVICULAR SUPINE:

Hypothenar/proximal clavicle with distraction; anterior-to-posterior glide (Figure 6-65)

Covered thumb/proximal clavicle; superior-to-inferior glide (Figure 6-66)

Covered thumb/proximal clavicle with knee extension; inferior-to-superior glide (Figure 6-67)

Digital/proximal clavicle with distraction; posterior-to-anterior glide (Figure 6-68)

Thenar/distal clavicle, thenar manubrium; long-axis distraction (Figure 6-69)

BOX 6-5 Shoulder Adjustive Techniques—Cont'd**STERNOCLAVICULAR SITTING:**

Reinforced thenar/proximal clavicle; inferior-to-superior glide (Figure 6-70)

Digital/proximal clavicle, thenar/manubrium; long-axis distraction (Figure 6-71)

SCAPULOCOSTAL SIDE POSTURE:

Bimanual thumb thenar/lateral scapula; lateral-to-medial glide (Figure 6-72)

Crossed bilateral mid-hypothenar (knife-edge)/scapula; medial-to-lateral glide (Figure 6-73)

Bimanual digital thenar grasp/scapula; rotation—inferior angle lateral to medial (Figure 6-74)

Bimanual digital thenar grasp/scapula; rotation—inferior angle medial to lateral (Figure 6-75)



Figure 6-48 Adjustment for long-axis distraction of the left glenohumeral joint.



Figure 6-49 Adjustment for anterior-to-posterior glide of the left glenohumeral joint in the supine position.

DP: Stand at the side of the table and straddle the affected arm so that the patient's epicondyles are held between your knees.

SCP: Proximal humerus.

CP: With both hands, grasp the proximal humerus with thumbs/thenars together in the midline.

VEC: A-P.

P: With your knees, provide slight distraction while applying an impulse thrust anteriorly to posteriorly with both hands.

Interlaced Digital/Proximal Humerus; Superior-to-Inferior Glide in Flexion (Figure 6-50)

IND: Loss of accessory movements in inferior glide in flexion, superior misalignment of the humerus.

PP: The patient is supine, with the involved arm raised to 90 degrees flexion and the elbow bent so that the hand rests on the shoulder.

DP: Stand on the involved side in a lunge position, facing cephalad and allowing the patient's elbow to rest against your shoulder.

SCP: Proximal humerus.

CP: Grasp the proximal humerus with both hands, using interlaced fingers over the superior aspect of the glenohumeral joint.

VEC: S-I.

P: Using the patient's elbow on your shoulder as a pivot point, apply S-I joint distraction with both hands, finishing with an S-I impulse thrust.



6-50

Figure 6-50 Adjustment for inferior glide in flexion of the left glenohumeral joint in the supine position.

Index/Proximal Humerus; Superior-to-Inferior Glide in Abduction (Figure 6-51)

IND: Loss of accessory movements in inferior glide in abduction, superior misalignment of the humerus.

PP: The patient is supine, with the involved arm abducted to 90 degrees.

DP: Stand on the involved side at the head of the table, facing caudal.

SCP: Superior aspect of the proximal humerus.

CP: Establish a web contact over the superior aspect of the proximal humerus with the cephalic hand.

IH: With your caudal hand, grasp the distal aspect of the patient's humerus.

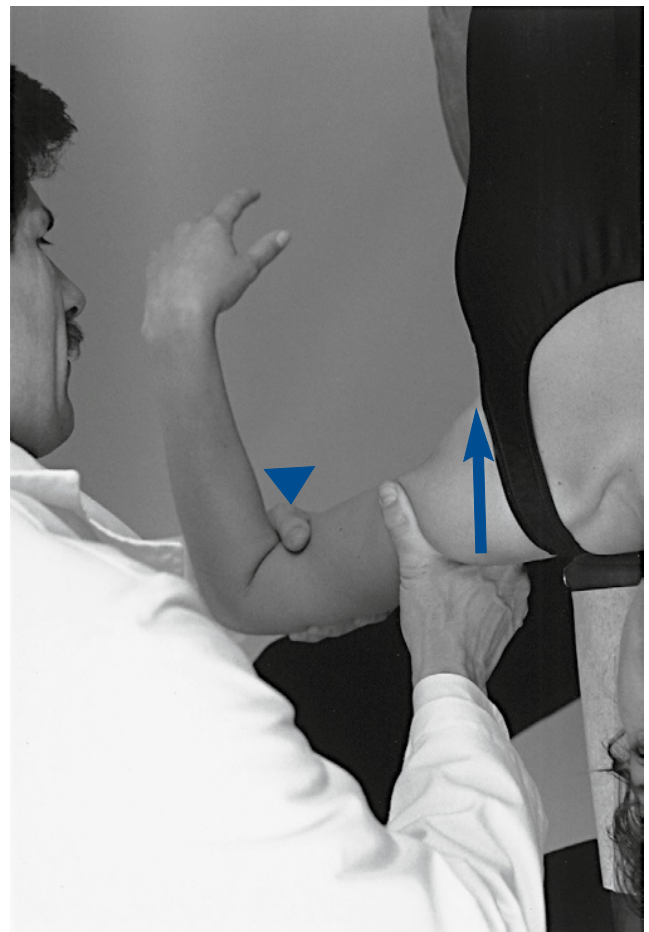
VEC: S-I.

P: Your IH serves as a pivot point, stabilizing the distal humerus and elbow, and your cephalic hand removes articular slack, finishing with an impulse-type thrust in an S-I direction.

Bimanual Thumb Thenar Grasp/Proximal Humerus with Knee Extension; Internal Rotation (Figure 6-52)

IND: Restricted internal rotation accessory joint movement, external rotation misalignment of the humerus.

PP: The patient is supine, with the affected arm abducted slightly away from the patient's body and the edge of the table in internal rotation.



6-51

Figure 6-51 Adjustment for inferior glide in abduction of the left glenohumeral joint in the supine position.

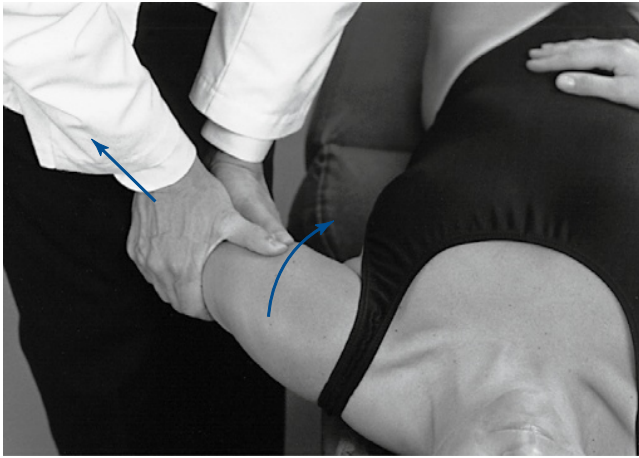


Figure 6-52 Adjustment for internal rotation of the left glenohumeral joint with distraction. External rotation is done with the same procedure, using external rotation prestress and thrust.

DP: Stand on the involved side, facing cephalad and straddling the patient's affected arm so that your knees can squeeze the distal humerus just above the epicondyles.

SCP: Proximal humerus.

CP: Grasp the patient's proximal humerus with interlaced fingers of both hands.

VEC: Rotational-internal rotation.

P: Your hand contacts first turn the humerus into internal rotation, removing articular slack. Simultaneously straighten both knees, applying a long-axis distraction to the glenohumeral joint.

Bimanual Thumb Thenar Grasp/Proximal Humerus with Knee Extension; External Rotation (see Figures 6-52 and 6-39)

IND: Restricted external rotation accessory joint movement, internal rotation misalignment of the humerus.

PP: The patient is supine, with the affected arm abducted slightly away from his or her body and the edge of the table, holding the arm in external rotation.

DP: Stand on the involved side, facing cephalad and straddling the patient's affected arm so that your knees can squeeze the distal humerus just above the epicondyles.

SCP: Proximal humerus.

CP: With your hand, grasp the patient's proximal humerus with interlaced fingers.

VEC: Rotational-external rotation.

P: Use both hands to turn the humerus into external rotation. Simultaneously straighten both knees to create a long-axis distraction to the glenohumeral joint.

Bimanual Thumb Thenar Grasp/Proximal Humerus Grasp; Mobilization with Distraction (Figure 6-53)

IND: Intracapsular adhesions in the glenohumeral joint and mobilization of the shoulder.

PP: The patient is supine, with the affected arm outstretched.

DP: Stand in a lunge position on the affected side, facing the head of the table.

SCP: Humerus.

CP: With your inside hand, grasp the patient's arm to hold the patient's forearm against your thoracic cage.

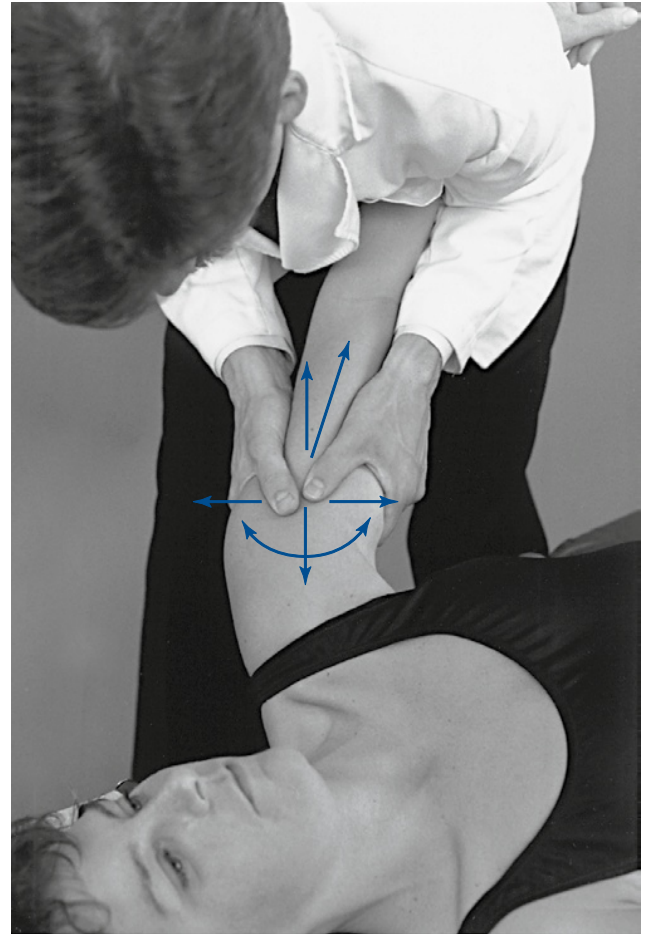


Figure 6-53 Glenohumeral mobilization in the supine position.

IH: With your outside hand, make a palmar contact on the posterior aspect of the shoulder and scapula to provide support and lift during the mobilization.

VEC: Circumduction and distraction.

P: Use your body weight to assist in producing a mild distraction and circumduction movement of the shoulder in all directions.

Bimanual Grasp/Hand; Pendular Abduction Mobilization (Figure 6-54)

IND: Intracapsular adhesions in the glenohumeral joint, mobilization of the shoulder, and adhesive capsulitis.

PP: The patient is supine, with the affected arm slightly abducted and the forearm flexed to 90 degrees, pointing upward.

DP: Stand at the side of the table on the involved side, facing the patient.

SCP: The hand.

CP: With both hands, grasp the patient's hand.

VEC: S-I with passive rocking.

P: Instruct the patient to relax the arm as much as possible. Raise the arm away from the table so that it can swing freely. Induce a pendular motion in the glenohumeral joint by rocking the forearm cephalad and caudal, increasing the arc of abduction motion as tolerated.

Prone:

Bimanual Thumb Thenar Grasp/Proximal Humerus with Knee Extension; Posterior-to-Anterior Glide (Figure 6-55)



Figure 6-54 Glenohumeral mobilization using pendular abduction.



Figure 6-55 Adjustment for posterior-to-anterior glide of the right glenohumeral joint in the prone position.

IND: Loss of P-A accessory movements; or posterior misalignment of the humerus.

PP: The patient lies in the prone position, with the involved arm in slight abduction and the glenohumeral joint positioned off the edge of the table.

DP: Stand at the side of the table and straddle the patient's affected arm, with the epicondyles held between your knees.

SCP: Proximal humerus.

CP: With both of your hands, grasp the proximal humerus with thumbs together in the midline.

VEC: P-A.

P: With your knees, provide slight distraction while applying an impulse thrust posteriorly to anteriorly with both hands.

Bimanual Thumb Thenar Grasp/Proximal Humerus; Mobilization with Distraction (Figure 6-56)

IND: Intercapsular adhesions and mobilization of the shoulder.

PP: The patient lies prone, with the affected arm hanging down and off the side of the table.

DP: Kneel at the side of the table, facing the patient.

SCP: Proximal humerus.

CP: Grasp the patient's proximal humerus with both hands, with your thumbs together on the posterior aspect of the humerus while your fingers wrap around and into the axilla on the underside of the humerus.

VEC: Circumduction.

P: Using both hands, first distract the glenohumeral joint in the long axis of the humerus, and then move the humerus toward and away from you, cephalad and caudal, in a figure-8 motion.

Standing:

Interlaced Digital/Proximal Humerus; Superior-to-Inferior Glide in Flexion (Figure 6-57)

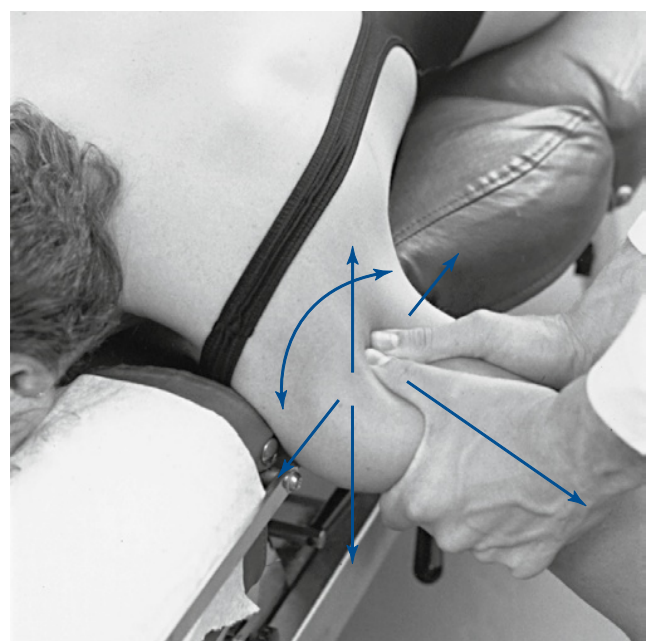


Figure 6-56 Glenohumeral mobilization in the prone position.



6-57

Figure 6-57 Adjustment for inferior glide in flexion of the right glenohumeral joint in the standing position.

IND: Loss of accessory movements in inferior glide in flexion; superior misalignment of the humerus.

PP: The patient stands, with feet spread at least shoulder-distance apart (or farther if the patient is taller than the doctor). The involved arm is flexed to 90 degrees, and the elbow is flexed so that the hand rests on the patient's shoulder.

DP: Stand in front of the patient and to the affected side. Your legs should be spread appropriately for balance, as well as to align to the patient's height.

SCP: Proximal humerus.

CP: First place the patient's elbow on your shoulder, then, using both hands, grasp the proximal humerus with your fingers interlaced on the superior aspect of the joint capsule while your thumbs wrap into the axilla.

VEC: S-I.

P: First draw away from the patient, creating a joint separation, and then apply a downward pressure to remove articular slack. Give a thrust in the S-I direction.

Interlaced Digital/Proximal Humerus; Superior-to-Inferior Glide in Abduction (Figure 6-58)

IND: Loss of accessory movements in inferior glide in abduction; superior misalignment of the humerus.

PP: The patient stands with legs at least shoulder-distance apart, with the involved arm abducted to 90 degrees and the elbow flexed so that the hand rests on the patient's shoulder.

DP: Stand with legs apart so that the patient's elbow can rest on your shoulder.

SCP: Proximal humerus.

CP: Grasp the proximal humerus, with interlaced fingers on the superior aspect and thumbs in the axilla.

VEC: S-I.

P: Back away from the patient to distract the joint while applying a downward pressure with the hands to remove articular slack. Give an impulse thrust in the S-I direction.

Sitting:

Reinforced Palmar/Olecranon; Anterior-to-Posterior Glide (Figure 6-59)



6-58

Figure 6-58 Adjustment for inferior glide in abduction of the right glenohumeral joint in the standing position.

IND: Loss of A-P accessory movement, anterior misalignment of the humerus.

PP: The patient sits with the arm in forward flexion, the elbow bent, and the hand resting on the opposite shoulder if internal rotation is also desired or on the same shoulder if external rotation is also desired.

DP: Stand behind the patient, slightly to the side of involvement, stabilizing the patient's shoulder girdle against the torso.



6-59

Figure 6-59 Adjustment for anterior-to-posterior glide of the right glenohumeral joint in the sitting position.

SCP: The olecranon process.

CP: With your ipsilateral hand, use a palmar contact to cup the patient's elbow.

IH: With your other hand, reinforce the CP.

VEC: A-P.

P: Using both hands, remove the articular slack and give a very quick and shallow thrust primarily in the axis of the humerus.

Acromioclavicular Joint

Supine:

Index/Distal Clavicle; Superior-to-Inferior Glide (Figure 6-60)

IND: Restricted S-I accessory movement of the distal clavicle, superior misalignment of the distal clavicle.

PP: The patient is supine, with the affected arm abducted to 90 degrees.

DP: Stand at the head of the table, facing caudal, to the side of the affected arm.

SCP: Superior aspect of the distal clavicle.

CP: Establish an index contact with the inside hand over the superior aspect of the distal clavicle.

IH: With your outside hand, grasp the humerus at midshaft.

VEC: S-I.

P: As your IH draws the humerus into long-axis distraction and abduction, apply an S-I impulse thrust with your contact hand.

Covered Thumb/Distal Clavicle; Inferior-to-Superior Glide (Figure 6-61)

IND: Loss of I-S accessory joint movement of the distal clavicle, inferior misalignment of the distal clavicle.

PP: The patient is supine, with the affected arm straight and slightly abducted.

DP: Stand at the side of the table, straddling the patient's affected arm so that your knees can grasp the distal humerus above the patient's epicondyles.

SCP: Inferior aspect of the distal clavicle.

CP: With your outside hand, apply a thumb contact on the inferior aspect of the proximal clavicle.



Figure 6-60 Adjustment for superior-to-inferior glide of the left acromioclavicular joint in the supine position.



Figure 6-61 Adjustment for inferior-to-superior glide of the right acromioclavicular joint.

IH: With your inside hand, place a pisiform contact over the thumbnail of the contact hand.

VEC: I-S.

P: As you straighten your knees to create a long-axis distraction of the shoulder joint, use both hands to deliver an I-S impulse thrust to the distal clavicle.

Hypothenar/Distal Clavicle with Distraction; Anterior-to-Posterior Glide (Figure 6-62)

IND: Restricted A-P accessory joint movement of the distal clavicle, anterior misalignment of the distal clavicle.

PP: The patient is supine, with the affected arm straight and forward flexed to approximately 60 degrees.

DP: Stand at the side of the table, opposite the involved side.

SCP: Anterior aspect of the distal clavicle.

CP: With your cephalic hand, establish a pisiform hypothenar contact over the anterior aspect of the distal clavicle.

IH: With your IH, grasp the outer aspect of the distal forearm.

VEC: A-P.

P: As you distract the shoulder anteriorly and inferiorly with your IH, apply an A-P impulse thrust to the distal clavicle with your contact hand.

Digital/Distal Clavicle with Distraction; Posterior-to-Anterior Glide (Figure 6-63)

IND: Restricted P-A accessory joint movement of the distal clavicle, posterior misalignment of the distal clavicle.

PP: The patient is supine, with the affected arm straight, flexed to approximately 60 degrees, and slightly abducted.

DP: Stand at the side of the table on the affected side, facing cephalad, between the patient's affected arm and the table.

SCP: Posterosuperior aspect of the distal clavicle.

CP: With your inside hand, place the digital contact of the index and middle fingers over the posterosuperior aspect of the distal clavicle.

IH: With your outer hand, grasp the patient's distal forearm.

VEC: P-A.

P: With your IH, distract the shoulder anteriorly and while maintaining distraction, flex the arm, raising it past 90 degrees. As the articular slack is taken out, use your contact hand to



Figure 6-62 Adjustment for anterior-to-posterior glide of the right acromioclavicular joint.

deliver a very quick and shallow P-A impulse thrust to the distal clavicle (lifting the distal clavicle).

Sitting:

Web/Distal Clavicle; Superior-to-Inferior Glide (Figure 6-64)

IND: Restricted S-I accessory movement of the distal clavicle, superior misalignment of the distal clavicle.

PP: The patient sits, with the affected arm abducted.

DP: Stand behind the patient and to the side of the affected arm.

SCP: Superior aspect of the distal clavicle.

CP: With your inside hand, apply a web contact over the superior aspect of the distal clavicle.

IH: With your outside hand, grasp the patient's distal forearm.

VEC: S-I.

P: While your IH uses the patient's forearm as a lever to distract and abduct the shoulder joint, deliver an S-I impulse thrust with your contact hand.

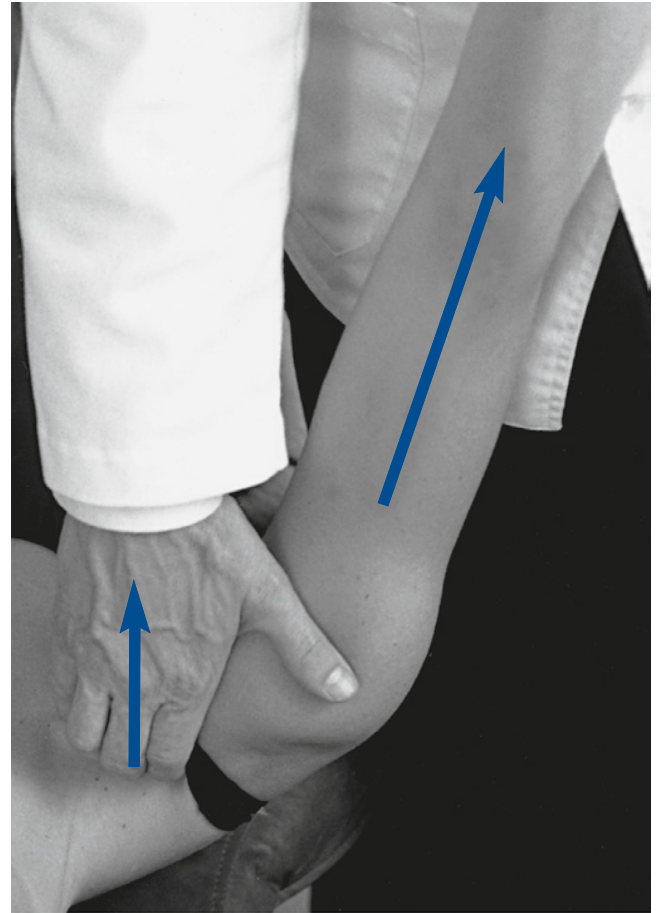


Figure 6-63 Adjustment for posterior-to-anterior glide of the right acromioclavicular joint.



Figure 6-64 Adjustment for superior-to-inferior glide of the right acromioclavicular joint in the seated position.

Sternoclavicular Joint

Supine:

Hypothenar/Proximal Clavicle with Distraction; Anterior-to-Posterior Glide (Figure 6-65)

IND: Restricted A-P accessory movement of the proximal clavicle, anterior misalignment of the proximal clavicle.



Figure 6-65 Adjustment for anterior-to-posterior glide of the right sternoclavicular joint.

PP: The patient is supine, with the involved arm flexed forward to approximately 60 degrees.

DP: Stand on the side of the table on the side of involvement, facing cephalad.

SCP: Anterior aspect of the proximal clavicle.

CP: Use your inside hand to apply a pisiform-hypothenar contact over the anterior aspect of the proximal clavicle.

IH: With your outside hand, grasp the outer aspect of the distal humerus at the epicondyles.

VEC: A-P.

P: With your IH, distract the shoulder anteriorly, raising the shoulder and scapula off the adjusting table. As the articular slack is removed, deliver an A-P impulse thrust over the proximal clavicle with the contact hand.

Covered Thumb/Proximal Clavicle; Superior-to-Inferior Glide (Figure 6-66)

IND: Loss of S-I accessory motion of the proximal clavicle, superior misalignment of the proximal clavicle.

PP: The patient is supine, with the involved arm abducted to 90 degrees and the hand placed under the head.

DP: Stand at the head of the table, facing caudal.

SCP: Superior aspect of the proximal clavicle.

CP: With your ipsilateral hand, place a thumb contact on the superior aspect of the proximal clavicle.

IH: With your contralateral hand, place a pisiform-hypothenar contact over the thumb contact.

VEC: S-I.

P: Deliver an impulse thrust with both hands in an S-I direction on the proximal clavicle.



Figure 6-66 Adjustment for superior-to-inferior glide of the left sternoclavicular joint.

Covered Thumb/Proximal Clavicle with Knee Extension; Inferior-to-Superior Glide (Figure 6-67)

IND: Loss of I-S accessory joint movement of the proximal clavicle, inferior misalignment of the proximal clavicle.

PP: The patient is supine, with the affected arm slightly abducted.

DP: Stand on the affected side, straddling the patient's arm and grasping the distal humerus between your knees.

SCP: Inferior aspect of the proximal clavicle.

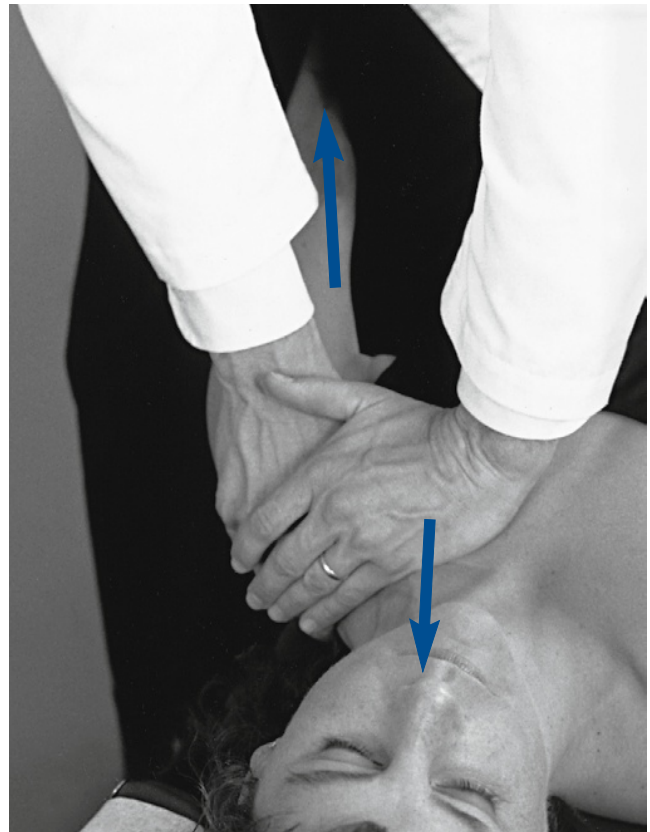


Figure 6-67 Adjustment for inferior-to-superior glide of the left sternoclavicular joint in the supine position.

CP: With your outside hand, place a thumb contact on the inferior aspect of the proximal clavicle.

IH: With your inside hand, place a pisiform-hypothenar contact over the thumb contact for reinforcement.

VEC: I-S.

P: Use your knee contact on the patient's distal humerus to distract the shoulder girdle caudally. When articular slack has been removed, apply an impulse thrust through both hands in an I-S direction on the proximal clavicle.

Digital/Proximal Clavicle with Distraction; Posterior-to-Anterior Glide (Figure 6-68)

IND: Loss of P-A accessory joint movement of the proximal clavicle, posterior misalignment of the proximal end of the clavicle.

PP: The patient is supine.

DP: Stand at the side of the table on the affected side, facing cephalad.

SCP: Posterosuperior aspect of the proximal clavicle.

CP: With your inside hand, apply the digital contact of the index and middle fingers over the posterosuperior aspect of the proximal clavicle.

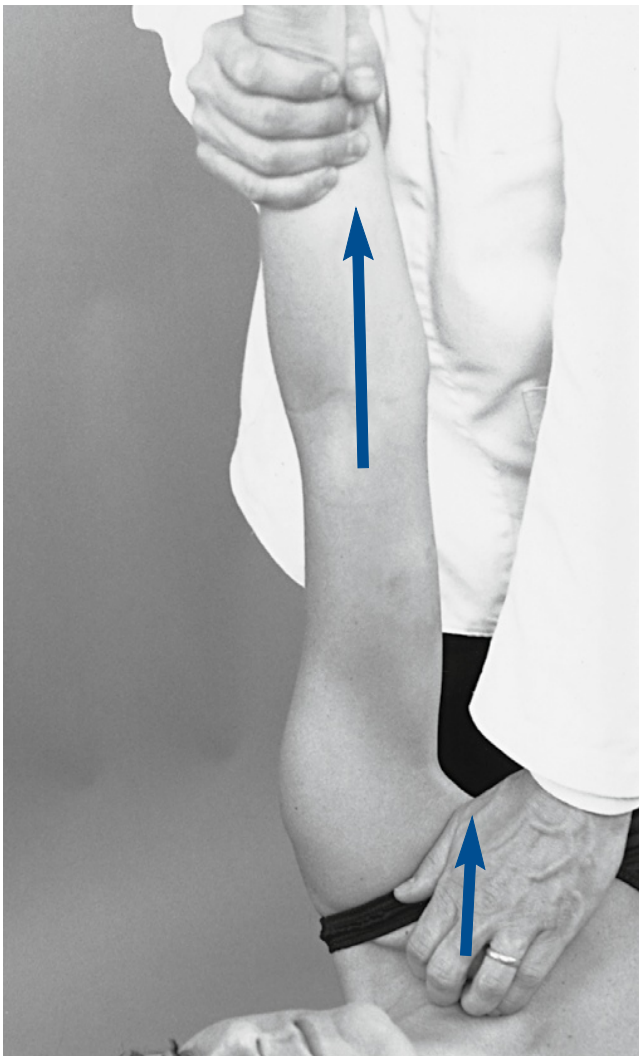


Figure 6-68 Adjustment for posterior-to-inferior glide of the left sternoclavicular joint.

IH: Grasp the patient's distal forearm with your outer hand.

VEC: P-A.

P: With your IH, distract the shoulder anteriorly and while maintaining distraction, flex the arm, raising it past 90 degrees. As the articular slack is taken out, use your contact hand to deliver a very quick and shallow P-A impulse thrust to the proximal clavicle (lifting the proximal clavicle).

Thenar/Distal Clavicle, Thenar/Manubrium; Long-Axis Distraction (Figure 6-69)

IND: Generalized decrease in movement of the sternoclavicular joint, and displacement of the intraarticular meniscus.

PP: The patient is supine, with a rolled towel or small cylindrical pillow placed under the upper thoracic spine. The affected arm is abducted to approximately 90 degrees.

DP: Stand on the affected side in a lunge position, facing cephalad.

SCP: Distal clavicle.

CP: With your outside hand, place a thenar contact over the distal clavicle and grasp the deltoid area.

IH: With your inside hand, place a thenar contact over the manubrium of the sternum, with the thumb pointing cephalad and the fingers pointing laterally across the contralateral clavicle.

VEC: Distraction.

P: With your IH, stabilize the patient's manubrium and opposite shoulder against the table, applying a downward pressure. The pillow or rolled towel serves as a fulcrum as you apply a shallow impulse thrust to the distal clavicle and shoulder to distract the proximal clavicle from the manubrium. Alternatively, the clinician can use crossed arms, applying hypothenar knife-edge contacts on the clavicle and manubrium.

Sitting:

Reinforced Thenar/Proximal Clavicle; Inferior-to-Superior Glide (Figure 6-70)

IND: Loss of I-S accessory joint movement of the proximal clavicle, inferior misalignment of the proximal clavicle.

PP: The patient sits, with arms relaxed.

DP: Stand behind the patient.



Figure 6-69 Distraction of the right sternoclavicular joint in the supine position. The patient lies on a sternal roll or rolled towel, which serves as a fulcrum for distraction.

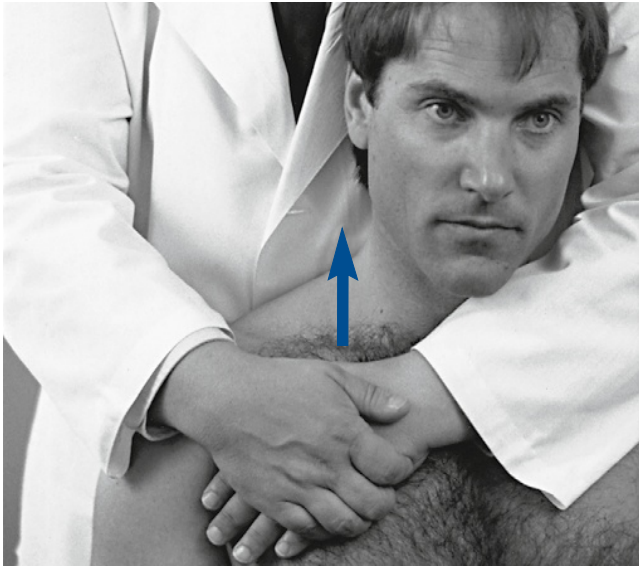


Figure 6-70 Adjustment for inferior-to-superior glide of the right sternoclavicular joint in the seated position.

SCP: Inferior aspect of the proximal clavicle.

CP: With your contralateral hand, establish a thenar contact on the inferior aspect of the proximal clavicle.

IH: With your ipsilateral hand, take a calcaneal contact over the thenar contact for reinforcement.

VEC: I-S.

P: Stabilize the patient's torso against the back of the chair or your body. Deliver an impulse thrust with both hands in an I-S direction.

Digital Proximal/Clavicle, Thenar/Manubrium; Long-Axis Distraction (Figure 6-71)

IND: Generalized decrease in movement of the sternoclavicular joint, and displacement of the intra-articular meniscus.

PP: The patient sits, with the affected arm abducted to approximately 90 degrees.

DP: Stand behind the patient and slightly to the side of involvement.

SCP: Proximal clavicle.

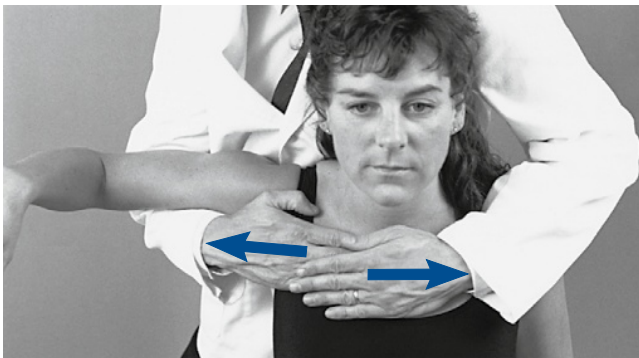


Figure 6-71 Distraction of the right sternoclavicular joint in the seated position. The clinician's body is used as a fulcrum for separation.

CH: With your ipsilateral hand, reach under the patient's affected arm to support the patient's arm on your forearm. Make digital contact with the index and middle fingers on the proximal end of the clavicle.

IH: With your contralateral hand, make a thenar contact over the manubrium of the sternum, with the forearm lying across the contralateral clavicle.

VEC: Distraction.

P: With your IH, stabilize the manubrium and opposite shoulder girdle against the back of the chair or your body while your contact hand draws the affected clavicle medially to laterally and your arm draws the patient's affected shoulder slightly anteriorly to posteriorly. When articular slack is removed, give a quick and shallow impulse thrust, separating the proximal clavicle from the manubrium.

Scapulocostal Articulation

Side posture:

Bilateral Thumb Thenar/Lateral Scapula; Lateral-to-Medial Glide (Figure 6-72)

IND: Loss of L-M glide movement of the scapulocostal articulation, dysfunctional scapulohumeral rhythm, subscapular adhesions.

PP: The patient is in a side-lying position, with the affected side up and the arm resting on the side.

DP: Stand at the side of the table, facing the patient.

SCP: Lateral border of the scapula.

CP: With both hands, establish a thumb, thenar, and calcaneal contact over the axillary (lateral) border of the scapula, with the fingers pointing toward the spine.

VEC: L-M.

P: Draw the scapula laterally to medially, and when the end of passive movement is reached, give an L-M impulse thrust.

Crossed Bilateral Mid-Hypothenar (Knife-Edge)/Medial Scapula; Medial-to-Lateral Glide (Figure 6-73)

IND: Loss of medial-to-lateral glide movement of the scapulocostal articulation, dysfunctional scapulohumeral rhythm, subscapular adhesions.

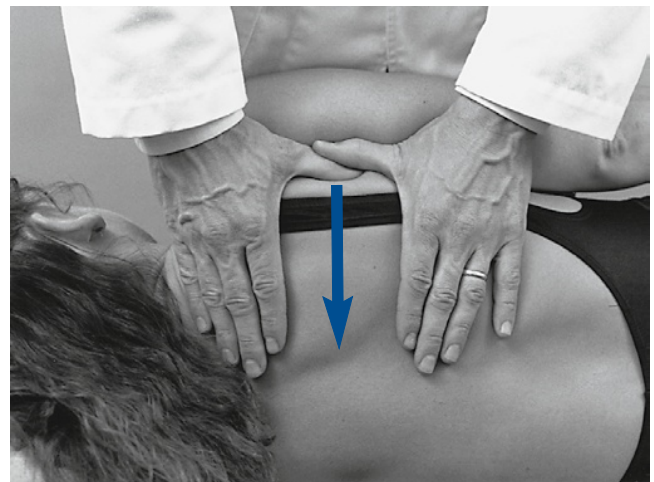
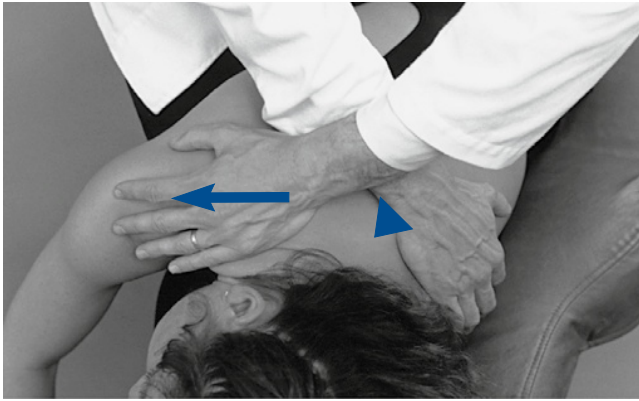


Figure 6-72 Manipulation for lateral-to-medial glide of the right scapulocostal articulation.



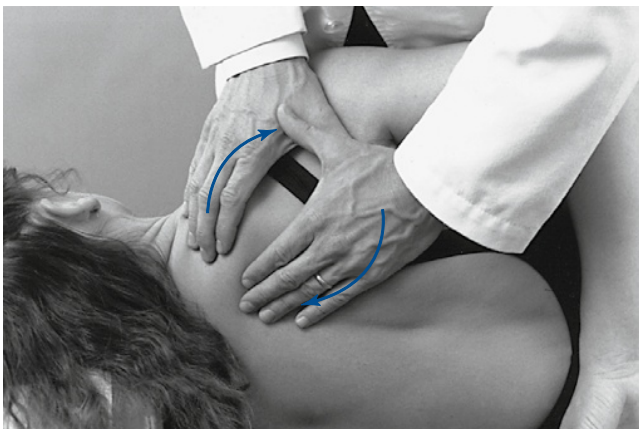
6-73

Figure 6-73 Manipulation for medial-to-lateral glide of the right scapulocostal articulation.

- PP:** The patient is in a side-lying position, with the affected arm hanging forward in front of the table.
- DP:** Stand at the side of the table and to the front of the patient in a lunge position (fencer stance), facing cephalad.
- SCP:** Medial (vertebral) border of the scapula.
- CP:** Use your caudal hand to apply a metacarpophalangeal (MP) (knife-edge) contact over the vertebral (medial) border of the affected scapula, with the fingers over the spine and body of the scapula.
- IH:** With your cephalic hand, establish a calcaneal contact over the vertebral border of the other scapula, with the fingers over the body of the scapula.
- VEC:** M-L.
- P:** Use both hands in opposing directions to draw passive movement medially to laterally and administer an impulse thrust primarily through the contact hand medially to laterally.

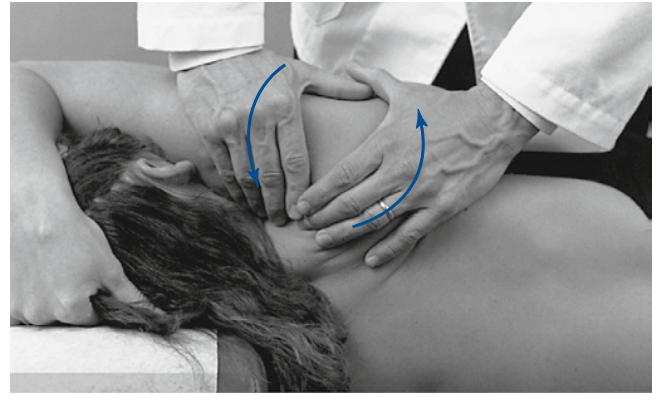
Bimanual Digital Thenar Grasp/Scapula; Rotation—Inferior Angle Lateral to Medial (Figure 6-74)

- IND:** Loss of rotational movement of the scapulocostal articulation, dysfunctional scapulohumeral rhythm, subscapular adhesions.



6-74

Figure 6-74 Manipulation for rotation of the right scapulocostal articulation, moving the inferior angle in an lateral-to-medial direction (clockwise).



6-75

Figure 6-75 Manipulation for rotation of the right scapulocostal articulation, moving the inferior angle in an medial-to-lateral direction (counterclockwise).

- PP:** The patient is in a side-lying position, with the affected side up and the affected arm placed behind the back, with the fist in the small of the back.
- DP:** Stand at the side of the table, facing the patient.
- SCP:** Lateral aspect of the inferior angle of the scapula.
- CP:** With your caudal hand, apply a thenar contact on the lateral aspect of the inferior angle of the scapula, with the fingers lying across the scapula and pointing toward the spine.
- IH:** With your cephalic hand, place a thenar contact on the superior aspect of the spine of the scapula, with the fingers pointing toward the inferior angle.
- VEC:** Rotational.
- P:** Use both hands to induce a rotational, twisting action, using an impulse-type thrust to drive the inferior angle of the scapula laterally to medially.

Bimanual Digital Thenar Grasp/Scapula; Rotation—Inferior Angle Medial to Lateral (Figure 6-75)

- IND:** Loss of rotational movement of the scapulocostal articulation, dysfunctional scapulohumeral rhythm, subscapular adhesions.
- PP:** The patient is in a side-lying position, with the affected side up and the affected arm abducted, with the hand behind the head.
- DP:** Stand at the side of the table, facing the patient.
- SCP:** Medial aspect of the inferior angle of the scapula.
- CH:** With your caudal hand, establish a pisiform-hypothenar contact on the medial aspect of the inferior angle of the scapula, with the fingers pointing toward the axilla.
- IH:** With your cephalic hand, grasp the spine of the scapula.
- VEC:** Rotational.
- P:** Use both hands to create a rotational, twisting action, using an impulse-type thrust to drive the inferior angle medially to laterally.

ELBOW

Although outwardly the elbow appears to be a simple singular joint, it is actually an intricate mechanism that depends on the integrated action of three bones that form four distinct articulations. This peripheral joint complex must work together to enable

the movements of flexion and extension (humero-ulnar and humero-radial joints), as well as pronation and supination (proximal and distal radioulnar joints) of the forearm and hand. Performance of the unique manual skills of the upper extremity depends largely on the proper functioning of the bones, ligaments, and muscles around the elbow joint.

FUNCTIONAL ANATOMY

Osseous Structures

The cylindrical shaft of the humerus becomes flattened and spreads out distally to form the medial and lateral epicondyles. The distal end of the humerus contains two articular surfaces: the trochlea, which resembles an hourglass on its side, and the capitulum, which is spherical (Figure 6-76). The radial fossa and coronoid fossa on the anterior surface and the olecranon fossa on the posterior surface allow an increased range of flexion and extension by delaying impact of the respective bony prominences on the humeral shaft.¹⁶ The proximal end of the ulna contains the coronoid process and olecranon process, with the trochlear notch lying between. This surface of the ulna articulates with the trochlea of the humerus. The trochlear articular surface is asymmetric, which directs the ulna, and hence the forearm, into an abducted position on full extension. The forearm forms an angle with the arm ranging from 5 to 15 degrees, which is commonly referred to as the *carrying angle* (Figure 6-77).

The superior surface of the radial head is concave to accept the spherical capitulum. The radial or bicipital tuberosity protrudes anteriorly and medially, just distal to the head of the radius.

Ligamentous Structures

Besides the joint capsule that encloses the three-joint complex (humero-ulnar joint, radiohumeral joint, and radioulnar joint), three primary ligaments stabilize the elbow. The annular ligament encircles the head of the radius and maintains its contact with the ulna. It is lined with articular cartilage so that the radial head has an articular surface with the ulna, humerus, and annular ligament (Figure 6-78). The medial and lateral collateral ligaments reinforce the joint capsule of the elbow. They restrict medial and lateral angulation and glide of the ulna on the humerus. Each collateral ligament spreads from its respective epicondyle attachment to reinforce the annular ligament anteriorly while providing medial and lateral stability through attachments to the radius and ulna, respectively.

Musculature

Several significant muscle groups cross the elbow joint and, therefore, serve as predominant stabilizers of the elbow (Table 6-7). The wrist flexors cross the elbow to attach at the medial epicondyle, and the wrist extensors cross the elbow to attach to the lateral epicondyle. Tendons from both of these muscles blend into the fibers of their respective collateral ligaments. Although these muscles cross the elbow joint, their primary function is wrist movement. Elbow flexion is accomplished by the brachialis,

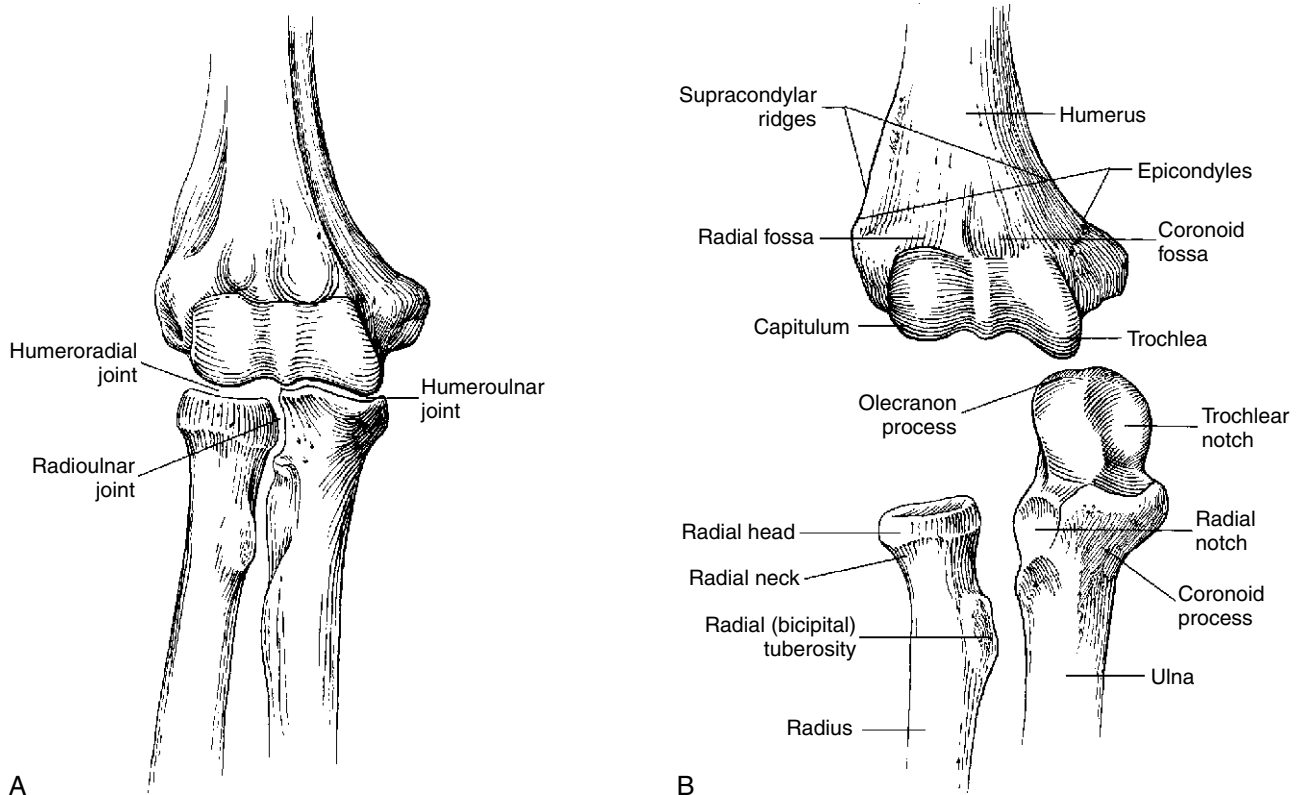


Figure 6-76 Anterior view of the right elbow. **A**, Three joints of the elbow. **B**, Osseous structures.

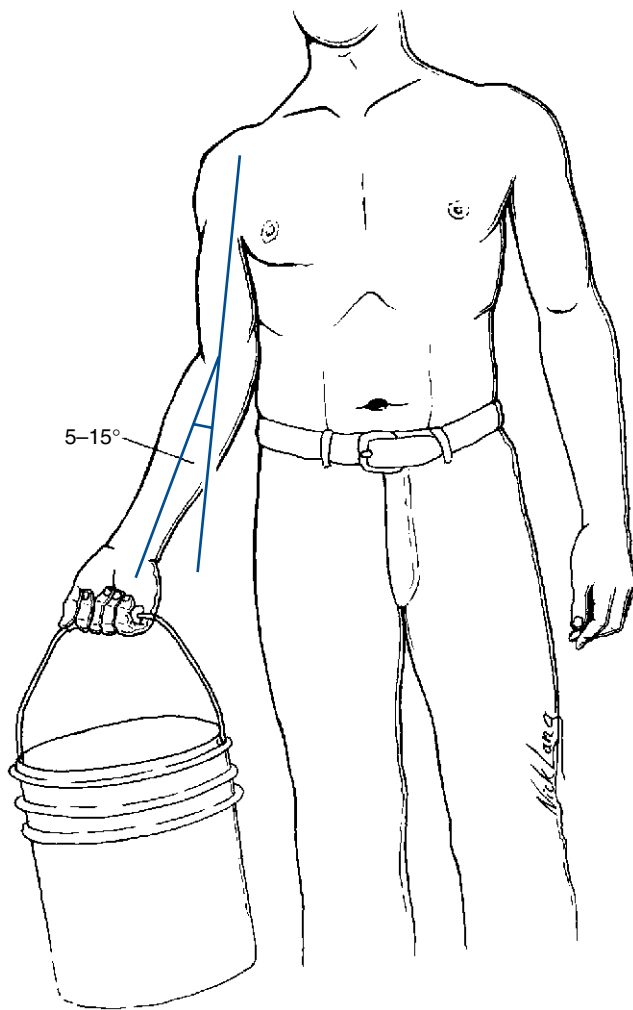


Figure 6-77 Carrying angle, a valgus angle ranging from 5 to 15 degrees.

brachioradialis, and biceps brachii muscles. Extension of the elbow occurs through the action on only one muscle, the triceps brachii, although the anconeus is thought to provide some extension. Supination of the forearm is produced by the contraction of the supinator and, to a lesser degree, the biceps. The pronator quadratus and pronator teres muscles provide the contractive force for the pronation of the forearm.

The olecranon bursa lies between the skin and the olecranon process to reduce friction between the large bony process and the skin (Figure 6-79). A bicipitoradial bursa lies between the tendon of the biceps muscle and the radius.

BIOMECHANICS

The elbow joint is a modified hinge, classified as a compound paracondylar joint in that one bone, the humerus, articulates with two others that lie side by side by way of two distinct facets.¹⁸ Flexion and extension movements occur between the trochlea of the humerus and the trochlear notch of the ulna. Within a single

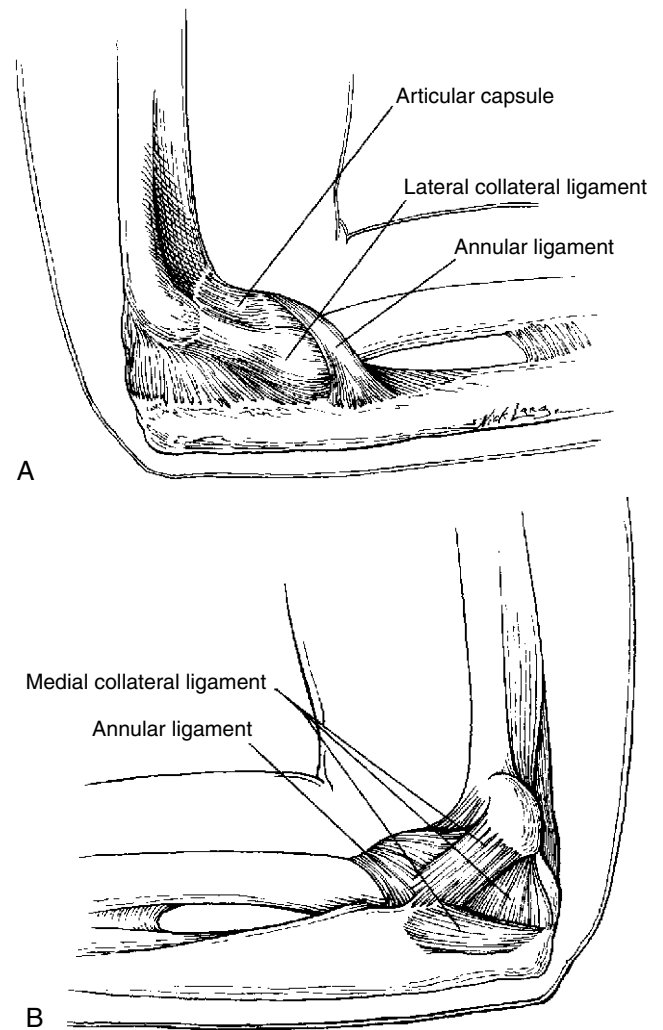


Figure 6-78 Ligaments of the right elbow. **A**, Lateral view. **B**, Medial view.

TABLE 6-7 Actions of the Muscles of the Elbow

Action	Muscles
Flexion	Brachialis
Flexion in supination	Biceps
Rapid flexion or flexion with loads	Brachioradialis
Extension	Triceps and anconeus
Supination	Supinator and biceps
Pronation	Pronator quadratus
Rapid pronation or pronation with loads	Pronator teres
Medial stability and some extension	Wrist flexors
Lateral stability and some flexion	Wrist extensors

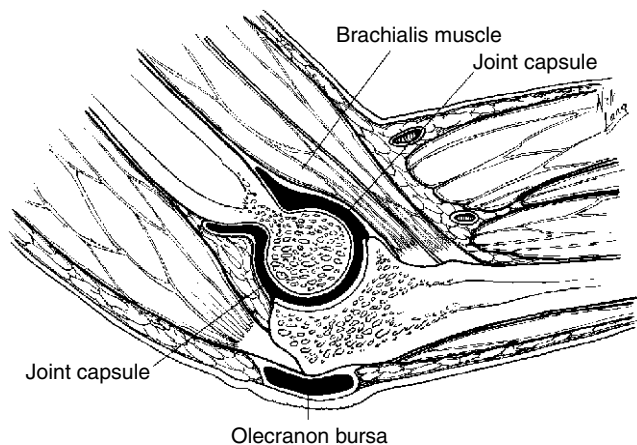


Figure 6-79 Sagittal section through the elbow, showing the olecranon bursa.

joint cavity lie the hinged (ginglymus) humeroulnar articulation, the gliding (plane) humeroradial articulation, and the pivotal (trochoid) superior radioulnar articulation.¹⁵ Table 6-8 shows the normal ROMs for the elbow joint.

Flexion and extension movements occur around an axis that passes through the centers of arcs described by the trochlear sulcus and the capitulum, permitting 145 degrees of active and 160 degrees of passive movement from full extension to flexion (Figure 6-80). Extension with supination is the closed-packed position for the humeroulnar joint, whereas the closed-packed position for the humeroradial joint is at 90 degrees of flexion with 5 degrees of supination. For the elbow to move from extension to flexion, the radius and ulna must undergo roll and glide movements in relation to the capitulum and the trochlea, respectively. Active flexion is limited by compression of the soft tissues in the anterior aspect of the forearm and arm, with passive flexion being limited by the tension of the posterior joint capsule and tension in the triceps. Extension is limited by contact of the olecranon process in the olecranon fossa.

The anatomic and mechanical arrangement of the proximal radioulnar joint accounts for the uniqueness of this joint, but also adds difficulty to its treatment. A fibro-osseous ring, which is part of the annular ligament, as opposed to true articular cartilage, composes 80% of the articular surface. The distal radioulnar joint has a convex ulnar head fitting into a shallow concavity on the radius. The distal ulnar also articulates with the triangular fibrocartilage forming part of the ulnocarpal joint.

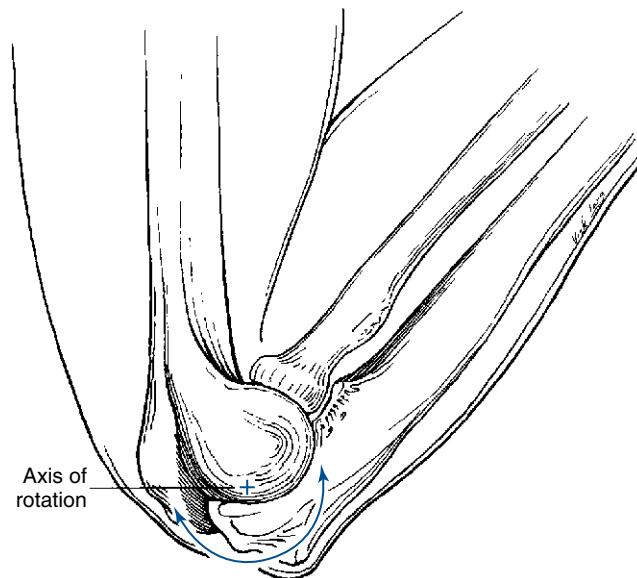


Figure 6-80 The axis of movement for flexion and extension of the elbow.

The mechanical axis for forearm pronation and supination movements is a line passing from the center of the radial head to near the styloid process of the ulna distally. Normally, between 175 and 180 degrees of movement occur from pronation to supination. This motion is primarily the function of the proximal and distal radioulnar joint with some rotational movement occurring at the humeroradial joint (Figure 6-81). During pronation, the radius (and hand) cross over a relatively fixed ulna. The ulna is essentially stationary through the movements of supination and pronation. The radius and the ulna are bound together by the interosseous membrane. Compressive and distractive forces along the length of the forearm place stress on the attachments of the interosseous membrane that can be a source of deep aching pain in the forearm. The distal radioulnar joint will be considered with the wrist and hand.

As mentioned, when the arm is extended in its anatomic position, the longitudinal axis of the arm and forearm forms a lateral (valgus) angle at the elbow, known as the *carrying angle*. The normal carrying angle measures approximately 5 degrees in men and between 10 and 15 degrees in women. An increased angulation can be caused by epiphyseal damage secondary to

TABLE 6-8 Arthrokinematic and Osteokinematic Movements of the Elbow Joints

Osteokinematic Movements	Degrees	Arthrokinematic Movements
Flexion	135 to 165	Roll and glide
Extension	0 to 5	Roll and glide
Supination	90	Rotation and glide
Pronation	90	Rotation and glide

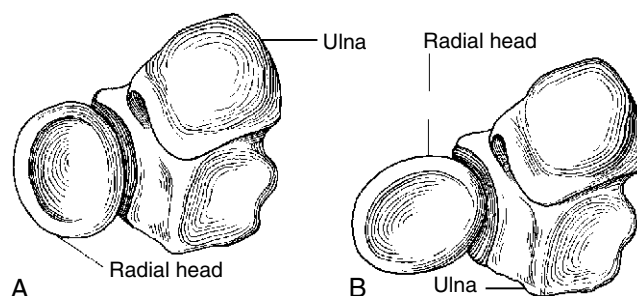


Figure 6-81 Transverse section through the proximal radioulnar articulation. A, Pronation. B, Supination.

TABLE 6-9 Close-Packed and Loose-Packed (Rest) Positions for the Elbow Joints

Articulation	Close-Packed Position	Loose-Packed Position
Humeroulnar	Full extension in supination	70 degrees of flexion with 10 degrees of supination
Humeroradial	90 degrees of flexion with 5 degrees of supination	Full extension and supination
Proximal radioulnar	5 degrees of supination	35 degrees of supination with 70 degrees of flexion

lateral epicondylar fracture and may cause a delayed nerve palsy, which presents as an ulnar nerve distribution in the hand. A decrease in the carrying angle, called a *gunstock deformity*, is often the result of trauma, such as a supracondylar fracture in childhood.

Arthrokinematics of the elbow are dictated by the spiral shape of the articular surface of the trochlea. Flexion and extension movements are impure swing movements that couple adduction with flexion and abduction with extension. The close-packed and loose-packed positions for the elbow joints are identified in Table 6-9.

EVALUATION

The elbow is exposed to numerous traumatic events that can lead to joint injury and dysfunction. A common cause of elbow problems is a result of muscle activity across the joint. Lateral epicondylitis results from such activity of the wrist extensors. The extensor mass, and especially the deeply located extensor carpi radialis, rubs and rolls over the lateral epicondyle and radial head during forced contraction of the muscle. The forced contractions of the muscle group produce tugs on the origin, resulting in microtears in the tendon and a pulling away of the periosteum. This, coupled with the irritation of soft tissue rubbing over bony prominences, results in a painful elbow condition. Typically, a pain pattern extending down the forearm, following the extensor muscle group, and point tenderness over the lateral epicondyle both occur. The pain is intensified by resisted extension of the wrist and fingers, as well as by shaking hands. The pain may progress to the point that the patient has difficulty picking up a coffee cup or turning a door-knob. The action of the backhand stroke in tennis has been a frequent cause, hence the name *tennis elbow*.

Medial epicondylitis occurs because of forced muscle activity of the wrist flexors. The clinical picture is much the same as for lateral epicondylitis, but the pain is medial and follows the wrist flexors. It has been referred to as *golfer's elbow*, being associated with forced flexion during a golf swing.

Subluxation of the radial head can occur in a young person who is forcibly pulled up from the floor by grabbing the wrist. This action creates traction on the annular ligament on one side by the pull of the arm and on the other side by the pull of the body. This condition, called *pulled elbow* or *nursemaid's elbow*, results in a limitation of supination and tenderness over the radial head. The patient will usually refuse to use the arm, and it will hang limp at the side, with the hand in pronation.

Trauma to the posterior aspect of the elbow, either by a fall on the flexed elbow or by recurrent irritation, can lead to inflammation of the olecranon bursa. Swelling will be visible and palpable and will result in pain on palpation and movement. Olecranon bursitis is frequently seen in individuals such as students who lean on their elbows on a hard surface for long periods. The ligamentous stability of the elbow can be breached, causing an elbow sprain in hyperextension, hyperabduction, and hyperadduction.

The ulnar nerve is vulnerable to trauma as it passes through the ulnar groove at the medial aspect of the elbow. It can be contused by a direct blow, stretched by a valgus force to the elbow, trapped in scar tissue after trauma to the elbow, and irritated by bone spurs. Any or all of these processes can create a peripheral entrapment of the ulnar nerve known as *cubital tunnel syndrome*. Elbow pain may or may not be associated with this problem. The cardinal symptoms of ulnar nerve injury are tingling and burning of the little finger and ulnar half of the ring finger. Motor function of the opponens digiti minimi and interosseous muscles also may be impaired.

The effective treatment and management of patients with elbow problems obviously depends on first establishing the nature and extent of the lesion, being aware of which anatomic structures have been potentially injured. The elbow is largely derived from C6 and C7 and may therefore be a site of referred pain, as well as a source for referral of pain to other structures from these segments (Figure 6-82).

To begin the evaluation of the elbow, observe the elbow for evidence of swelling, asymmetry of contours, posture, and attitude. Also note functional use of the arm during gait, position changing, and other activities. Evaluate the carrying angle by having the patient straighten his or her supinated elbows (anatomic position) and measuring the angle from the junction of the longitudinal axis of the upper arm and forearm.

Identify osseous symmetry and pain production through static palpation of the radial head, medial epicondyle, lateral epicondyle, olecranon process, and fossa. Structural integrity of the elbow joint can be evaluated through the relationship of the olecranon process to the humeral epicondyles. With the elbow extended and viewed from the posterior, the three landmarks should lie in a horizontal line. Then flex the elbow to 90 degrees; the three landmarks should form an isosceles triangle, with the apex pointing downward (Figures 6-83 and 6-84). Any appreciable deviation from this alignment may indicate some anatomic problem and may require further investigation.¹⁹

Identify tone, texture, and tenderness changes through soft tissue palpation of the olecranon bursa, collateral ligaments, annular ligament, ulnar nerve, wrist flexor muscle group, wrist extensor muscle group, triceps, brachial radialis, and biceps. Evaluate accessory joint motions for the elbow articulations (Box 6-6) to

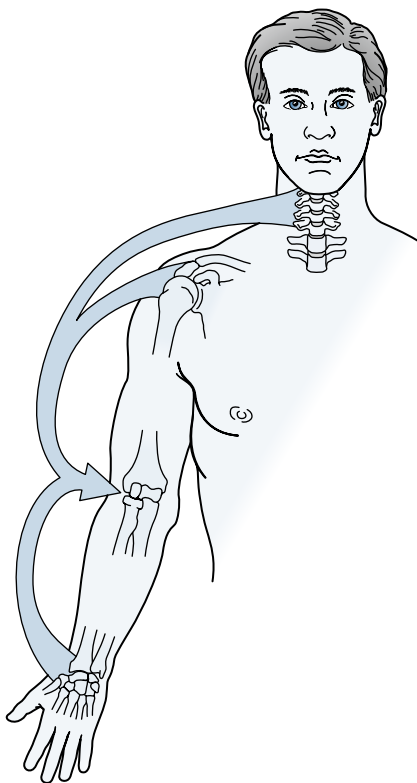


Figure 6-82 The elbow may be the site of referred pain, as well as the source of referral. (From Magee DJ: *Orthopedic Physical Assessment*, ed 5, St Louis, 2008, Saunders.)

determine the presence of joint dysfunction. Assess long-axis distraction, primarily of the humeroulnar joint, with the patient sitting or supine and his or her elbow bent slightly. Stand to the side of involvement, facing the patient, and use your inside hand to stabilize the humerus while your outside hand grasps the distal

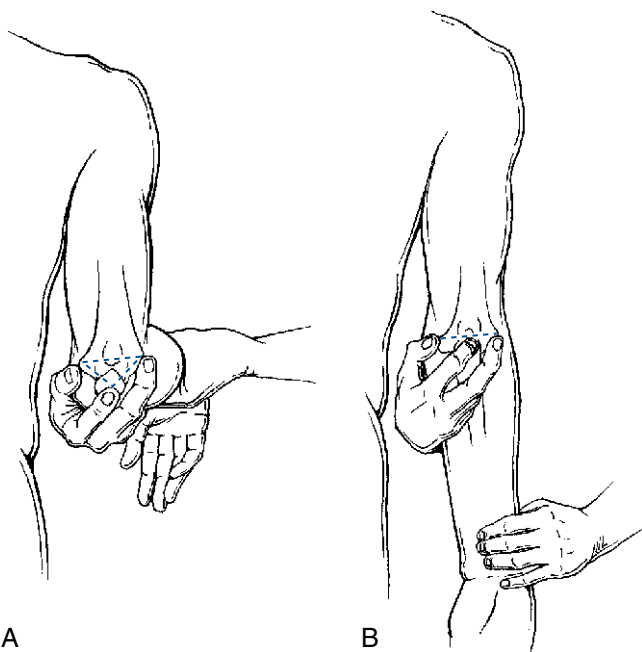


Figure 6-83 Palpatory relationship between the epicondyles and olecranon process in a normal elbow.

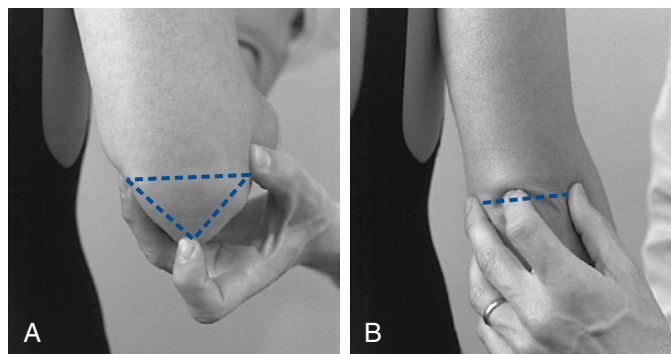


Figure 6-84 Palpatory relationship between the epicondyles and olecranon process. **A**, Flexion landmarks form an isosceles triangle. **B**, Extension landmarks form a straight line. Deviations indicate a structural problem.

BOX 6-6 Accessory Joint Movements of the Elbow Joint

- Long-axis distraction
- Medial-to-lateral glide
- Lateral-to-medial glide
- Posterior-to-anterior glide in extension
- Anterior-to-posterior glide (radioulnar joint)
- Posterior-to-anterior glide (radioulnar joint)
- Posterior-to-anterior glide (radioulnar joint in pronation)

forearm. Then stress the forearm along its long axis, feeling for a springing end feel (Figure 6-85).

Evaluate M-L glide of the humeroradial and humeroulnar joints with the patient seated and the affected arm extended at the elbow and flexed at the shoulder. Stand and face the patient on the medial side of the affected arm. Stabilize the patient's arm against your body with your outer arm while your inside hand takes a calcaneal contact over the medial aspect of the elbow joint. With the forearm stabilized, stress the elbow from medial to lateral, assessing for the presence of a springing joint play movement (Figure 6-86).

Assess L-M glide of the humeroradial and humeroulnar joint with the patient in a seated position and the affected arm extended at the elbow and flexed at the shoulder. Stand and face the patient on

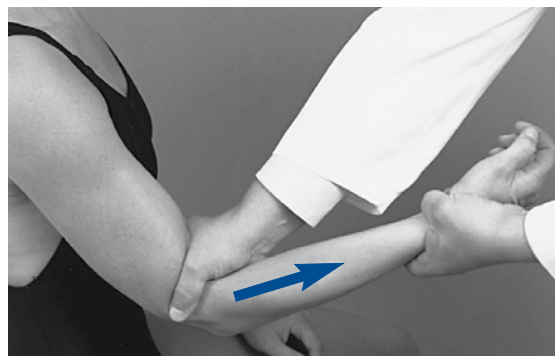


Figure 6-85 Assessment of long-axis distraction of the right humeroulnar joint.

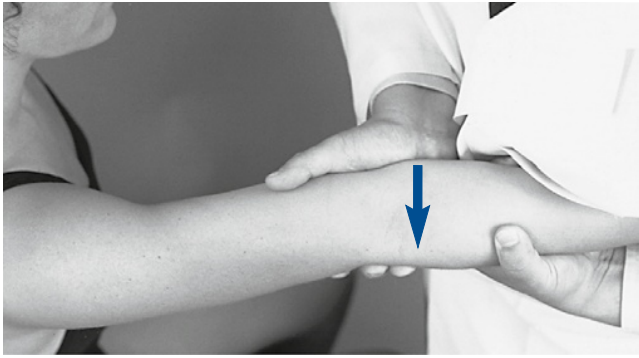


Figure 6-86 Assessment of medial-to-lateral glide of the right humeroulnar joint.

the lateral aspect of the affected arm. Stabilize the patient's forearm, using the inside arm to hold the patient's arm against your body. Your outside arm takes a calcaneal contact over the lateral aspect of the elbow joint. With the patient's forearm stabilized against your body, stress the elbow laterally to medially, determining the presence of a springing joint play movement (Figure 6-87).

Evaluate P-A glide of the humeroulnar joint in extension, with the patient sitting and the affected arm extended at the elbow and flexed at the shoulder. Stand and face the patient on the lateral side



Figure 6-87 Assessment of lateral-to-medial glide of the right humeroulnar joint.

of the affected arm. Form a ring with your thumb and index finger of your outside hand and place it over the posterior aspect of the olecranon process. Rest your other hand on the anterior aspect of the distal forearm. With very little downward pressure on the distal forearm, apply a gentle P-A stress to the olecranon process, looking for a springing joint play movement (Figure 6-88).

Assess A-P and P-A glide of the radioulnar joint, with the patient in the seated position and the affected arm extended at the elbow and flexed at the shoulder. Stand and face the patient on the lateral aspect of the affected arm. With your inside arm, stabilize the patient's forearm against your body and grasp the distal humerus and proximal ulna. With your outside hand, hold the radial head between the thumb and index finger. While stabilizing the ulna and humerus, stress the radial head from anterior to posterior and from posterior to anterior, determining the presence of a springing joint play movement (Figure 6-89).

Evaluate P-A glide of the radioulnar joint in pronation with the patient in the seated position and the affected arm extended at the elbow and flexed at the shoulder. Stand and face the lateral aspect of the affected arm. With your outside hand, grasp the distal forearm, with digital contacts of the index, middle, and ring fingers on the posterior aspect of the radius. With your inside hand, place a thumb contact on the posterior aspect of the radial head. Use your outside hand to pronate the forearm. At the contact over the

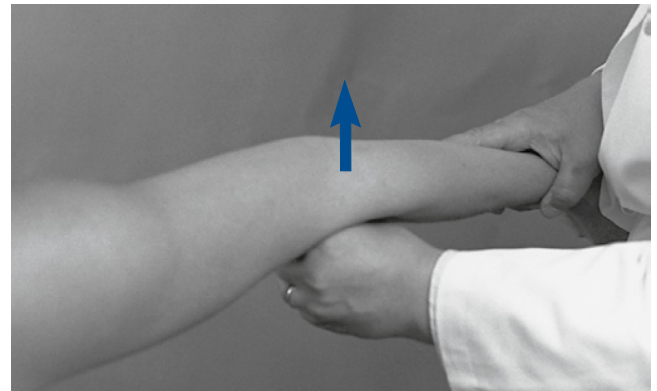


Figure 6-88 Assessment of posterior-to-anterior glide of the right humeroulnar joint.

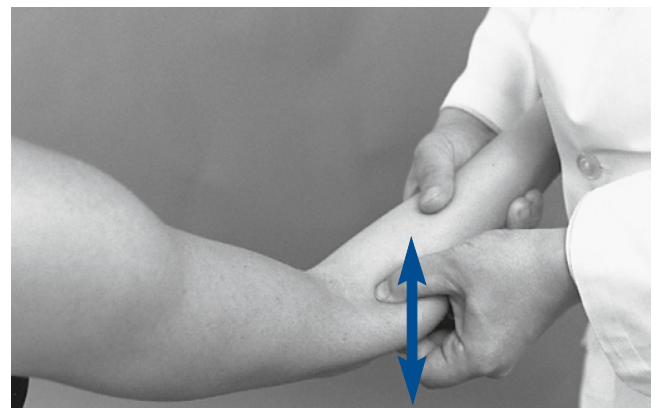


Figure 6-89 Assessment of anterior-to-posterior and posterior-to-anterior glide of the right radioulnar joint.

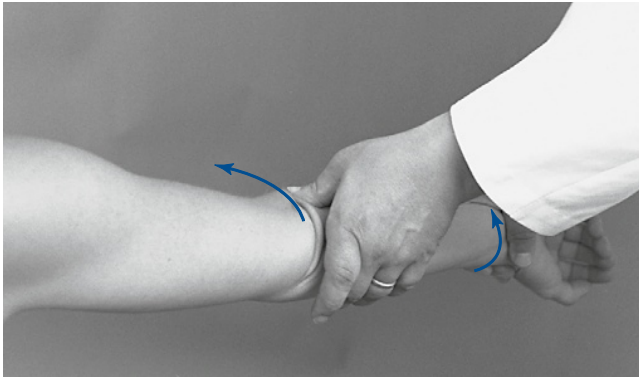


Figure 6-90 Assessment for posterior-to-anterior glide in pronation of the right radial head.

radial head, you should first perceive a rotational movement of the radial head, and at the end point of movement, apply a P-A stress to the radial head to determine the presence of a springing end-feel movement (Figure 6-90).

ADJUSTIVE PROCEDURES

The manipulative techniques used to treat elbow disorders aim to restore normal joint mechanics, which will then ideally allow full pain-free functioning of the elbow joints. Box 6-7 identifies the adjustive procedures for the joints of the elbow.

BOX 6-7 Elbow Adjustive Techniques

ELBOW SITTING:

Web/distal humerus, forearm grasp pull; long-axis distraction (Figure 6-91)

Calcaneal/proximal ulna, forearm stabilization; medial-to-lateral glide (Figure 6-92)

Calcaneal/proximal radius, forearm stabilization; lateral-to-medial glide (Figure 6-93)

Reinforced hypothenar/proximal radius pull; anterior-to-posterior glide (Figure 6-94)

Thumb/radius push, distal forearm grasp; posterior-to-anterior glide in pronation (Figure 6-95)

Mid-hypothenar (knife-edge)/proximal ulna, elbow flexion; anterior-to-posterior glide (Figure 6-96)

Hypothenar/radius push, ulnar stabilization; posterior-to-anterior glide in supination (Figure 6-97)

Thumb index/olecranon; posterior-to-anterior glide in full extension (Figure 6-98)

ELBOW SUPINE:

Bimanual grasp/distal humerus with knee extension; long-axis distraction (Figure 6-99)

Web/proximal ulna push with knee extension; medial-to-lateral glide (Figure 6-100)

Web/proximal radius push with knee extension; lateral-to-medial glide (Figure 6-101)

Elbow

Sitting:

Web/Distal Humerus, Forearm Grasp Pull; Long-Axis Distraction (Figure 6-91)

IND: Loss of long-axis distraction of the elbow (radiohumeral and ulnohumeral joints).

PP: The patient sits, with the affected arm in slight elbow flexion.

DP: Stand and face the patient.

SCP: Proximal forearm.

CP: Grasp the patient's distal forearm with your inside hand.

IH: Apply a web contact over the distal humerus with your outside hand.

VEC: Long axis of the forearm.

P: With your outside hand, stabilize the humerus while your contact hand delivers an impulse-type thrust in the long axis of the forearm. This procedure can also be used to mobilize the elbow by applying sustained traction followed by pronation and supination movements.

Calcaneal/Proximal Ulna Forearm Stabilization; Medial-to-Lateral Glide (Figure 6-92)

IND: Loss of M-L accessory joint movement, olecranon process displaced toward the medial epicondyle.

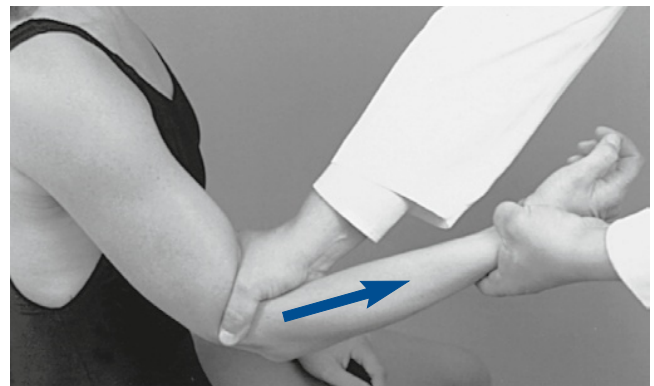


Figure 6-91 Adjustment for long-axis distraction of the right humeroradial joint in the seated position.



Figure 6-92 Adjustment for medial-to-lateral glide of the right elbow in the seated position.

PP: The patient sits, with the affected arm flexed at the shoulder.
DP: Stand and face the patient at the medial aspect of the patient's affected arm.

SCP: Medial aspect of the proximal ulna.

CP: With your inside hand, establish a calcaneal contact over the medial aspect of the proximal ulna, just distal to the medial aspect of the elbow joint space. The fingers will rest in the ante-cubital fossa and over the proximal anterior forearm.

IH: With your outside hand, contact the lateral aspect of the forearm so that your arm can stabilize the patient's forearm against your body.

VEC: M-L.

P: As your IH stabilizes the forearm, drawing it into slight distraction and elbow extension, induce an M-L impulse thrust with your contact hand.

Calcaneal/Proximal Radius Forearm Stabilization; Lateral-to-Medial Glide (Figure 6-93)

IND: Loss of L-M accessory joint motion, olecranon process displaced toward the lateral epicondyle.

PP: The patient sits, with the affected arm flexed at the shoulder.

DP: Stand and face the patient on the lateral side of the patient's affected arm.

SCP: Lateral aspect of the elbow joint and proximal radius.

CP: With your outer hand, establish a calcaneal contact over the radial head and lateral aspect of the elbow joint.

IH: With your inner hand, grasp the patient's proximal forearm so that the patient's forearm is held against your body.

VEC: L-M.

P: With your IH and body, stabilize the forearm and apply a slight distractive force while delivering an impulse thrust in an L-M direction with your contact hand.

Reinforced Hypothenar/Proximal Radius Pull; Anterior-to-Posterior Glide (Figure 6-94)

IND: Decreased A-P glide of the radial head, anterior misalignment of radius.

PP: The patient sits on a chair or on the edge of an adjusting table, sitting on the palmar aspect of the hand on the affected side.

DP: Either straddle the table, facing the patient on the affected side, or squat next to the patient, who is seated in a chair.

SCP: Anterior aspect of the radial head.



Figure 6-94 Adjustment for anterior-to-posterior glide of the right radial head.

CP: Using your forward hand, establish a pisiform-hypothenar contact on the anterior aspect of the radial head.

IH: Reinforce your contact hand with your posterior hand.

VEC: A-P.

P: The patient's body weight on the patient's hand stabilizes the upper extremity while the doctor delivers an A-P impulse thrust with both hands to the radial head. The patient can be asked to hold the end of the table for torso stabilization as needed.

Thumb/Radius Push, Distal Forearm Grasp; Posterior-to-Anterior Glide in Pronation (Figure 6-95)

IND: Loss of P-A accessory joint movement of the radial head in pronation, posterior misalignment of the radius.

PP: The patient sits, with the affected arm flexed at the elbow and pronated.

DP: Stand on the affected side of the patient.

SCP: Posterior aspect of the radial head.

CP: Use your proximal hand to apply a thumb contact on the posterior aspect of the radial head, with your fingers lying across the posterior aspect of the elbow.

IH: Grasp the patient's distal forearm, with a digital contact of all fingers on the posterior aspect of the radius.

VEC: P-A.

P: Use your IH to move the elbow from a flexed and supinated position to an extended and pronated position. Repeat this procedure several times so that the patient relaxes the elbow. When the patient's elbow is fully extended and fully pronated,



Figure 6-93 Adjustment for lateral-to-medial glide of the right elbow in the seated position.

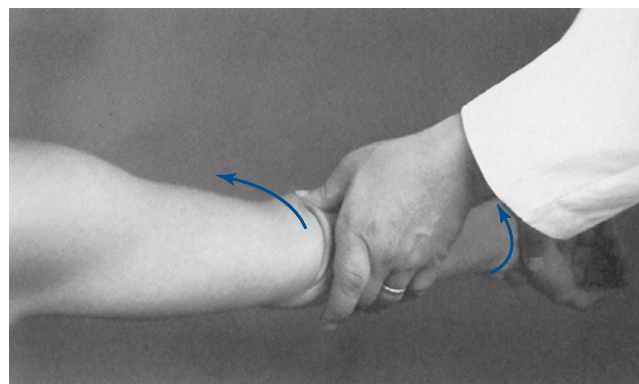


Figure 6-95 Adjustment for posterior-to-anterior glide in pronation of the right radial head.

deliver a very shallow but quick impulse thrust in a P-A direction against the radial head with your contact hand.

Mid-Hypothenar (Knife-Edge)/Proximal Ulna Elbow Flexion; Anterior-to-Posterior Glide (Figure 6-96)

IND: Decreased A-P movement of the humeroulnar joint, anterior misalignment of the radius.

PP: The patient sits, with the affected arm flexed at the shoulder and at the elbow.

DP: Stand and face the patient.

SCP: Proximal ulna.

CP: With your inside hand, establish a mid-hypothenar (knife-edge) contact over the proximal ulna in the antecubital fossa.

IH: Grasp the proximal forearm with your outer hand.

VEC: A-P.

P: Using your IH, flex the patient's elbow over your contact hand until articular slack is removed. Accomplish the thrust with your IH, moving the forearm toward the patient's shoulder while your contact hand delivers an A-P thrust over the proximal ulna.

Hypothenar/Radius Push Ulnar Stabilization; Posterior-to-Anterior Glide in Supination (Figure 6-97)

IND: Loss of P-A accessory joint movement of the radial head, posterior misalignment of the radius.

PP: The patient sits on a chair or on the edge of an adjusting table, sitting on the palmar aspect of the hand on the affected side.

DP: Either straddle the table, facing the patient on the affected side, or squat next to the patient, who is seated on a chair.

SCP: Posterior aspect of the radial head.

CP: With your forward hand, establish a pisiform-hypothenar contact on the posterior aspect of the radial head.

IH: Grasp the proximal ulna with your posterior hand.

VEC: P-A.

P: The patient's body weight on the patient's hand stabilizes the upper extremity. While your IH stabilizes the ulna, deliver a P-A impulse thrust to the radial head with the contact hand.



Figure 6-96 Adjustment for anterior-to-posterior glide of the right ulna.



Figure 6-97 Adjustment for posterior-to-anterior glide of the right radial head.

Thumb Index/Olecranon Push; Posterior-to-Anterior Glide in Full Extension (Figure 6-98)

IND: Loss of P-A glide of the humeroulnar joint.

PP: The patient sits, with the affected arm extended at the elbow and flexed at the shoulder.

DP: Stand and face the patient on the lateral side of the affected arm.

SCP: The olecranon process.

CP: Form a ring with the thumb and index finger of your outside hand and place it over the posterior aspect of the olecranon process.

IH: Rest your other hand on the anterior aspect of the distal forearm.

VEC: P-A.

P: With very little downward pressure on the distal forearm, apply a gentle P-A stress to the olecranon process, finishing with a very shallow impulse thrust.

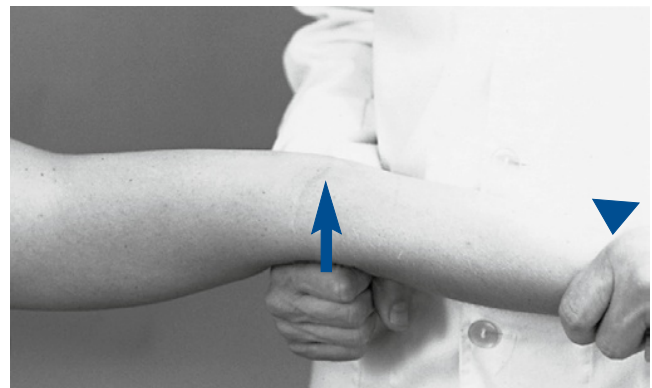


Figure 6-98 Adjustment for posterior-to-anterior glide of the right ulna in extension.

Supine:**Bimanual Grasp/Distal Humerus with Knee Extension; Long-Axis Distraction** (Figure 6-99)

IND: Loss of joint separation in the long axis.

PP: The patient is supine, with the affected arm slightly abducted.

DP: Stand on the affected side, facing cephalad, and straddle the patient's forearm so that your knees can grasp the patient's distal forearm.

SCP: Distal humerus.

CP: Use both hands to grasp the patient's distal humerus.

VEC: Long axis of the forearm.

P: Use both hands to stabilize the humerus, and then straighten both knees to create long-axis distraction at the elbow.

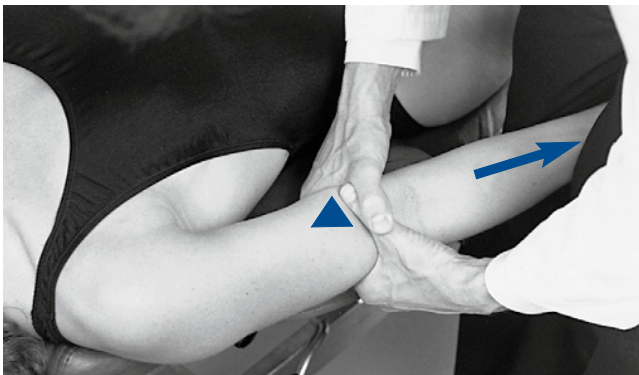
Web/Proximal Ulna Push with Knee Extension; Medial-to-Lateral Glide (Figure 6-100)

Figure 6-99 Adjustment for long-axis distraction of the right humeroradial joint in the supine position.



Figure 6-100 Adjustment for medial-to-lateral glide of the right elbow in the supine position.

IND: Loss of M-L accessory joint movement, olecranon process displaced toward the medial epicondyle.

PP: The patient is supine, with the affected arm abducted slightly.

DP: Stand at the side of the table facing cephalad and straddling the patient's affected arm so that your knees can grasp the patient's distal forearm.

SCP: Lateral aspect of the proximal ulna.

CP: Use your inside hand to apply a web contact over the lateral aspect of the proximal ulna, just distal to the medial elbow joint space. Wrap your fingers around the posterior aspect of the elbow with the thumb in the antecubital fossa.

IH: With your outside hand, grasp the lateral aspect of the forearm, distal to the other contact.

VEC: M-L.

P: Straighten your knees to create distraction at the elbow joint, simultaneously applying an M-L thrust with your contact hand as you supply an opposite vector with your IH.

Web/Proximal Radius Push with Knee Extension; Lateral-to-Medial Glide (Figure 6-101)

IND: Loss of L-M accessory joint motion or olecranon process displaced toward the lateral epicondyle.

PP: The patient is supine, with the affected arm abducted slightly at the shoulder.

DP: Stand and face cephalad, straddling the patient's affected arm so that your knees can squeeze the distal forearm.

SCP: Lateral aspect of the elbow joint and proximal radius.

CP: Using your outside hand, apply a web contact over the lateral aspect of the proximal radius, just distal to the lateral elbow joint space. Wrap your fingers around the posterior aspect of the elbow with the thumb in the antecubital fossa.

IH: With your inside hand, grasp the medial aspect of the forearm, distal to the other contact.

VEC: L-M.

P: Straighten your knees to create distraction at the elbow joint, simultaneously applying an L-M thrust with your contact hand as you supply an opposite vector with your IH.

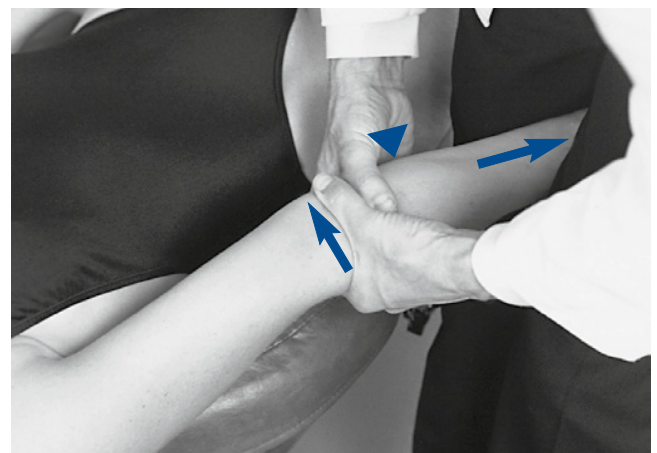


Figure 6-101 Adjustment for lateral-to-medial glide of the right elbow in the supine position.

WRIST AND HAND

An intricate interaction of numerous structures in the wrist and hand is necessary to produce the remarkable dexterity and precision that characterizes this joint complex.¹⁵ The entire upper limb is apparently subservient to the hand in its use as a tactile organ, a means of expression, and a weapon. The study of the hand is inseparable from that of the wrist and the forearm, which function as a single physiologic unit, with the wrist being the key joint.³ The wrist serves as a flexible spacer between the forearm and hand and is critical in setting the length-tension relationship for the extrinsic finger muscles. By far the most important musculoskeletal function of the hand is its ability to grasp objects. However the hand is also an important sensory organ (kinesthetic) and helps to express emotion through gestures, touch, and art. Because of its biomechanical complexity, the function of the hand involves a disproportionately large region of the cortex of the brain. Therefore, dysfunction of the hand can create equally disproportionate disabilities. The hand is the main manipulative organ of the body, performing many different types of functions, and it should not be overlooked in the evaluation for dysfunction.

FUNCTIONAL ANATOMY

Osseous Structures

Interestingly, although the ulna plays a highly significant role in the function of the elbow, it is secondary in the wrist, whereas the radius, having a secondary role in the elbow, has a dominant part in the wrist. The radius flares to become much larger at the distal end, terminating with a lateral extension, called the *radial styloid process*. The distal end of the ulna also ends in a styloid process, but it is much smaller in comparison with the radial styloid. The distal aspects of the radius and ulna form an articulation with the proximal row of carpal bones directly in the radius and indirectly via an intracapsular disc in the ulna. The eight carpal bones that make up the wrist are arranged in two rows that greatly enhance the hand's mobility. The proximal row consists of the scaphoid, lunate, triquetrum, and pisiform (in order from medial to lateral). The pisiform overlies the triquetrum, which forms an articulation with the proximal ulna via the interarticular disc. The scaphoid and lunate articulate directly with the radius. The distal row of carpals consists of the trapezium, trapezoid, capitate, and hamate (in order from lateral to medial). The proximal and distal rows of carpal bones collectively form an intercarpal joint, although some movement also occurs between the individual carpal bones (Figure 6-102).

The base of each of the five metacarpals articulates with the distal row of carpals. Five proximal phalanges articulate with each of the metacarpals, followed by a middle and distal phalanx for each of the fingers and a distal phalanx for the thumb.

Ligamentous Structures

The numerous ligaments of the wrist, many of which are unnamed, are not all separate entities. They form a crisscross pattern of

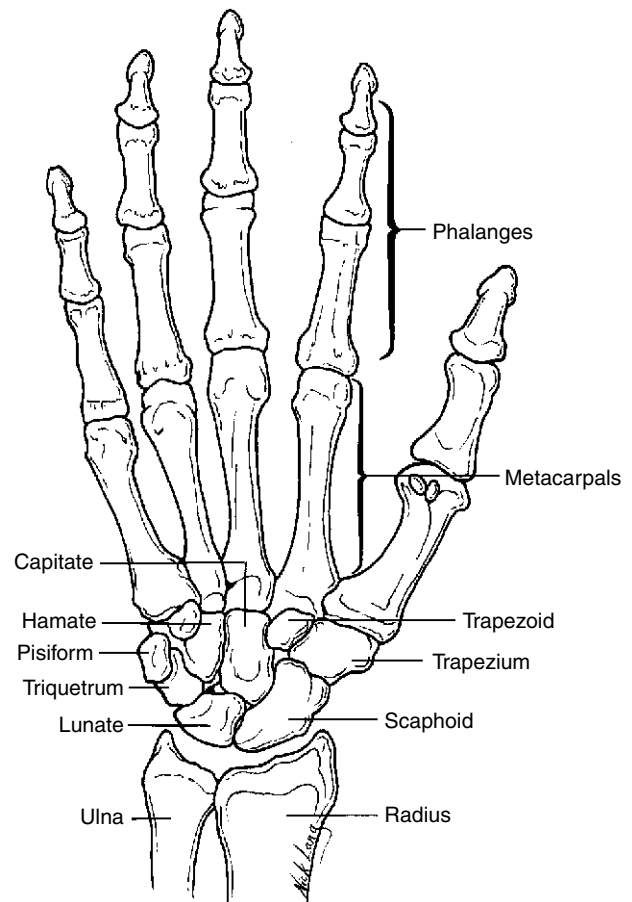


Figure 6-102 Palmar view of the osseous structures of the right wrist and hand.

connections between the radius and ulna to the carpals, between the carpals, from the carpals to the metacarpals, and between the metacarpals (Figure 6-103). The volar radiocarpal and radioulnar carpal ligaments strengthen the joint capsule and wrist anteriorly, while the dorsal radiocarpal ligament provides support posteriorly (Figure 6-104). Radial collateral and ulnar collateral ligaments stabilize the wrist laterally and medially, respectively. Collateral ligaments also support the MP and the interphalangeal joints (Figure 6-105).

Musculature

Extrinsic and intrinsic muscles function for the wrist and hand (Table 6-10). The wrist flexors and extensors are located in the forearm, attached to the epicondyles of the humerus. As the muscles head distally, their tendons are enclosed in sheaths that offer a smooth environment for sliding. Intrinsic muscles include the interosseous and lumbricales muscles, as well as those responsible for the movements of the thumb and little finger. Six passageways transport the extensor tendons through fibro-osseous tunnels. Fibrous bands running from the retinaculum to the carpal bones form the tunnels (Figure 6-106). The flexor retinaculum spans the scaphoid, trapezium, hamate, and pisiform. It forms a tunnel out of the carpal arch to allow passage of the median nerve and the flexor tendons (Figure 6-107).

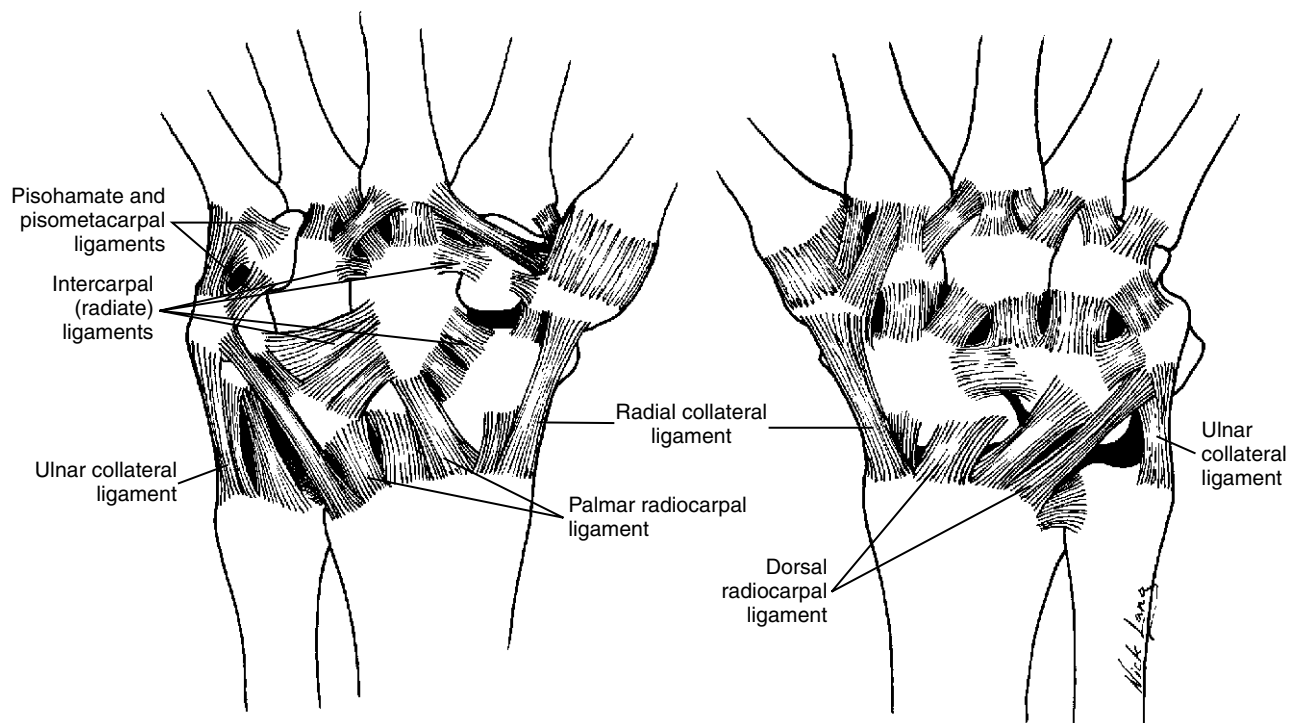


Figure 6-103 Ligaments of the wrist. **A**, Palmar view. **B**, Dorsal view. (Modified from Hertling D, Kessler RM: *Management of common musculoskeletal disorders: Physical therapy principles and methods*, ed 2, Philadelphia, 1990, JB Lippincott.)

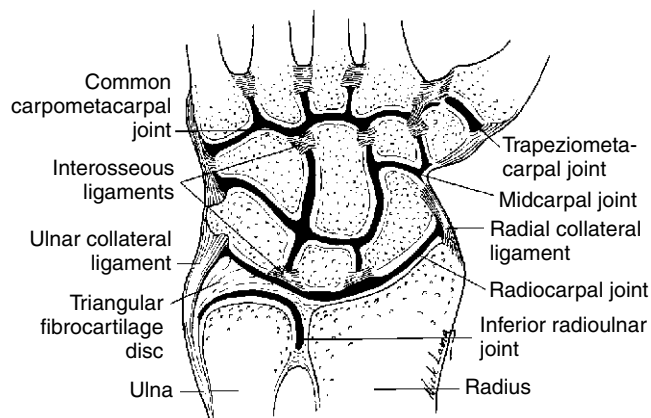


Figure 6-104 Coronal section through right wrist, showing intercarpal joints and ligaments.

BIOMECHANICS

The complex movements of the wrist are accomplished by the distal radioulnar joint, the radiocarpal joint, and the midcarpal joint. The radiocarpal and midcarpal joints produce the motion at the wrist joint. Wrist flexion and extension, as well as radial and ulnar deviation, are thought to occur around an axis of movement that passes through the capitate (Figure 6-108). However, the multiplicity of wrist articulations and the complexity of joint motion make it difficult to calculate the precise instantaneous axis of motion.¹² The close-packed position for the wrist is full extension (Table 6-11). The wrist can undergo approximately 160 degrees of flexion and extension, with extension being slightly greater. Radial and ulnar deviation are possible to 60 degrees, and ulnar deviation is almost twice as great

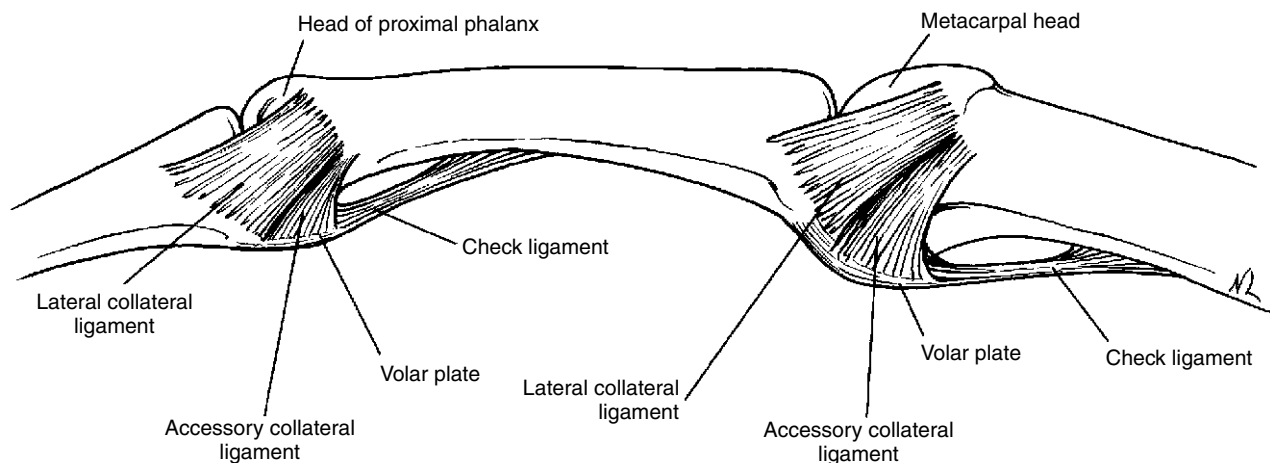
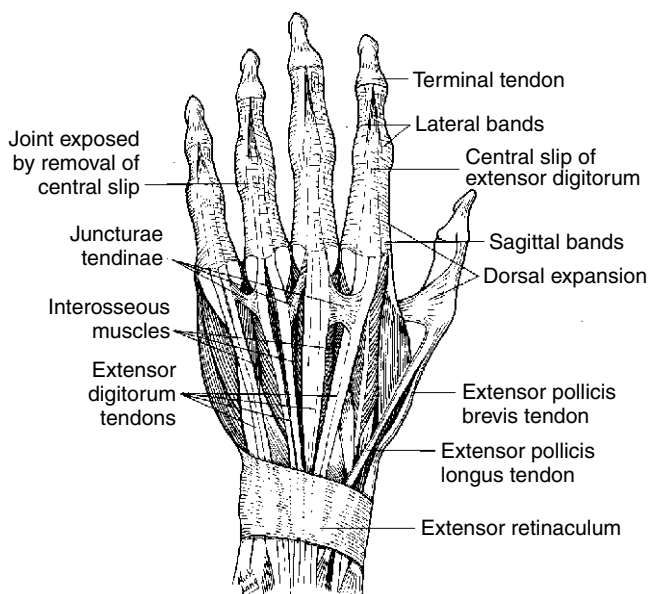


Figure 6-105 Lateral view of the ligaments of the finger.

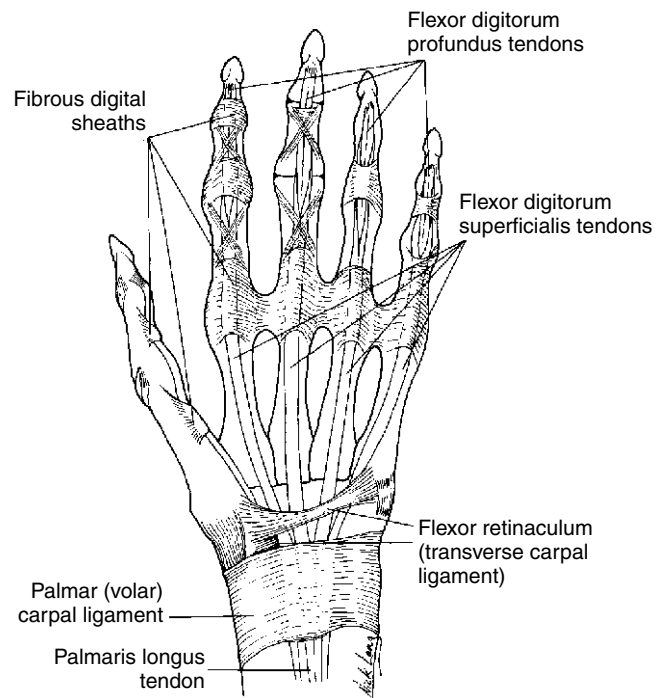
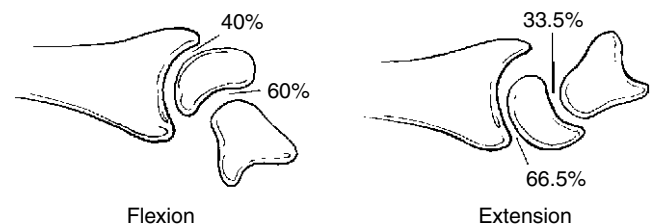
TABLE 6-10 Actions of the Muscles of the Wrist and Hand

Actions	Muscles
Wrist flexion	Flexor carpi radialis, abductor pollicis longus, palmaris longus, flexor pollicis longus, flexor carpi ulnaris, and flexor digitorum superficialis and profundus
Wrist extension	Extensor carpi radialis, extensor digitorum, extensor carpi ulnaris, and extensor pollicis longus
Wrist adduction (ulnar deviation)	Extensor carpi ulnaris and flexor carpi ulnaris
Wrist abduction (radial deviation)	Extensor carpi radialis, abductor pollicis longus, and extensor pollicis longus and brevis
Finger flexion	Flexor digitorum superficialis and profundus
Finger extension	Extensor digitorum, extensor digiti minimi, and extensor indicis
Finger abduction	Interosseous muscles

**Figure 6-106** Dorsal view of the left hand, showing the location of extensor tendons and dorsal interosseous muscles.

as radial deviation (Figure 6-109). Radial deviation is limited by contact of the scaphoid against the radial styloid process. ROMs for the wrist and hand are listed in Table 6-12.

With dorsiflexion of the wrist, a supinatory rotation of the carpal bones also occurs, which is mostly a result of the scaphoid

**Figure 6-107** Palmar view of the left hand, showing flexor tendons.**Figure 6-108** Most wrist flexion occurs at the intercarpal joint, and most wrist extension occurs in the radiocarpal joint.

moving more with respect to the radius while the lunate and triquetrum relate to the ulna. Furthermore, when moving from flexion to dorsiflexion, the distal row of carpals becomes close-packed with respect to the scaphoid first. This results in the scaphoid moving with the distal row into dorsiflexion, necessitating movement between the scaphoid and lunate as full dorsiflexion is approached.

TABLE 6-11 Close-Packed and Loose-Packed (Rest) Positions for the Wrist and Hand Joints

	Closed-Packed Position	Loose-Packed Position
Wrist	Full dorsiflexion	Palmar flexion with slight ulnar deviation
Hand	Full extension	Flexion with slight ulnar deviation

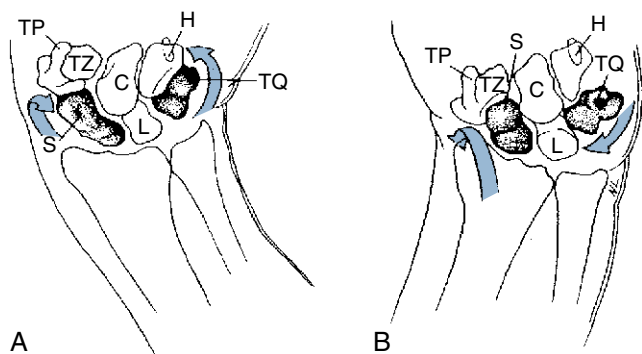


Figure 6-109 Dorsal view of the right wrist. **A**, With ulnar deviation, some extension of proximal carpals occurs. **B**, With radial deviation, some flexion of the proximal carpal occurs. **C**, Capitate; **H**, hamate; **L**, lunate; **S**, scaphoid; **TP**, trapezium; **TQ**, triquetrum; **TZ**, trapezoid.

TABLE 6-12 Arthrokinematic and Osteokinematic Movements of the Wrist and Hand Joints

Osteokinematic Movements	Degrees	Arthrokinematic Movements
Wrist flexion	80	Roll and glide
Wrist extension	70	Roll and glide
Ulnar deviation	30	Roll and glide
Radial deviation	20	Roll and glide
MCP flexion	90	Roll and glide
MCP extension	30–45	Roll and glide
PIP flexion	100	Roll and glide
PIP extension	0	Roll and glide
DIP flexion	90	Roll and glide
DIP extension	10	Roll and glide
Finger abduction	20	Roll and glide

DIP, Distal interphalangeal joint; *MCP*, metacarpophalangeal joint; *PIP*, proximal interphalangeal joint.

With extension of the wrist, the proximal row of carpals rolls and glides anteriorly with respect to the radius and ulna, and the distal row of carpals moves similarly with respect to the proximal row of carpals. The converse is true of wrist flexion; the proximal row moves posteriorly relative to the radius and ulna as the proximal row moves posteriorly relative to the proximal row of carpals.

Radial and ulnar deviation involve rotary movements between the proximal row of carpals and the radius, as well as between the proximal row and distal row of carpals. Moreover, during radial deviation, the proximal row combines pronation, flexion, and ulnar glide movements with respect to the radius as the distal row combines supination, extension, and ulnar glide movements with respect to the proximal row. Ulnar deviation has the opposite movements.^{3,16}

The hand must be able to change its shape to grasp objects. Three physiologic functional arches running in different directions provide the means for the wrist and hand to conform to a position for grasping (Figure 6-110). Transversely, an arch through the carpal region corresponds to the concavity of the wrist, and

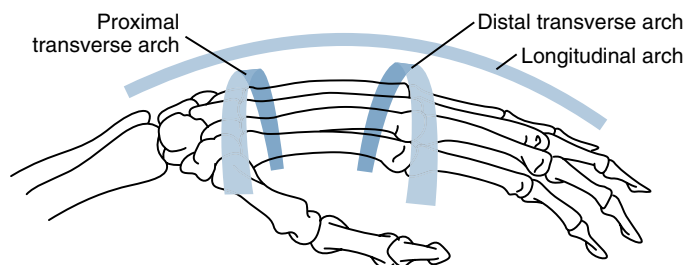


Figure 6-110 The three physiologic arches of the wrist and hand. (Modified from Nordin M, Frankel VH: *Basic biomechanics of the musculoskeletal system*, ed 2, Philadelphia, 1989, Lea & Febiger.)

distally, the metacarpal arch is formed by the metacarpal heads. Longitudinal arches are formed along each finger by the corresponding metacarpal bone and phalanges. Obliquely, arches are formed by the thumb during opposition with the other fingers. These arches allow coordinating synergistic digital flexion and opposition of the thumb and little finger.

The wrist provides a stable base for the hand, and its position controls the length of the extrinsic muscles to the digits. The muscles stabilize the wrist, as well as provide for the fine movements of the hand, to place it in its functioning position. The positioning of the wrist has a significant influence on the strength of the fingers. For most effective action of the extrinsic muscles of the fingers, the wrist usually must move in a direction opposite the movement of the fingers.

The naturally assumed position of the hand to grasp an object, or the position from which optimal function is most likely to occur, is termed the *functional position* (Figure 6-111). The functional position occurs when the wrist is extended 20 degrees and the ulna is deviated 10 degrees, the fingers are flexed at all of their joints, and the thumb is in a midrange position, with the MP joint moderately flexed and the interphalangeal joints slightly flexed. Prehensile functions of the hand are unique and fundamental characteristics (Figure 6-112).

EVALUATION

The wrist and hand are prone to injuries from trauma, commonly a fall on the outstretched hand. The radial side of the wrist and hand tends to take the majority of the force from such a trauma. Displacement, instability, and rotary subluxation of the scaphoid often occur, creating pain on dorsiflexion of the wrist and limited ROM. Palpable tenderness will be present over the joint space between the scaphoid and

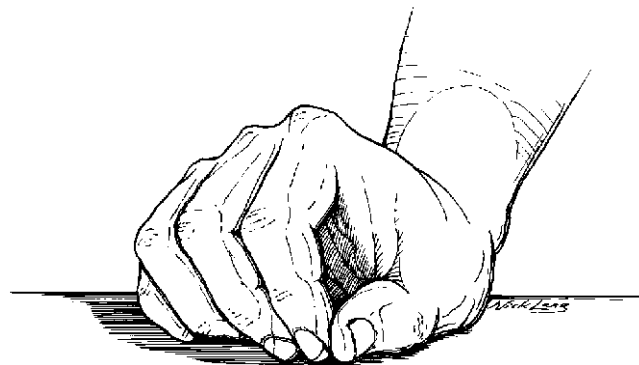


Figure 6-111 The functional position of the hand.

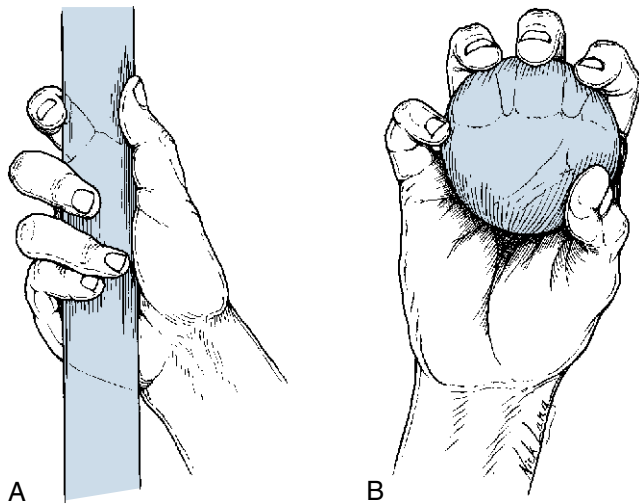


Figure 6-112 The fundamental patterns of prehensile hand function. **A**, Power grip. **B**, Precision maneuver.

the lunate. Occasionally, the scaphoid can be felt slipping as the wrist is moved, or a painful click may be perceived. A space of more than 3 mm between the scaphoid and lunate may be seen on a closed-fist supinated radiographic view of the wrist.

Trauma to an outstretched hand that is forcefully flexed or extended may cause a fracture to the radius. A Colles fracture occurs when the wrist is in dorsiflexion and the forearm is in pronation. Local pain and tenderness to palpation and vibration are important physical findings; however, the radiograph is the most important tool for determining the presence of a fracture. Manipulative therapy to this area is then contraindicated.

Singular trauma, such as a fall, or repetitive activities may lead to a sprain of the ligaments of the wrist. Moreover, when the wrist is subjected to sudden increases in workload, such as in gripping or lifting, or racquet games that require flexion and extension of the wrist, the tendons crossing the wrist can become inflamed, resulting in tendinitis. In addition, a possible response to repeated twisting and straining is a localized nodular swelling, called a *ganglion*. Likely a defense mechanism, the ganglion is characterized by a fibrous outer coat that covers a thick gelatinous fluid derived from the synovium lining the tendon sheaths.

Undoubtedly, the most noted condition affecting the wrist and hand is carpal tunnel syndrome, a peripheral entrapment neuropathy involving the median nerve. The median nerve lies superficial to the flexor tendons beneath the tense transverse carpal ligament (flexor retinaculum), making the carpal tunnel just barely adequate to accommodate these structures. In the act of grasping an object, particularly with the wrist in flexion, the flexor tendons are displaced forward and can compress the nerve against the unyielding ligament. Narrowing of the carpal tunnel can occur through bony deformity after fracture, degenerative joint disease, synovial swelling of a tendon or of the wrist joint ligaments, and thickening of the transverse carpal ligament (Figure 6-113). Most often, however, no definitive local cause for nerve compression can be detected. Moreover, Upton and McComas²⁰ has identified the possibility that peripheral nerves can be compressed at more than one spot along their course, creating “double-,” “triple-,” and “quadruple-crush” syndromes. Therefore, although the patient’s

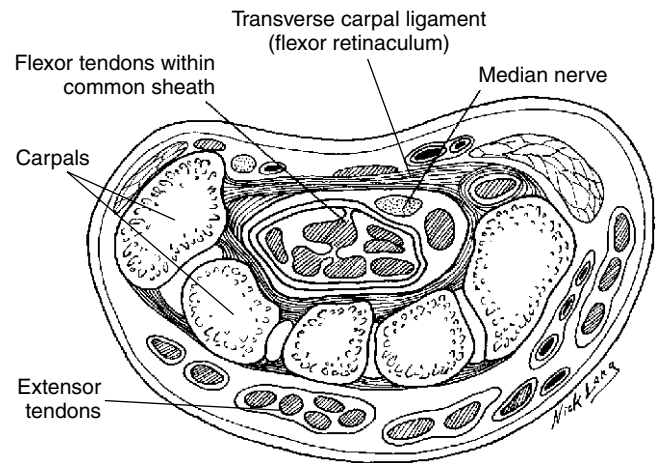


Figure 6-113 Cross-section of the wrist, showing the relationship between the carpal bones, tendons, flexor retinaculum, and median nerve.

clinical picture may be defined as *carpal tunnel syndrome*, the nerve compression may not necessarily be at the wrist but may be at the elbow, shoulder, or neck. This syndrome occurs more often in women, with onset commonly between 40 and 50 years of age. Slight paresthesias may precede the onset of the acute symptoms for several months. Then, paroxysms of pain, paresthesia, and numbness occur in the area of the median nerve distribution. The patient is often awakened at night by numbness or pain that can be described as burning, aching, prickly, or pins-and-needles. Motor weakness of the thumb adductor or opposer may be found. The patient may describe relief from dangling the hand over the side of the bed, shaking the hand vigorously, or rubbing it.

Because the wrist structures are innervated primarily from segments C6 through C8, lesions affecting structures of similar derivation may refer pain to the wrist and vice versa. Symptoms experienced at the wrist and hand must always be suspected as possibly having a more proximal origin (Figure 6-114).

Observe the wrist and hand for general posture and attitude. In the resting attitude of the hand, the MP and interphalangeal joints are held in a position of slight flexion. Observe the arm and hand for natural swing when the patient walks. Also, note functional activities of the hand and wrist, including the firmness of the person’s handshake, as well as temperature and moisture of the hand. The dominant hand should be determined. Sometimes this can be done by noting the hand with more developed musculature, but is most easily done simply by asking the patient.

To begin the evaluation of the wrist and hand, osseous symmetry, bony relationships, and pain production are identified through static palpation of the wrist and hand (Figure 6-115). Palpate the radius and ulna distally, identifying each of their styloid processes. Just distal to the radial styloid and in the anatomic snuffbox, the scaphoid can be palpated. Wrist flexion will facilitate the palpation of the lunate, which lies next to the scaphoid. The triquetrum and pisiform overlie one another and are just distal to the ulnar styloid. The trapezium can be identified at the base of the first metacarpal. The trapezoid lies at the base of the second metacarpal. The capitate is found between the base of the third metacarpal and the lunate. The hook of the hamate, and hence the hamate, can be

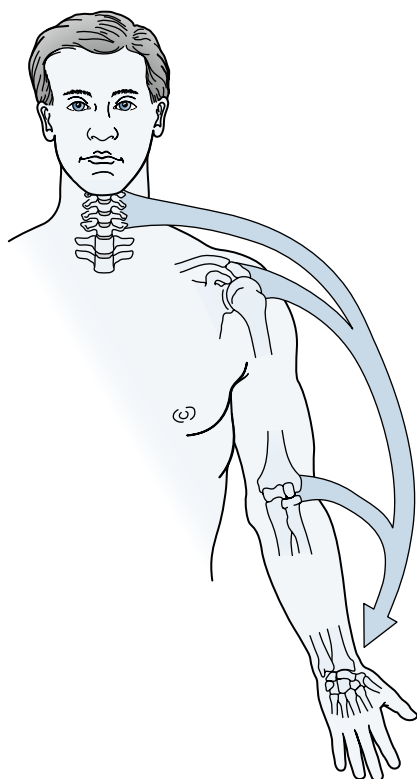


Figure 6-114 Symptoms in the hand and wrist must be suspected of having a more proximal origin. (From Magee DJ: *Orthopedic Physical Assessment*, ed 5, St Louis, 2008, Saunders.)

found on the palmar surface, just distal and to the thumb side of the pisiform. Then palpate the metacarpals through the palm of the hand, with the fingers along the shaft of the metacarpal on the palmar surface while the thumb is over the dorsal surface. Finally, palpate the 14 phalanges (2 for the thumb and 3 for each finger).

Identify tone, texture, and tenderness changes through soft tissue palpation of the flexor and extensor tendons, the thenar eminence, and the hypothenar eminence. Determine patency of the radial and ulnar arteries (Allen test) and take a radial pulse.

Evaluate accessory joint motions for the wrist and hand articulations to determine the presence of joint dysfunction (Table 6-13). Assess A-P and P-A glide of the distal radioulnar joint with the patient supine or sitting. Grasp the distal radius with one hand and the distal ulna with the other. Apply an opposing A-P and P-A shearing stress between the radius and ulna (Figure 6-116).

Conduct M-L compression of the distal radioulnar joint with the patient supine or sitting, and use both hands to encircle the distal radius and ulna. Use both hands to apply an M-L compression stress to the distal radius and ulna (Figure 6-117).

In evaluating long-axis distraction of the intercarpal joint, the patient can be seated or supine. Grasp the distal forearm with one hand and the distal wrist with the other. While stabilizing the forearm, distract the wrist in the long axis (Figure 6-118).

Perform M-L tilt and glide of the intercarpal joints with the patient seated and the affected arm raised in forward flexion. Stand on the affected side, facing the lateral aspect of the arm. Grasp the distal radius and ulna with your proximal hand while grasping the patient's distal wrist with your distal hand. Use

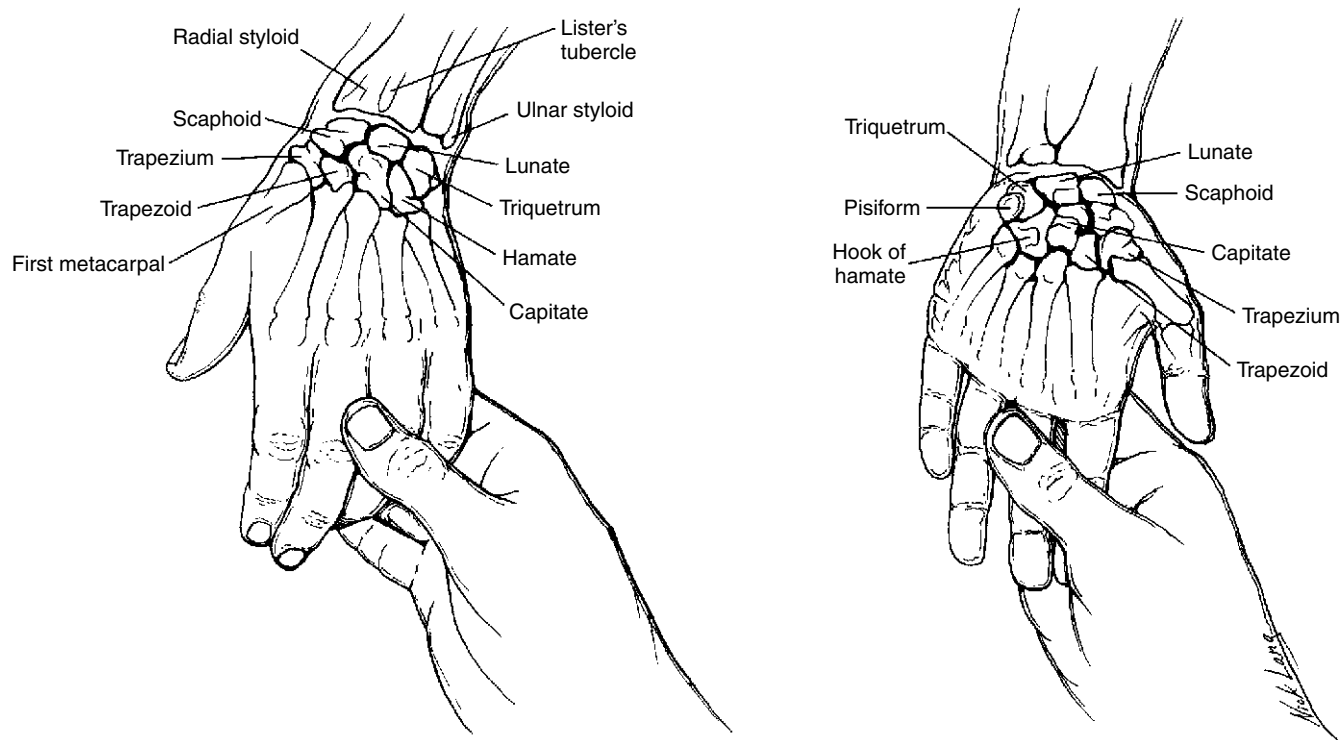


Figure 6-115 Localization of osseous structures of the left wrist.

TABLE 6-13 Accessory Joint Movements of the Wrist and Hand Joints

Joint	Movement
Distal radioulnar	A-P glide P-A glide
Intercarpal	M-L compression Long-axis distraction M-L tilt M-L glide A-P glide P-A glide
Individual carpals	A-P glide P-A glide
Intermetacarpal	A-P glide P-A glide
Metacarpophalangeal and interphalangeal	Long-axis distraction M-L glide L-M glide A-P glide P-A glide Internal rotation External rotation

A-P, Anterior-to-posterior; *L-M*, lateral-to-medial; *M-L*, medial-to-lateral; *P-A*, posterior-to-anterior.

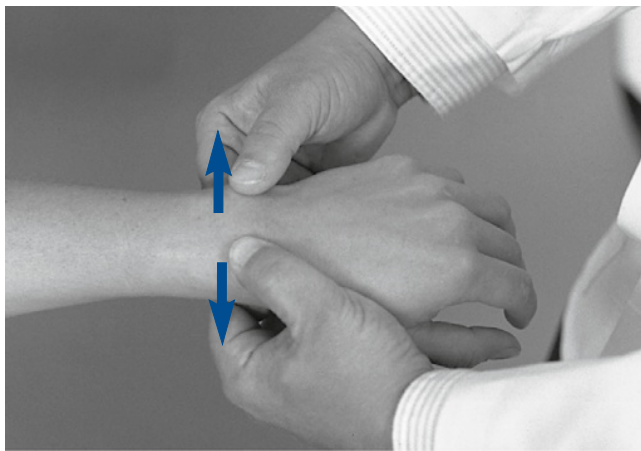


Figure 6-116 Assessment of anterior-to-posterior and posterior-to-anterior glide of the distal left radioulnar joint.

both hands to create opposing forces, creating a shearing stress (M-L glide) (Figure 6-119) and radial and ulnar deviation stress (M-L tilt) (Figure 6-120).

Assess A-P and P-A glide of the intercarpal joint, with the patient seated and arm raised in forward flexion. Stand on the affected side. Grasp the distal radius and ulna with your proximal hand while grasping the patient's distal wrist with your distal hand. Using both hands to create opposing forces, stress the intercarpal joints in an A-P and P-A glide, looking for a springing joint play movement (Figure 6-121).



Figure 6-117 Assessment of medial-to-lateral compression of the distal left radioulnar joint.

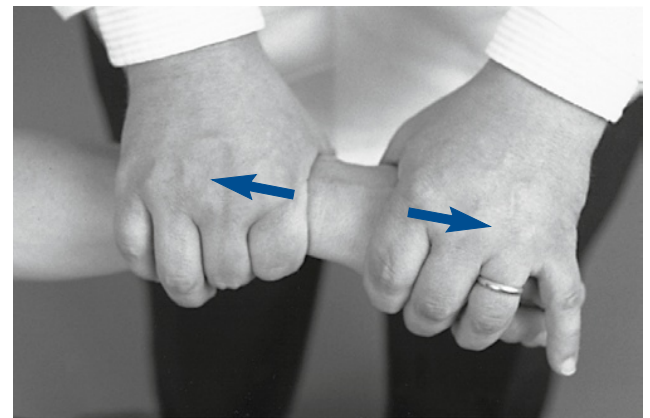


Figure 6-118 Assessment of long-axis distraction of the left intercarpal joint.

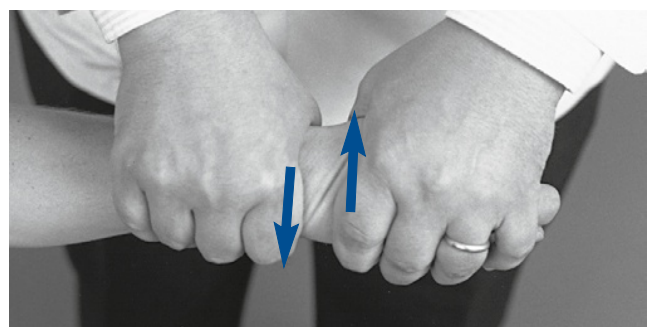


Figure 6-119 Assessment of medial-to-lateral and lateral-to-medial glide of the left intercarpal joint.

Assess A-P and P-A glide of the individual carpal bones with the patient seated and the affected arm raised in forward flexion. Stand and face the patient. Use your thumb and index or middle fingers to contact the anterior and posterior surfaces of the carpal bone to be evaluated while using your other hand to stabilize the rest of the wrist. Apply an A-P and P-A stress to each individual carpal bone, looking for a springing joint play movement (Figure 6-122, *A* and *B*).

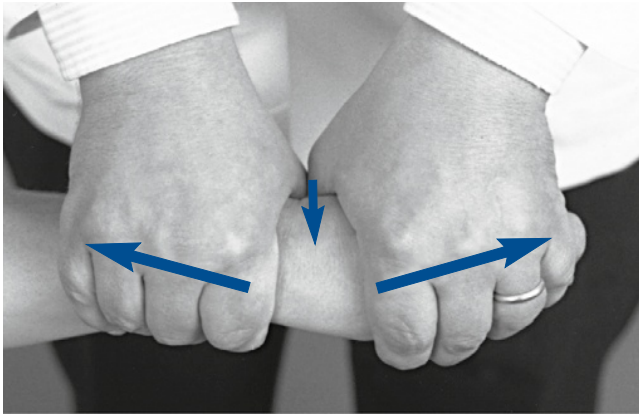


Figure 6-120 Assessment of medial-to-lateral and lateral-to-medial tilt of the left intercarpal joint.

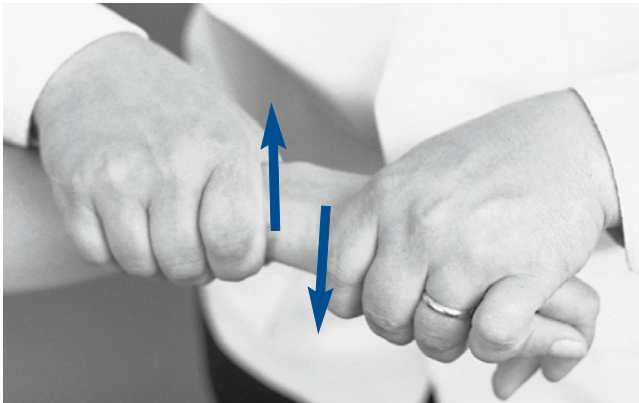


Figure 6-121 Assessment of anterior-to-posterior and posterior-to-anterior glide of the left intercarpal joint.

Assess A-P and P-A glide of the intermetacarpal joints with the patient seated and the affected arm raised in forward flexion. Stand and face the patient, grasp the adjacent metacarpals with both hands, and stress them in an A-P and P-A glide (Figure 6-123).

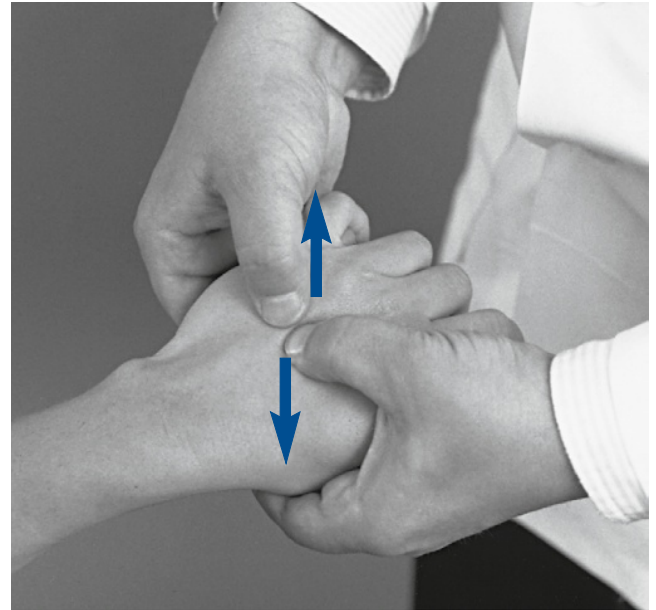


Figure 6-123 Assessment of anterior-to-posterior and posterior-to-anterior glide of the left intermetacarpal joints.

Evaluate the MP and interphalangeal joints in a similar fashion. With the patient seated, grasp the proximal member of the joint to be tested with one hand while grasping the distal member of the joint being tested with the other hand. Then stress each MP or interphalangeal joint with long-axis distraction, A-P and P-A glide, L-M and M-L glide, and internal and external rotation (Figures 6-124 and 6-125).

ADJUSTIVE PROCEDURES

The application of an impulse thrust often can be performed using the accessory joint motion test procedure and adding the impulse thrust at the end. Although this is true of any joint in the body, fewer adjustive procedures are unique or different from the testing procedure for the wrist and hand. Box 6-8 identifies the adjustive procedures for the joints of the wrist and hand.

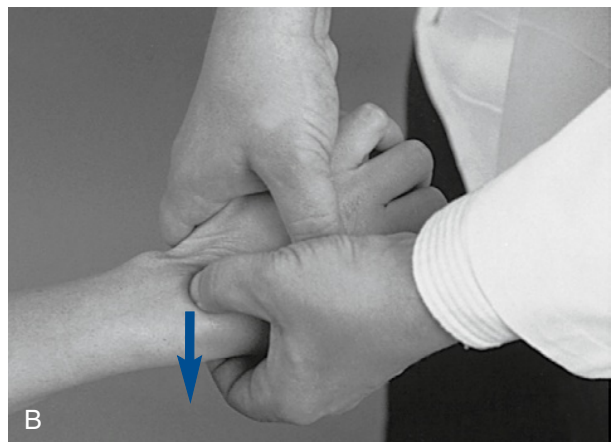
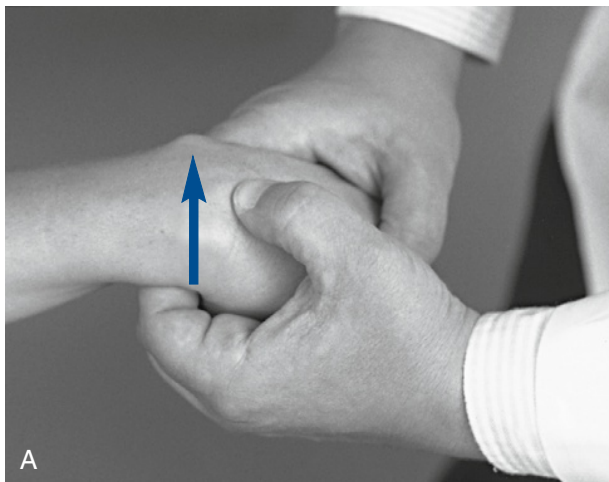
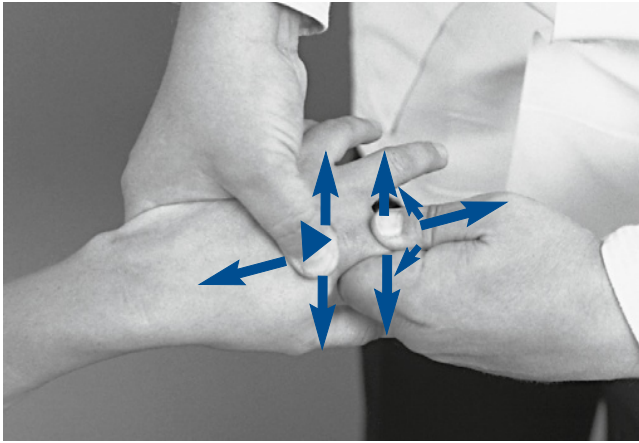
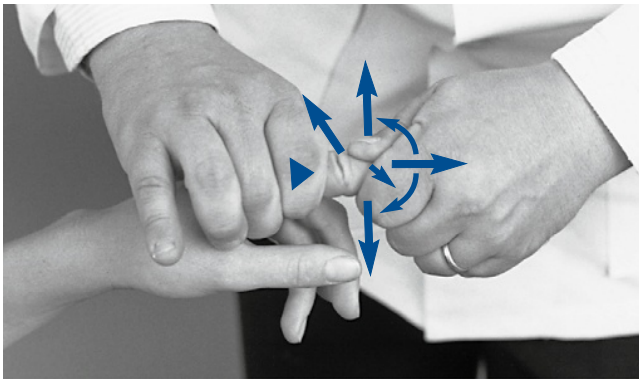


Figure 6-122 Assessment of posterior-to-anterior (A) and anterior-to-posterior (B) glide of the individual carpals of the left wrist.



6-124

Figure 6-124 Assessment of long-axis distraction, internal and external rotation, and anterior-to-posterior, posterior-to-anterior, lateral-to-medial, and medial-to-lateral glide of the left metacarpophalangeal joints.



6-125

Figure 6-125 Assessment of long-axis distraction, internal and external rotation, and anterior-to-posterior, posterior-to-anterior, lateral-to-medial, and medial-to-lateral glide of the left interphalangeal joints.

Wrist

Supine or sitting:

Bimanual Thumb-Index Radius and Ulna Shear; Anterior-to-Posterior and Posterior-to-Anterior Glide (Figure 6-126)

IND: Loss of A-P or P-A glide of the radius and ulna.

PP: The patient is supine or sitting.

DP: Stand and face the patient on the involved side.

SCP: Distal radius and distal ulna.

CP: Grasp the distal radius with one hand and the distal ulna with the other.

P: Apply an opposing A-P and P-A shearing thrust between the radius and ulna.

Sitting:

Reinforced Hypothenar/Radius; Medial-to-Lateral Compression (Figure 6-127)

IND: Loss of radial and ulnar compressibility.

PP: The patient is seated on the adjusting table, with the affected arm resting on the headrest so that the ulnar surface of the forearm is down and the radial aspect is up.

BOX 6-8

Wrist and Hand Adjustive Techniques

WRIST SUPINE OR SITTING:

Bimanual thumb index/radius and ulna shear; anterior-to-posterior or posterior-to-anterior glide (Figure 6-126)

WRIST SITTING:

Reinforced hypothenar/radius; medial-to-lateral compression (Figure 6-127)

Hand-grasp pull with forearm stabilization; long-axis distraction (Figure 6-128)

Bimanual palmar grasp/hand with arm axillary stabilization; long-axis distraction (Figure 6-129)

Bimanual grasp/distal forearm hand; medial-to-lateral or lateral-to-medial glide; medial-to-lateral or lateral-to-medial tilt (Figure 6-130)

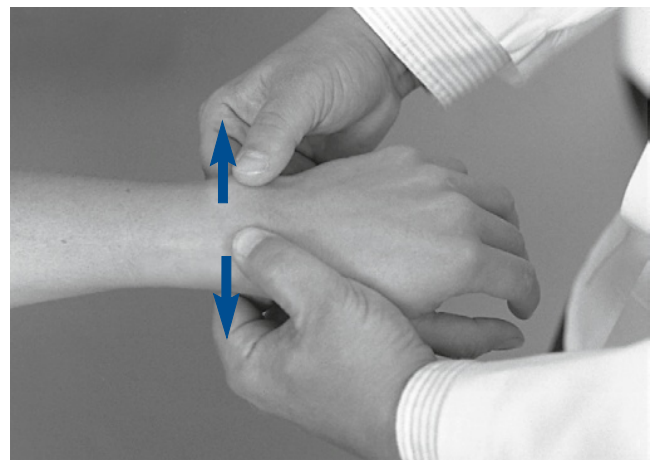
Bimanual grasp/distal forearm hand; anterior-to-posterior or posterior-to-anterior glide (Figure 6-131)

Reinforced thumbs/carpal; anterior-to-posterior or posterior-to-anterior glide (Figure 6-132)

HAND SITTING:

Bimanual thumbs digits/metacarpals; anterior-to-posterior or posterior-to-anterior glide (Figure 6-133)

Thumb index grasp/metacarpophalangeal (or interphalangeal) with hand stabilization; long-axis distraction; internal or external rotation; anterior-to-posterior or posterior-to-anterior glide; lateral-to-medial or medial-to-lateral glide (Figure 6-134)



6-126

Figure 6-126 Adjustment for anterior-to-posterior and posterior-to-anterior glide of the distal left radioulnar joint.

DP: Stand at the head end of the table, facing the patient.

SCP: Distal radius.

CP: Establish a pisiform contact with your cephalic hand over the patient's distal radius.

IH: Place your caudal hand pisiform contact in the contact hand's anatomic snuff box.

VEC: Compressive approximation.



Figure 6-127 Adjustment for lateral-to-medial compression of the distal right radioulnar joint.

P: With both arms, deliver an extension thrust, creating an impulse movement and compressing the radius and ulna. This procedure can be augmented with the use of a mechanical drop headpiece.

Hand Grasp Pull with Forearm Stabilization; Long-Axis Distraction (Figure 6-128)

IND: Loss of long-axis accessory movements.

PP: The patient is seated, with the affected arm raised in forward flexion.

DP: Stand and face the patient.

SCP: The hand.

CP: Using your inside hand, grasp the patient's hand as though to give a handshake.

IH: Grasp the distal forearm with your outside hand.

VEC: Long-axis distraction.

P: While you stabilize the forearm with your IH, distract the wrist in the long axis with your contact hand.

Bimanual Palmar Grasp/Hand with Arm Axillary Stabilization; Long-Axis Distraction (Figure 6-129)

IND: Loss of long-axis accessory movements.

PP: The patient is seated, with the affected arm slightly flexed at the shoulder and slightly flexed at the elbow.

DP: Stand or sit on the affected side.

SCP: Proximal to the hypothenar and thenar eminences.

CP: Position your inside arm so that the inferior aspect of your arm rests in the patient's antecubital fossa and a calcaneal contact can be placed just proximal to the patient's hypothenar and thenar eminences.

IH: With your outside hand, place a calcaneal contact over the dorsal aspect of the metacarpal heads, with a palmar contact over the back of the patient's hand.

VEC: Long-axis distraction.

P: Squeeze both the contact hand and the IH together to maintain contact while flexing the patient's elbow. The lever action will create and maintain a long-axis distraction at the wrist.

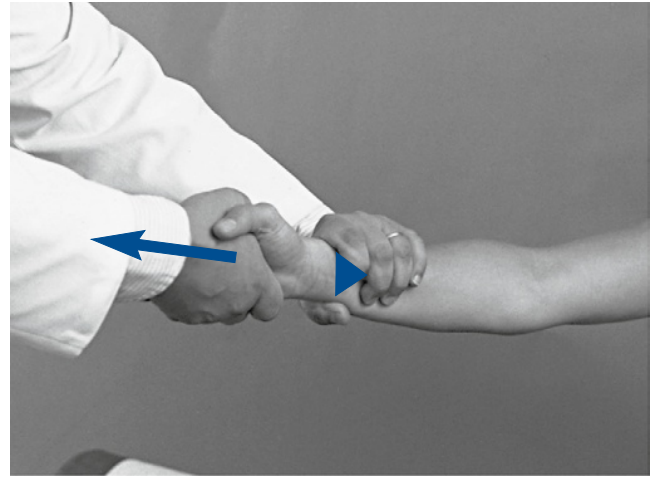
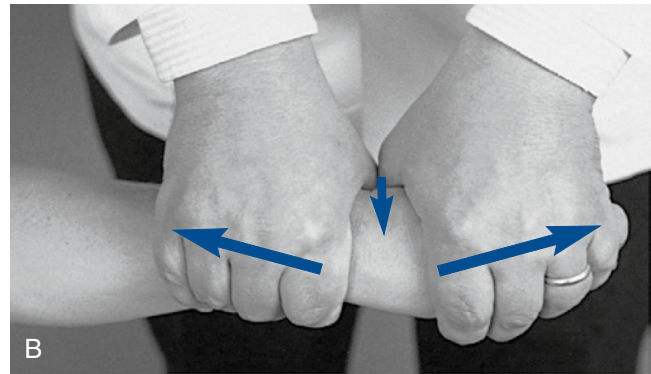
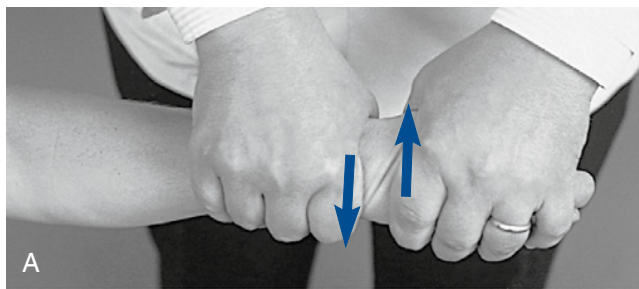


Figure 6-128 Adjustment for long-axis distraction of the right intercarpal joint.



Figure 6-129 Manipulation for sustained long-axis distraction of the right intercarpal joint.



6-130

Figure 6-130 Adjustment for medial-to-lateral and lateral-to-medial glide (A) and medial-to-lateral and lateral-to-medial tilt (B) of the left intercarpal joint.

Bimanual Grasp/Distal Forearm Hand; Medial-to-Lateral or Lateral-to-Medial Glide; Medial-to-Lateral or Lateral-to-Medial Tilt (Figure 6-130)

IND: Decreased M-L or L-M glide movements.

PP: The patient sits, with the affected arm raised in forward flexion.

DP: Stand on the affected side, facing the lateral aspect of the arm.

SCP: Distal radius and ulna.

CP: Using your proximal hand, grasp the distal radius and ulna.

IH: With your distal hand, grasp the patient's distal wrist.

P: Use both hands to develop opposing forces and deliver an impulse thrust, creating a shearing stress (M-L glide) or radial and ulnar deviation stress (M-L tilt).

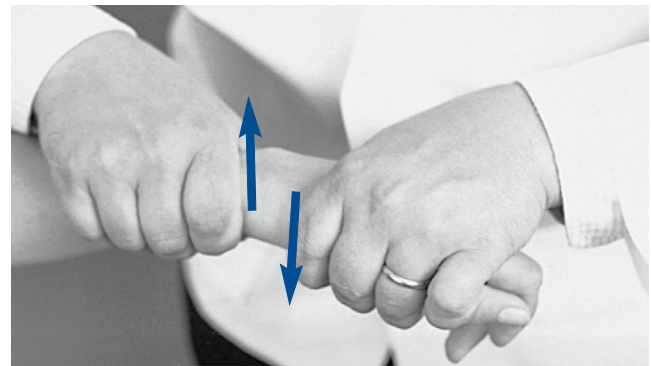
Bimanual Grasp/Distal Forearm Hand; Anterior-to-Posterior or Posterior-to-Anterior Glide (Figure 6-131)

IND: Loss of A-P and P-A accessory joint movements.

PP: The patient sits, with the affected arm raised in forward flexion.

DP: Stand on the affected side.

SCP: Distal radius and ulna.



6-131

Figure 6-131 Adjustment for anterior-to-posterior and posterior-to-anterior glide of the left intercarpal joint.

CP: With your proximal hand, grasp the distal radius and ulna.

IH: With your distal hand, grasp the patient's distal wrist over the metacarpal-carpal joints.

P: Using both hands to create opposing forces, deliver an impulse thrust, stressing the intercarpal joints in either an A-P or P-A direction.



6-132

Figure 6-132 Adjustment for anterior-to-posterior (A) and posterior-to-anterior (B) glide of the individual carpals of the left wrist.

Reinforced Thumbs/Carpal; Anterior-to-Posterior or Posterior-to-Anterior Glide (Figure 6-132)

IND: Restricted glide motions of the carpals, anterior or posterior misalignment of the individual carpals.

PP: The patient is seated, with the affected arm raised in slight forward flexion.

DP: Stand and face the patient.

SCP: Carpal bone.

CP: Establish a thumb contact over the affected carpal.

IH: Using your other hand, apply a thumb contact over the contact thumb to reinforce it.

VEC: A-P or P-A.

P: Use both hands to remove articular slack and deliver an impulse thrust with both thumbs to create A-P or P-A glide.

Hand

Sitting:

Bimanual Thumbs Digits/Metacarpals; Anterior-to-Posterior or Posterior-to-Anterior Glide (Figure 6-133)

IND: Restricted intermetacarpal glide movements.

PP: The patient is seated, with the affected arm in forward flexion and the elbow flexed so that the palm of the hand faces outward.

DP: Stand and face the patient.

SCP: Metacarpal bone.

CP: Establish a thumb contact on the palmar aspect of a metacarpal bone. With your fingers, hold the same metacarpal shaft on the dorsal surface of the hand.

IH: Make the same contacts on the adjacent metacarpal.

VEC: A-P or P-A.

P: Use both hands to create an A-P and P-A shear between the two metacarpals.

Thumb Index Grasp/Metacarpophalangeal (or Interphalangeal) with Hand Stabilization; Long-Axis Distraction; Internal or External Rotation; Anterior-to-Posterior or Posterior-to-Anterior Glide; Lateral-to-Medial or Medial-to-Lateral Glide (Figure 6-134)



Figure 6-133 Adjustment for anterior-to-posterior and posterior-to-anterior glide of the left intermetacarpal joints.

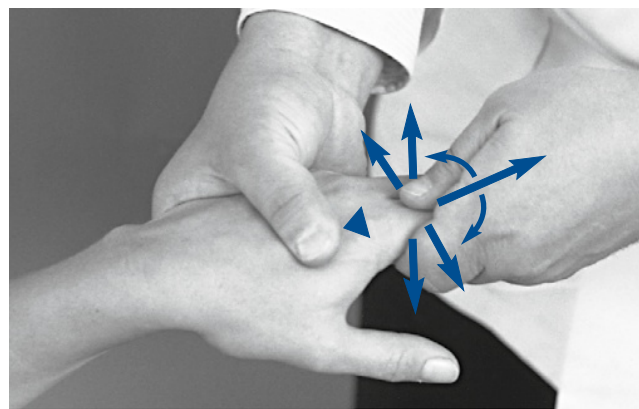


Figure 6-134 Adjustment for long-axis distraction, internal and external rotation, and anterior-to-posterior, posterior-to-anterior, lateral-to-medial, and medial-to-lateral glide of the left metacarpophalangeal joints (interphalangeal joints are adjusted in the same way).

IND: Lack of accessory joint movement in the finger joints, misalignment of the finger joints.

PP: The patient is seated.

DP: Stand and face the patient.

SCP: Distal component of the affected joint.

CP: Grasp the distal member of the joint to be adjusted with either hand.

IH: With your other hand, grasp the proximal member of the joint being adjusted.

VEC: Long-axis distraction, A-P and P-A glide, L-M and M-L glide, and internal and external rotation.

P: Apply an impulse thrust to the affected MP or interphalangeal joint, using long-axis distraction, A-P and P-A glide, L-M and M-L glide, and internal and external rotation.

HIP

The hip joint is one of the largest and most stable joints in the body.¹⁷ In contrast to the other extremity joints and, specifically, its counterpart in the upper extremity, the shoulder, the hip has intrinsic stability provided by its relatively rigid ball-and-socket configuration (Figure 6-135) and an extensive set of capsular ligaments. Although dysfunction of the hip is not as frequently encountered as dysfunction in the spine and other extremity joints, its identification and treatment are very important and often overlooked. A pathologic or traumatized hip can create a wide range of functional limitations, including difficulty in walking, dressing, driving a car, climbing stairs, and lifting and carrying loads. The hip joint must accommodate the great deal of mobility necessary for gait and the performance of daily activities. Furthermore, the hip joint is a multiaxial articulation that must form a stable link between the lower limb and the spine and pelvis.

FUNCTIONAL ANATOMY

Osseous Structures

The hip is a deep ball-and-socket joint with a spherical convex surface on the head of the femur and a concave articular surface formed by the acetabulum of the pelvis. The acetabulum is

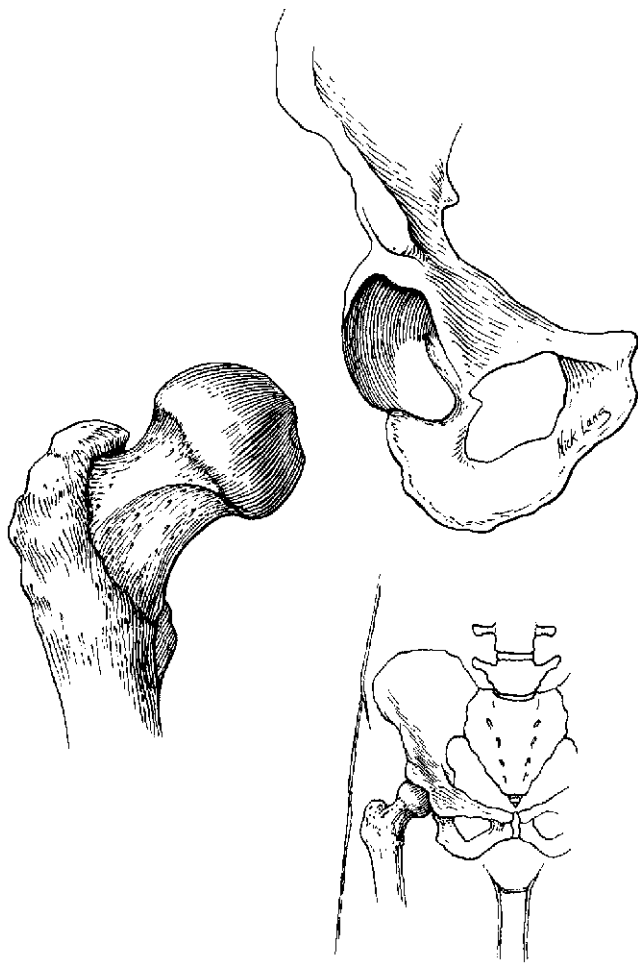


Figure 6-135 Ball-and-socket configuration of the hip joint.

formed by a fusion of the three bones that make up the innominate: the ilium (superior), the ischium (posteroinferior), and the pubic bone (anteroinferior) (Figure 6-136). A fibrocartilaginous acetabular labrum surrounds the rim of the acetabulum, effectively deepening it and serving to protect the acetabulum against

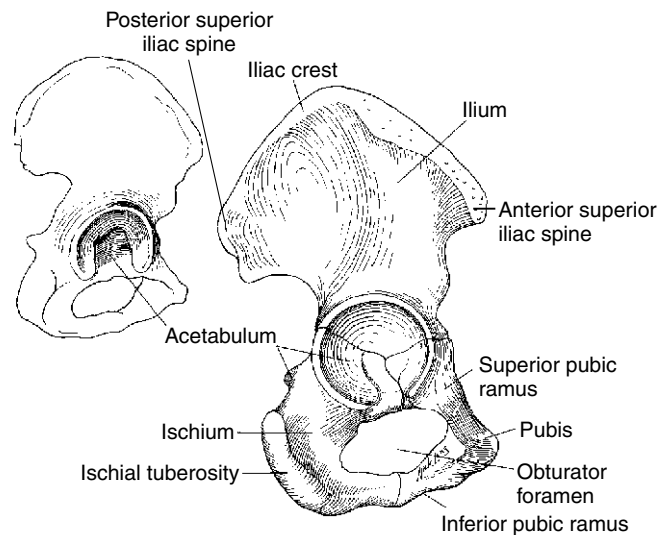


Figure 6-136 Structures of the innominate and acetabulum.

the impact of the femoral head in forceful movements. Hyaline cartilage lines the horseshoe-shaped surface of the acetabulum. The center of the acetabulum is filled in with a mass of fatty tissue covered by a synovial membrane. The cavity of the acetabulum is directed obliquely anteriorly, laterally, and inferiorly. The inferior component is important because of the transference of weight from the upper body through the sacroiliac joints into the head of the femur and down its shaft.

The femur is the longest bone in the body, as well as one of the strongest. It must withstand not only weight-transmission forces but also those forces developed through muscle contraction. The femoral head is completely covered with hyaline cartilage, except for a small pit near its center, known as the *fovea capitis*. The cartilage is thicker above and tapers to a thin edge at the circumference.

The femoral neck forms two angular relationships with the femoral shaft that influence hip function (Figure 6-137). The angle of inclination of the femoral neck to the shaft in the frontal plane (the neck-to-shaft angle) is approximately 125 degrees

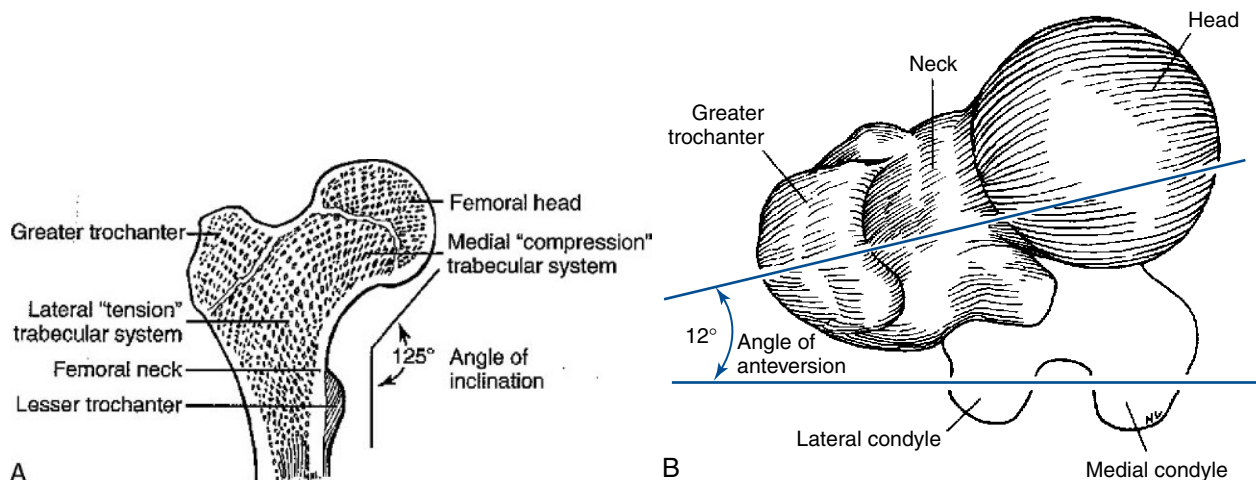


Figure 6-137 The proximal femur. **A**, Coronal section, showing the trabecular patterns and the angle of inclination of the femoral neck. **B**, Apical view of left femur, showing the angle of anteversion.

(90 to 135 degrees). This angle offsets the femoral shaft from the pelvis laterally and facilitates the freedom of motion for the hip joint. An angle of more than 125 degrees produces a coxa valga, whereas an angle of less than 125 degrees results in coxa vara. A deviation either way can alter the force relationships of the hip joint. The angle of anteversion is the second angle associated with the femoral neck and is formed as a projection of the long axis of the femoral head and the transverse axis of the femoral condyles. This angle should be approximately 12 degrees but has a great deal of variation, of which 10 to 30 degrees is considered within normal limits. Any increase in this anterior angulation is called *excessive anteversion* and results in a toe-in posture and gait. An angle that is less than ideal produces a *retroversion* and an externally rotated leg posture (toe-out) and gait.

The atmospheric pressure holding the head of the femur in the acetabulum is approximately 18 kg. This could support the entire limb without ligamentous or muscular assistance, although capsular ligament and muscular tension do help keep the head of the femur stable in the acetabulum.

A unique trabecular pattern corresponding to the lines of force through the pelvis, hip, and lower extremity are developed through the course of the femoral neck (Figure 6-138). Tension trabeculae are more superior and run from the femoral head to the trochanteric line. Compression trabeculae are inferior and run from the trochanteric area to the femoral head. The epiphyseal plates are at right angles to the tension trabeculae, which likely places them perpendicular to the joint reaction force on the

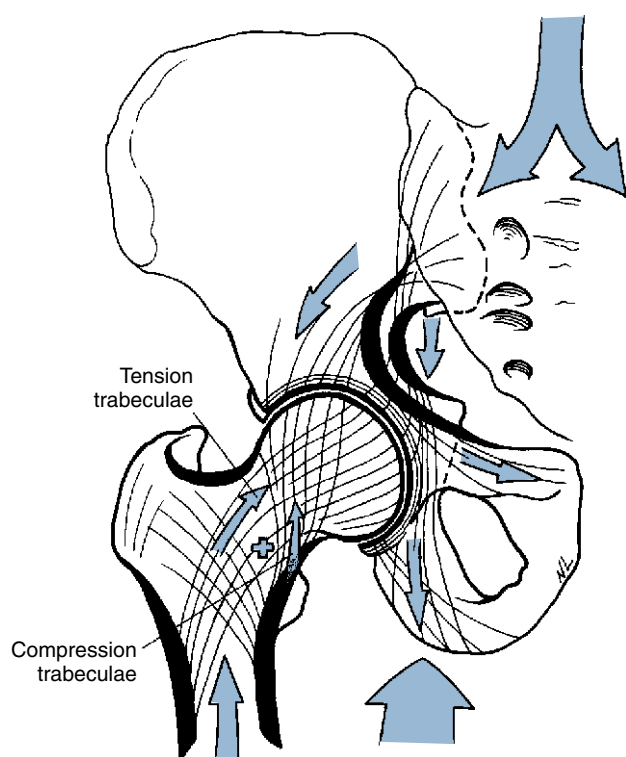


Figure 6-138 Hip joint, showing tension and compression trabeculation, as well as the forces transmitted from the ground and gravity. (Modified from Kapandji IA: *The physiology of the joints*, ed 2, vol 1, Edinburgh, 1970, Churchill Livingstone.)

femoral head. Aging produces degenerative changes that gradually cause the trabeculae to resorb, predisposing the femoral neck to fracture.

Ligamentous Structures

The hip joint is completely covered by an articular capsule that attaches to the rim of the acetabulum, to the femoral side of the intratrochanteric line, and to parts of the base of the neck and adjacent areas. The joint capsule is a cylindrical structure resembling a sleeve running between its attachment around the peripheral surface of the acetabular labrum to the femoral neck (Figure 6-139). It therefore encloses not only the head of the femur but the neck as well. From its femoral attachments, some of the fibers are reflected upward along the neck as longitudinal bands, termed *retinacula*. The capsule is thicker toward the upper and anterior part of the joint, where the greatest amount of resistance is required. Some deep fibers of the distal portion of the capsule are circular, coursing around the femoral neck and forming the *zona orbicularis*. They form somewhat of a sling, or collar, around the neck of the femur.

The joint capsule is reinforced and supported by strong ligaments named for the bony regions to which they are attached (Figure 6-140). The *iliofemoral ligament* lies anteriorly and superiorly and forms an inverted Y from the lower part of the anteroinferior iliac spine to the trochanteric line of the femur. It prevents posterior tilt of the pelvis during erect standing, limits extension of the hip joint, and is responsible for the so-called “balancing on the ligaments” maneuver that occurs in the absence of muscle contraction.

The *ischiofemoral ligament* consists of a triangular band of strong fibers extending from the ischium below and behind the acetabulum to blend with the circular fibers of the joint capsule and attaching to the inner surface of the greater trochanter. It reinforces the posterior portion of the capsule and limits excessive medial rotation, abduction, and extension.

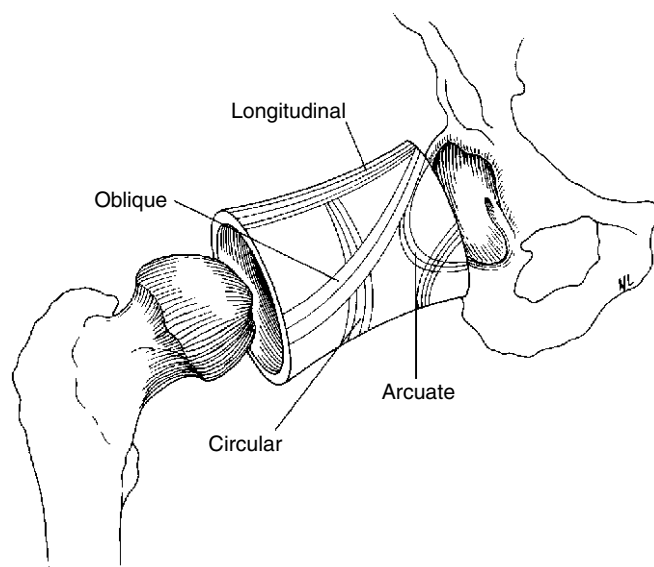


Figure 6-139 Diagrammatic representation of the cylindrical joint capsule, showing the orientation of fibers to resist stresses.

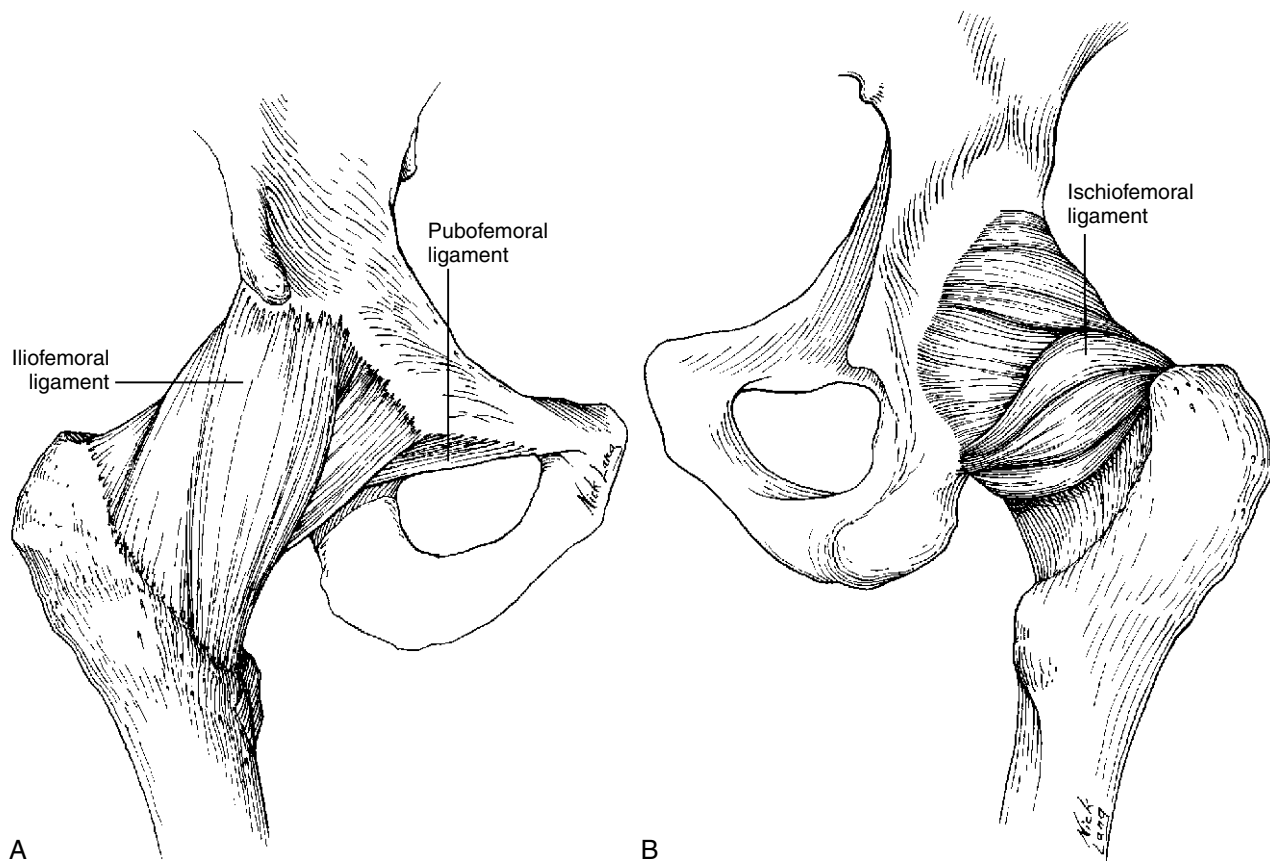


Figure 6-140 The ligamentous structure of the right hip. **A**, Anterior view. **B**, Posterior view.

The *pubofemoral ligament* is attached above to the obturator crest and the superior ramus of the pubis, and below it blends with the capsule and with the deep surface of the vertical band of the iliofemoral ligament. It reinforces the medioinferior portion of the joint capsule and limits excessive abduction, lateral rotation, and extension.

The apex of the *ligamentum teres* attaches to the fovea capitis femoris, and its base attaches by two bands, one into either side of the acetabular notch (Figure 6-141). This ligament does not truly contribute to the support of the joint, although it becomes taut when the thigh is semiflexed and then adducted or externally rotated. It sometimes contains and offers some protection for a nutrient artery that supplies the femoral head. The *ligamentum teres* is lined with synovium and is believed to play a role in assisting with joint lubrication. As such, it may act somewhat like the meniscus in the knee by spreading a layer of synovial fluid over the articular surface of the head of the femur. The *transverse ligament* crosses the acetabular notch, converting the notch into a foramen through which the artery that supplies the head of the femur runs.

Musculature

The hip is supported by strong muscles on all four sides (Table 6-14). The posterior musculature, composed of the gluteus maximus, posterior fibers of the gluteus medius, hamstrings, and piriformis, provides posterior stability for the hip joint. Anterior joint

stability is provided by the iliopsoas, sartorius, and rectus femoris muscles. The tensor fascia lata, gluteus medius, and gluteus minimus provide lateral stability. Medial stability comes from the pectineus, adductors, and gracilis muscles. With the amount of movement and soft tissues lying in the different planes associated with the hip, many bursae exist; however, only three have important clinical significance. The iliopectineal bursa lies between the iliopsoas muscle and the hip joint capsule. It sometimes communicates with the joint cavity itself, and excess fluid from trauma may spill into it. The combined motions of hip flexion and adduction or excessive extension can compress the inflamed bursa, creating pain. The trochanteric bursa separates the tendon of the gluteus maximus and the iliotibial band from the greater trochanter. Direct trauma to this area or overuse of the joint may irritate the bursa, causing it to become inflamed. A third bursa, the ischiogluteal bursa, lies superficially over the ischial tuberosity. This bursa will become inflamed in those who sit for prolonged periods, and they need to flex and extend their hips periodically.

Because of the hip's role in posture and gait, the combined actions of many powerful muscles are required. Hip flexion is accomplished primarily by the iliopsoas and is assisted by the rectus femoris, pectineus, adductor longus, gracilis, tensor fascia lata, and sartorius. Hip extension occurs through the contraction of the gluteus maximus muscle, easily one of the most powerful muscles in the body. Hip extension is assisted by the hamstrings and the posterior fibers of the gluteus medius. The gluteus medius and minimus,

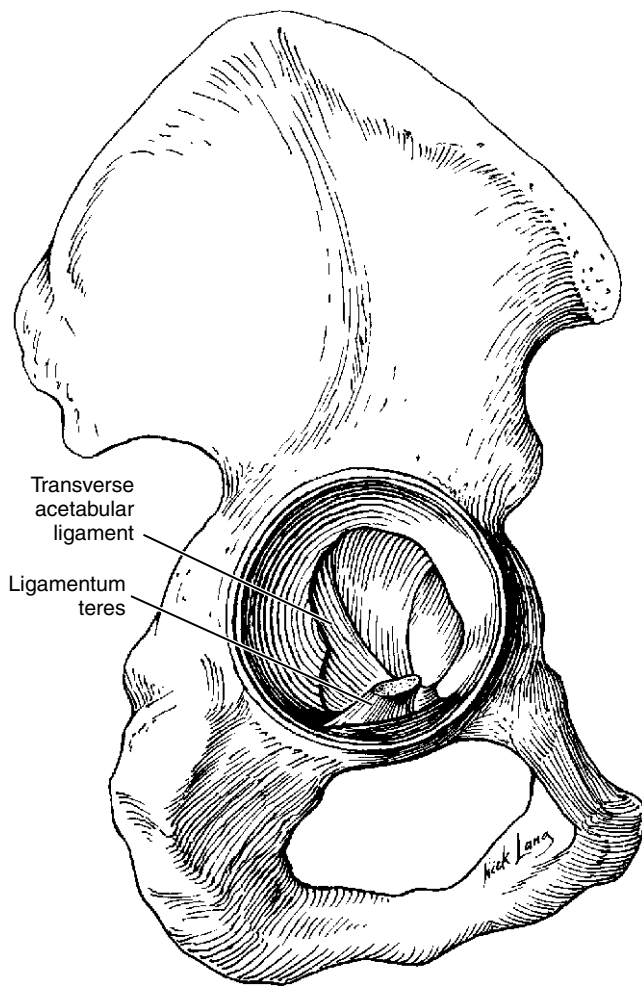


Figure 6-141 Ligamentum teres and transverse acetabular ligaments are not true supportive ligaments.

the tensor fascia lata, and to a lesser extent, the piriformis create hip abduction. Hip adduction is the primary role of the adductor muscles, the gracilis, and the pectineus, with some influence from the hamstrings. External rotation of the hip occurs through contraction of the piriformis, obturators, gemelli, and quadratus femoris muscles. Internal rotation of the hip is accomplished by the tensor fascia lata, gluteus medius, gluteus minimus, and gracilis muscles.

Several of the muscles that act at the hip joint also act with equal or greater effectiveness at the knee joint. These are known as *two-joint muscles* of the lower extremity. The location and line of pull or action of the muscles make it relatively easy to understand the mechanics of testing any individual muscle.

BIOMECHANICS

The movements of the femur are similar to those of the humerus but not as free because of the depth of the acetabulum. In the standing position, the shaft of the femur slants somewhat in a medial direction and is not vertically straight. This places the center of motion of the knee joint more nearly under the center of motion of the hip joint. Therefore, the mechanical axis of the femur is almost vertical. The degree of slant of the femoral shaft depends on both the angle between the neck and the shaft and the width of the pelvis. Seen from the side, the shaft of the femur bows forward. These orientations of the femur are provisions for resisting the stresses and strains sustained in walking and jumping and for ensuring proper weight transmission.

Pelvic rotation about the hip accounts for a significant portion of forward bending. Trunk flexion from the erect posture through approximately the first 45 to 60 degrees involves primarily the lumbar spine, with further forward bending occurring because of the pelvis rotating about the hip (Figure 6-142). The iliofemoral and ischiofemoral ligaments twist as they go from the pelvic attachment to the femur. In the erect neutral position, these ligaments are under moderate tension. Thigh extension “winds” these ligaments around the neck of the femur and tightens them.

TABLE 6-14 Actions of the Muscles of the Hip Joint	
Action	Muscles
Extension	Gluteus maximus, gluteus medius, and hamstrings
Flexion	Iliopsoas, sartorius, rectus femoris, tensor fascia lata, gracilis, and pectineus
Abduction	Tensor fascia lata, gluteus medius and minimus, and piriformis
Adduction	Adductors, pectineus, and gracilis
External rotation	Piriformis, gemelli, obturators, and quadratus femoris
Internal rotation	Tensor fascia lata, gluteus medius and minimus, and gracilis

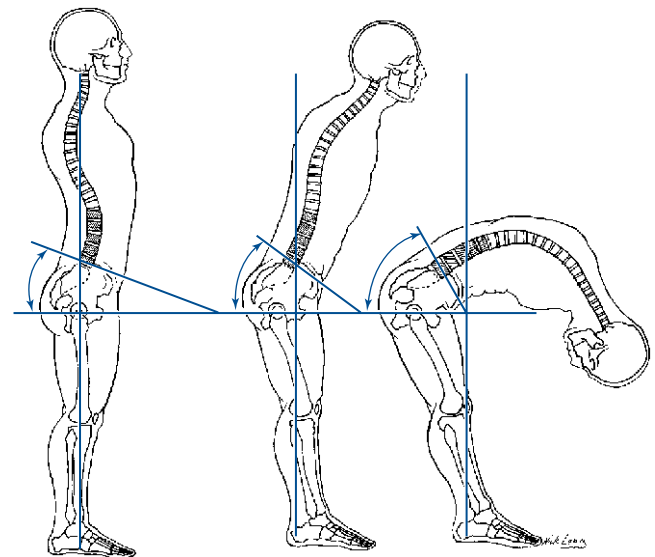


Figure 6-142 Trunk flexion begins with lumbar spine flexion, followed by pelvic flexion at the hip joints.

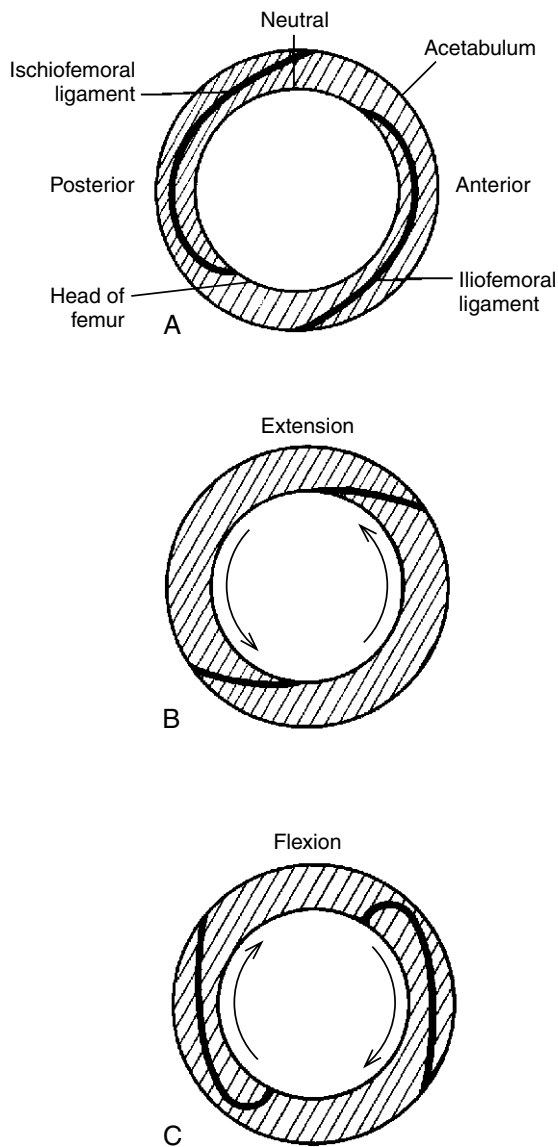


Figure 6-143 Diagrammatic representation of the effects of flexion and extension on the ischiofemoral and iliofemoral ligament. **A**, Right hip in the neutral position. **B**, Extension tightens ligaments. **C**, Flexion slackens ligaments.

Furthermore, during posterior tilting of the pelvis, these ligaments are taut and therefore are responsible for maintaining optimal pelvic position (Figure 6-143). Anterior hip and thigh pain may occur as a result of tension in these ligaments from excessive posterior pelvic tilting. In contrast, flexion of the hip “unwinds” these ligaments. Moreover, anterior pelvic tilting is not prevented by these ligaments, and the hip extensors must play an important role in stabilizing the pelvis in the anteroposterior direction. The twisting of these ligaments, as well as the twisting that occurs within the joint capsule, draws the joint surfaces into a close-packed position through a “screw-home” movement of the joint surfaces. The close-packed position of the hip is in extension, abduction, and internal rotation (Box 6-9). According to Kapandji,²¹ erect posture tilts the pelvis posteriorly, relative to the femur, causing these ligaments to become coiled around the femoral neck.

BOX 6-9 Close-Packed and Loose-Packed (Rest) Positions for the Hip Joint

CLOSE-PACKED POSITION

Full extension, internal rotation, and abduction

LOOSE-PACKED POSITION

30 degrees of flexion, 30 degrees of abduction, and slight external rotation

During flexion, a forward movement of the femur occurs in the sagittal plane. If the knee is straight, the movement is restricted by the tension of the hamstrings. In extreme flexion, the pelvis tilt supplements the movement at the hip joint. Extension is a return movement from flexion. Hyperextension, however, is a backward movement of the femur in the sagittal plane. This movement is extremely limited. In most people, this is possible only when the femur is rotated outward. The restricting factor is the iliofemoral ligament at the front of the joint. The advantage of restriction of this movement is that it provides a stable joint for weight-bearing without the need for strong muscular contraction. The movements are mostly rotary actions. *Abduction* is described as a sideward movement of the femur in the frontal plane, with the thigh moving away from the midline of the body. A greater range of movement is possible when the femur is rotated outward. *Adduction* is a return movement from abduction, whereas hyperadduction is possible when the other leg is moved out of the way. Abduction and adduction motions are a combination of roll and glide. Internal rotation and external rotation are rotary movements of the femur around its longitudinal axis, resulting in the knee turning inward and outward, respectively (Figure 6-144). Circumduction is a combination of flexion, abduction, extension, and adduction performed sequentially in either direction (Table 6-15).

When the hip is externally rotated, the anterior ligaments become taut while the posterior ligaments relax. The converse is true when the hip is internally rotated (Figure 6-145). During adduction the inferior part of the joint capsule becomes slack while the superior portion becomes taut. The opposite is true during abduction; the inferior part of the capsule becomes taut, and the superior portion relaxes and folds on itself (Figure 6-146). During abduction the iliofemoral ligament becomes taut, and the pubofemoral ligament and ischiofemoral ligament slacken. Again, during adduction, the opposite occurs; the pubofemoral ligament and the ischiofemoral ligament become taut, and the iliofemoral ligament slackens.

Pelvic stability in the coronal plane is secured by the simultaneous contraction of the ipsilateral and contralateral adductors and abductors. When these antagonistic actions are properly balanced, the pelvis is stabilized in the position of symmetry (Figure 6-147). If, however, an imbalance exists between the abductors and the adductors, the pelvis will tilt laterally to the side of adductor predominance. If the pelvis is supported by only one limb, stability is provided only by the action of the ipsilateral abductors. An insufficiency in the abductor muscles and,

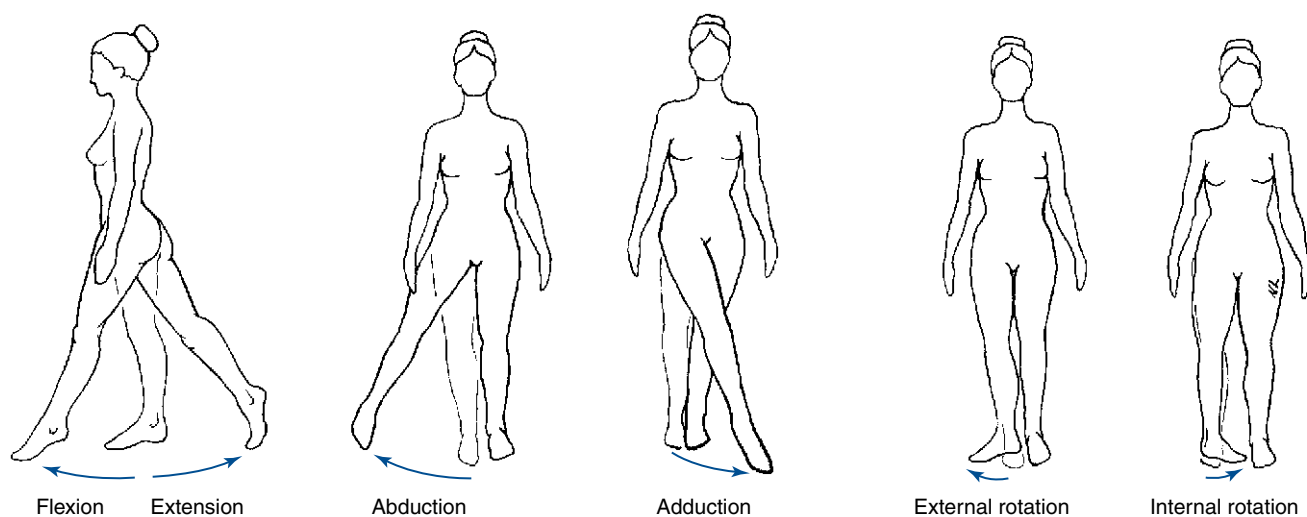


Figure 6-144 Hip joint movements.

TABLE 6-15 Arthrokinematic and Osteokinematic Movements of the Hip Joint

Osteokinematic Movements	Degrees	Arthrokinematic Movements
Flexion	120	Rotation
Extension	30	Rotation
Abduction	45–50	Roll and glide
Adduction	20–30	Roll and glide
Internal rotation	35	Roll and glide
External rotation	45	Roll and glide

specifically, the gluteus medius results in the body weight not being counterbalanced, resulting in a pelvic tilt to the opposite side. The severity of muscular insufficiency relates directly to the degree of lateral pelvic tilting. Furthermore, during standing on one leg, the femoral head must support more than the weight of the body. The total force acting vertically at the femoral head is equal to the force produced by the pull of the abductors plus the force produced by the body weight or up to three times the body weight.³

The resting, or loose-packed, position of the hip, or that in which the joint capsule is totally slack, is 10 degrees of flexion, 10 degrees of abduction, and 10 degrees of external rotation. This position will often be assumed to accommodate swelling. Pathomechanical changes and degenerative processes can alter the resting position. A joint posture of flexion, adduction, and external rotation is the classic capsular pattern of the hip.

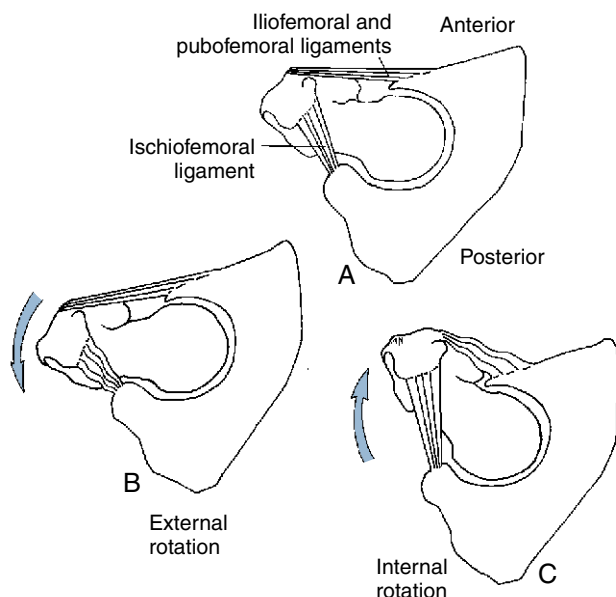


Figure 6-145 Transverse section through the left hip, demonstrating effects of internal and external rotation on the ischiofemoral, iliofemoral, and pubofemoral ligaments. **A**, Neutral position. **B**, External rotation slackens posterior ligaments and stretches anterior ligaments. **C**, Internal rotation slackens anterior ligaments.



Figure 6-146 Coronal section through the right hip viewed from anterior to posterior, demonstrating the effects of abduction and adduction of the joint capsule. **A**, Neutral. **B**, Adduction tightens superior fibers and slackens inferior fibers. **C**, Abduction tightens inferior fibers and slackens superior fibers.

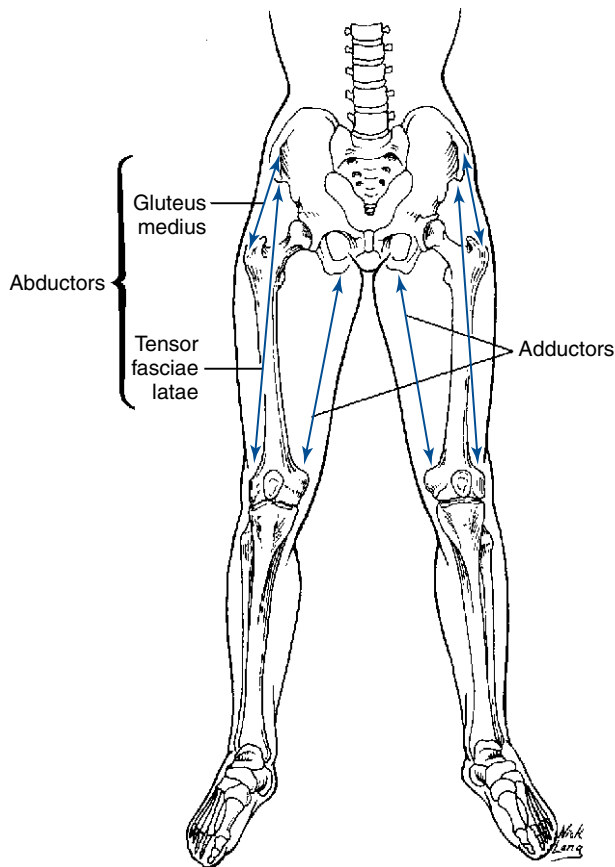


Figure 6-147 Pelvic stability in the coronal plane is produced by a balance between the abductors and adductors.

EVALUATION

Although the hip joint exhibits 3 degrees of freedom of motion and is analogous to the glenohumeral joint, the hip is intrinsically a much more stable joint. The hip, however, is still quite prone to pathomechanic changes and, as such, is often overlooked as a source for mechanical joint dysfunction. Clinically, pain originating in the hip joint is primarily perceived as involving the L3 segment, although derivation of the hip joint is from segments L2 to S1. Hip pain can be the result of referral from the facets of the lower lumbar spine. Moreover, the knee also refers pain to the hip area, and the hip can refer pain to the knee (Figure 6-148).

The muscles working across the hip joint are subject to strain, either through overuse (chronic strain) or overstress (acute strain or trauma). Tenderness is usually localized to the involved muscle, and the pain increases with resisted contraction. Commonly strained muscles include the sartorius, rectus femoris, iliopsoas, hamstrings, and adductors.

Trochanteric bursitis, a result of overuse or direct injury, presents as pain felt primarily over the lateral hip region, which is often aggravated by going up stairs. The pain is usually described as deep and aching pain that began insidiously. Getting in and out of a car is sometimes listed as a precipitating factor. Point tenderness is found over the inflamed bursa at the posterolateral aspect of the greater trochanter.

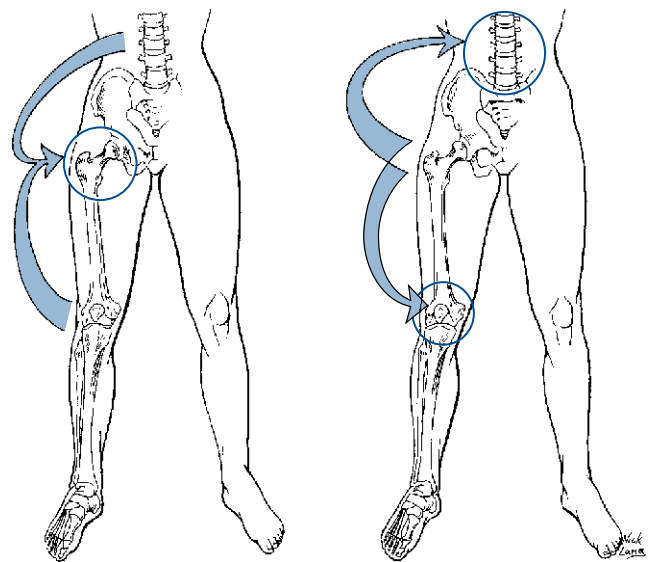


Figure 6-148 Hip pain can be referred from the knee or lumbar spine, and hip disorders can refer pain to the lumbar spine and knee.

Entrapment of several peripheral nerves can occur in association with hip dysfunction. The femoral nerve lies close to the femoral head, and trauma or hematoma may produce entrapment, causing weakness of the hip flexors and local tenderness in the groin. The sciatic nerve, which passes deep to or through the piriformis muscle, may be compressed with contraction of the piriformis muscle. A sciatic radiculopathy with concomitant motor and sensory changes may result. The lateral femoral cutaneous nerve is prone to entrapment near the anterior superior iliac spine (ASIS), where the nerve passes through the lateral end of the inguinal ligament. Entrapment creates a condition called *meralgia paresthetica* and is characterized by a burning pain in the anterior and lateral portions of the thigh. The condition may be associated with a biomechanical dysfunction of the lumbopelvic complex and postural unleveling of the pelvis.²²

Use radiographic examination of children with hip pain (anteroposterior and frog leg) to evaluate the integrity of the capital femoral epiphysis. A slipped capital femoral epiphysis may occur, creating hip or knee pain. On examination, the hip will tend to swing into external rotation instead of flexion. A referral for a surgical consult is indicated.

An unrecognized or improperly treated slipped capital femoral epiphysis or a reactive synovitis may occlude the blood supply to the femoral head, and part or all of the femoral head may die as a result of avascular necrosis. These avascular changes usually involve the superoanterior weight-bearing part of the femoral head, and in later stages, this area becomes irregular, collapsed, and sclerotic. Examine radiographs for rarefaction of the femoral head, characteristic of Legg-Calvé-Perthes avascular necrosis.

To begin evaluation of the hip, observe the joint for the presence of any skin lesions associated with trauma, signs of inflammation, and the presence of pelvic obliquity. Observe gait patterns, although usually a pathomechanic hip dysfunction will not be severe enough to create a noticeable change in gait. However, toeing-in or toeing-out may be identified.

BOX 6-10 Accessory Joint Movements of the Hip Joint

Long-axis distraction
Anterior-to-posterior glide
Posterior-to-anterior glide
Internal rotation
External rotation
Inferior glide in flexion

Note osseous symmetry and relationships between the greater trochanters, ASIS and posterior superior iliac spine, iliac crests, ischial tuberosities, and pubic symphysis. Identify tone, texture, and tenderness changes through soft tissue palpation of the bursa, inguinal ligament, hip flexors, hip extensors, hip adductors, and hip abductors.

Evaluate accessory joint motions for the hip joint for the presence of joint dysfunction (Box 6-10). Evaluate long-axis distraction of the iliofemoral joint, with the patient supine and the affected side close to the edge of the table. Straddle the patient's distal thigh, grasping the area just proximal to the epicondyles with your knees. With your outside hand, palpate the greater trochanter while you stabilize the pelvis at the ASIS with your inside hand. By straightening your legs, you can induce a long-axis distraction into the hip joint and perceive a springing joint play movement with the contact on the greater trochanter (Figure 6-149).

Evaluate internal and external rotation, with the patient supine with the affected hip flexed to 90 degrees and the knee flexed to 90 degrees. Stand on the affected side, facing cephalad and using your outside hand to palpate the hip joint and greater trochanter while grasping the patient's calf and thigh area with your inside arm. Then induce internal and external rotational stresses while evaluating for the presence of a springy end feel-type motion (Figure 6-150).

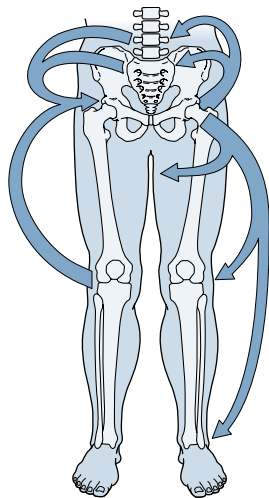
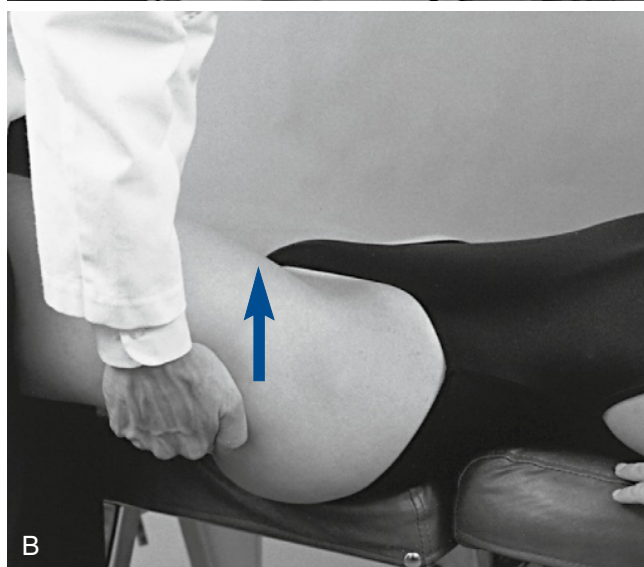
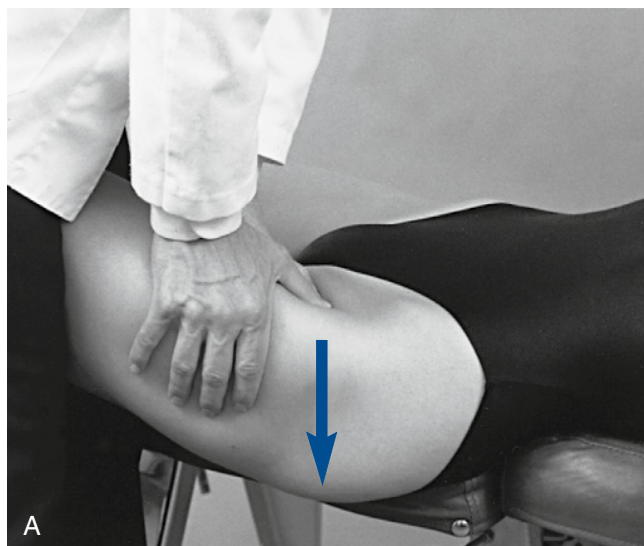


Figure 6-149 Referred pain around the hip. Right side demonstrates referral to the hip. Left side shows referral from hip. (Modified from Magee DJ: *Orthopedic physical assessment*, ed 5, St Louis, 2008, Saunders.)

Determine A-P and P-A glide movement, with the patient supine and the involved leg slightly abducted. Straddle the patient's thigh just above the knee. Grasp the proximal thigh with both hands and induce an A-P and P-A stress, feeling for the presence of a springing joint play movement (Figure 6-151).



Figure 6-150 Assessment of external (A) and internal (B) rotation of the left hip joint.



6-151

Figure 6-151 Assessment of anterior-to-posterior (A) and posterior-to-anterior (B) glide of the left hip joint.

Evaluate inferior glide of the hip in flexion, with the patient supine and the involved knee flexed to 90 degrees and the hip flexed to 90 degrees. Stand on the involved side, facing the patient and bending over so that the patient's calf can rest over your shoulder. Grasp the anterior aspect of the proximal thigh and create a caudal stress toward the foot end of the table, evaluating for the presence of a springing end-feel movement (Figure 6-152).

ADJUSTIVE PROCEDURES

The manipulative techniques used to treat hip disorders aim to restore normal joint mechanics, which will then ideally allow full pain-free functioning of the hip joint. Box 6-11 identifies the adjustive procedures for the hip.



6-152

Figure 6-152 Assessment of inferior glide in flexion of the left hip joint.

BOX 6-11 Hip Adjustive Techniques

HIP SUPINE:

- Bimanual grasp/distal tibia pull; long-axis distraction (Figure 6-153)
- Towel wrap grasp/distal tibia pull; long-axis distraction (Figure 6-154)
- Bimanual grasp/proximal femur; internal rotation (Figure 6-155)
- Bimanual grasp/proximal femur; external rotation (Figure 6-156)
- Hypothenar/proximal femur, palmar distal femur grasp; anterior-to-posterior glide (Figure 6-157)
- Bimanual grasp/proximal femur; inferior glide in flexion (Figure 6-158)

HIP SIDE POSTURE:

- Hypothenar/trochanter push; long-axis distraction (Figure 6-159)

HIP PRONE:

- Hypothenar/proximal femur, palmar distal femur grasp; posterior-to-anterior glide (Figure 6-160)

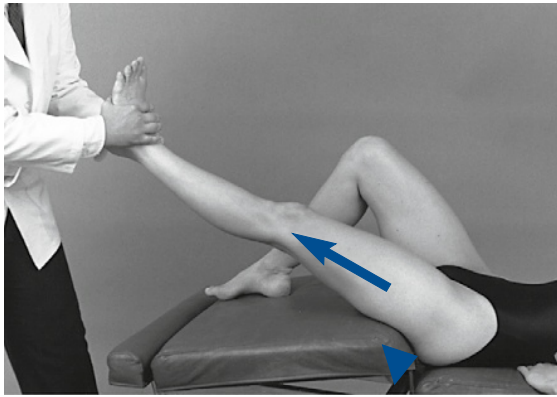


Figure 6-153 Adjustment for long-axis distraction of the left hip joint.

Hip

Supine:

Bimanual Grasp/Distal Tibia Pull; Long-Axis Distraction (Figure 6-153)

IND: Loss of long-axis distraction accessory movement.

PP: The patient is supine, with the ischial tuberosities just cephalad of the slightly raised pelvic piece.

DP: Stand at the foot of the table.

SCP: Distal tibia.

CP: With both hands, grasp the distal tibia just above the ankle.

VEC: Long-axis distraction.

P: Use the raised pelvic piece to stabilize the patient's pelvis on the table while using both hands to produce long-axis distraction at the hip joint. It is difficult to perceive whether the distraction has affected the hip, the knee, or the ankle, and it likely affects all three. A modification to this technique is made by wrapping a towel around the patient's ankle and grasping the towel with both hands (**Supine Towel Wrap Grasp/Distal Tibia Pull to Induce Long-Axis Distraction**) [Figure 6-154].

Bimanual Grasp/Proximal Femur; Internal Rotation (Figure 6-155)

IND: Loss of internal rotation accessory joint movement of the hip joint, external rotation misalignment of the proximal femur.

PP: The patient is supine, with the affected hip flexed to 90 degrees and the knee flexed to 90 degrees.

DP: Stand on the side of involvement, facing cephalad.

SCP: Femur midshaft.

CP: With your cephalic hand, grasp the proximal aspect of the affected femur.

IH: With your caudal hand, grasp the proximal aspect of the affected femur, reinforcing the CP.

VEC: Internal rotation.

P: Using your IH, induce internal rotation, then deliver an impulse-type thrust with the contact hand.

Bimanual Grasp/Proximal Femur; External Rotation (Figure 6-156)

IND: Loss of external rotational accessory joint movement or the hip joint, internal rotation misalignment of the proximal femur.

PP: The patient is supine, with the affected hip flexed to 90 degrees and abducted slightly and the knee flexed to 90 degrees.

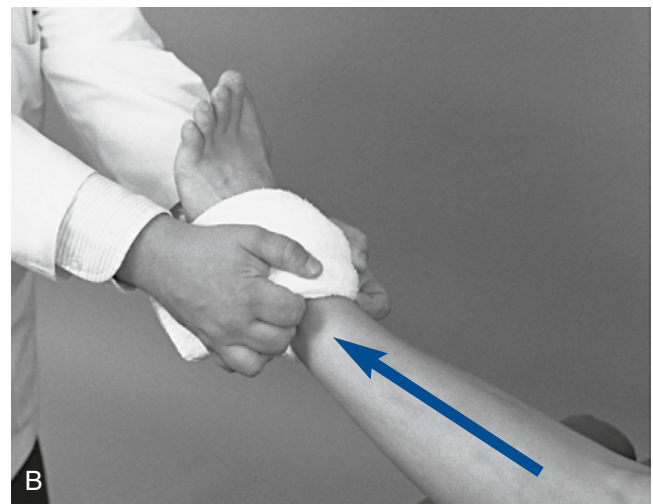


Figure 6-154 Modification for long-axis distraction using a towel. **A**, Wrapping the towel. **B**, Grasping the towel to apply the thrust.

DP: Stand on the side of involvement between the patient's leg and the adjusting table, facing outward.

SCP: Medial aspect of the proximal femur.

CP: With your cephalic hand, grasp the medial aspect of the proximal femur.

IH: With your caudal hand, grasp the proximal aspect of the affected femur, reinforcing the CP.

VEC: External rotation.

P: Using your IH, stress the femur into external rotation, then supply an impulse thrust with the contact hand.

Hypothenar/Proximal Femur, Palmar/Distal Femur Grasp; Anterior-to-Posterior Glide (Figure 6-157)

IND: Loss of A-P glide accessory joint movements, anterior misalignment of the proximal femur.

PP: The patient is supine, with the hip and knee flexed slightly.

DP: Stand at the side of the table opposite the involved leg.

SCP: Anterior aspect of the proximal femur.

CP: With your cephalic hand, establish a knife-edge contact over the anterior aspect of the proximal femur.

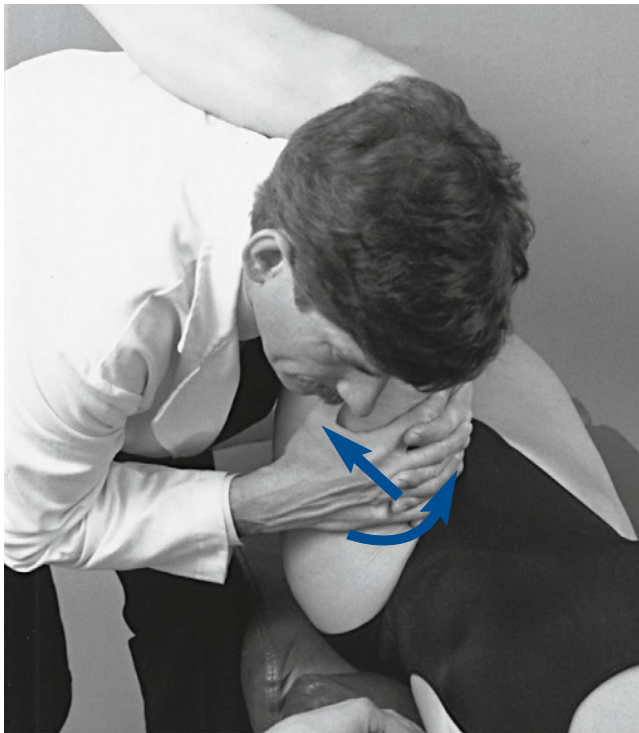


Figure 6-155 Adjustment for internal rotation of the left hip joint.



Figure 6-156 Adjustment for external rotation of the left hip joint.

IH: With your caudal hand, grasp the distal femur with the fingers in the popliteal fossa.

VEC: A-P.

P: Using your IH, flex the hip while delivering an A-P impulse thrust to the proximal femur with the contact hand.



Figure 6-157 Adjustment for anterior-to-posterior glide of the left hip joint.

Bimanual Grasp/Proximal Femur; Inferior Glide in Flexion (Figure 6-158)

IND: Loss of inferior glide in flexion accessory joint movements of the hip joint.

PP: The patient is supine, with the involved knee flexed to 90 degrees and the hip flexed to 90 degrees.

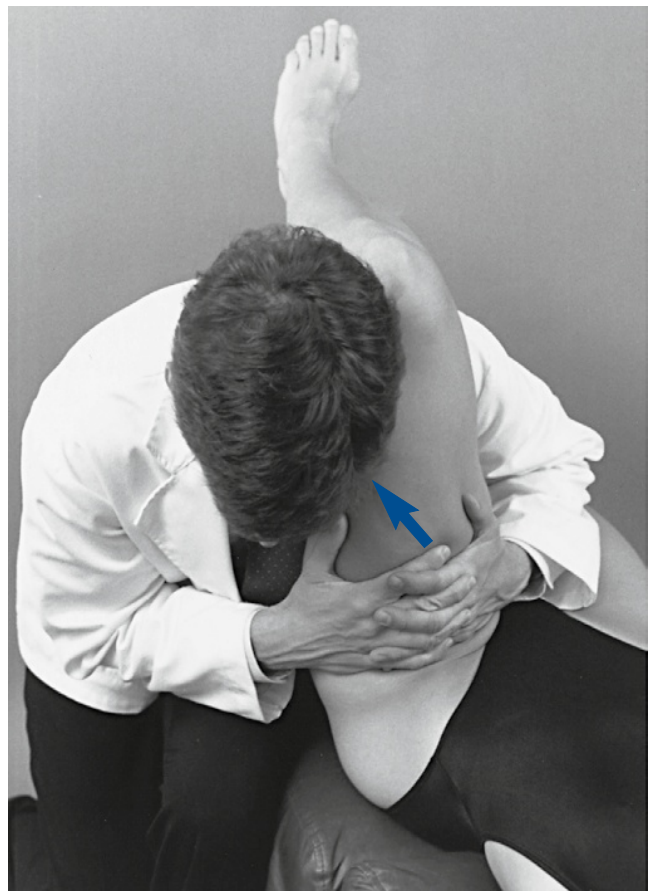
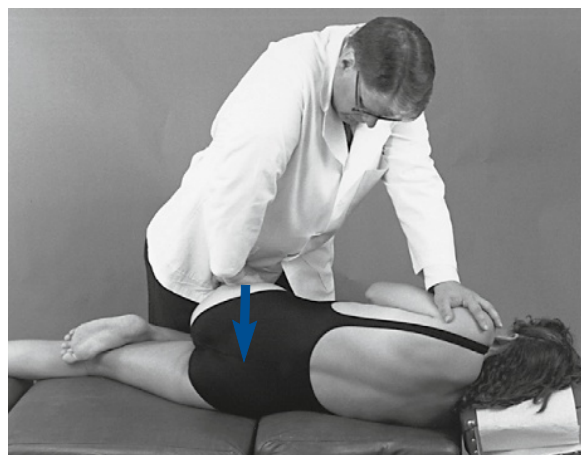


Figure 6-158 Adjustment for inferior glide in flexion of the left hip joint.



6-159

Figure 6-159 Adjustment for long-axis distraction, as well as inferior glide in flexion or internal rotation, of the left hip joint in the side-posture position.

DP: Stand on the involved side, facing the patient, flexed forward, with the patient's calf resting over your shoulder.

SCP: Anterior aspect of the proximal femur.

CP: Grasp the anterior aspect of the proximal thigh with both hands.

VEC: S-I.

P: With both hands, deliver an impulse thrust caudal.

Side Posture:

Hypothenar/Trochanter Push; Long-Axis Distraction (Figure 6-159)

IND: Loss of long-axis distraction accessory movement of the hip joint.

PP: The patient lies in a basic side-posture position, with the involved side up, the hip flexed to approximately 60 degrees, and the knee bent to 90 degrees, with the dorsum of the foot in the popliteal fossa of the other leg.

DP: Stand in front of the patient, straddling the patient's involved leg.

SCP: Posterosuperior aspect of the greater trochanter.

CP: With your caudal hand, establish a pisiform hypothenar contact on the posterosuperior aspect of the greater trochanter.

IH: Use your cephalic hand to apply a palmar contact over the patient's shoulder.

VEC: Long-axis distraction.

P: With your IH, stabilize the patient's trunk while using your contact hand to create long-axis distraction to the hip joint. This same procedure can also be used for a loss of internal rotation and inferior glide in flexion.

Prone:

Hypothenar/Proximal Femur, Palmar Distal Femur Grasp; to Induce Posterior-to-Anterior Glide (Figure 6-160)

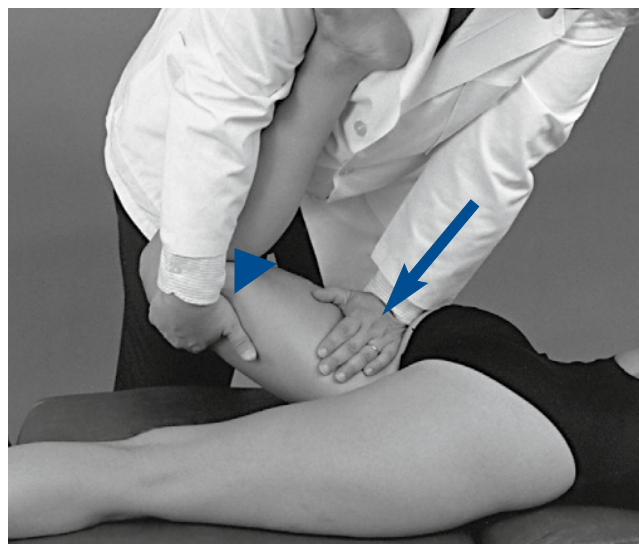
IND: Loss of P-A glide accessory joint movements, posterior misalignment of the proximal femur.

PP: The patient is prone.

DP: Stand at the side of the table on the involved side.

SCP: Posterior aspect of the proximal femur.

CP: With your cephalic hand, apply a knife-edge contact to the posterior aspect of the proximal femur.



6-160

Figure 6-160 Adjustment for posterior-to-anterior glide of the left hip joint.

IH: With your caudal hand, grasp the distal femur from the medial aspect (the patient's knee can be bent and cradled against your IH and forearm).

VEC: P-A.

P: With your IH, draw the hip into extension by raising the knee off the table. Deliver a P-A impulse thrust with your contact hand.

KNEE

The distal end of the femur and the proximal end of the tibia are connected by numerous ligaments and stabilized by strong muscles to form the very complicated knee joint. This joint is situated between the body's two longest lever arms and therefore must be able to transmit significant loads as it sustains high forces through upright posture and gait. Three articular complexes are typically discussed in conjunction with the knee: the tibiofemoral, patellofemoral, and tibiofibular articulations. However, only the tibiofemoral and patellofemoral articulations participate in knee joint activity. The tibiofibular articulation does not actually contribute to the actions of the knee. Instead it is part of the ankle joint complex, moving with inversion and eversion as well as dorsiflexion and plantar flexion. However, dysfunctional processes in the proximal tibiofibular articulation can affect other knee functions and can be a source of knee pain.

The knee joints are located between the ends of each supporting column of the body and are therefore subjected to severe stress and strain in the combined function of weight-bearing and locomotion. For adaptation to weight-bearing stresses, the knee has large condyles that are padded by the intra-articular menisci. The articular structure allows a wide ROM to facilitate locomotion, and the knee has strong ligaments on its sides to resist lateral stresses. To combat the downward pull of gravity and to meet the demands of violent locomotor activities, such as running and jumping, the knee is provided with powerful musculature. Injuries to the ligaments and cartilage of the knee are two common consequences of the large function demands placed on the knee.²³

FUNCTIONAL ANATOMY

Osseous Structures

The femoral shaft lies in oblique alignment with the lower leg, which produces a physiologic valgus angle of approximately 170 to 175 degrees (Figure 6-161). The distal end of the femur is expanded to form a large, convex, U-shaped articular surface (Figure 6-162). The medial and lateral femoral condyles lie on the end of the U shape and are separated by the intercondylar fossa. Anteriorly, the articular surface of the femoral condyles forms the patellar groove. The proximal end of the tibia is flattened to create a plateau with a bifid, nonarticulating intracondylar eminence, dividing the plateau into medial and lateral sections to accommodate the medial and lateral femoral condyles. The tibial tuberosity projects from the anterior surface of the tibia, serving as the point of insertion of the quadriceps tendon (Figure 6-163). The patella, the largest sesamoid bone in the body, lies embedded within the quadriceps tendon. It is triangular in shape, with its apex directed inferiorly. The anterior surface is nearly flat, and a longitudinal

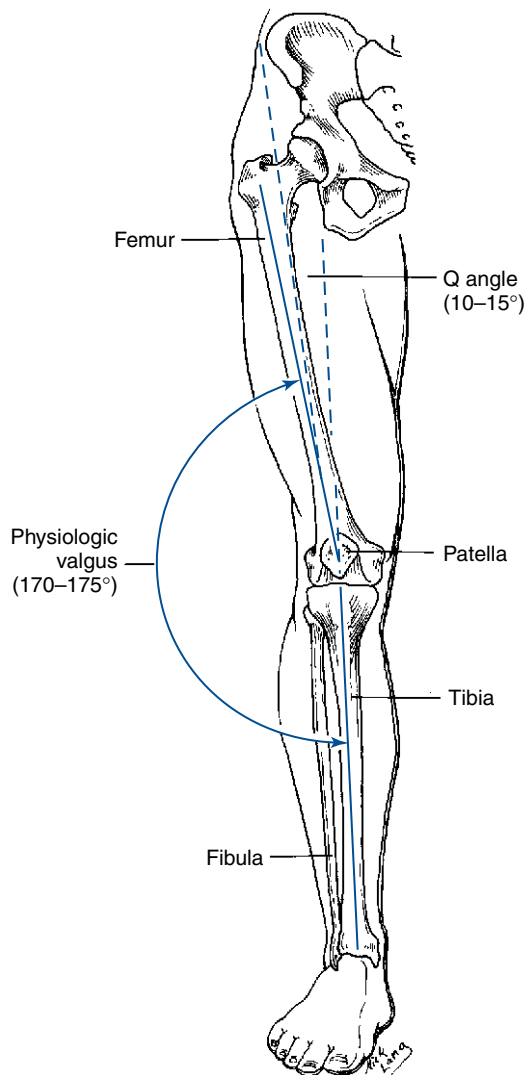


Figure 6-161 The physiologic valgus tilt of the lower extremity places the knee under the hip.

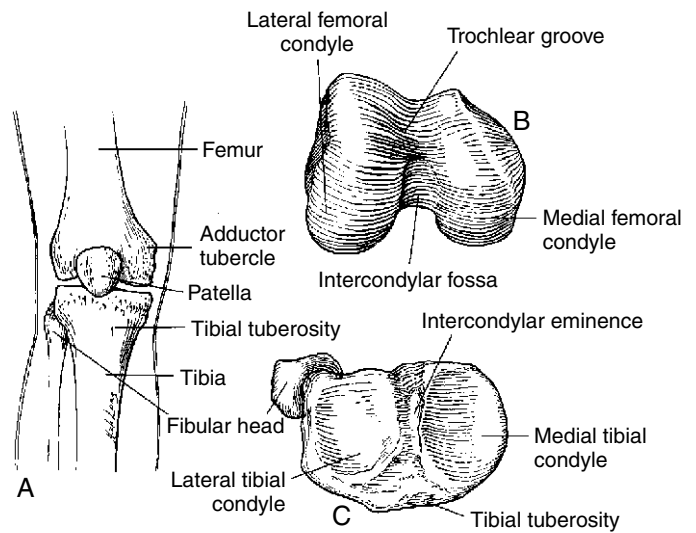


Figure 6-162 Osseous structures of the right knee. **A**, Anterior view. **B**, Articular surface of the distal femur. **C**, Articular surface of the proximal tibia.

ridge divides the posterior surface into medial and lateral articulating facets. The longitudinal ridge fits into the patellar groove of the femur. The proximal head of the fibula is expanded and contains a single facet that corresponds with a facet on the posterolateral aspect of the rim of the tibial condyle.

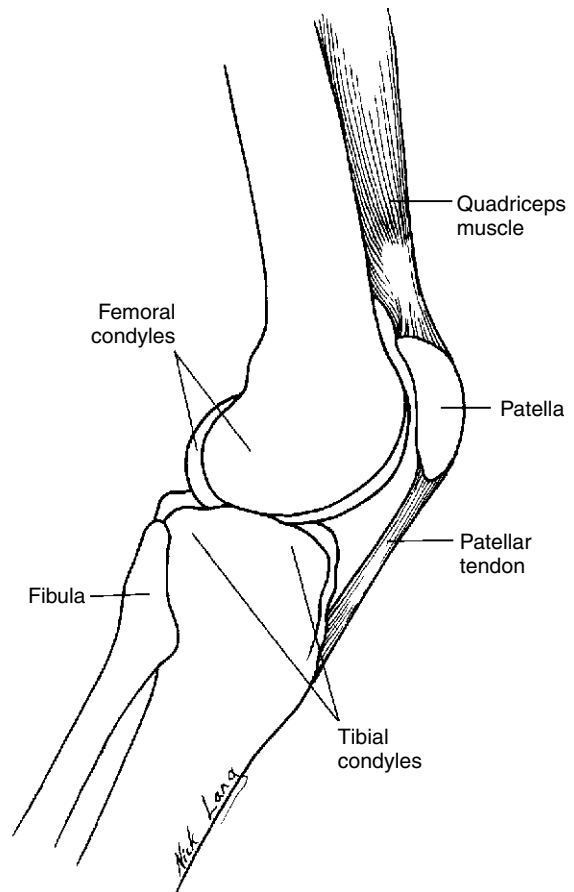


Figure 6-163 Lateral view of the knee.

Ligamentous Structures

Internal to the joint are the cruciate ligaments, arranged in a criss-cross manner, providing A-P, as well as M-L, stability at the knee. They prevent excessive medial rotation of the tibia and help maintain contact between the articular surfaces of the tibia and femur (Figures 6-164 to 6-167). The anterior cruciate ligament extends from the anterior aspect of the intercondylar eminence of the tibia and runs posteriorly and superiorly to the medial side of the lateral

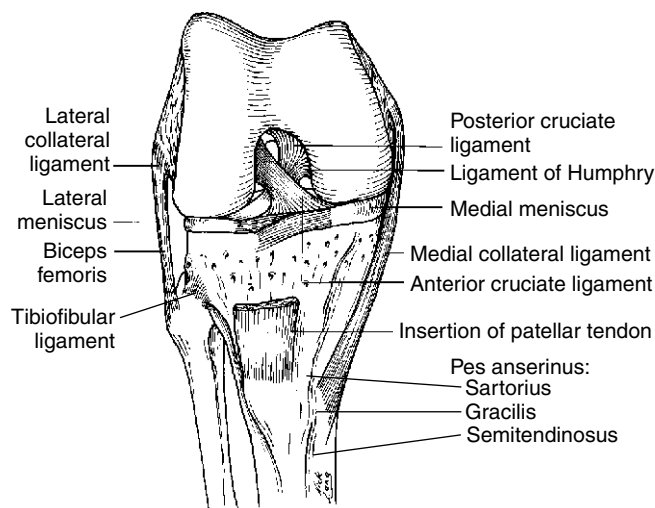


Figure 6-164 Anterior view of the right knee ligaments.

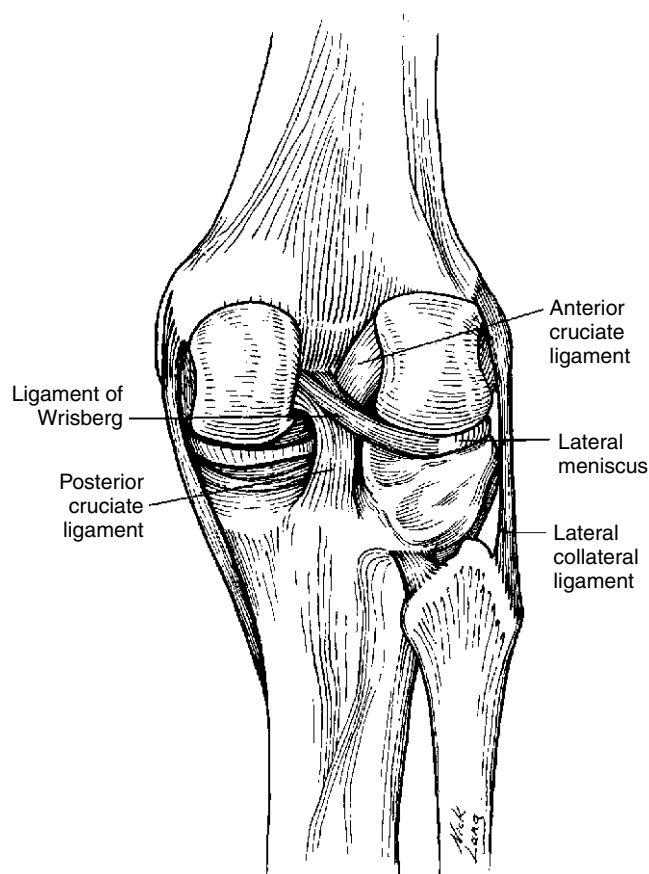


Figure 6-165 Posterior view of the right knee ligaments.

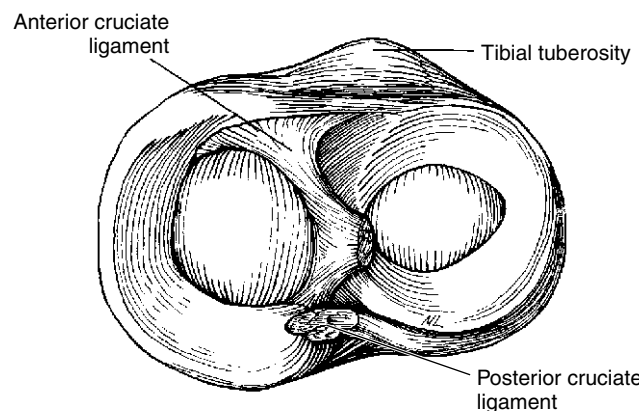


Figure 6-166 Superior aspect of the proximal right tibia, showing origin of the cruciate ligaments.

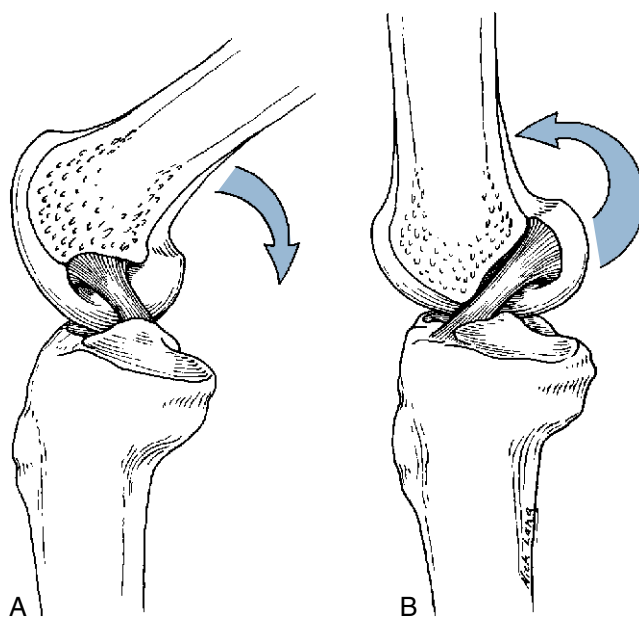


Figure 6-167 A, Anterior cruciate ligament becomes taut while in flexion. B, Posterior cruciate ligament becomes taut on knee extension.

condyle of the femur. The posterior cruciate ligament attaches from the posterior intercondylar eminence of the tibia, extending anteriorly and superiorly to the lateral side of the medial condyle of the femur. The anterior cruciate ligament resists anterior displacement of the tibia and checks extension movements. In contrast, the posterior cruciate ligament primarily checks posterior displacement of the tibia and resists internal rotation of the tibia. The posterior cruciate ligament, lying medial to the anterior cruciate ligament, is the strongest of the knee ligaments. It is especially important for providing M-L stability of the knee when in extension.

The collateral ligaments provide M-L stability and support for the knee while also preventing excessive external rotation of the tibia. The medial, or tibial, collateral ligament attaches from the medial epicondyle of the femur to the medial aspect of the shaft of the tibia. This ligament becomes taut on extension of the knee, abduction of the tibia on the femur, and external rotation of the tibia on the femur. The medial collateral ligament provides some

help in preventing anterior displacement of the tibia on the femur. The lateral, or fibular, collateral ligament attaches from the lateral epicondyle of the femur to the head of the fibula. This ligament becomes taut on extension, adduction, and external rotation of the tibia on the femur. The tendon of the biceps femoris almost completely covers the lateral collateral ligament, and the popliteus tendon runs beneath it and separates it from the meniscus.

Surrounding the external aspect of the joint is the fibrous joint capsule attaching at the margins of the articular cartilage. The inferior portion of the capsule has been referred to as the *coronary ligament*. A substantial thickening in the medial portion of the joint capsule has fibrous attachments to the periphery of the medial meniscus, thereby binding it firmly to the femur and loosely to the tibia. The lateral aspect of the joint capsule has a similar thickening and has fibrous attachments to the lateral meniscus.

The posterior fibers of the medial collateral ligament blend with the joint capsule and the attachments to the medial meniscus. The patellofemoral ligaments form as thickenings of the anterior joint capsule and extend from the middle of the patella to the medial and lateral femoral condyles. Their function is to stabilize the patella in the patellar groove. The posterior thickening of the joint capsule arches over the popliteus tendon, attaches to the base of the fibular head, and becomes taut in hyperextension. Also blending with the posterior joint capsule is the oblique popliteal ligament, which forms as an expansion of the semimembranosus tendon and runs obliquely in a superior and lateral direction to attach to the lateral femoral condyle. It also becomes tight in hyperextension.

Lying between the femur and the tibia are the two semilunar cartilages called *menisci* (Figure 6-168). The menisci are shaped so that the more peripheral portions are thicker than the central portion. This serves to deepen the articular surface on the tibial plateau and provide additional stability to the joint. Because they increase the surface area of the joint, the menisci help to share the load in weight-bearing across the joint by distributing weight over a broader area. They also aid in the lubrication and nutrition of the joint and, coupled with their shock-absorbing capabilities, help to decrease cartilage wear. The periphery of each meniscus attaches to the joint capsule, and the inner edge remains free. The medial meniscus is C-shaped, and the posterior portion is larger than the anterior. The anterior horn inserts on the intercondylar area of the tibia, and the posterior horn inserts just anterior to the attachment of the posterior cruciate ligament. The lateral meniscus is almost a complete circle, with the tips of each horn quite close to one another. The lateral meniscus is more mobile than the medial meniscus.

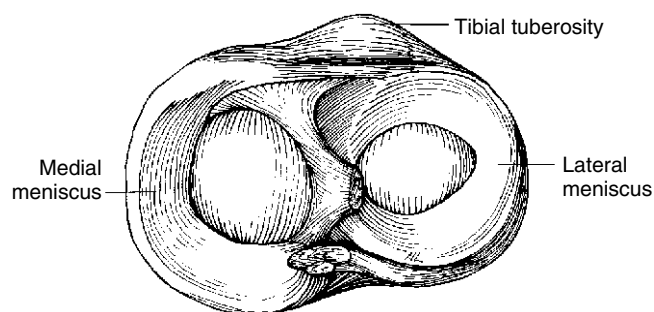


Figure 6-168 Menisci of the right knee.

TABLE 6-16 Actions of the Muscles of the Knee Joint

Action	Muscles
Extension	Quadriceps
Flexion	Hamstrings, gracilis, sartorius, tensor fascia lata, and popliteus
Internal rotation	Sartorius, gracilis, semitendinosus, semimembranosus, and popliteus
External rotation	Biceps femoris and tensor fascia lata (iliotibial tract)

Musculature

Stabilizing the knee are the many muscles that cross it (Table 6-16). Laterally, the iliotibial band attaches to the lateral condyle of the tibia, providing anterolateral reinforcement and stabilization against excessive internal rotation of the tibia on the femur. Crossing the anterior aspect of the knee is the quadriceps tendon. It is formed by the junction of the four heads of the quadriceps muscle, which consist of the vastus lateralis, the vastus medius, the vastus medialis, and the rectus femoris. The quadriceps musculature functions to extend the knee. Balanced activity between the vastus medialis and lateralis maintains optimal orientation of the patella within the patellofemoral groove. The sartorius and gracilis muscles provide medial stability to the joint. The sartorius also assists in knee and hip flexion, external rotation of the femur, and internal rotation of the tibia, depending on whether the extremity is weight-bearing. The gracilis acts to adduct the femur and assists in knee flexion and internal rotation of the tibia. Posteromedial reinforcement of the joint is supplied by the pes anserinus tendons (semitendinosus, gracilis, and sartorius), and the semimembranosus tendon. These help to prevent external rotation, abduction, and anterior displacement of the tibia. Posterolateral support from the biceps femoris tendon helps to check excessive internal rotation and anterior displacement of the tibia. The hamstring muscle is the primary knee flexor; the biceps femoris also provides some external rotation. Posterior reinforcement of the knee joint is provided by the gastrocnemius muscle and the popliteus muscle (Figure 6-169). The gastrocnemius is a primary ankle plantar flexor but also assists in knee flexion. The popliteus internally rotates and flexes the tibia when the limb is not bearing weight. The converse occurs when the limb is bearing weight.

Because the knee is exposed to a variety of demands in human locomotion, numerous bursae are located in relationship to the knee joint and to the synovial cavity. The synovial membrane of the knee joint is the most extensive of any in the body. The suprapatellar pouch, or quadriceps bursa, is actually an extension of the synovial sac that runs from the superior aspect of the patella upward beneath the quadriceps tendon and then folds back on itself to form a pouch inserting on the distal femur above the condyles (Figure 6-170). The prepatellar bursa is relatively large but superficial and lies between the skin and the patella. It can

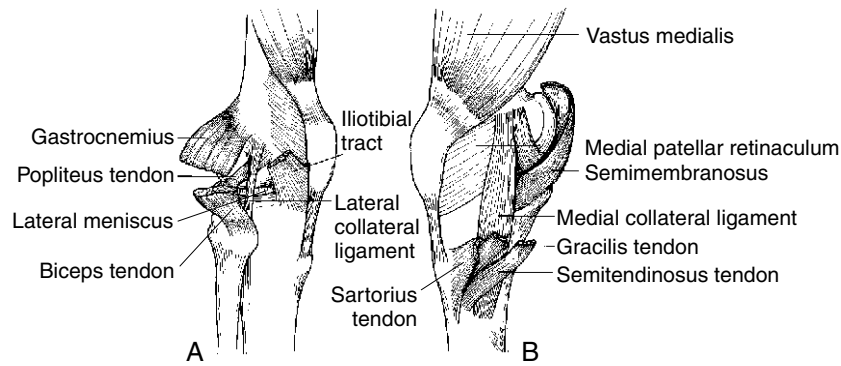


Figure 6-169 Lateral (A) and medial (B) aspects of the right knee, demonstrating muscular attachments.

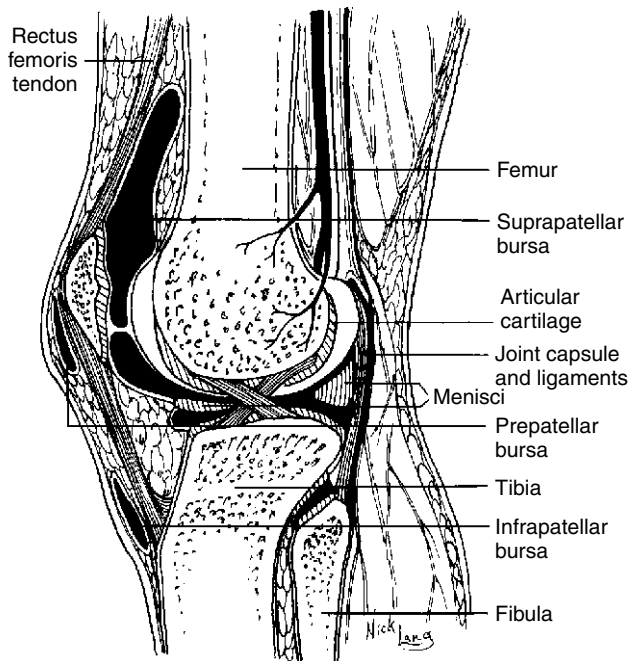


Figure 6-170 Sagittal section through the knee, demonstrating numerous bursae.

become inflamed with prolonged kneeling activities. The deep and superficial infrapatellar bursae lie just under and over the patellar tendon, respectively. Bursal sacs also lie between the semimembranosus tendon and the medial head of the gastrocnemius muscle, and two bursae separate the mediolateral heads of the gastrocnemius muscle from the joint capsule. (A Baker cyst occurs with effusion of the medial gastrocnemius and the semimembranosus bursae.) A bursa also lies under the pes anserine tendon, separating it from the tibial collateral ligament.

BIOMECHANICS

The knee joint must provide a broad ROM while maintaining its stability. It must react to rotational forces, as well as absorb shock, and then immediately prepare for propulsion. The knee functions as a modified hinge joint, with flexion and extension being its primary motions. Limited rotation occurs, especially when the joint is not in the closed-packed position (extension) (Box 6-12).

BOX 6-12 Close-Packed and Loose-Packed (Rest) Positions for the Knee Joint

CLOSE-PACKED POSITION

Full extension with full external rotation

LOOSE-PACKED POSITION

25 degrees flexion

Flexion and extension of the knee are combinations of roll, slide, and spin movements, which effectively shift the axis of movement posteriorly as the knee moves from extension into flexion (Figure 6-171). Similar to elbow flexion, knee flexion is limited by soft tissue of the calf and posterior thigh, and extension is limited by the locking of the joint from bony and soft tissue elements in the joint's close-packed position. Moreover, the so called *screw-home mechanism*, which is a combination of external rotation of the tibia occurring with knee extension, further approximates the osseous structures and tightens the ligamentous structures to stabilize the joint (Figure 6-172). The marked incongruent positions of the tibiofemoral joint are reduced by the fibrocartilaginous menisci. These also help to distribute the forces of compressive loading over a greater area and reduce compressive stresses to the joint surfaces of the knee. ROMs of the knee are listed in Table 6-17.

The patellofemoral joint plays an active role in flexion and extension of the knee joint. This joint has a gliding motion, moving caudal approximately 7 cm when going from full flexion to full extension. The articular surface of the patella never makes complete contact with the femoral condyles; the joint space decreases with flexion, and at full flexion, the patella sinks into the intracondylar groove (Figure 6-173). This characteristic is important in helping to increase weight-bearing capabilities in a flexed position (squatting). Therefore, the patella plays two important biomechanical roles for the knee. It primarily aids in knee extension by producing anterior displacement of the quadriceps tendon, thereby lengthening the lever arm of the quadriceps muscle force. In addition, it allows a wider distribution of compressive force on the femur, especially in a fully flexed position.

The patella has an optimal position in relationship to the knee joint in both the vertical and sagittal planes. On a lateral view of the knee, the length of the patella is compared with the

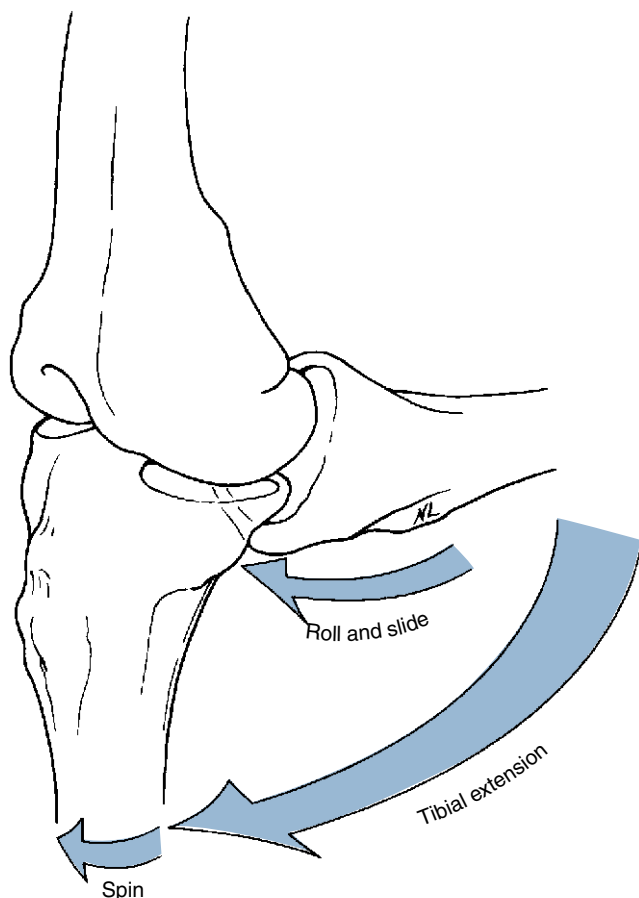


Figure 6-171 Flexion and extension movements are combinations of roll, slide, and spin.

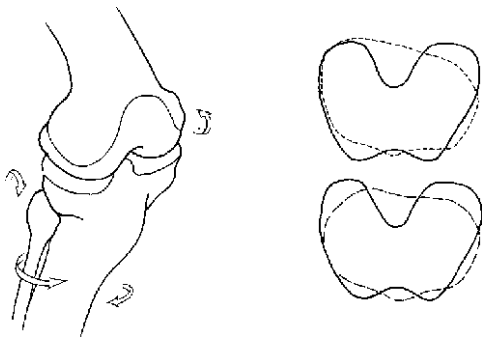


Figure 6-172 “Screw-home” mechanism of the knee, combining external rotation with extension, which maximally approximates the joint surfaces. (Modified from Nordin M, Frankel VH: *Basic biomechanics of the musculoskeletal system*, ed 2, Philadelphia, 1989, Lea & Febiger.)

distance from the inferior pole of the patella to the tibial tubercle (Figure 6-174). If the difference of these two numbers is greater than 1 cm, the position of the patella is either high (alta) or low (baja), depending on which distance is greater. To determine if the patella is aligned properly in the femoral groove, the Q angle can be determined. After locating the center of the patella, extend a line up the center of the patellar tendon through the center point. Then draw a line from the center of the patella toward the ASIS (Figure 6-175). The resultant angle is the Q angle. The normal

TABLE 6-17 Arthrokinematic and Osteokinematic Movements of the Knee Joint

Osteokinematic Movements	Degrees	Arthrokinematic Movements
Flexion	130	Roll and glide
Extension	10	Roll and glide
Internal rotation	10	Rotation
External rotation	10	Rotation

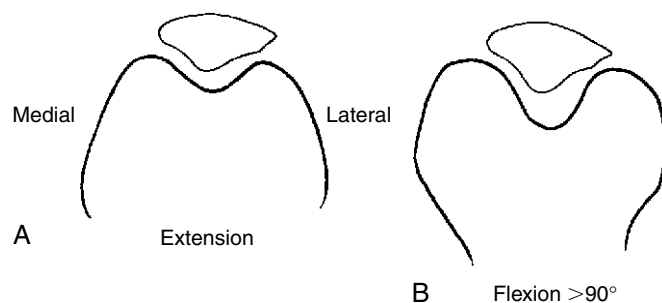


Figure 6-173 The relationship of the patella to the femur in extension (A) and flexion (B) of the knee.

Q angle is 10 to 15 degrees, being slightly greater in females. Muscle imbalance and rotational disrelationships of the tibia and femur produce changes in the Q angle.

The superior tibiofibular joint is mechanically linked to the ankle, but a dysfunctional process in this joint presents clinically as pain in and about the knee. Therefore, inclusion of it in a discussion of the knee is clinically relevant. The superior tibiofibular joint allows superior and inferior movement, as well as internal and external rotation of the fibula. During ankle dorsiflexion the fibula internally rotates and rises superiorly. The addition of ankle eversion causes some posterior displacement of the fibular head. Ankle plantar flexion draws the fibula inferiorly and creates external rotation. The addition of ankle inversion draws the fibular head anteriorly.

EVALUATION

The knee joint is affected by a wide variety of clinical syndromes. Trauma is by far the most common causative agent. Injuries to the collateral ligaments usually require some type of traumatic force. The mechanism of most medial collateral ligament injuries is a twisting external rotation strain while the knee is flexed or a valgus (abduction) blow to the knee. A lateral collateral ligament injury may occur with a varus (adduction) blow to the knee, and when internal rotation and hyperextension occur with this force, the fibular head may avulse.

The anterior cruciate ligament is commonly injured by forced internal rotation of the femur on a fixed tibia while the knee is abducted and flexed. The posterior cruciate ligament can be torn when a traumatic force is delivered to the front of the flexed tibia, driving it posteriorly under the femur. Forced external rotation of the femur on the tibia while the foot is fixed and the knee is abducted and flexed will also injure the posterior cruciate ligament.

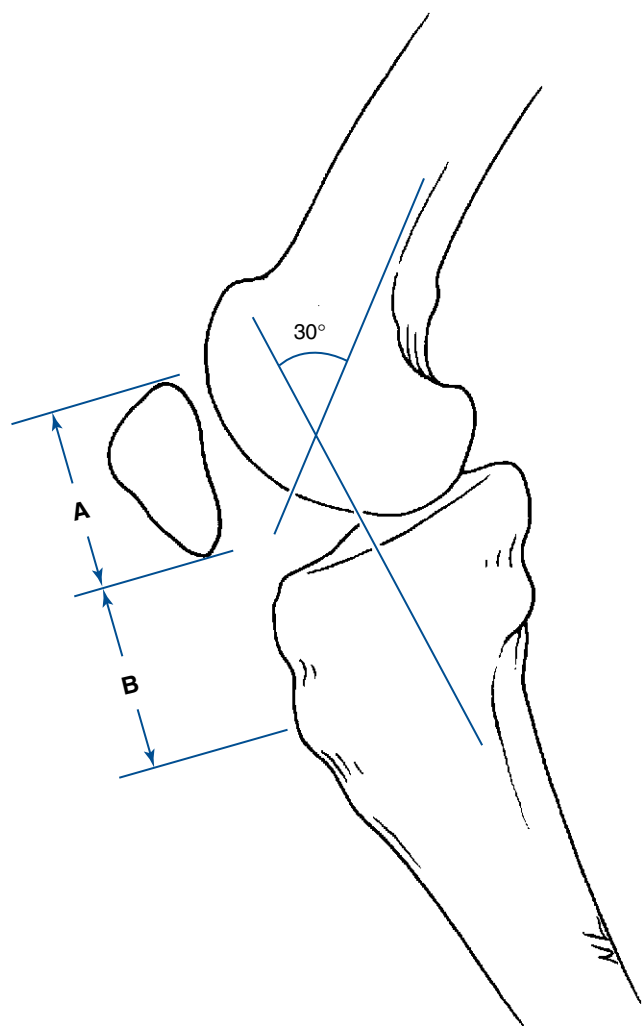


Figure 6-174 Identification of the position of the patella. If A–B is greater than 1 cm, a low (baja) patella exists; if B–A is greater than 1 cm, a high (alta) patella exists.

After traumatic injuries to the knee causing ligamentous injury, joint instabilities are likely. The knee can undergo translational or rotational instabilities. Orthopedic stress tests are performed to identify the presence of joint instability.

The menisci are another site of possible injury. A trauma that couples rotation or violent extension of the knee may cause an isolated longitudinal or transverse tear in the meniscus.

Problems involving the patellofemoral joint complex are common and may be more frequent than ligamentous or meniscus disorders. A patient complaining of vague aching pain about the knee that is aggravated by going up or down stairs likely has patellofemoral joint dysfunction. Patellar tracking problems can occur primarily from injuries to the knee and quadriceps mechanism or secondarily in response to problems affecting the ankle or hip. Chondromalacia patella, an erosion and fragmentation of the subpatellar cartilage, may be secondary to trauma, recurrent subluxation, pronated feet, postural instability, short leg syndrome, or excessive femoral torsion with resultant irregular Q angle.

An avulsion fracture with resulting aseptic necrosis of the tibial tuberosity may occur from a sudden contraction of the quadriceps

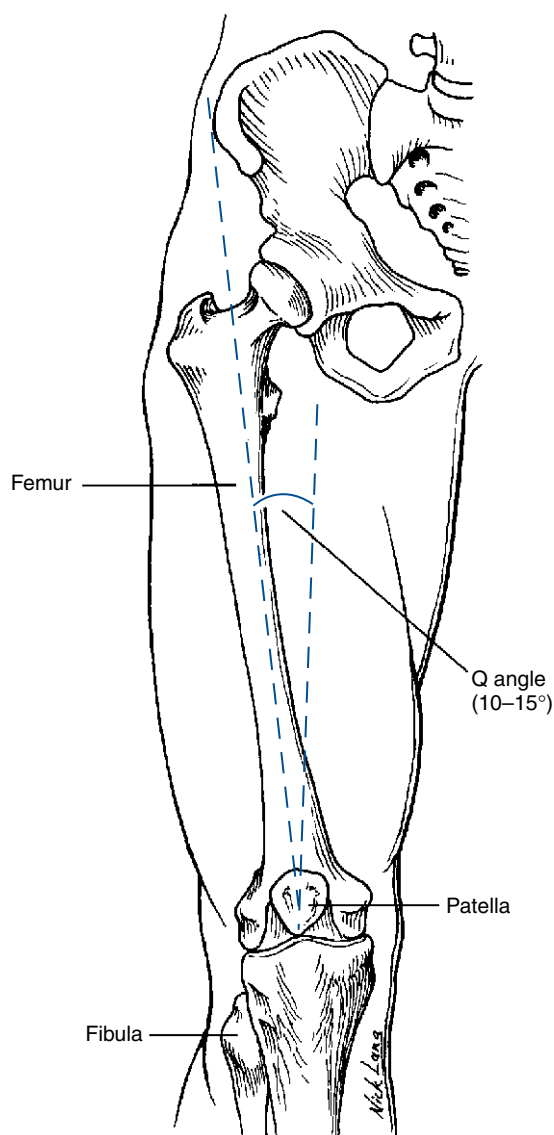


Figure 6-175 Q angle, the angle formed from the intersection of lines from the center of the patella to the anterior superior iliac spine and along the quadriceps tendon.

femoris. This condition is called *Osgood-Schlatter's disease* and is more common in boys.

The knee joint is innervated by segments L3–S1, and therefore in cases of pain of nontraumatic onset, lesions situated elsewhere in segments L3–S2 must be ruled out. The lumbar spine, hip, and foot are sources of referred pain to the knee (Figure 6-176).

To begin the evaluation of the knee, observe the knee for evidence of swelling, asymmetry of contours, and postural changes (valgus, varus, or recurvatum). Movements of the knee during gait should be smooth and rhythmic, with the knee bent during the swing phase and fully extended at heel strike.

Identify osseous symmetry and pain production through static palpation of the tibial plateau, tibial tubercle, adductor tubercle, femoral condyles, femoral epicondyles, fibular head, patella, and trochlear groove (Figure 6-177). Determine the Q angle.

Identify tone, texture, and tenderness changes through soft tissue palpation of the quadriceps muscle, the infrapatellar tendon,

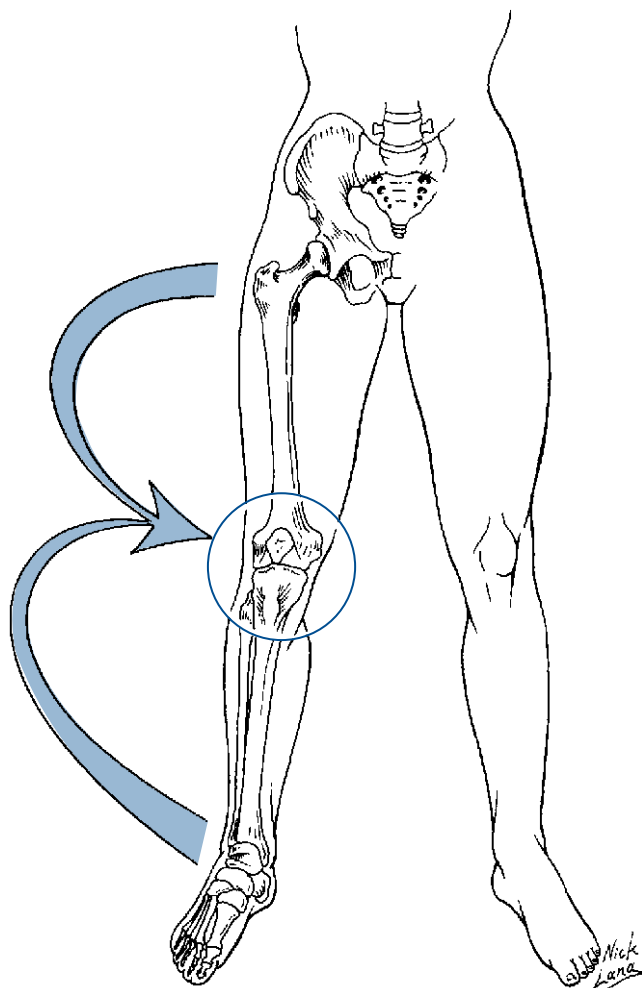


Figure 6-176 The ankle and hip can refer pain to the knee.

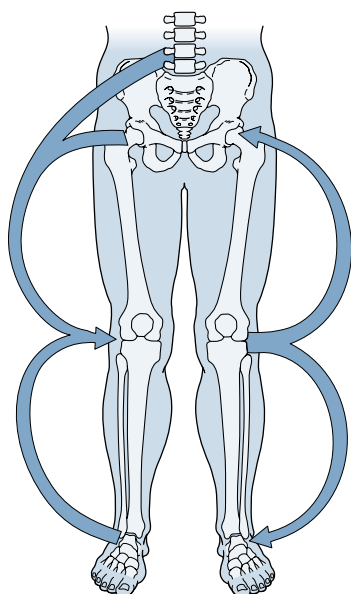


Figure 6-177 Patterns of referred pain to and from the knee. (Modified from Magee DJ: *Orthopedic physical assessment*, ed 5, St Louis, 2008, Saunders.)

TABLE 6-18 Accessory Joint Movements of the Knee Joint

Joint	Movement
Tibiofemoral	Long-axis distraction
	A-P glide
	P-A glide
	Internal rotation
	External rotation
	M-L glide
Patellofemoral	L-M glide
	S-I glide
	I-S glide
	L-M glide
Tibiofibular	M-L glide
	A-P glide
	P-A glide
	I-S glide
	S-I glide

A-P, Anterior-to-posterior; *I-S*, inferior-to-superior; *L-M*, lateral-to-medial; *M-L*, medial-to-lateral; *P-A*, posterior-to-anterior; *S-I*, superior-to-inferior.

the collateral ligaments, the pes anserine tendons, the peroneal nerve, the tibial nerve, the popliteal artery, the hamstring muscles, and the gastrocnemius and soleus muscles.

Evaluate accessory joint motions for the knee articulations to determine the presence of joint dysfunction (Table 6-18). Assess long-axis distraction with the patient supine and the affected leg slightly abducted. Stand and face the patient, straddling the affected leg so that your knees can grasp the patient's distal leg just proximal to the malleoli. Use both hands to palpate the knee joint at its medial and lateral aspects and use your legs to create a long-axis distraction while palpating with your hands for a springy end feel (Figure 6-178). Alternatively, one hand may stabilize the patient's femur on the table while the other hand palpates for end feel.

Evaluate A-P and P-A glide with the patient supine and the involved knee flexed to 90 degrees with the foot flat on the table. Either kneel or sit on the patient's foot for stability while grasping the proximal tibia with both hands. Stress the proximal tibia in an A-P and P-A direction, looking for a springing end feel (Figure 6-179).



6-178

Figure 6-178 Assessment of long-axis distraction of the left tibiofemoral joint.

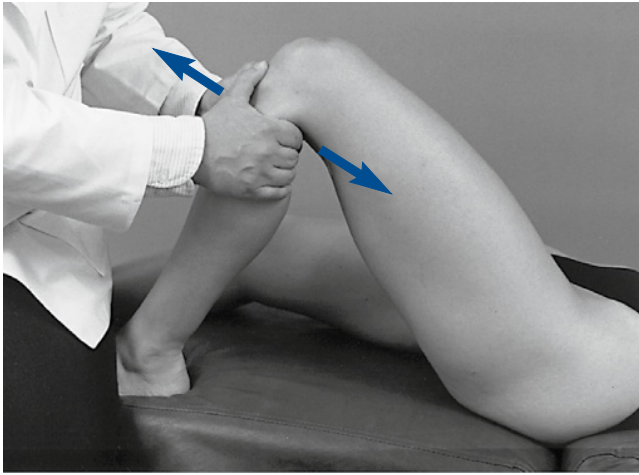


Figure 6-179 Assessment of anterior-to-posterior and posterior-to-anterior glide in flexion of the left tibiofemoral joint.

To evaluate internal and external rotation, use the same positions as for A-P and P-A glide. Stress the proximal tibia internally and externally to feel for a springing end feel (Figure 6-180).

Evaluate M-L and L-M glide with the patient supine and the involved leg abducted beyond the edge of the table. Then straddle the patient's involved leg just proximal to the ankle while grasping the proximal tibia with both hands. Apply an M-L and L-M stress to the knee joint to identify a springing end feel. Alternatively, grasp the patient's involved leg with the tibia held between your arm and body, with one hand on the tibia and one hand on the femur. The two hands can then create an M-L or L-M stress action (Figures 6-181 and 6-182).

Evaluate the patellofemoral articulation for M-L glide (Figure 6-183), L-M glide (Figure 6-184), S-I glide (Figure 6-185), and I-S glide (Figure 6-186), with the patient lying supine and the involved leg straight in passive knee extension. Contact the borders of the patella with both thumbs and apply a stress to the patella in M-L, L-M, S-I, and I-S directions, feeling for a comparative amount of movement from side to side, as well as a springing quality of movement.

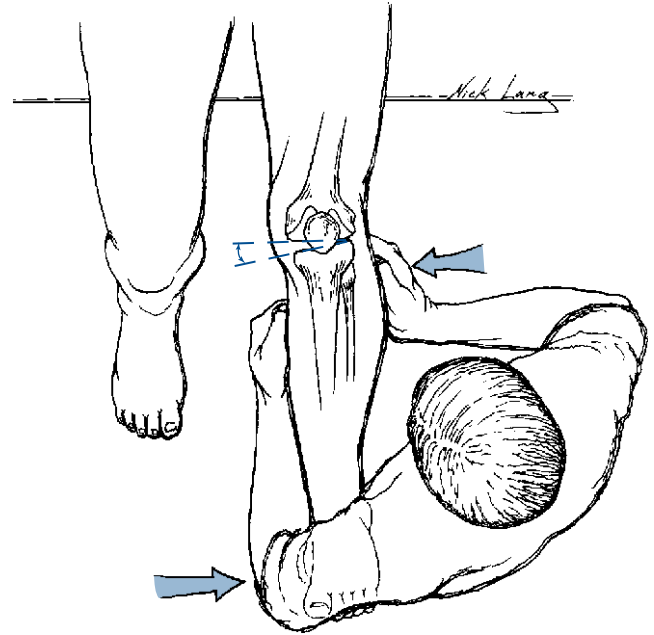


Figure 6-181 Lateral-to-medial glide of the left tibiofemoral joint.

Evaluate A-P and P-A glide of the tibiofibular articulation with the patient supine and the affected knee bent to 90 degrees and the foot flat on the table. Either kneel or sit on the patient's foot to stabilize it and grasp the proximal fibula with the outside hand while stabilizing the proximal tibia with the inside hand. Then stress the fibula in P-A and A-P directions, looking for a springing end feel (Figures 6-187 and 6-188).

Perform I-S and S-I glide of the tibiofibular articulation with the patient supine, the affected leg straight, and the knee in the relaxed extension. Use a digital contact of the cephalic hand to palpate the proximal fibula while grasping the patient's foot with your caudal hand. Then passively invert (with plantar flexion) and evert (with dorsiflexion) the patient's ankle while palpating for the fibula to move it superiorly and inferiorly (Figures 6-189 and 6-190).

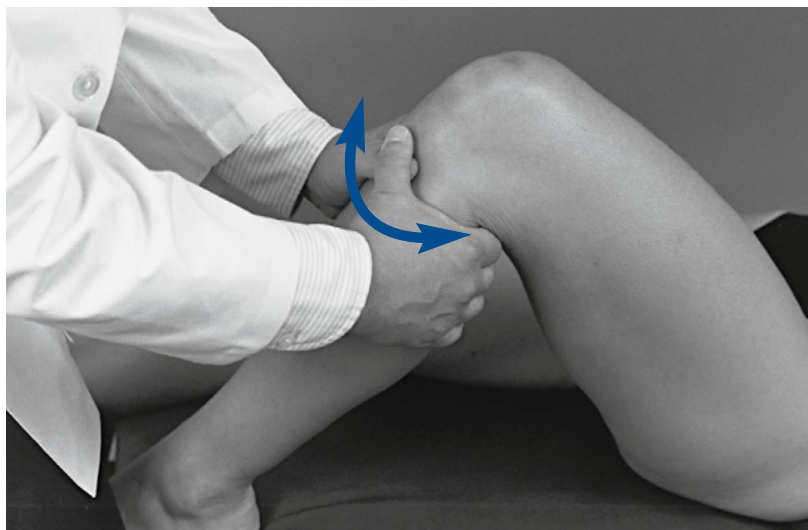


Figure 6-180 Assessment of external and internal rotation in flexion of the left tibiofemoral joint.



Figure 6-182 Assessment of medial-to-lateral glide of the left tibiofemoral joint.



Figure 6-183 Assessment of medial-to-lateral glide of the left patellofemoral joint.

ADJUSTIVE PROCEDURES

The manipulative techniques used to treat knee disorders aim to restore normal joint mechanics, which will then ideally allow full pain-free functioning of the knee joints. The three joints associated with the knee should be evaluated for characteristics of dysfunction when there are knee symptoms present. Box 6-13 identifies the adjustive procedures for the joints of the knee.

Femorotibial Joint

Supine:

Bimanual Grasp/Proximal Tibia with Knee Extension; Long-Axis Distraction (Figure 6-191)

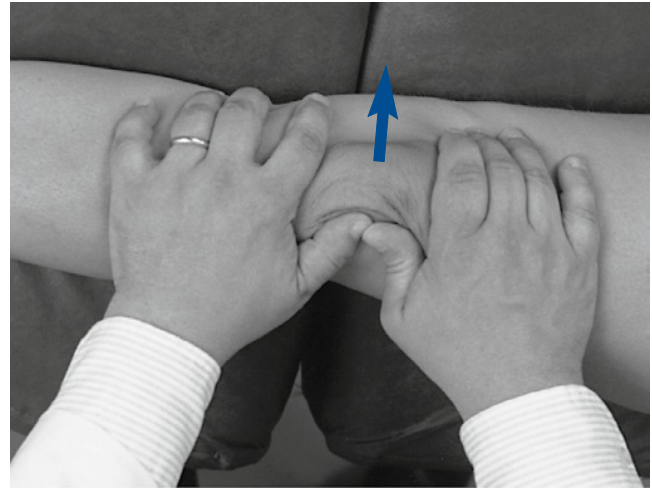


Figure 6-184 Assessment of lateral-to-medial glide of the left patellofemoral joint.

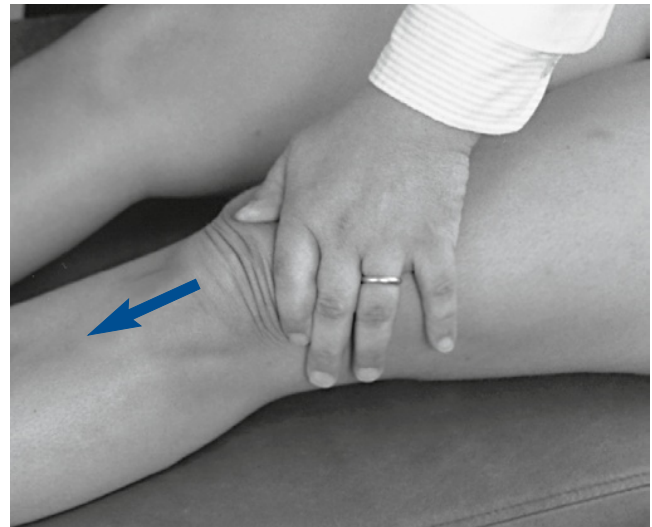


Figure 6-185 Assessment of superior-to-inferior glide of the left patellofemoral joint.



Figure 6-186 Assessment of inferior-to-superior glide of the left patellofemoral joint.



Figure 6-187 Assessment of posterior-to-anterior glide of the left tibiofibular joint.



Figure 6-188 Assessment of anterior-to-posterior glide of the left tibiofibular joint.

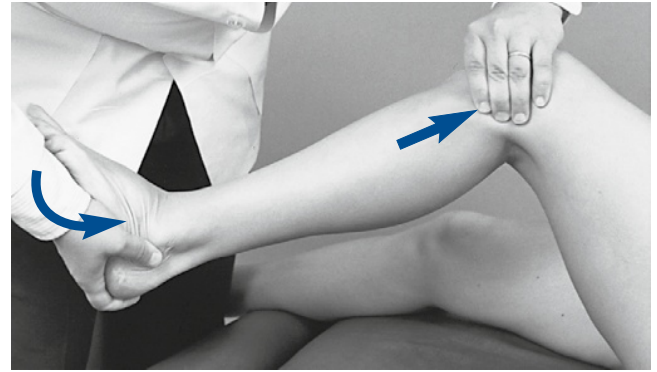


Figure 6-190 Assessment of inferior-to-superior glide of the left tibiofibular joint.

IND: Loss of long-axis distraction accessory movements of the femorotibial joint.

PP: The patient is supine, with the involved leg abducted beyond the edge of the adjusting table.

DP: Straddle the patient's involved leg, grasping the patient's distal tibia above the malleoli with your knees. If the adjusting table has an elevating pelvic piece, raise it to form a ledge for the patient's buttocks.

SCP: Proximal tibia.

CP: Use both hands to grasp the proximal tibia.

VEC: Long-axis distraction.

P: Straighten your knees while simultaneously using your hands to pull the proximal tibia into a long-axis distraction movement.

Reinforced Web/Proximal Tibia Push; Anterior-to-Posterior Glide in Flexion (Figure 6-192)

IND: Loss of A-P tibial glide, anterior misalignment of the proximal tibia.

PP: The patient is supine, with the affected leg flexed to 90 degrees at the hip and the knee.

DP: Stand on the affected side, with your caudal foot placed on the table so that the patient's affected leg will rest over your thigh.

SCP: Anterior aspect of the proximal tibia.

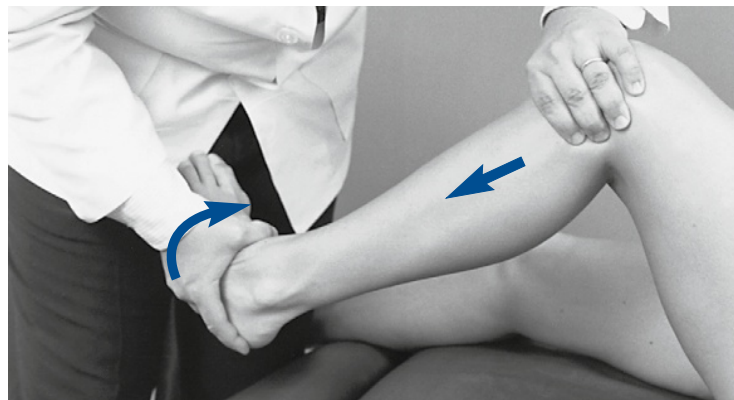


Figure 6-189 Assessment of superior-to-inferior glide of the left tibiofibular joint.

BOX 6-13 Knee Adjustive Techniques**FEMOROTIBIAL SUPINE:**

- Bimanual grasp/proximal tibia with knee extension; long-axis distraction (Figure 6-191)
- Reinforced web/proximal tibia push; anterior-to-posterior glide in flexion (Figure 6-192)
- Bimanual grasp/proximal tibia with knee extension; internal or external rotation in extension (Figure 6-193)
- Hypothenar/proximal tibia with leg stabilization; medial-to-lateral glide (Figure 6-194)
- Hypothenar/proximal tibia with leg stabilization; lateral-to-medial glide (Figure 6-195)

FEMOROTIBIAL PRONE:

- Reinforced mid-hypothenar (knife-edge) proximal tibia pull; posterior-to-anterior glide in flexion (Figure 6-196)
- Bimanual grasp/distal tibia with knee thigh stabilization; internal or external rotation in flexion (Figure 6-197)

PATELLOFEMORAL SUPINE:

- Bimanual web/patella; superior medial-to-inferior lateral glide; superior lateral-to-inferior medial glide; inferior medial-to-superior lateral glide; inferior lateral-to-superior medial glide (Figure 6-198)

TIBIOFIBULAR SUPINE:

- Index/proximal fibula, palmar ankle push; posterior-to-anterior glide in flexion (Figure 6-199)
- Reinforced thumbs/proximal fibula; anterior-to-posterior glide in flexion (Figure 6-200)

TIBIOFIBULAR PRONE:

- Reinforced mid-hypothenar (knife-edge)/proximal fibula pull; posterior-to-anterior glide in flexion (Figure 6-201)

TIBIOFIBULAR SIDE POSTURE:

- Reinforced mid-hypothenar (knife-edge)/proximal fibula push; inferior-to-superior glide in eversion (Figure 6-202)
- Reinforced mid-hypothenar (knife-edge)/proximal superior fibula push; superior-to-inferior to glide in inversion (Figure 6-203)

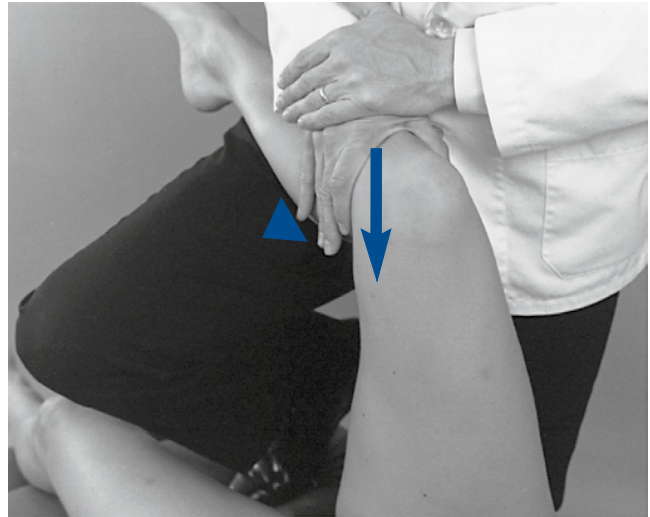


Figure 6-192 Adjustment for anterior-to-posterior glide of the right tibiofemoral joint.

CP: Use your cephalic hand to apply a web contact over the anterior aspect of the proximal tibia.

IH: With your caudal hand, place a knife-edge contact, reinforcing the contact hand.

VEC: A-P.

P: Use both hands to create an A-P impulse thrust on the proximal tibia.

Bimanual Grasp/Proximal Tibia with Knee Extension; Internal or External Rotation in Extension (Figure 6-193)

IND: Loss of rotational accessory movements of the tibia, internal or external misalignment of the proximal tibia.

PP: The patient is supine, with the affected leg abducted off the edge of the table.

DP: Stand and face the patient, straddling the affected leg and grasping the patient's ankle between the knees.

SCP: Proximal tibia.

CP: Using both hands, grasp the proximal tibia with the thumbs along the sides of the tibial tuberosity.

VEC: Rotation.

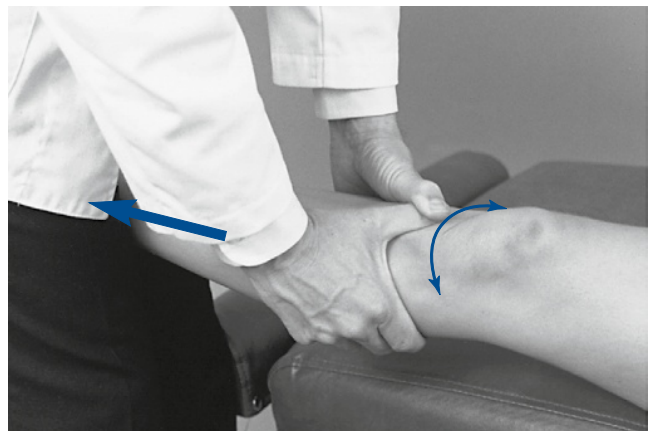


Figure 6-193 Adjustment for external and internal rotation of the left tibiofemoral joint in the supine position.



Figure 6-191 Adjustment for long-axis distraction of the left tibiofemoral joint.



Figure 6-194 Adjustment for medial-to-lateral glide of the left tibiofemoral joint.

P: Extend your knees and hop caudal to create some long-axis distraction while simultaneously twisting the proximal tibia, either internally or externally, with your hands.

Hypothenar/Proximal Medial Tibia with Leg Stabilization; Medial-to-Lateral Glide (Figure 6-194)

IND: Loss of M-L glide movement, medial misalignment of the proximal tibia.

PP: The patient is supine, with the involved hip flexed to approximately 45 degrees.

DP: Stand on the side opposite the involved leg, grasping the distal tibia with your caudal arm and axilla.

SCP: Medial aspect of the proximal tibia.

CP: With your cephalic hand, establish a pisiform-hypothenar contact on the medial aspect of the proximal tibia.

IH: Use your caudal arm to grasp the proximal tibia and hold it against your torso.

VEC: M-L.

P: Create articular tension by using your body on the distal tibia as a lever, pivoting against the contact hand. When all joint movement is removed, deliver an M-L impulse thrust.

Hypothenar/Proximal Lateral Tibia with Leg Stabilization; Lateral-to-Medial Glide (Figure 6-195)

IND: Loss of L-M tibial glide movement, lateral misalignment of the proximal tibia.

PP: The patient is supine, with the involved hip flexed to approximately 45 degrees.

DP: Stand on the involved side, with your caudal arm grasping the patient's distal tibia in the axilla.

SCP: Lateral aspect of the proximal tibia.



Figure 6-195 Adjustment for lateral-to-medial glide of the right tibiofemoral joint.

CP: Using your cephalic hand, apply a pisiform-hypothenar contact on the lateral aspect of the proximal tibia.

IH: Use your caudal arm to grasp the proximal tibia and hold it against your torso.

VEC: L-M.

P: Using your contact hand as a pivot, tilt the distal tibia with your indifferent arm to remove articular slack, and then use the contact hand to deliver an L-M impulse thrust to the proximal tibia.

Prone:

Reinforced Mid-Hypothenar (Knife-Edge)/Proximal Tibia Pull; Posterior-to-Anterior Glide in Flexion (Figure 6-196)

IND: Loss of P-A tibial glide, posterior misalignment of the proximal tibia.

PP: The patient is prone, with the involved leg flexed to just less than 90 degrees.

DP: Stand at the foot end of the table and bend over so that the patient's dorsum of the foot can rest on your inside shoulder.

SCP: Posterior aspect of the proximal tibia.

CP: With your inside hand, establish a knife-edge contact on the posterior aspect of the proximal tibia.



6-196

Figure 6-196 Adjustment for posterior-to-anterior glide of the left tibiofemoral joint.

IH: Using your outside hand, reinforce the contact hand.

VEC: P-A.

P: Use both hands to remove articular slack and thrust in the long axis of the femur, creating a P-A glide of the proximal tibia.

Bimanual Grasp/Distal Tibia with Knee Thigh Stabilization; Internal or External Rotation in Flexion (Figure 6-197)

IND: Loss of rotational accessory movements of the tibia, internal or external rotation misalignment of the proximal tibia.

PP: The patient is prone, with the affected leg flexed to just less than 90 degrees.

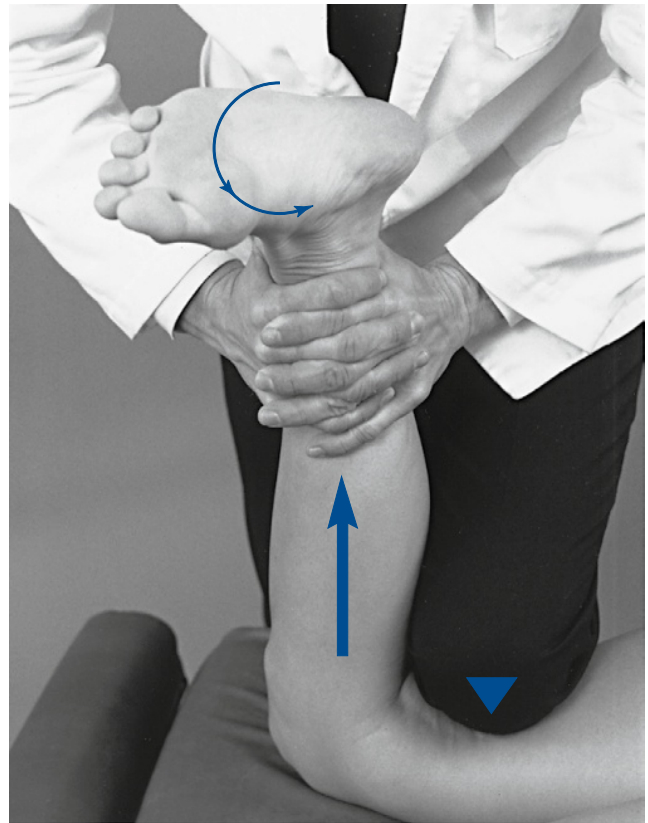
DP: Stand at the side of the table on the affected side, placing your cephalic proximal tibia on the patient's distal femur.

SCP: Distal tibia.

CP: Using both hands, grasp the patient's distal tibia with your fingers interlaced.

VEC: Rotation.

P: Stabilize the patient's thigh on the table, use both hands to apply long-axis distraction, and then impart an impulse thrust, creating internal or external rotation of the tibia.



6-197

Figure 6-197 Adjustment for external rotation of the left tibiofemoral joint in the prone position.

Patellofemoral Joint

Prone:

Bimanual Web/Patella; Superior Medial-to-Inferior Lateral Glide; Superior Lateral-to-Inferior Medial Glide; Inferior Medial-to-Superior Lateral Glide; Inferior Lateral-to-Superior Medial Glide (Figure 6-198)

IND: Patellar tracking problems, restricted patellar movements.

PP: The patient is supine, with the involved knee in relaxed extension.

DP: Stand on the involved side.

SCP: Patella.

CP: Use both hands to apply thumb web contacts over the borders of the patella.

VEC: Inferolateral, inferomedial, superolateral, and superomedial.

P: Depending on the direction of dysfunctional movement and involved soft tissues, give a thrust to the patella in a down-and-in, down-and-out, up-and-in, or up-and-out direction.

Tibiofibular Joint

Supine:

Index/Proximal Fibula, Palmar Ankle Push

IND: Loss of P-A fibular movement, posterior misalignment of the fibula. (Figure 6-199)

PP: The patient is supine, with the involved leg flexed at the hip and knee.



6-198

Figure 6-198 Adjustment for superior and medial (A) or inferior and lateral (B) glide of the right patellofemoral joint.



6-199

Figure 6-199 Adjustment for P-A glide of the left tibiofibular joint in the supine position.

DP: Stand on the involved side.

SCP: Posterior aspect of the proximal fibula.

CP: Using your superior hand, apply the palmar aspect of the index contact on the posterior aspect of the proximal fibula.

IH: With your caudal hand, grasp the distal tibia.

VEC: P-A.

P: Use your IH to flex the leg, pushing the heel toward the buttock, while giving a lifting motion with the contact hand, creating P-A movement of the proximal fibula.

Reinforced Thumbs/Proximal Fibula; Anterior-to-Posterior Glide in Flexion (Figure 6-200)

IND: Loss of P-A fibular movement, anterior misalignment of the fibula.

PP: The patient is supine, with the affected knee bent to 90 degrees and the foot flat on the table.

DP: Either kneel or sit on the patient's foot to stabilize it.

SCP: Anterior aspect of the proximal fibula.

CP: With your outside hand, establish a thumb or pisiform-hypothenar contact over the anterior aspect of the proximal fibula.

IH: With your inside hand, grasp the proximal tibia and reinforce your contact.

VEC: A-P.

P: Using your IH, stabilize the tibia while using the contact hand to deliver an A-P impulse thrust to the proximal fibula.



6-200

Figure 6-200 Adjustment for anterior-to-posterior glide of the left tibiofibular joint.



6-201

Figure 6-201 Adjustment for posterior-to-anterior glide of the left tibiofibular joint in the prone position.

Prone:

Reinforced Mid-Hypothenar (Knife-Edge)/Proximal Fibula Pull; Posterior-to-Anterior Glide in Flexion (Figure 6-201)

IND: Loss of P-A fibular movement, posterior misalignment of the fibula.

PP: The patient is prone, with the knee flexed to just less than 90 degrees.

DP: Stand at the foot end of the table, flexed at the waist so the dorsum of the patient's foot can rest on your inside shoulder.

SCP: Posterior aspect of the proximal fibula.

CP: With your outside hand, establish a pisiform-hypothenar contact on the posterior aspect of the proximal fibula.

IH: Use your inside hand to reinforce the contact.

VEC: P-A.

P: Using both hands, deliver a P-A impulse thrust to the proximal fibula.

Side Posture:

Reinforced Mid-Hypothenar (Knife-Edge)/Proximal Fibula Push; Inferior-to-Superior Glide in Eversion (Figure 6-202)



6-202

Figure 6-202 Adjustment for inferior-to-superior glide of the left tibiofibular joint.

IND: Loss of I-S fibular movement, inferior misalignment of the fibula.

PP: The patient is side-lying, with the affected leg up and positioned anterior to the unaffected side. Both knees should be slightly flexed.

DP: Stand at the foot end of the table so that the patient's involved foot rests against your thigh, maintaining the patient's ankle in eversion.

SCP: Lateral and inferior aspect of the proximal fibula.

CP: Using your anterior hand, establish a knife-edge contact on the inferior aspect of the proximal fibular head.

IH: With your posterior hand, reinforce the contact hand.

VEC: I-S.

P: While maintaining the ankle in eversion, deliver a thrust to the proximal fibula in an I-S direction. This movement can be achieved in a similar fashion by placing a thenar contact under the inferior aspect of the lateral malleolus, reinforcing the contact with the other hand, and delivering an I-S thrust.

Reinforced Mid-Hypothenar (Knife-Edge)/Proximal Superior Fibula Push; Superior-to-Inferior to Glide in Inversion (Figure 6-203)

IND: Loss of S-I fibular movement, superior misalignment of the fibula.

PP: The patient is side-lying, with the involved side up and involved leg resting on the table behind the uninvolved leg so that the ankle of the involved leg can hang in inversion off the edge of the adjusting table.

DP: Stand at the side of the table behind the patient, facing caudal.

SCP: Posterosuperior aspect of the proximal fibula.

CP: With your outside hand, establish a knife-edge contact over the posterosuperior aspect of the proximal fibula.

IH: Using your inside hand, reinforce the contact hand.

VEC: S-I.

P: Using both hands, apply an S-I impulse thrust to the fibular head.

ANKLE AND FOOT

The ankle and foot can be discussed together because they are intimate components of a very intricately functioning unit. Together they make up a significant component in a kinetic chain responsible for propulsion and balance. These joints may be



6-203

Figure 6-203 Adjustment for superior-to-inferior glide of the left tibiofibular joint.

viewed as initial supports for the musculoskeletal frame, because they form the base on which all other osseous and muscular mechanisms reside. To the contrary, these joints may also be viewed as a terminal segment, in that they must translate and carry out the messages from the central nervous system through the hip and knee. This joint complex must attenuate weight-bearing forces, support and propel the body, and maintain equilibrium.¹⁵ The feet and ankles must therefore provide the two paradoxical qualities of stability and pliability. They achieve these requirements through an interaction of interrelated joints, connective tissues, and muscles. Certainly, this part of the lower extremity is subject to a multiplicity of traumatic and postural disorders, leading to numerous joint dysfunction syndromes.

FUNCTIONAL ANATOMY

Osseous Structures

The distal tibia and fibula join with the talus to form a mortise-type hinge joint called the *talocrural joint*. The calcaneus, the largest tarsal bone, articulates with the talus, forming the subtalar joint. The navicular articulates with the talus proximally and cuneiforms distally. The cuboid articulates with the calcaneus proximally and

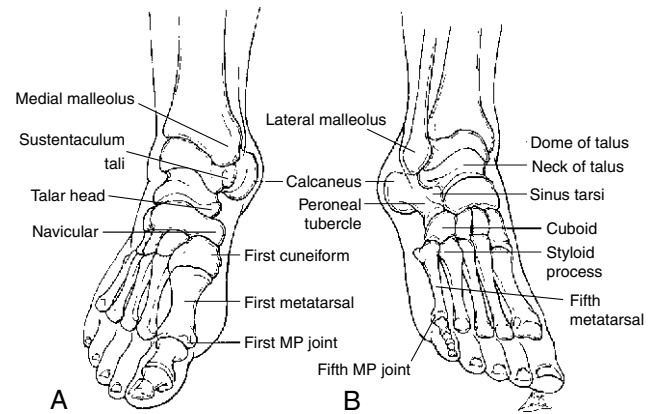


Figure 6-204 Osseous structures of the right foot and ankle. **A**, Medial aspect. **B**, Lateral aspect.

with the fourth and fifth metatarsals distally (Figure 6-204). It also articulates with the navicular and third cuneiform medially. The first, or medial, cuneiform articulates with the first metatarsal; the second, or intermediate, cuneiform articulates with the second metatarsal; and the third, or lateral, cuneiform articulates with the third metatarsal. Two phalanges complete the structure of the great toe, and three phalanges complete the bony structures of each of the other four toes. The function of the tibia is to transmit most of the body weight to and from the foot, and although the fibula plays a very important role in ankle stability, it is not directly involved in the transmission of weight-bearing forces. Functionally, the talus serves as a link between the leg and the foot.

Ligamentous Structures

Although many ligaments and joint capsules are associated with the foot and the ankle, some are more important to localize, palpate, and functionally understand (Figures 6-205, 6-206 to 6-207). The deltoid ligament, composed of four parts, provides medial stability to the ankle by attaching from the medial malleolus to the talus anteriorly and posteriorly, as well as to the navicular and calcaneus. Laterally, the ankle is secondarily stabilized by five fibular ligaments: the anterior and posterior tibiofibular ligaments,

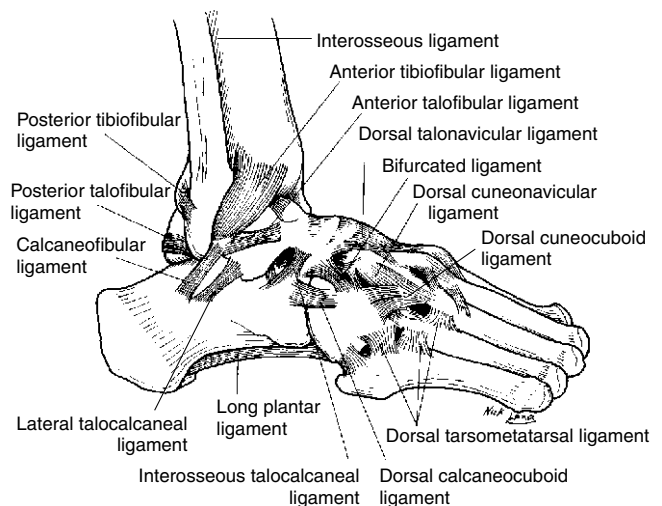


Figure 6-205 Ligaments on the lateral aspect of the right ankle.

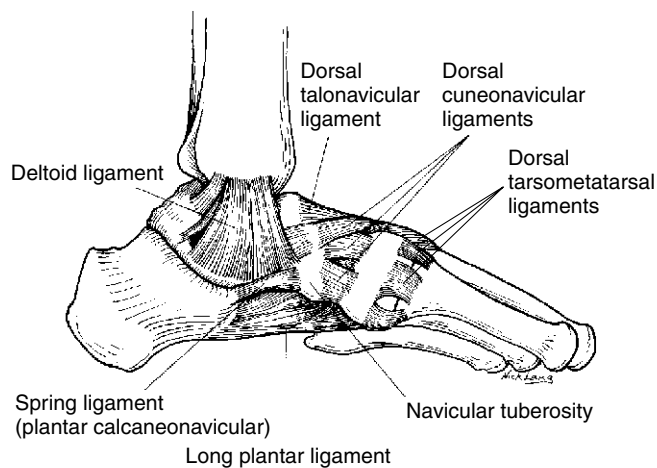


Figure 6-206 Ligaments on the medial aspect of the left ankle.

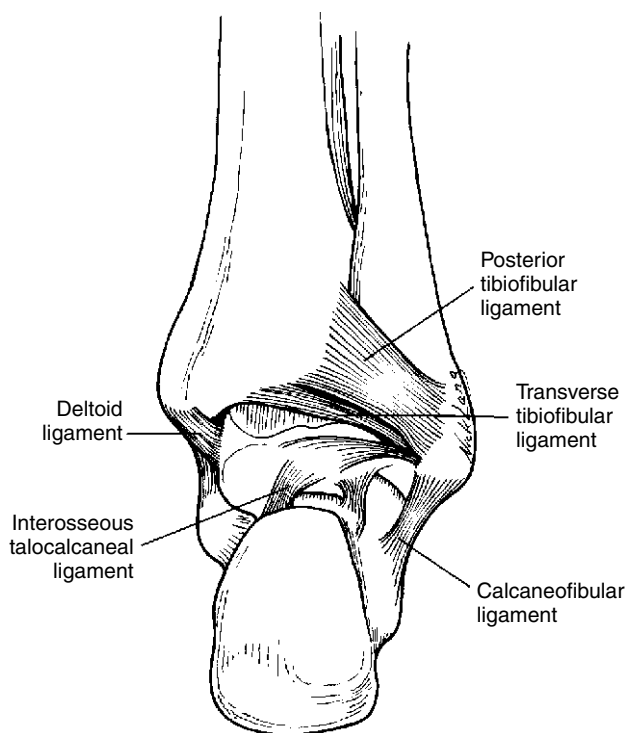


Figure 6-207 Ligaments on the posterior aspect of the right ankle.

the anterior and posterior talofibular ligaments, and the calcaneofibular ligament. The plantar calcaneonavicular (spring) ligament attaches from the sustentaculum tali to the navicular (Figure 6-208). The function of this ligament is to keep the medial aspect of the forefoot and hindfoot in apposition and, in so doing, help to maintain the arched configuration of the foot.

Musculature

Similar to the way the muscles of the wrist are located in the arm, the muscles of the ankle are located in the calf. Posteriorly, the large calf muscle group (gastrocnemius and soleus) attaches from the femoral condyles, proximal fibula, and tibia to the calcaneus, providing plantar flexion of the ankle. The tendon of the tibialis posterior passes under the medial malleolus to attach to the plantar surfaces of the navicular; cuneiforms; the cuboid; and the

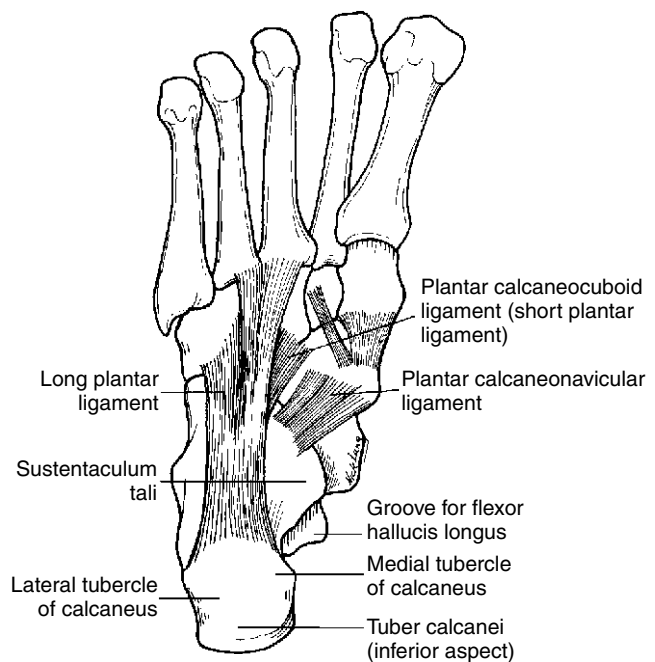


Figure 6-208 Ligaments on the plantar aspect of the left foot.

second, third, and fourth metatarsals. As such, it serves primarily as an inverter and adductor, or supinator, of the foot. The flexor hallucis longus and flexor digitorum longus muscles also have tendons that pass under the medial malleolus, with each inserting on the distal phalanx of each toe, thereby creating flexion of the toes. Anteriorly are the extensor digitorum longus, tibialis anterior, and extensor hallucis longus muscles. The extensor digitorum longus attaches to the dorsal surfaces of each of the distal phalanges, primarily extending the four toes, but also serving to dorsiflex, evert, and abduct the foot. The extensor hallucis longus is the primary extensor of the big toe. The tendon of the tibialis anterior passes over the ankle joint and across the medial side of the dorsum of the foot, and inserts into the medial and plantar surface of the medial cuneiform bone and the base of the first metatarsal. It functions as the primary dorsiflexor of the ankle, but because of its insertion, it also inverts and adducts the foot. On the lateral aspect is the peroneus muscle group. The tendons of the peroneus longus and brevis pass under the lateral malleolus and cross the cuboid to insert into the medial cuneiform and base of the first metatarsal. The chief action, then, of both peronei muscles is to evert the foot. The peroneus tertius is continuous, with the origin of the extensor digitorum longus muscle; its tendon diverges laterally to insert into the dorsal surface of the base of the fifth metatarsal bone. It works with the extensor digitorum longus to dorsiflex, evert, and abduct the foot.

The ankle musculature can be divided into positional groups and divided according to the actions they perform (Table 6-19). The gastrocnemius, soleus, and plantaris muscles lie posteriorly and are responsible for plantar flexion of the foot and ankle. The extensor hallucis longus, extensor digitorum longus, peroneus tertius, and tibialis anterior muscles lie anteriorly and serve primarily to dorsiflex the foot and extend the toes. The peroneus longus and brevis muscles are situated laterally and pronate and evert the foot. The tibialis posterior, flexor digitorum longus,

TABLE 6-19 Actions of the Muscles of the Foot and Ankle

Action	Muscles
Ankle plantar flexion	Gastrocnemius, soleus, and plantaris
Ankle dorsiflexion	Extensor digitorum longus, tibialis anterior, peroneus tertius, and extensor hallucis longus
Ankle inversion (adduction)	Tibialis posterior and tibialis anterior
Ankle eversion (abduction)	Extensor digitorum longus, peroneus longus and brevis, and peroneus tertius
Toe flexion	Flexor digitorum longus and flexor hallucis longus
Toe extension	Extensor digitorum longus and extensor hallucis longus

and flexor hallucis longus muscles are medial and function to invert the foot and flex the toes. The intrinsic muscles of the foot lie in layers and generally perform the actions indicated by the muscle names.

BIOMECHANICS

The functional biomechanics of the foot and ankle must include the ability to bear weight and allow flexible locomotion. The ankle joint is a uniplanar hinge joint, with talus motion occurring primarily in the sagittal plane about a transverse axis. The lateral side of the transverse axis is skewed posteriorly from the frontal plane (Figure 6-209). The primary movement at the ankle mortise is dorsiflexion (20 to 30 degrees) and plantar flexion (30 to 50 degrees) (Table 6-20). Through the normal gait pattern, however, only 10 degrees of dorsiflexion and 20 degrees of plantar flexion are required.²⁴

The subtalar joint formed between the talus and calcaneus is also a hingelike joint. The axis of movement passes through all three cardinal planes of movement, thereby allowing movement to some extent in all three planes. Movement of this joint, then, includes the complex movement of supination and pronation of the calcaneus under the talus. Supination is a combination of inversion, adduction, and plantar flexion, whereas pronation is a combination of eversion, abduction, and dorsiflexion (Figure 6-210). The subtalar joint has the important function of absorbing shock at heel strike and rotating the lower extremity in the transverse plane during the stance phase of gait.

The transverse or midtarsal joint is composed of the talonavicular and calcaneal cuboid articulations. The amount of movement occurring at these joints, which function in unison, depends on whether the foot is in pronation or supination. The supinated position creates a divergence of the axes of movement, which decreases the amount of movement, setting up a rigid

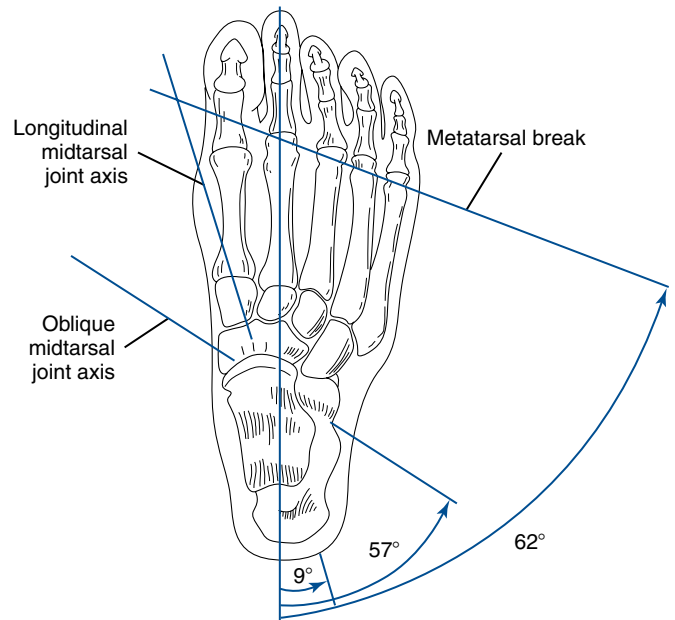


Figure 6-209 Dorsiflexion and plantar flexion in the foot and ankle occur around an oblique axis, and eversion and inversion occur around the longitudinal axis. (Modified from Wadsworth CT: *Manual examination and treatment of the spine and extremities*, Baltimore, 1988, Williams & Wilkins.)

TABLE 6-20 Arthrokinematic and Osteokinematic Movements of the Ankle and Foot Joints

Osteokinematic Movements	Degrees	Arthrokinematic Movements
Ankle dorsiflexion	20	Roll and glide
Ankle plantar flexion	50	Roll and glide
Subtalar inversion	5	Roll and glide
Subtalar eversion	5	Roll and glide
Forefoot abduction	10	Roll and glide
Forefoot adduction	10	Roll and glide

bony structure necessary for stability at the heel-strike stage of gait. Pronation, on the other hand, allows for the axes of motion to become aligned, which allows for increased mobility and decreased stability. The ligaments that run on the plantar surface between these tarsal bones are an essential component for absorbing stress and maintaining the longitudinal arch. Table 6-21 identifies the close-packed and loose-packed positions for the ankle and toe joints.

The individual tarsal joints, metatarsal-tarsal joints, metatarsophalangeal joints, and interphalangeal joints enhance the foot's stability or flexibility. They must provide a base for the stance phase, as well as the necessary hinges for flexion and extension during toe-off. Ligaments and tendons further enhance stability and flexibility by maintaining arches in the foot (Figure 6-211).

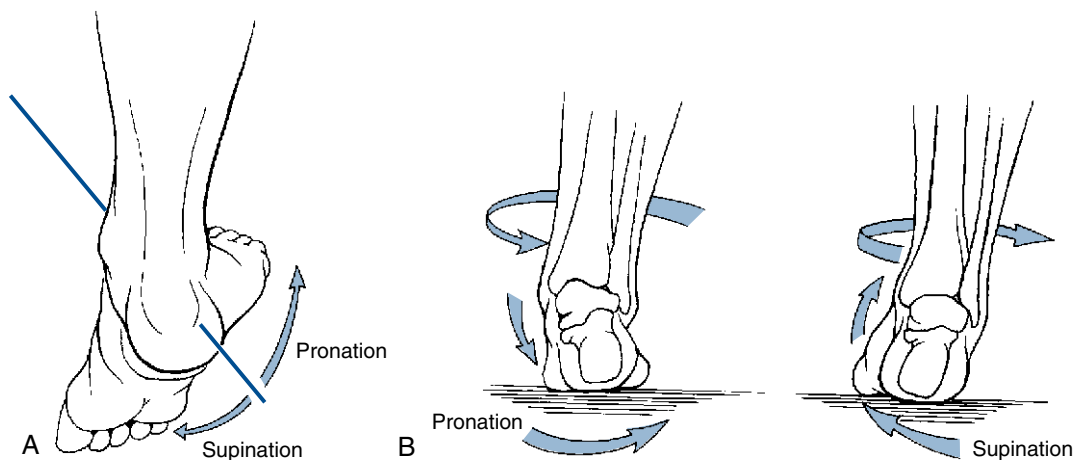


Figure 6-210 Pronation and supination movements in free swing action (open kinetic chain) (A) and with weight-bearing (closed kinetic chain) (B).

TABLE 6-21 Close-Packed and Loose-Packed (Rest) Positions for the Ankle and Foot Joints

Joint	Close-Packed Position	Loose-Packed Position
Tibiotalar articulation	Full dorsiflexion	10 degrees of plantar flexion midway between full inversion and eversion
Toes	Full extension	Flexion

During the gait cycle, two main phases are described. The first occurs when the foot is on the ground and is called the *stance phase*, and the second occurs when the foot is not contacting the ground and is called the *swing phase* (Figure 6-212). The stance phase is further divided into a contact component, mid-stance component, and a propulsive component. Movements

of the leg and foot change through the different components. Pronation of the subtalar joint occurs initially at heel strike while the tibia internally rotates. This is followed by supination of the subtalar joint and external rotation of the tibia through mid-stance and propulsive stages, as well as through the swing phase of gait. This creates a shifting area of weight-bearing across the foot, starting from the posterolateral aspect of the calcaneus and curving over to the first metatarsophalangeal joint. Abnormal supination or pronation of the subtalar joint will result in altered gait patterns, as well as weight-bearing stresses on the plantar surface of the foot.

EVALUATION

With the concentrated stress that occurs to the foot and ankle during bipedal static and dynamic postures, these areas are susceptible to many injuries. Commonly, ankle injuries have an acute traumatic onset, whereas the foot is more likely to develop chronic and insidious onset disorders from stress overload. Pain and paresthesias arising from the lower lumbar or first sacral NRs should not be overlooked (Figure 6-213). Most foot and ankle pain, however, arises from local disease or pathomechanic processes.

The most common traumatic injury to this area is the inversion sprain of the ankle, causing a separation to the lateral compartment with damage to the anterior talofibular ligament and possibly to the calcaneofibular ligament. Rarely does inversion occur alone, because usually plantar flexion of the ankle, as well as external rotation of the leg, also occur.

An eversion sprain involving trauma to the medial aspect of the ankle and affecting the deltoid ligament usually occurs when the foot is fixed in an excessive amount of pronation and the individual turns forcefully toward the opposite foot. The stress is applied first to the anterior tibiofibular ligament.

Shin splints refer to a generalized, deep aching or, sometimes, sharp pain along the tibia. It is considered an overuse or abuse syndrome occurring commonly because of running or jumping on a hard surface. This activity causes the talus to be driven upward into the mortise, forcing the tibia and fibula to separate. Stress to the interosseous membrane results and may cause a periostitis.

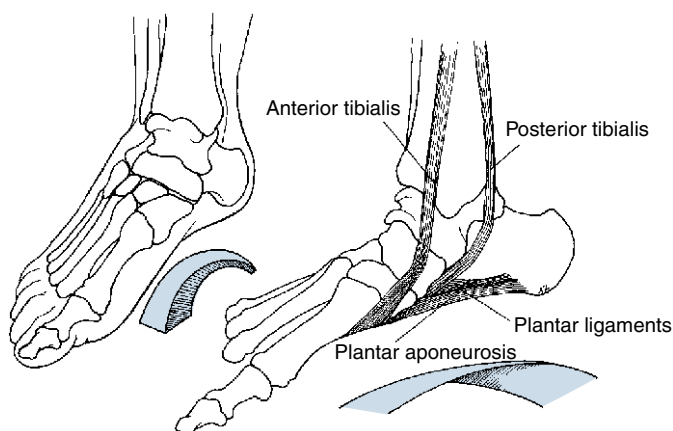


Figure 6-211 The longitudinal arch of the right foot formed by the tibialis anterior and posterior.

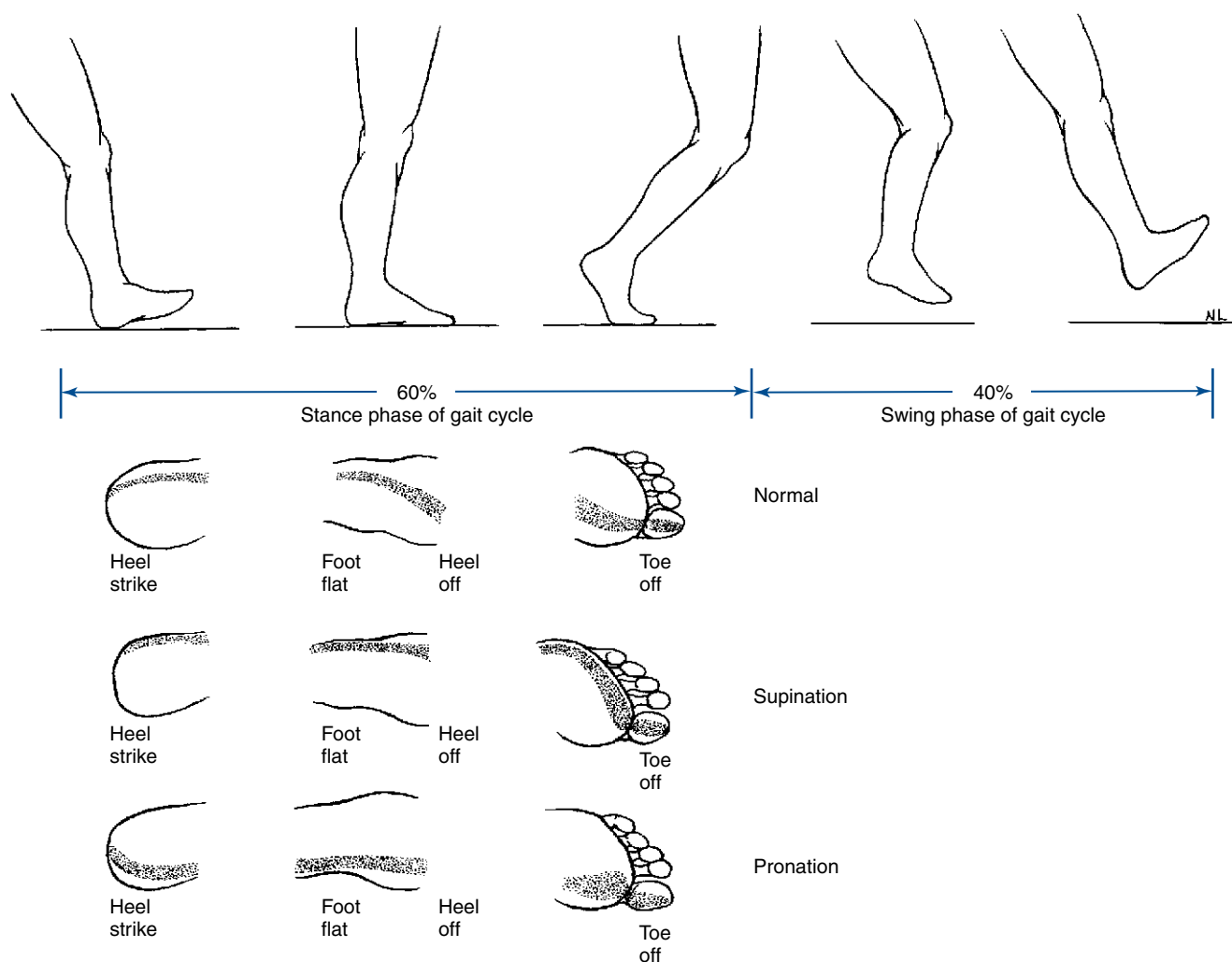


Figure 6-212 Gait pattern demonstrating that 60% of the gait is the stance phase, and 40% is the swing phase. Also shown is the pattern of weight-bearing on the plantar surface of the foot normally and in pronation and supination.

Furthermore, activity of the anterior tibialis may result in fluid becoming entrapped within the fascial covering, creating a compartment syndrome.

Plantar fasciitis results as a strain to the plantar fascia on the sole of the foot. This may be a result of standing on hard surfaces, quick acceleration or deceleration, repeated shocks, standing on ladders, or long periods of pronation. A calcaneal heel spur may eventually occur as the fasciitis continues or worsens. The fascia will pull the periosteum off the calcaneus, creating a painful periostitis, and bone will be laid down at the site of stress.

Hallux valgus is a lateral deviation of the big toe, usually with a concomitant metatarsal varum. Improperly fitting footwear, as well as an unstable and pronated foot, has been blamed for this condition.

The evaluation of the foot and ankle begins with observation during static posture, as well as gait for symmetry, arches, toe deformities, and soft tissue swelling. Inspect the plantar surface of the foot for signs of weight-bearing asymmetry in the form of callous formation. Identify osseous symmetry and pain production through static palpation of the distal tibia and fibula (malleoli), dome of the talus, navicular, cuboid, calcaneus, cuneiforms, metatarsals, and phalanges.

Identify tone, texture, and tenderness changes through soft tissue palpation of the medial and lateral ligaments, Achilles tendon, and plantar fascia, as well as the musculature that controls movement of the foot and ankle. Additionally, palpate the posterior tibial artery and dorsalis pedis artery.

Evaluate accessory joint movements for the foot and ankle articulations to determine the presence of joint dysfunction (Table 6-22). Assess long-axis distraction of the tibiotalar articulation or ankle mortise joint with the patient supine, the knee flexed to approximately 90 degrees, and the hip flexed and abducted. Sit on the table between the patient's legs and face caudal. Place web contacts over the dome of the talus and superior aspect of the calcaneus, applying a distraction force through both hands (Figure 6-214).

Evaluate A-P and P-A glide of the ankle mortise joint with the patient supine and the hip and knee both slightly flexed so that the calcaneus rests on the table. Stand at the side of the table and place a web contact of your cephalic hand over the anterior aspect of the distal tibia while placing a web contact with your caudal hand over the anterior aspect of the dome of the talus. With both hands, grasp the respective structures and maintain the

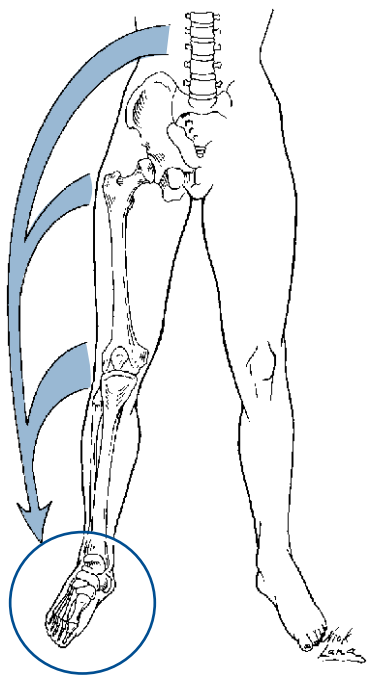


Figure 6-213 The low back, hip, and knee can refer pain to the ankle and foot area.

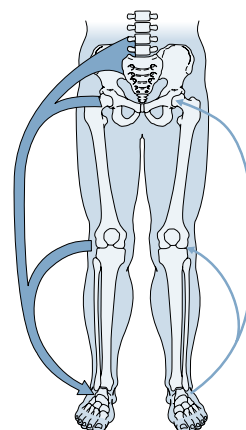


Figure 6-214 Patterns of referred pain to and from the ankle. (Modified from Magee DJ: *Orthopedic physical assessment*, ed 5, St Louis, 2008, Saunders.)



Figure 6-215 Assessment of anterior-to-posterior and posterior-to-anterior glide of the right tibiotalar joint.

TABLE 6-22 Accessory Joint Movements of the Foot and Ankle Joints

Joint	Movement
Tibiotalar joint	Long-axis distraction A-P glide P-A glide M-L tilt (inversion) L-M tilt (eversion)
Subtalar joint	A-P glide P-A glide M-L tilt (inversion) L-M tilt (eversion)
Tarsals (cuboid, navicular, and cuneiforms)	A-P glide P-A glide
Intermetatarsal joints	A-P glide P-A glide
Metatarsophalangeal and interphalangeal joints	Long-axis distraction A-P glide P-A glide M-L glide and tilt L-M glide and tilt Internal rotation External rotation

A-P, Anterior-to-posterior; L-M, lateral-to-medial; M-L, medial-to-lateral; P-A, posterior-to-anterior.

joint in its neutral position. Apply an A-P and P-A translational force through both hands, working in opposite directions, looking for a springing joint play movement (Figure 6-215).

Assess M-L and L-M glide of the tibiotalar articulation with the patient supine. Stand at the foot of the table, facing cephalad. Grasp the dome of the talus with the fingers of both hands, using the thumbs to grasp under the plantar surface of the foot. Then stress the talus in an M-L and L-M direction, feeling for a springing joint play movement (Figure 6-216).

Evaluate subtalar joint glide with the patient lying in the prone position and the knee flexed to approximately 60 degrees. Stand at the foot of the table, facing cephalad, with the plantar surface of the patient's toes resting against your abdomen. Then grasp the calcaneus with palmar contacts while interlacing your fingers in a "praying-hands" position. Use both hands to create A-P and P-A glide, as well as M-L and L-M glide movements (Figure 6-217).

Perform A-P and P-A glide of the navicular, cuboid, and cuneiforms by grasping the specific tarsal bone while stabilizing the proximal tarsal and creating an A-P and P-A glide movement (Figure 6-218).

Perform A-P and P-A shear of the intermetatarsals by grasping adjacent metatarsals with each hand and creating an A-P and P-A shear (Figure 6-219).

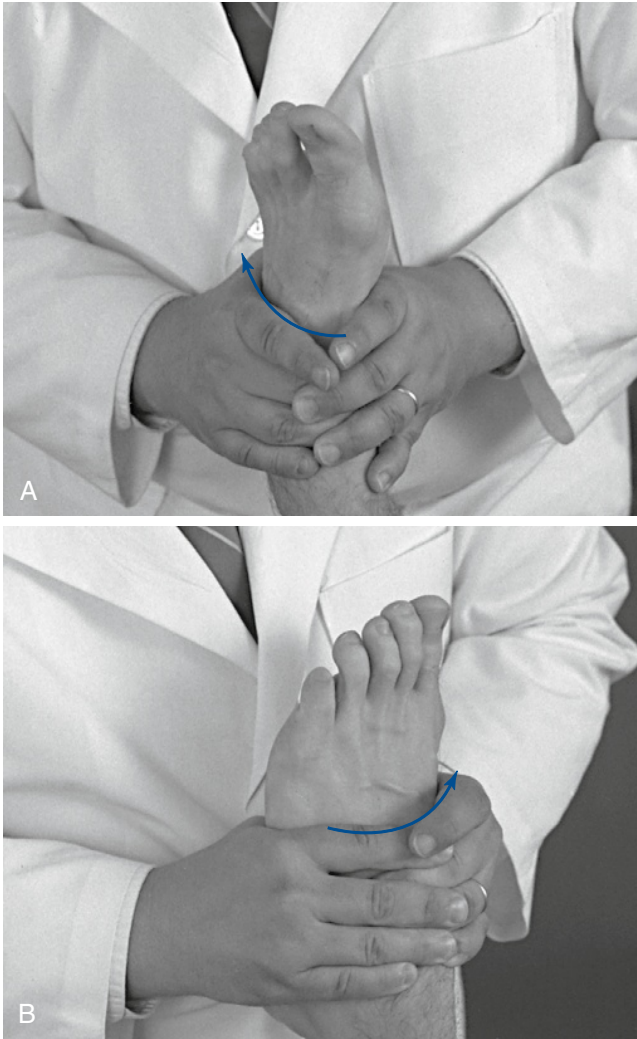


Figure 6-216 Assessment of medial-to-lateral (inversion) (A) and lateral-to-medial (eversion) (B) glide of the left tibiotalar joint.

Evaluate the metatarsophalangeal and interphalangeal joints for A-P and P-A glide, M-L and L-M glide, axial rotation, and long-axis distraction by grasping the metatarsals with one hand for stabilization and placing the specific phalanx between the index and middle fingers of the other hand (Figure 6-220).

ADJUSTIVE PROCEDURES

The manipulative techniques used to treat ankle and foot disorders aim to restore normal joint mechanics, which will then ideally allow full pain-free functioning of ankle and foot hip joints. Box 6-14 identifies the adjustive procedures for the ankle and foot.

Tibiotalar Joint

Supine:

Bimanual Reinforced Interphalangeal/Anterior Talus Pull; Long-Axis Distraction (Figure 6-221)



Figure 6-217 Assessment of anterior-to-posterior, posterior-to-anterior, medial-to-lateral, and lateral-to-medial glide of the right subtalar joint.

IND: Loss of long-axis distraction joint play movement of the tibiotalar joint.

PP: The patient is supine on the table, with the pelvic section raised and the buttocks resting against the raised pelvic piece.

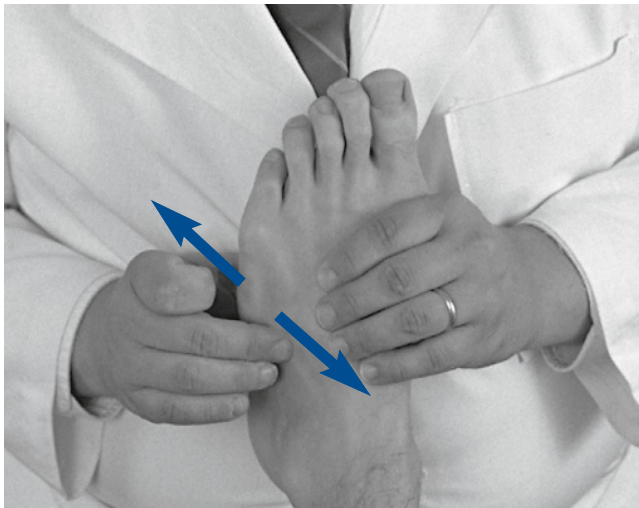
DP: Stand at the foot end of the table, facing cephalad.

SCP: Dome of the talus.

CP: Use either hand to apply proximal interphalangeal contact, with the middle finger over the dome of the talus.

IH: With your other hand, use a middle finger contact over the contact hand to reinforce it. With the thumbs of both hands, grasp the plantar surface of the foot.

VEC: Long-axis distraction.



6-218

Figure 6-218 Assessment of anterior-to-posterior and posterior-to-anterior glide of the left cuboid (same procedure used for the navicular and cuneiforms).



6-219

Figure 6-219 Assessment of anterior-to-posterior and posterior-to-anterior shear between the left metatarsals.



6-220

Figure 6-220 Assessment of long-axis distraction, internal and external rotation, and anterior-to-posterior, posterior-to-anterior, medial-to-lateral, and lateral-to-medial glide of the left metatarsophalangeal joints (same procedure for the interphalangeal joints).

BOX 6-14 Ankle and Foot Adjustive Techniques

TIBIOTALAR SUPINE:

Bimanual reinforced interphalangeal grasp/anterior talus pull; long-axis distraction (Figure 6-221)

Reinforced webs/anterior talus push anterior-to-posterior glide (Figure 6-222)

Reinforced middle interphalangeal/talus pull; lateral-to-medial glide (eversion) or medial-to-lateral glide (inversion) with long-axis distraction (Figure 6-223)

Web/talus, mid-hypothenar (knife-edge)/calcaneus; long-axis distraction with either inversion or eversion (Figure 6-224)

TIBIOTALAR PRONE:

Reinforced webs/talus push; posterior-to-anterior glide (Figure 6-225)

SUBTALAR PRONE:

Reinforced web/calcaneus; long-axis distraction (Figure 6-226)

Interlaced bimanual grasp/calcaneus; lateral-to-medial glide; medial-to-lateral glide; anterior-to-posterior glide; posterior-to-anterior glide (Figure 6-227)

TARSOMETATARSAL PRONE:

Hypothenar/cuboid with forefoot distraction; plantar-to-dorsal glide (Figure 6-228)

Hypothenar/navicular (cuneiforms) with forefoot distraction; plantar-to-dorsal glide (Figure 6-229)

Reinforced thumbs/cuneiform (cuboid, navicular) with forefoot distraction; plantar-to-dorsal glide (Figure 6-230)

TARSOMETATARSAL SUPINE:

Reinforced hypothenar/navicular (cuboid, cuneiforms); anterior-to-posterior glide (Figure 6-231)

Reinforced middle interphalangeal/cuneiform (navicular, cuboid) pull; anterior-to-posterior glide (Figure 6-232)

INTERTARSAL SUPINE:

Bimanual webs/tarsals; long-axis distraction (Figure 6-233)

INTERMETATARSAL SUPINE:

Bimanual thenar/metatarsal grasp shear; anterior-to-posterior and posterior-to-anterior glide (Figure 6-234)

METATARSOPHALANGEAL SUPINE

Thumb metatarsal/thumb phalanx shear; plantar-to-dorsal glide (Figure 6-235)

Thumb index grasp/phalanx; long-axis distraction (Figure 6-236)

FIRST METATARSOPHALANGEAL SUPINE:

Web metatarsal/finger grasp phalanx; medial-to-lateral glide with pendular distraction (Figure 6-237)

INTERPHALANGEAL SUPINE:

Thumb index grasp/phalanx; long-axis distraction; internal or external rotation; anterior-to-posterior or posterior-to-anterior glide; lateral-to-medial or medial-to-lateral glide (Figure 6-238)



Figure 6-221 Adjustment for long-axis distraction of the left tibiotalar joint.

P: Maintain the ankle in dorsiflexion and apply a long-axis distraction with both hands. To induce subtalar long-axis distraction, move the knife-edge or web contact of your IH to the posterosuperior aspect of the calcaneus.

Reinforced Webs/Anterior Talus Push; Anterior-to-Posterior Glide (Figure 6-222)

IND: Loss of A-P glide tibiotalar joint, and anterior misalignment of the talus.



Figure 6-222 Adjustment for A-P glide of the left tibiotalar joint.

PP: The patient is supine on the table, with the heel just off the end of the table.

DP: Stand at the foot end of the table, facing cephalad.

SCP: Dome of the talus.

CP: With your outside hand, establish a web contact over the dome of the talus, grasping the foot with your thumb and fingers.

IH: With your other hand, either reinforce the contact hand or grasp the distal tibia for stabilization.

VEC: A-P.

P: Apply an A-P translational thrust to the talus.

Reinforced Middle Interphalangeal/Talus Pull; Lateral-to-Medial Glide (Eversion) or Medial to Lateral Glide (Inversion) with Long Axis Distraction (Figure 6-223)



Figure 6-223 Adjustment for lateral-to-medial (A) and medial-to-lateral (B) glide of the right tibiotalar joint.

IND: Loss of medial or lateral glide at the tibiotalar articulation, medial or lateral misalignment of the talus.

PP: The patient is supine, with the leg straight and the foot off the end of the table.

DP: Stand at the foot end of the table, facing cephalad.

SCP: Dome of the talus.

CP: To produce eversion, use your inside hand to establish a middle finger distal interphalangeal contact over the dome of the talus, drawing tissue and articular slack medially. To produce inversion, use your outside hand to apply the middle finger contact over the dome of the talus, drawing tissue and medial slack laterally.

IH: With your other hand, grasp the posterior aspect of the calcaneus.

VEC: L-M or M-L.

P: Use both hands to distract in the long axis and give a thrust, drawing the dome of the talus in an L-M or M-L direction.

Web/Talus, Mid-Hypothenar (Knife-Edge)/Calcaneus; Long-Axis Distraction with Either Inversion or Eversion (Figure 6-224)

IND: Loss of long-axis distraction joint play movement of the tibiotalar joint.

PP: The patient is supine on the table, with the pelvic section raised and the buttocks resting against the raised pelvic piece.

DP: Stand at the foot end of the table, facing the affected ankle.

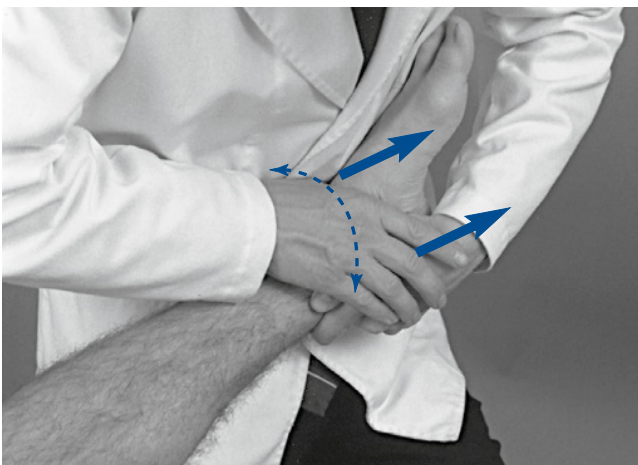
SCP: Dome of the talus.

CP: Using your cephalic hand, establish a web contact over the dome of the talus with the forearm along the line of the tibia.

IH: With your caudal hand, grasp the distal tibia while applying a mid-hypothenar (knife-edge) contact over the superior aspect of the calcaneus.

VC: Long-axis distraction.

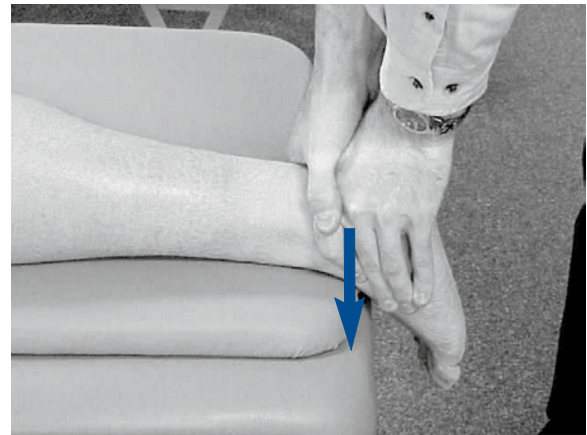
P: Use both hands to deliver an impulse thrust in the long axis of the tibia. M-L glide (inversion) or L-M glide (eversion) can be produced as well.



6-224

sion or eversion.

Figure 6-224 Adjustment for distraction of the left tibiotalar joint, which can combine either inversion or eversion.



6-225

Figure 6-225 Adjustment for posterior-to-anterior glide of the left tibiotalar joint.

Prone:

Reinforced Webs/Talus Push; Posterior-to-Anterior Glide (Figure 6-225)

IND: Loss of P-A glide movement, posterior misalignment of the talus.

PP: The patient is prone, positioned with the distal tibia at the edge of the table.

DP: Stand at the foot end of the table at the side of the table, facing the side of involvement.

SCP: Posterior aspect of the talus.

CP: With your caudal hand, establish a web contact over the posterior aspect of the talus.

IH: With your cephalic hand, grasp the distal tibia for stabilization.

VEC: P-A.

P: With your contact hand, apply a thrust, creating P-A glide of the talus.

Subtalar Joint

Prone:

Reinforced Web/Calcaneus; Long-Axis Distraction (Figure 6-226)

IND: Loss of subtalar long axis distraction.

PP: The patient lies prone, with the dorsum of the foot resting on the edge of the table, maintaining the ankle in plantar flexion.

DP: Stand on the affected side, facing caudal, in a lunge position.

SCP: Posterosuperior aspect of the calcaneus.

CP: With your cephalic hand, establish a calcaneal contact on the posterosuperior aspect of the calcaneus.

IH: With your caudal hand, reinforce the contact hand.

VEC: Distraction.

P: Use both hands to deliver a thrust caudal, drawing the calcaneus away from the talus.

Interlaced Bimanual Grasp/Calcaneus; Lateral-to-Medial Glide; Medial-to-Lateral Glide; Anterior-to-Posterior Glide; Posterior-to-Anterior Glide (Figure 6-227)



Figure 6-226 Adjustment for distraction of the left subtalar joint.

IND: Loss of subtalar glide movements (P-A, A-P, L-M, M-L), misalignment of the calcaneus (anterior, posterior, medial, or lateral).

PP: The patient lies prone, with the knee flexed to approximately 45 degrees.



Figure 6-227 Adjustment for lateral-to-medial glide of the left subtalar joint.

DP: Stand at the foot end of the table, facing cephalad, so that the plantar aspect of the patient's foot can rest against your abdomen.

SCP: Calcaneus.

CP: With both hands, grasp the calcaneus, interlacing the fingers in a "praying-hands" position.

VEC: A-P, P-A, L-M or M-L.

P: While stabilizing the patient's foot against your abdomen, the calcaneus can be moved medially, laterally, anteriorly, or posteriorly.

Tarsometatarsal Joint

Prone:

Hypothenar/Cuboid with Forefoot Distraction; Plantar-to-Dorsal Glide (Figure 6-228)

IND: Loss of plantar-to-dorsal movement of the cuboid, inferior misaligned cuboid.

PP: The patient is prone, with the knee bent to 90 degrees.

DP: Stand between the patient's legs, facing the affected side at its medial aspect.

SCP: Plantar aspect of the cuboid.

CP: Use your cephalic hand to apply a pisiform/hypothenar contact over the plantar aspect of the cuboid, wrapping your fingers around the lateral aspect of the foot.

IH: With your caudal hand, cradle the dorsum of the foot or interlace the fingers with the contact hand.

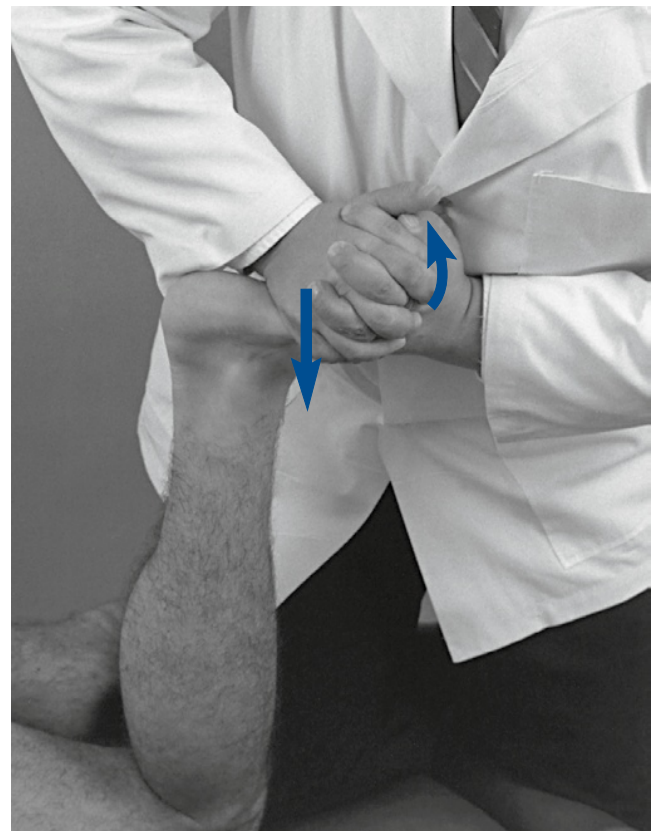


Figure 6-228 Adjustment for plantar-to-dorsal glide of the left cuboid.



Figure 6-229 Adjustment for plantar-to-dorsal glide of the right navicular.

VEC: Plantar-to-dorsal.

P: Use your IH to accentuate the longitudinal arch against the pressure applied to the plantar surface of the cuboid. Then give a thrust through the contact hand in a plantar-to-dorsal direction on the cuboid.

Hypothenar/Navicular (Cuneiforms) with Forefoot Distraction; Plantar-to-Dorsal Glide (Figure 6-229)

IND: Loss of plantar-to-dorsal accessory movement, inferior misalignment of the navicular.

PP: The patient is prone, with the knee bent to 90 degrees.

DP: Stand at the affected side, facing the lateral aspect of the foot.

SCP: Plantar aspect of the navicular.

CP: With your cephalic hand, establish a pisiform/hypothenar contact over the plantar aspect of the navicular, wrapping your fingers around the medial aspect of the foot.

IH: With your caudal hand, cradle the dorsum of the foot or interlace your fingers with the fingers of your contact hand.

VEC: Plantar-to-dorsal.

P: Using your IH, increase the longitudinal arch against the pressure applied to the navicular while applying a plantar-to-dorsal thrust to the navicular.

Reinforced Thumbs/Cuneiform (Cuboid, Navicular) with Forefoot Distraction; Plantar-to-Dorsal Glide (Figure 6-230)

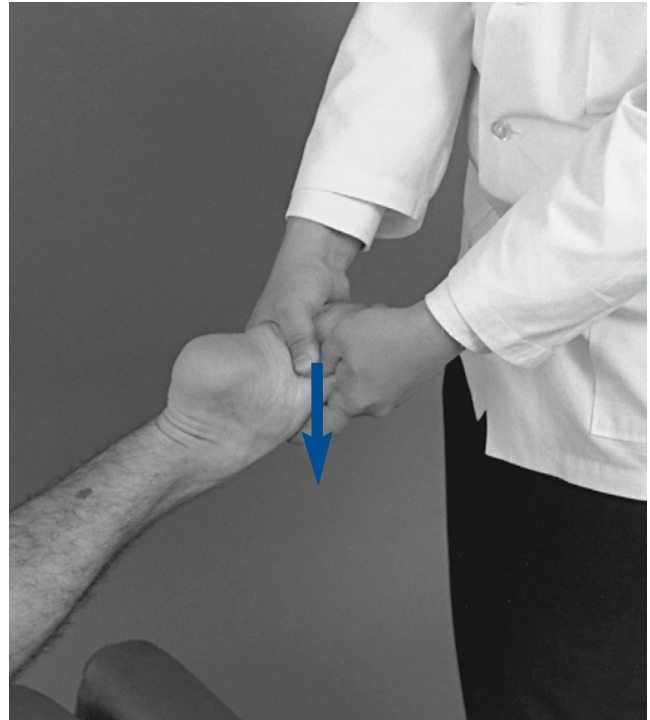


Figure 6-230 Adjustment for plantar-to-dorsal glide of the right first cuneiform.

IND: Loss of plantar-to-dorsal accessory joint movement of the cuneiforms, plantar misalignment of the cuneiforms.

PP: The patient is prone, with the knee flexed to approximately 45 degrees.

DP: Stand at the foot end of the table, facing cephalad.

SCP: Plantar aspect of a cuneiform.

CP: Use your inside hand to apply a thumb contact over the plantar aspect of the cuneiform, wrapping your fingers around the dorsum of the foot.

IH: With your outside thumb, reinforce the contact.

VEC: Plantar-to-dorsal.

P: With both thumbs, deliver a plantar-to-dorsal snapping-type thrust, being careful not to take the ankle into complete plantar flexion. This procedure can also be used for the navicular and cuboid.

Tarsometatarsal Joint

Supine:

Reinforced Hypothenar/Navicular (Cuboid, Cuneiforms); Anterior-to-Posterior Glide (Figure 6-231)

IND: Loss of dorsal-to-plantar accessory joint movement, dorsal misalignment of a tarsal bone.

PP: The patient is supine, with the knee and hip flexed so that the plantar aspect of the foot rests on the table.

DP: Stand at the foot of the table, facing cephalad.

SCP: Dorsal aspect of a tarsal bone (cuboid, navicular, or cuneiform).



Figure 6-231 Adjustment for dorsal-to-plantar glide of the tarsals (left navicular shown).

- CP:** Use either hand to establish a pisiform contact over the dorsal aspect of the involved tarsal bone.
- IH:** With your other hand, reinforce the contact.
- VEC:** Dorsal-to-plantar.
- P:** Deliver a very quick dorsal-to-plantar impulse or recoil-type thrust. A mechanical drop section can be used to enhance this procedure.

Reinforced Middle Interphalangeal/Cuneiform (Navicular, Cuboid) Pull; Anterior-to-Posterior Glide (Figure 6-232)

- IND:** Loss of dorsal-to-plantar accessory joint movement, dorsal misalignment of a tarsal bone.
- PP:** The patient is supine, with the affected leg straight.
- DP:** Stand at the foot of the table, facing cephalad.
- SCP:** Dorsal aspect of a tarsal bone (cuboid, navicular, or cuneiform).
- CP:** Use either hand to apply a middle finger distal interphalangeal contact over the dorsal aspect of the affected tarsal.
- IH:** With your other hand, reinforce the contact, wrapping both hands around the plantar aspect of the foot with the thumbs just distal to the point of contact.
- VEC:** Dorsal-to-plantar.
- P:** Use both hands to apply a dorsal-to-plantar pressure, while the thumbs apply an opposite plantar-to-dorsal stress. Then apply a quick dorsal-to-plantar thrust through the contact hand.

Intertarsal Joint

Supine:

Bimanual Web/Tarsals; Long-Axis Distraction (Figure 6-233)

- IND:** Metatarsal varum, hypomobility of the medial tarsal articulations.

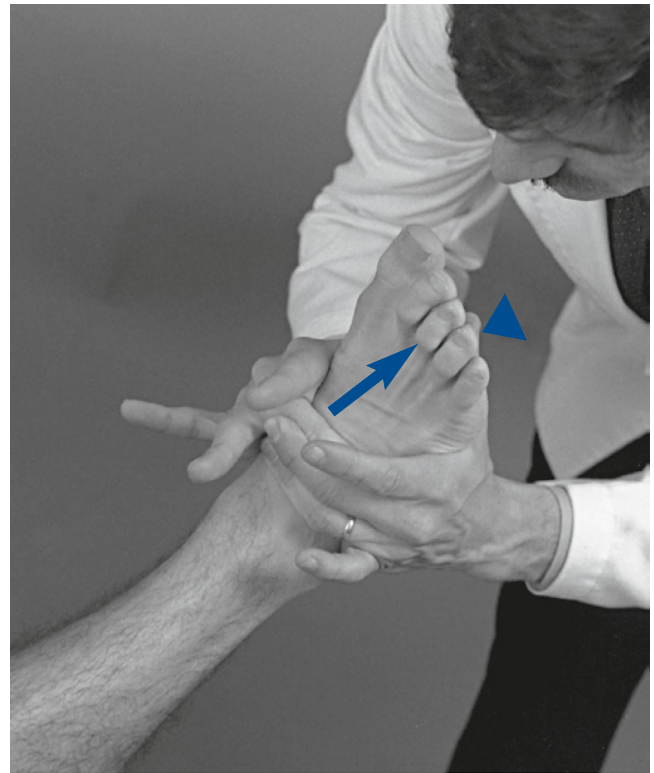


Figure 6-232 Adjustment for dorsal-to-plantar glide of the tarsals (right cuneiform shown).

- PP:** The patient is supine, with the leg externally rotated and abducted off the table.
- DP:** Stand on the affected side, facing caudal, with the inside foot on the table so that the lateral aspect of the patient's affected foot can rest on the doctor's thigh.
- SCP:** Medial aspect of the navicular.



Figure 6-233 Distraction between the navicular, first cuneiform, and first metatarsal joints of the left foot.

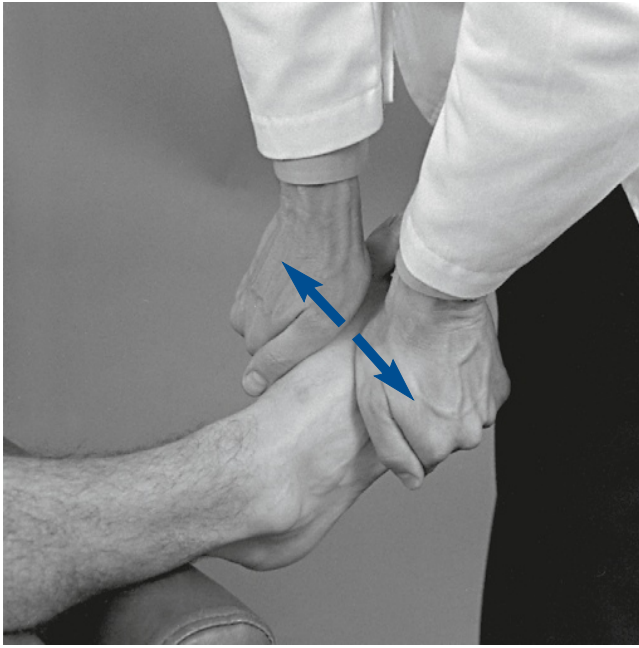


Figure 6-234 Adjustment for anterior-to-posterior and posterior-to-anterior glide between the right metatarsals.

CP: With your inside hand, establish a web contact over the medial aspect of the navicular.

IH: Use your outside hand to apply a web contact over the medial aspect of the proximal metatarsal.

VEC: Distraction.

P: Using your thigh as a fulcrum, thrust both hands away from each other, effectively separating the navicular from the first cuneiform and the first cuneiform from the proximal metatarsal.

Intermetatarsal Joint

Supine:

Bimanual Thenar/Metatarsal Grasp Shear; Anterior-to-Posterior and Posterior-to-Anterior Glide (Figure 6-234)

IND: Restricted intermetatarsal glide movements.

PP: The patient is supine, with the affected leg in slight flexion.

DP: Stand at the foot of the table, facing cephalad.

SCP: Metatarsal bone.

CP: Establish a thumb-thenar contact on the palmar aspect of a metatarsal bone, and with your fingers, hold the same metatarsal shaft on the dorsal surface of your hand.

IH: Make the same contacts on the adjacent metatarsal.

VEC: A-P or P-A.

P: Use both hands to create an A-P and P-A shear between the two metatarsals.

Metatarsophalangeal Joint

Supine:

Thumb Metatarsal/Thumb Phalanx Shear; Plantar-to-Dorsal Glide (Figure 6-235)

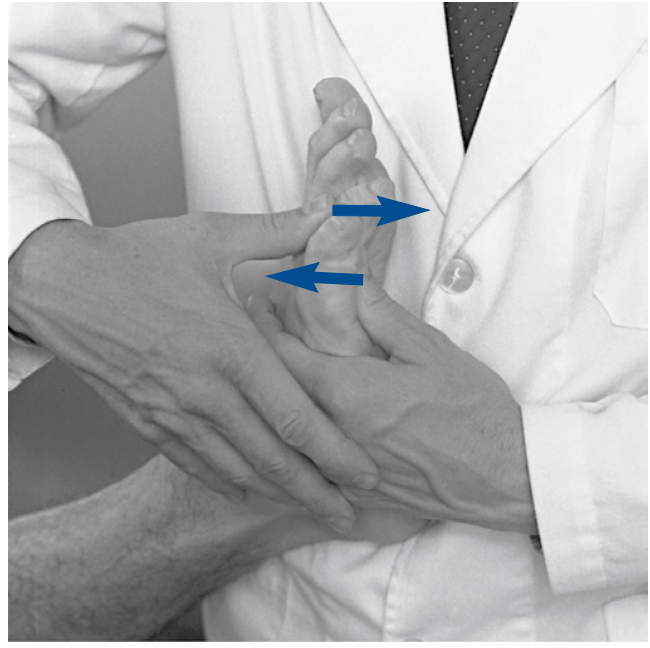


Figure 6-235 Adjustment for plantar-to-dorsal glide of the right metatarsophalangeal joints.

IND: Loss of plantar-to-dorsal accessory joint movement of a metatarsal, plantar misalignment of a metatarsal.

PP: The patient is supine, with the leg straight and resting on the table.

DP: Stand at the foot of the table, facing cephalad.

SCP: Plantar aspect of a metatarsal bone.

CP: Use your outside hand to apply a thumb contact over the plantar aspect of the affected metatarsal.

IH: With your inside hand, establish a thumb contact over the dorsal aspect of the phalanx just distal to the metatarsal contact.

VEC: Plantar-to-dorsal.

P: Use both thumbs to create a simultaneous shear, emphasizing the plantar-to-dorsal component on the metatarsal.

Thumb Index Grasp/Phalanx; Long-Axis Distraction (Figure 6-236)

IND: Loss of long axis accessory joint movement in the metatarsophalangeal or interphalangeal joints.

PP: The patient is supine, with the affected foot extending off the end of the table.

DP: Stand at the foot of the table, facing cephalad.

SCP: Individual phalanges.

CP: Use either hand and loosely curl the index finger, applying its radial aspect against the plantar surface of the metatarsophalangeal joint. With the thumb, then grasp the phalanges from above (dorsal aspect) and distal to the index contact on the plantar surface.

IH: With the other hand, grasp the foot to stabilize it.

VEC: Long-axis distraction.

P: With the thumb, draw the phalanges over the index contact, and apply a dorsal-to-plantar distractive thrust.



Figure 6-236 Adjustment for long-axis distraction of the right metatarsophalangeal joints (same procedure for the interphalangeal joints).

First Metatarsophalangeal Joint

Supine:

Web Metatarsal/Finger Grasp Phalanx; Medial-to-Lateral Glide with Pendular Distraction (Figure 6-237)

IND: Hallux valgus, bunions, loss of M-L movement of the first metatarsophalangeal joint.

PP: The patient is supine, with the affected foot off the end of the table.

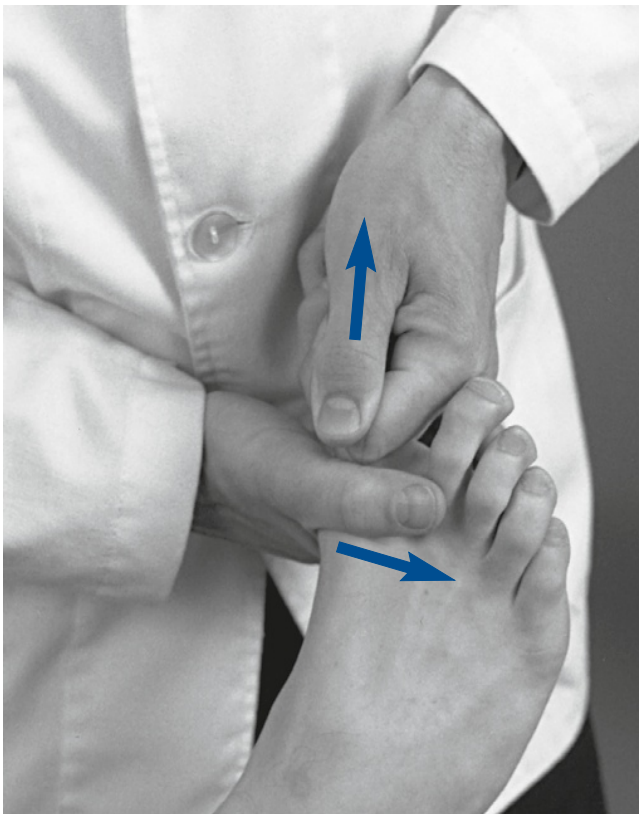


Figure 6-237 Adjustment for medial-to-lateral glide of the right first metatarsophalangeal joint.

DP: Stand at the foot of the table, facing cephalad.

SCP: Proximal phalanx of the great toe.

CP: With your outside hand, grasp the proximal phalanx between your index and middle finger.

IH: Using your inside hand, apply a web contact over the medial aspect of the metatarsophalangeal joint.

VEC: M-L.

P: With the contact hand, elevate the foot, using gravity to create long-axis distraction at the metatarsophalangeal joint. Induce side-to-side rocking with the contact hand to mobilize the joint initially. A very shallow M-L thrust can then be applied by the IH.

Interphalangeal Joint

Supine:

Thumb Index Grasp/Phalanx; Long-Axis Distraction; Internal or External Rotation; Anterior-to-Posterior or Posterior-to-Anterior Glide; Lateral-to-Medial or Medial-to-Lateral Glide (Figure 6-238).

IND: Loss of accessory joint movement in the toe joints, misalignment of the toes joints.

PP: The patient is supine.

DP: Stand and face the patient.



Figure 6-238 Adjustment for internal and external rotation and anterior-to-posterior, posterior-to-anterior, lateral-to-medial, and medial-to-lateral glide of the right metatarsophalangeal joints (same procedure for the interphalangeal joints).

SCP: Distal component of the affected joint.

CP: Grasp the distal member of the joint to be adjusted with either hand.

IH: With the other hand, grasp the proximal member of the joint being adjusted.

VEC: A-P and P-A glide, L-M and M-L glide, and internal and external rotation.

P: Apply an impulse thrust to the affected metatarsophalangeal or interphalangeal joint, using A-P and P-A glide, L-M and M-L glide, and internal and external rotation.

The use of manipulative or adjustive techniques with peripheral joint problems is a valuable aspect in chiropractic practice, requiring no more or no less skill than techniques for the spine.

NONTHRUST PROCEDURES: MOBILIZATION, TRACTION, AND SOFT TISSUE TECHNIQUES

OUTLINE			
JOINT MOBILIZATION	381	McKENZIE METHOD	387
Definition	381	Treatment Principles	388
Primary Goal	382	Three Syndromes: Postural, Dysfunctional, and Derangement	389
Various Types	382	CRANIAL MANIPULATION	391
Selected Examples	383	Sagittal Suture Spread	392
MANUAL TRACTION-		Cranial Universal	392
 DISTRACTION	384	Parietal Lift	392
Definition	384	SOFT TISSUE MANIPULATION	393
Specific Procedures	386	Effects of Soft Tissue Manipulation	393
		Specific Techniques	394
		Ischemic Compression	408
		Body Wall Reflex Techniques	410
		Logan Basic Technique	413
		Spondylotherapy	417
		CONCLUSIONS	418

Whereas most chiropractic adjustive techniques impart a thrust, many manual therapy procedures are designed to affect physiologic processes without using a thrust. The principal procedures that do not incorporate a thrust include joint mobilization, traction, and soft tissue manipulation (STM). This chapter presents a description and overview of the practical application of many of the common nonthrust procedures applied in manual therapy and chiropractic.

The chiropractic profession did not develop most of the techniques and procedures discussed in this chapter. Therefore, they should not be considered unique to the chiropractic profession. Moreover, a small percentage of chiropractic professionals do not use them because of the exclusiveness of thrust techniques. However, they are important forms of manual therapy that can be used alone or in combination with thrust procedures. Procedures characterized by a high-velocity thrust (adjustment) may indeed have effects that are different than and, in certain clinical situations, superior to nonthrust procedures.^{1,2}

Nevertheless, both procedures share common physical attributes and overlapping potential positive clinical effects. Grieve³ contends that all mobilizations and manipulations are actually soft tissue techniques, because it is in the soft tissue that the lesions treated and the effects produced are found.

Chiropractors commonly encounter circumstances in which thrust manipulation is contraindicated or the primary neuromusculoskeletal (NMS) disorder is not the product of joint dysfunction or a pathologic condition. In such circumstances, it benefits both the doctor and the patient if the chiropractor is skilled in alternative nonthrust manual procedures. Indeed, various and numerous forms of manual therapy exist within the profession of chiropractic.⁴ Examples of circumstances in which nonthrust procedures might be more appropriate include the treatment of the older adults or patients with osteoporotic or extremely acute conditions, treatment of patients in the later stages of pregnancy, or treatment of patients with a diagnosis of myofascial pain syndrome (trigger point).

Nonthrust procedures are also viable options in circumstances in which the doctor is unable to produce a thrusting force that is capable of producing joint cavitation. If the doctor's size, strength, or ability to develop the needed speed and amplitude is inadequate to produce the necessary force, some other type of technique application may provide an effective alternative. This is suitable only in the circumstance in which both thrust and nonthrust procedures are assumed to have the same physical effects. All manual therapies are not equivalent simply because a wide variety of methods exist.⁵ Most of the procedures described in this chapter were developed empirically. Typically they are associated with a particular innovator or profession, but invariably development, refinement, and modification by subsequent innovators has led to an environment of multiple definitions, descriptions, and variations of the same procedure. The intention of this chapter is not to present a comprehensive discussion of the variations and nuances of each procedure, but to offer a fair representation of techniques and an overview of each method presented. Ideally, the descriptions of the procedures contained within this chapter will broaden your awareness of nonthrust manual procedures and stimulate further critical evaluation of those procedures that pique your interest.

JOINT MOBILIZATION

DEFINITION

Joint mobilization may be defined as a passive therapeutic movement up to but not exceeding the anatomic end range of joint movement. A nonthrust maneuver is not commonly applied beyond a joint's elastic barrier. Movement beyond the elastic barrier takes the joint into the parapsycho-physiologic joint space and is typically associated with an audible pop or click. Therefore mobilization is less commonly associated with an audible crack than manipulation. Grieve³ considers spinal mobilization a more

gentle, persuasive pressure performed within the available accessory range or at the end of the accessory range. However, mobilization can be applied over a wide range and can thus involve a series of movements (stages or grades). Mobilization is a coaxing, repetitive, rhythmic movement of a joint that can be resisted by the patient. Inherent within a nonthrust manipulation is a patient feedback mechanism. Because the motion is relatively slow, controlled, and gentle, the patient can report the effect of the technique during the application.

PRIMARY GOAL

The primary goal of mobilization is to restore optimal range of motion (ROM), quality of movement, and comfort to the joint being addressed. Indirect benefit is thought to accrue because of improved function within each part of the kinetic chain in need of treatment. For example, compensatory mechanical stress on adjacent structures may be reduced when a previously painful or restricted part of the chain is returned to normal function. Such return to comfortable, maximal ROM usually serves as a passive treatment end point.⁶ Mobilization can be performed in a physiologic direction (rotation, flexion, extension, or lateral flexion) or in a nonphysiologic direction (e.g., longitudinal traction or posterior-to-anterior [P-A] gliding). Furthermore, various types of mobilization can be used to restore segmental motion and perhaps reduce pain in the spine and extremities. Graded oscillation, progressive stretch, and continuous stretch mobilization form the basis for most types of mobilization techniques.

VARIOUS TYPES

Graded oscillation technique is a form of mobilization whereby alternate pressure (on and off) is delivered at different parts of the available range.⁷ The amplitude of the oscillation may also vary according to the purpose of the technique. Oscillatory technique is graded on a 1-to-4 scale based on the amplitude of the motion and part of the range being reached (Box 7-1). The vibratory nature of the graded oscillation technique is thought to activate sensory mechanoreceptors that may help reduce pain and improve proprioceptive function. Box 7-2 lists generalized procedural steps for the application of graded oscillatory mobilization.

Progressive stretch mobilization involves a series of successive short-amplitude, spring-type pressures or a series of short-amplitude stretch movements.⁸ The pressure or stretch is imparted at progressive increments of the range. Progressive stretch is graded on a 1-to-4 scale, as is graded oscillation. The major indication for the use of progressive stretch mobilization is mechanical or soft tissue restriction or both.

Continuous stretch is a sustained, gradually increased stretch or pressure without interruption. Maintaining a stretch or pressure throughout the mobilization procedure is recommended when immediate tissue feedback is desired. Adaptively shortened periarticular soft tissue structures are most likely to be affected using continuous stretch technique. Improving extensibility of the

BOX 7-1 Amplitude Grades for Oscillatory Technique

Grade I	A small-amplitude movement near the starting position of the range
Grade II	A larger-amplitude movement that carries well into the range, occupying any part of the range that is free of stiffness or muscle spasm
Grade III	A large-amplitude movement that moves into stiffness or muscle spasm
Grade IV	A small-amplitude movement that stretches into stiffness or muscle spasm

BOX 7-2 Generalized Graded Oscillatory Mobilization Procedure

1. Take the joint to tension (engage barrier or point of pain). This involves firm pressure until resistance is felt. However, it is important to avoid using heavy forces that might create reactive muscle spasm.
2. Hold gently against the barrier until a release of resistance occurs (3 to 10 seconds). This will be perceived as a “melting” or softening of resistance. Alternatively, repetitively and rhythmically mobilize until a release of resistance occurs.
3. Continue mobilizing until motion is normal (average is 3 to 10 mobilizations).
4. Stay just short of reproduction of the symptoms, barely engaging the point of pain and backing away.
5. If the amplitude is too small, the procedure will be less effective; if the amplitude is too great (going too far into the painful area), the symptoms will be aggravated.

periarticular soft tissues about a spinal joint by means of collagen fiber realignment and viscosity change allows for improved joint mobility.

Mobilization can be performed at general regions or at specific joint levels. The difference between these procedures rests in the localization of forces. To produce a specific mobilization, a particular segment must be placed in its most favorable position for movement, and contacts must be placed on or close to the segment being mobilized. A general or regional mobilization incorporates the use of longer levers or contacts placed at points distal to the area being mobilized. In addition, the arc of movement for a generalized or regional mobilization will be greater than the arc of motion used for the specific or segmental mobilization.

In addition, special attention should be given to the traction component of mobilization. Where anatomic relationships allow

traction to accompany mobilization, traction should be used as an integral part of the treatment program. In many cases of pain-induced failure of a mobilization technique, that is, when angular or even translational force vectors produce increased pain, pure traction techniques may still be useful. This fact reinforces the concept that traction is one of the least invasive mobilization methods.⁹

SELECTED EXAMPLES

The following is a representative sample of spinal and extremity mobilization procedures. These procedures have been selected because they represent a variety of methods, not because they demonstrate superior effectiveness as compared with other mobilization procedures.

Oscillatory Cervical Lateral Flexion (Figure 7-1)

The patient lies in the supine position, with the head and neck supported on the headrest and by the clinician's hands. The clinician sits or stands at the head end of the table. To produce left lateral flexion mobilization of the cervical spine, the clinician's right hand comfortably grasps the patient's chin while the forearm rests on the side of the head. The other hand supports the patient's occiput. Both hands support the patient's head and produce a repetitive, rhythmic left lateral flexion movement around the z-axis. An oscillatory movement is produced as the hands move reciprocally, and all excursions have equal value.

Oscillatory Atlas Lateral Glide (Figure 7-2)

The patient lies in the side-posture position with both knees bent to maintain pelvic stability, the upper arm resting along the side of the body and the hand grasping the thigh, and the head placed in a neutral position on an elevated headrest. The clinician sits or stands, facing the patient, and contacts the lateral aspect of the atlas transverse process with both thumbs, one on top of the other. A lateral-to-medial oscillatory movement is produced, developing a translational lateral glide movement.

Progressive Stretch Thoracic Extension (Figure 7-3)

The patient lies in the prone position with the hands interlaced behind the neck and the elbows together (Figure 7-3, *A*). The clinician stands on the side of the table and grasps the patient's arms from underneath with the cephalic hand. The caudal hand is used to establish a broad contact over the spine below the section to be mobilized. The clinician simultaneously and progressively raises the arms to the patient's tolerance while applying stabilizing pressure over the spine. This procedure may also be done in the seated position (Figure 7-3, *B*). There is powerful leverage produced in both positions, and caution must be used to prevent injury.

Oscillatory Posterior-to-Anterior Glide (Figure 7-4)

The patient lies in the prone position. The clinician stands on the side of the table facing cephalically and in a lunge position (fencer stance). The clinician establishes bilateral thumb contacts over the



Figure 7-1 Oscillatory cervical lateral flexion to produce left lateral flexion mobilization of the cervical spine.



Figure 7-2 Oscillatory atlas lateral glide developing a left-to-right translational lateral glide movement of the atlantoaxial articulation (C1-C2).

articular processes of a cervical segment, the transverse processes of a thoracic segment, or the mammillary processes of a lumbar segment. The thumbs may be on the same side of the patient or can cross the spine. The clinician produces a P-A oscillatory movement over the vertebra.

Progressive Stretch Lumbar Rotation (Figure 7-5)

The patient lies in a prone position over an elevated table or Dutchman roll. The clinician stands in a square stance at the side of the table opposite the area to be mobilized. The clinician's

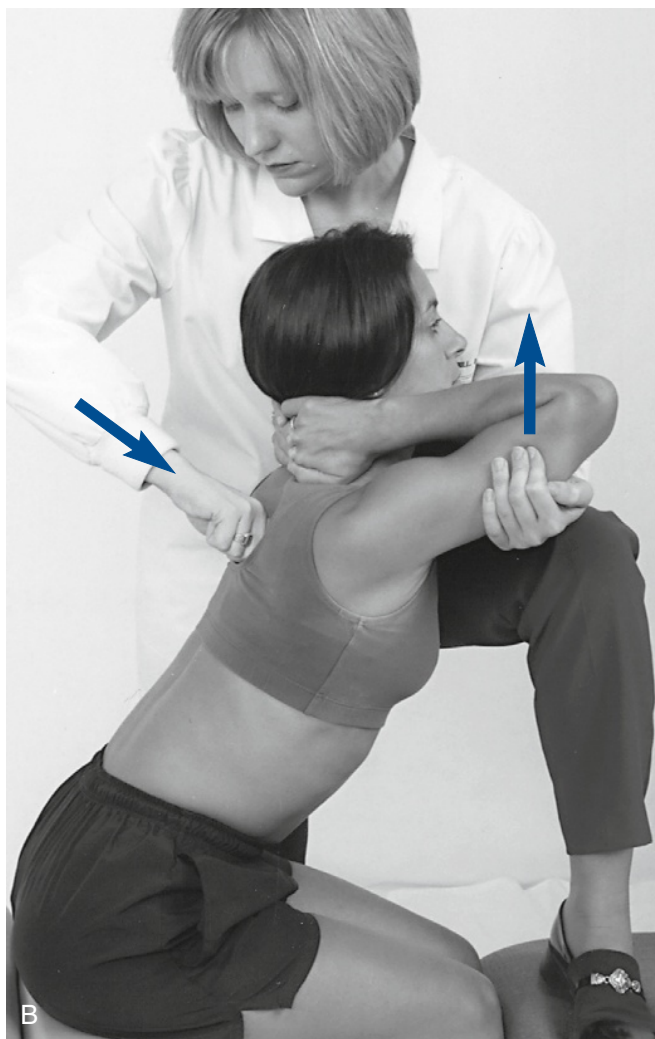


Figure 7-3 Progressive stretch thoracic extension. **A**, Prone. **B**, Sitting.

caudal hand grasps the anterior superior iliac spine while the cephalic hand establishes a broad contact over the lower rib cage on the side to be mobilized. The clinician raises and lowers the pelvis against the resistance provided by the cephalic hand, producing a rhythmic rocking lumbar rotation of small amplitude.

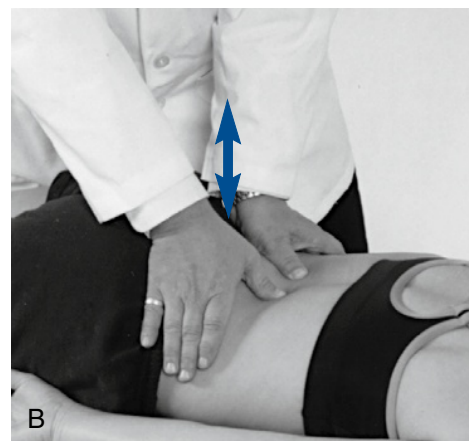
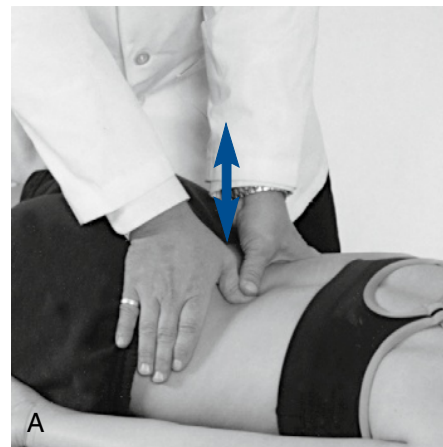


Figure 7-4 Oscillatory posterior-to-anterior (P-A) glide applied to the L3 vertebra. The patient lies in the prone position. The clinician stands on the side of the table, facing cephalically, in a lunge position (fencer stance). The clinician establishes bilateral thumb contacts over the articular processes of a lumbar segment. The thumbs may be on the same side of the patient or may cross the spine (**A**). A bilateral contact can also be established (**B**). A P-A oscillatory movement is produced over the vertebra.

Continuous Stretch Rotation (Figure 7-6)

The patient lies in a prone position over an elevated table or Dutchman roll. The clinician stands at the side of the table, facing cephalically, in a lunge position (fencer stance). The clinician establishes a thumb contact against the lateral aspect of a spinous process with the caudal hand. The cephalic hand is placed over the lateral aspect of the pelvis. A firm, continuous pressure to the patient's tolerance is applied against the L5 spinous process while the pelvis is stabilized. Continuous pressure is held for 8 to 12 seconds and repeated up to three times.

MANUAL TRACTION-DISTRACTION

DEFINITION

The term *traction* refers to the process of pulling one body in relationship to another, which results in separation of the two bodies. Traction is passive translational movement of a joint, which

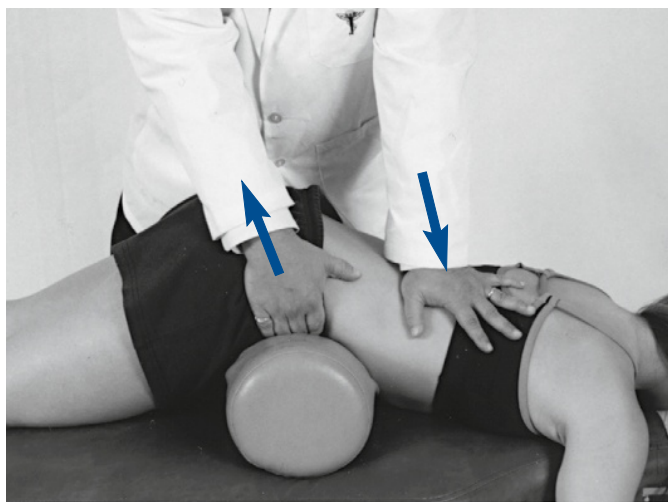


Figure 7-5 Progressive stretch lumbar rotation to mobilize the right side of the lumbar spine. The patient lies in a prone position over an elevated table or Dutchman roll. The clinician stands in a square stance at the side of the table opposite the area to be mobilized. The clinician's caudal hand grasps the anterior superior iliac spine while the cephalic hand establishes a broad contact over the lower rib cage on the side to be mobilized. The clinician raises and lowers the pelvis against the resistance provided by the cephalic hand, producing a rhythmic lumbar rotation of small amplitude.

occurs at right angles to the plane of the joint, resulting in separation of the joint surface. Kaltenborn¹⁰ graded manual traction by the three effects it produces. With the first effect, there is no appreciable joint separation because only enough traction force is applied to nullify the compressive forces acting on the joint. The compressive forces are the result of muscle tension, cohesive forces between articular surfaces, and atmospheric pressure. The second effect produces a tightening in the tissue surrounding the joint that is described as “taking up the slack.” The third grade of traction requires more traction force to produce a stretching effect into the tissues crossing the joint. The principal aim of treatment is restoration of normal painless ROM (Box 7-3).

Traction produces measurable separation of vertebral bodies and centripetal forces exerted by the tension applied to surrounding soft tissues. However, traction has other effects as well. Grieve¹¹ identifies some other effects that are the result of both sustained and rhythmic traction (Box 7-4).

Manual traction is not a unique and separate form of treatment but is simply one form of passive mobilization.¹² Traction can be varied in many ways; almost any form of passive handling may be used, with some form of oscillation or as a static hold. Therefore, a longitudinal movement may be performed as an oscillatory mobilization, as a slow rhythmic stretch, or as a static traction. Traction may be manual or mechanical, static or rhythmic, or fast or slow; the force applied may be strong or gentle, and it may be applied symmetrically or asymmetrically. These variations must be explored to determine which combination is most suitable for the patient's needs or the clinician's abilities. The effects of traction are not necessarily localized, but may be made more specific by careful positioning.

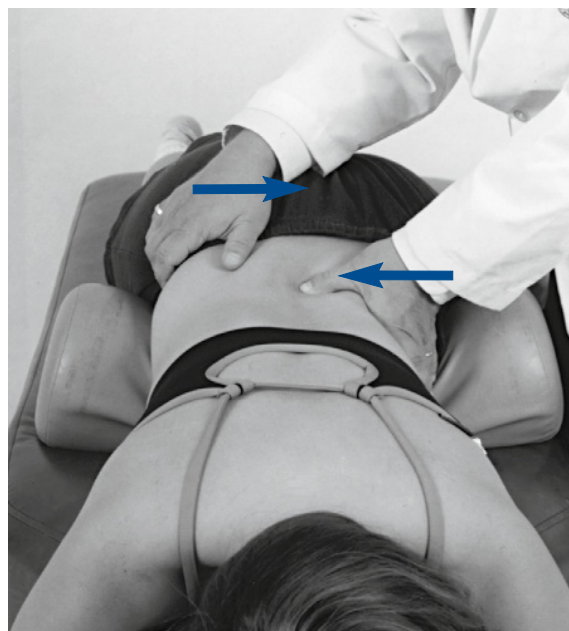


Figure 7-6 Continuous stretch rotation of L4, producing a left rotational mobilization of L4 over L5. The patient lies in a prone position over an elevated table or Dutchman roll. The clinician stands at the side of the table, facing cephalically, in a lunge position (fencer stance). The clinician establishes a thumb contact against the lateral aspect of the pelvis. A firm, continuous pressure to the patient's tolerance is applied against the L5 spinous process while the pelvis is stabilized. Continuous pressure is held for 8 to 12 seconds and repeated up to three times.

BOX 7-3 Treatment Aims of Manual Traction

1. Relief of pain and reduction of muscle spasm
2. Restoration of normal tissue-fluid exchange, soft tissue pliability and extensibility, and normal joint relationship and mobility
3. Correction of muscle weakness or imbalance
4. Stabilization of unstable segments
5. Restoration of adequate control of movement
6. Relief from chronic postural or occupational stress
7. Functional rehabilitation of the patient

BOX 7-4 Effects of Traction

Simple mobilization of joints with reversible stiffness
 Modification of the abnormal patterns of afferent impulses from joint mechanoreceptors
 Relief of pain by inhibitory effects on afferent neuron impulses subserving pain
 Reduction of muscle spasm
 Stretching of muscle and connective tissue
 Improvement of tissue-fluid exchange in muscle and connective tissue
 Likely improvement of arterial, venous, and lymphatic flow
 Physiologic benefit to the patient of rhythmic movement
 Lessening of compressive effects

For traction to achieve maximal success in a minimal amount of time, the patient must be positioned accurately, a minimal effective force must be used, and each patient's treatment must be based on his or her signs and symptoms rather than the diagnosis. The potential theoretic effects of traction on the spine include stretching of the muscles and ligaments, improving glide of the dural root sleeves, freeing fixation of articular facets, changing hydrostatic pressure in the discs and repositioning nuclear fragments, and improving the blood supply to the spine and its surrounding structures.¹³

SPECIFIC PROCEDURES

Manual Lumbar Flexion-Distraction (Cox Method)

Flexion-distraction is a mechanically assisted form of joint mobilization or distraction, blending osteopathic and chiropractic principles into one technique. Flexion-distraction has been advanced in the chiropractic profession largely by the work of chiropractor James Cox. Much of Cox's initial work in developing his technique of flexion-distraction was based on the work of the osteopath J.V. McManis. Moreover, the design of the early Cox table was a direct emulation of the McManis table of the early 1900s. This manual table provided an advantage to both the patient and the clinician, allowing a multiple-plane approach to distraction, including flexion-extension, lateral flexion, and rotation. The McManis table incorporated many of the features that appear on contemporary tables, including split headpieces, multiple sections adjustable for patient comfort, and positioning for various adjustable maneuvers.¹⁴

A number of lumbar disorders have been presented as conditions suitable for treatment with lumbar flexion-distraction. They include lumbar disc protrusion, spondylolisthesis, facet syndrome, subluxation, and scoliotic curves of a nonsurgical nature.¹⁵ The theoretic benefits that have been associated with this form of spinal manipulation are presented in Box 7-5.

The Cox method uses a process of analysis that incorporates physical examination, orthopedic and neurologic testing, and imaging as indicated to establish the presence of a disc lesion, facet syndrome, or any other condition affecting the low back. Cox distraction therapy typically consists of three 20-second flexion-distraction sessions. Once the patient is properly positioned on the table and the tolerance of the patient to flexion is determined, the sessions can begin. The hand is placed over the spinous process of the superior vertebra of the motion segment

to be distracted (e.g., to distract the L5–S1 segment, contact is on the L5 spinous process). The contact hand is slightly cupped, creating an indentation between the thenar and hypothenar eminence to receive the prominence of the spinous process without placing undue pressure and causing subsequent discomfort to the patient.¹⁶ The patient is encouraged to relax, and the clinician depresses the handle at the foot of the table. If there is no handle, the clinician depresses the foot of the table (pelvic section) by hand. The handle is an improvement over placing the hand on the caudal section of the table because it provides increased leverage and a better stance position for the clinician (Figure 7-7).

The pelvic section is then depressed until the clinician's hand detects that the musculature has reached a point of tautness and all tissue and joint play have been removed from the area under treatment. This point is maintained, and an additional 2 to 3 inches of table depression is achieved manually. The caudal section is then allowed to return to neutral, followed by another downward movement to the previous point over a 20-second period. This process creates a "pumping action" and is repeated three times, with a break of a few seconds between each 20-second session (Box 7-6).

A patient with a protruding disc may sense mild pain on traction, whereas a prolapsed disc does not usually produce such a sensation. Too much traction during the session should be avoided because it may produce further annular injury and impairment. In the case of disc involvement, the disc and motion segment are potentially sensitized to pain and mechanical stimuli, and the patient is probably sensitive and sore at the point of the injury. Therefore, it is better to undertreat the patient than to overtreat, and caution is encouraged in the application of the technique. Furthermore, if the patient does not tolerate the flexion-distraction movement or if the pain is peripheralized, the process should not continue. Further traction should concentrate on relieving the pain before the therapeutic distraction begins in earnest. Any intolerance should be viewed with caution and, although this does not become a clear contraindication for the treatment, it certainly should be treated with respect and restraint. In this instance, more is not better but, in fact, may make the patient's condition worse.

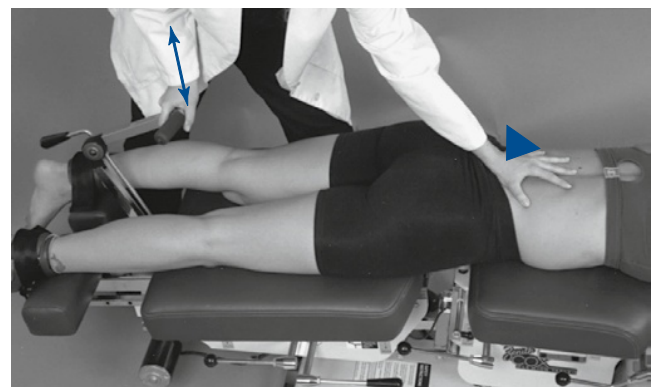


Figure 7-7 Cox flexion distraction for the L4 disc (L4–L5 segment) with a contact established over L4 spinous process as the caudal section is depressed, producing distraction.

BOX 7-5 Theoretic Benefits of Flexion-Distraction Technique

- Increasing IVD height
- Removal of pressure on the disc
- Centering of the nucleus of the disc, thus relieving disc pressure
- Restoration of normal motion to the spine
- Improvement of posture

BOX 7-6 Essential Steps of Flexion-Distraction Treatment

1. The patient is assisted into a prone position, with the ASIS positioned at the base of the thoracic section. The low back is then tested for tolerance to manual distraction. When tolerance has been tested and distraction is found to be tolerable to the patient, the ankle straps can be applied, increasing the traction force in the area of the proposed treatment.
2. Depression of the caudal section of the table is performed until tautness of the spinal musculature is felt by the clinician.
3. Contact is made and maintained on the spinous process of the vertebra immediately above the disc involvement.
4. Contact on the spinous process of the vertebra is to be maintained with one hand while the other contacts the handle or the foot of the table.
5. Traction is maintained by pressing on the caudal section of the table. Patient comfort should be maintained. The subsequent pumping of the caudal section creates a milking action of the disc and, according to Cox,¹⁵ speeds the recovery process.
6. The process outlined should be repeated to the patient's tolerance. The clinician may palpate a release at the noted vertebral level.
7. One more distraction session (the third) should be performed to patient tolerance for approximately 20 seconds.
8. Following treatment, the caudal section of the table is returned to the neutral position and secured, and the ankle straps are released.

ASIS, Anterior-superior iliac spine.

Motorized Lumbar Distraction (Leader Method)

A motorized traction table, such as the Leader table, can be used to assist in the production of lumbar traction (Figure 7-8). Traction to the lumbar spine is applied in the prone position while the pelvic section of the table produces continuous passive motion in the long axis of the spine. The clinician can apply a stabilizing pressure, using both hands over the spinous process of the segment to be distracted to produce a counterpressure against the distractive force produced by the table.

Manual Cervical Traction

Cervical traction can be applied manually or with mechanical assistance. Manual cervical traction is generally accomplished with the patient in the supine position. The clinician sits or stands at the head end of the table and establishes contacts with the fingers of both hands on the posterior aspect of the cervical spine. Contacts taken over the C5–C6 segment (Figure 7-9, A) will allow the neck to extend slightly to provide more distractive separation and stretch to the anterior structures (the disc and longus coli

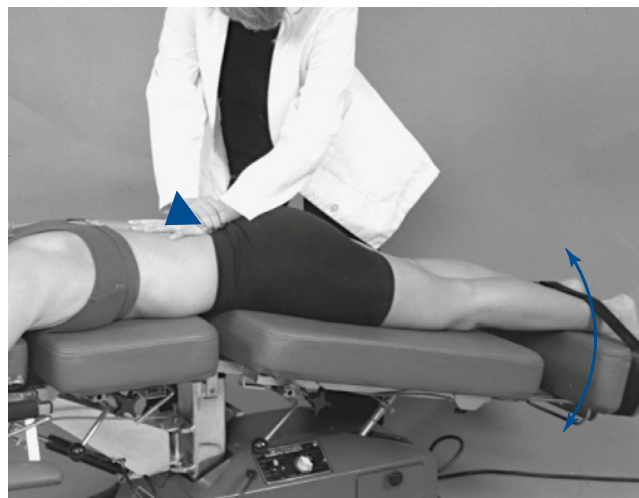


Figure 7-8 Leader lumbar distraction for the L4–L5 segment. Motorized pelvic section allows both hands to be used for contact.

muscles). Contacts taken under the base of the occiput (see Figure 7-9, B) will allow the neck to flex slightly to provide more distractive separation and stretch to the posterior structures (facets and paraspinal muscles). A towel may be substituted for hand contacts (see Figure 7-9, C).

Motorized Cervical Traction

A motorized traction table, such as the Leader table, can be used to assist in the production of cervical traction (Figure 7-10). Traction to the cervical spine is usually applied in the prone position while the pelvic section of the table produces continuous passive motion in the long axis of the spine. The clinician can apply a stabilizing pressure at the base of the occiput or anywhere in the cervical spine to produce a counterpressure against the distractive force produced by the table's moving pelvic piece (Box 7-7).

McKENZIE METHOD

The McKenzie method is most commonly associated with the promotion and application of lumbar extension exercises for the treatment of low back pain (LBP). Consequently, it is often viewed incorrectly as only a treatment method. The McKenzie method is both an evaluation and treatment approach to the management of painful spinal conditions. It is based on a structured and focused assessment of the effects of repeated movements and sustained postures on a patient's symptoms and spinal biomechanics. The information gained about the patient's symptomatic and mechanical responses to loading allows the clinician to determine which specific movements, positions, and activities to either pursue or avoid in the treatment plan. This information is specific to a particular patient at a specific point in time and provides reproducible objective and subjective criteria on which to base clinical decisions. It has been shown to reliably differentiate discogenic from nondiscogenic pain, and a competent from incompetent annulus.¹⁷ In comparison with magnetic resonance imaging, it demonstrates superior ability in distinguishing painful from non-painful discs.¹⁷

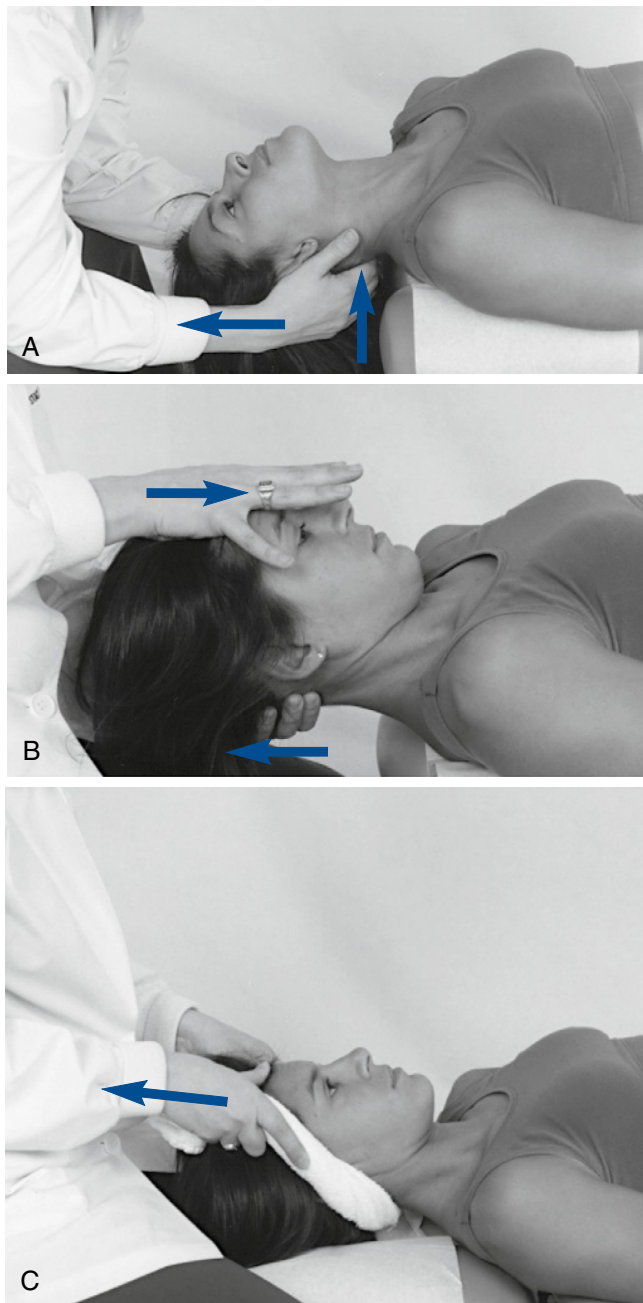


Figure 7-9 Manual cervical distraction. **A**, Contacts taken in the midcervical region to stretch anterior structures. **B**, Contacts taken at the base of the occiput to stretch suboccipital and posterior structures. **C**, Use of a towel as a substitute for hand contacts.

TREATMENT PRINCIPLES

The McKenzie approach to treatment is based on the application of mechanical forces and the patient's symptomatic and mechanical responses to those forces. The central premise is that those movements that decrease or "centralize" the patient's pain are the movements that should be applied in the treatment of the disorder. *Centralization* refers to the process of peripheral symptom resolution. With centralization, a patient's spine pain may persist

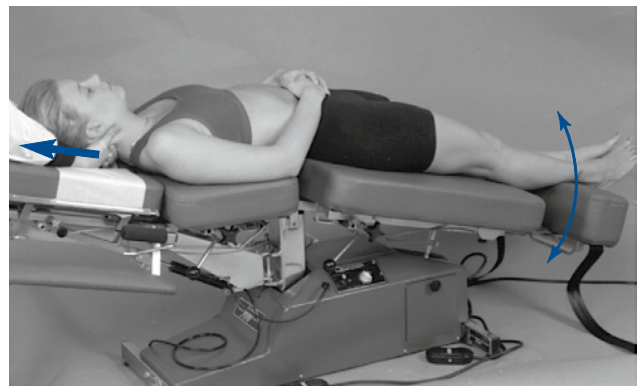


Figure 7-10 Motorized cervical distraction. Contact at base of occiput applying resistance to moving pelvic piece (may also be done in the prone position).

BOX 7-7 Motorized Cervical Traction

1. The patient lies in a supine or prone position on the table, with or without ankle straps fastened.
2. The clinician stands at the head end of the table if the patient is supine or at the side of the table, facing cephalically in a lunge position, if the patient is prone.
3. Contact is made at the desired level of the cervical spine or the base of the occiput.
4. The other hand reinforces the contact, maintaining the desired level.
5. The table is set in motion, producing flexion of the patient's pelvis and legs, creating traction to the cervical spine.
6. Resistance to the traction is maintained for three or four repetitions, and pressure is eased between tractions.
7. The table speed should be set relatively slow (10 to 12 cycles per minute).

or temporarily increase. Centralization has been shown to predict a beneficial clinical outcome.¹⁸ Increasing symptoms most distal to the spine are known as *peripheralization* and have been shown to correlate with a loss of competence of the disc annulus¹⁸ and predict a poor response to McKenzie treatment procedures. In the McKenzie treatment approach, there is a significant amount of self-applied therapy. The patient learns to appreciate the role of prophylactic procedures from the beginning of treatment and how to interpret which exercises, postures, or activities are beneficial or not. A primary consideration is that all applied procedures should be done in such away that the patient is able to self-treat more effectively. McKenzie¹⁹ suggests that clinician-applied forms of spinal manipulative therapy should not be performed on the entire population with back and neck pain, but rather on the minority of patients who have not been able to adequately resolve their symptoms through self-treatment.

THREE SYNDROMES: POSTURAL, DYSFUNCTIONAL, AND DERANGEMENT

Information obtained through the McKenzie evaluation allows for the classification of mechanical pain into one of three syndromes: postural, dysfunctional, or derangement. Each of the syndromes is considered a unique and separate disorder; however, they often coexist within the same individual. They are distinguished from one another by location of symptoms, presence or absence of acute spinal deformity, and effects of repeated movements and sustained end range positions altering pain patterns.²⁰

The postural syndrome characteristically has pain appearing after prolonged static loading, which causes overstretching and mechanical deformation of normal spinal tissue. Pain diminishes when the loading is removed. The aim of treatment is to correct posture, relieving the painful tension in normal tissues.

With the dysfunctional syndrome, pain appears immediately when shortened spinal tissues are mechanically deformed by overstretching. ROM is often decreased, and pain is increased at the end range of movement and decreased with the removal of end range stress.

The derangement syndrome is a product of anatomic disruption or displacement of the intervertebral segment. Pain may occur immediately with injury or develop over time and is often felt in the midrange of movement. The derangements are divided into one anterior and six posterior categories. Their history and repeated movement examination (Box 7-8) differentiate the three syndromes. Because the three identified syndromes of mechanical pain are separate entities, they each require a different approach to treatment.

Postural Syndrome

For those patients presenting characteristics of the postural syndrome, applying an understanding of the basic mechanics of postural stresses and their removal is fundamental in treatment. Once an abnormal posture has been identified as the source of the patient's symptoms, treatment is directed toward postural correction. The primary consideration is to develop within the patient the ability to control pain-producing mechanisms.

Slouch-Overcorrect Exercise (Figure 7-11). The patient sits in a slouched posture, provoking LBP. This posture is then corrected by forming an increased lumbar lordosis and retraction of the head. The patient should be able to feel that the pain can be abolished or at least reduced by changing the posture. Regular performance of the exercise encourages a postural awareness that reduces or eliminates pain of postural origins. The exercise can also be done in the standing position.

Dysfunctional Syndrome

Treatment of the dysfunction syndrome is directed at remodeling of adaptively shortened structures by stretching maneuvers. The most common pattern of the dysfunction syndrome is loss of spinal extension, except in the upper to middle cervical spine, where flexion is most likely to be limited.²¹ To achieve the required tissue response, end-range stretching procedures should be performed

BOX 7-8 Characteristics of McKenzie Syndromes

POSTURAL SYNDROME

Symptoms are often intermittent and worsen as the day goes on.

Pain is not produced on repeated movements.

Pain is produced with sustained load on normal tissues at the end of the full ROM.

Pain is present when the patient is stationary, and not present during repeated movements.

DYSFUNCTIONAL SYNDROME

The patient usually experiences a gradual onset of intermittent symptoms.

Pain is produced at the end of the ROM only.

The ROM is often decreased because of shortened tissues.

Pain stops on release of end range stress.

The pain pattern is fixed during testing (same end range pain).

Radiation of pain with nerve stretch is possible.

Condition is unchanged after testing.

No rapid and lasting changes result from testing.

DERANGEMENT SYNDROME

The patient often experiences rapid onset of acute symptoms.

Symptoms are produced or altered within movement range.

A painful arc of movement may exist.

The pain pattern varies during testing.

Progressive decrease or increase of pain during testing.

Centralization or peripheralization occurs during testing.

Condition becomes better or worse after testing.

Rapid and lasting changes result from testing.

ROM, Range of motion.

in a slow, repeated movement in sets of 10 to 15 every 2 hours throughout the day. The presence of significant degenerative changes or stretch irritation to neural structures may make the repetitive rate difficult to tolerate. The involved spinal region and the specific loss of movement pattern determine the movement chosen for treatment. In this case, correction of posture alone is not likely to enhance the lengthening of shortened structures. However, postural dysfunction, if present, must still be addressed because chronic poor posture often leads to tissue shortening and dysfunction. Moreover, when progress is slow or nonexistent, the use of manual procedures or other external forces is introduced. This can include the use of rhythmic end-range mobilizations designed to assist in the phenomenon of creep to lengthen soft tissue. However, these are used to augment, not replace, the exercise program. The repeated movements used in evaluation and treatment of the dysfunction syndrome include flexion, extension, and side-gliding in both the seated and lying positions (Figure 7-12). The treatment exercises will reproduce the patient's pain, but the increased pain should not persist for prolonged periods after treatment.

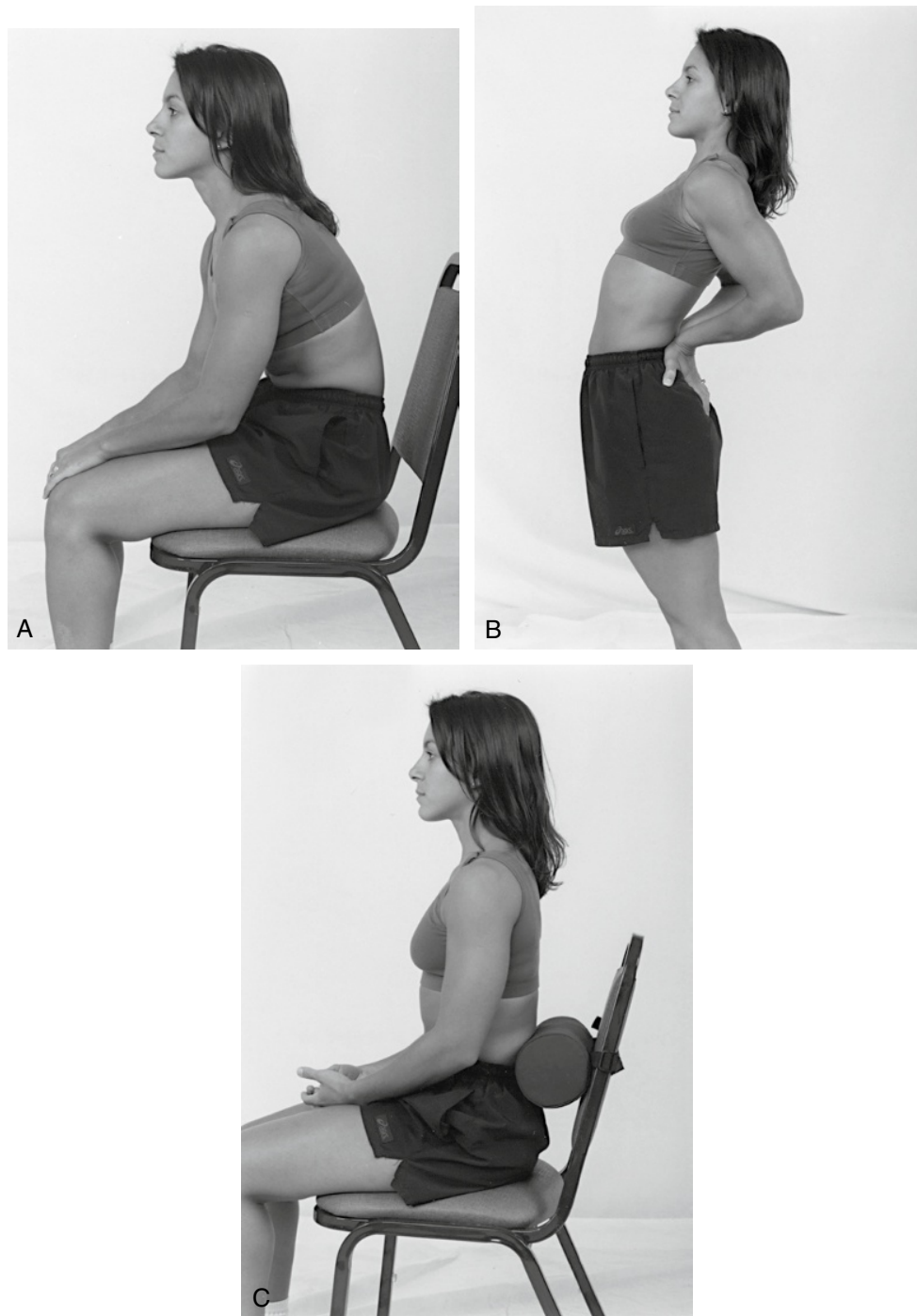


Figure 7-11 McKenzie method (slouch-overcorrect exercise). **A**, Patient slouches. **B**, Patient exaggerates the lumbar lordosis. **C**, Use of a lumbar roll while sitting.

Derangement Syndrome

For treatment of the derangement syndrome, a conceptual model of disc pathomechanics must be considered. The model is based on the premise that certain movements can be applied to reduce internal disc derangements and bulging intervertebral discs (IVDs). Movements that are successful in reducing disc protrusion are accompanied by a reduction in the patient's most distal symptoms (centralization) as the conflict between the protruding disc

and associated spinal nerve root is reduced. As symptoms centralize, it is also common for the patient's repeated ROM to increase with exercise. Using this approach, the suspected derangement patient is directed through a series of movements to ascertain which movements or positions will be most effective in reducing the segmental derangement. The evaluation results in the patient being classified into one of seven derangement categories. The first six derangements are variations of posterior disc disruptions,

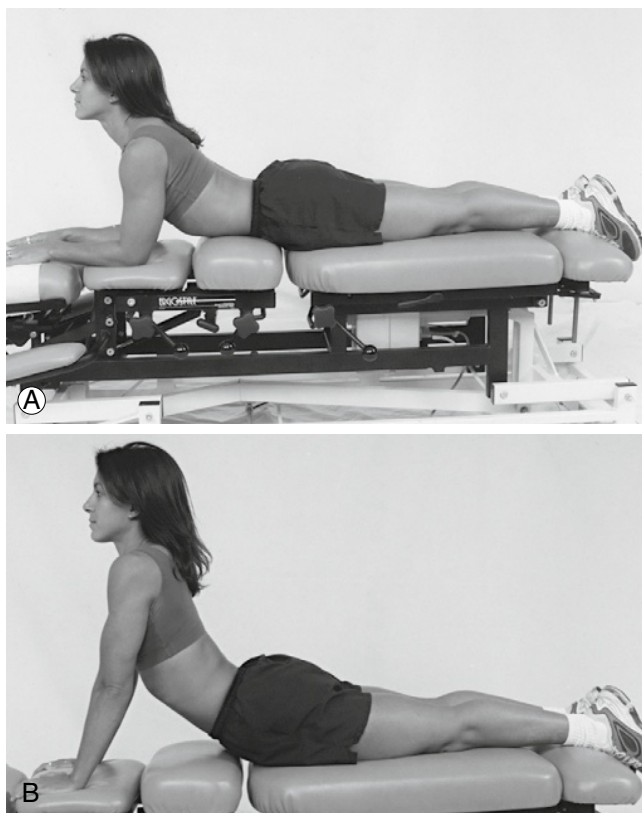


Figure 7-12 McKenzie method (prone lumbar extension exercises).
 A, Patient rises to forearms. B, Patient straightens arms.

often requiring extension reductions. In certain instances, coronal plane movements, such as side-gliding (Figure 7-13), or even rotational movements will be required for reduction. The seventh derangement is an anterior disc derangement, requiring a flexion reduction (Figure 7-14).

To complement the use of extension (or flexion) reduction exercises, avoidance of stressful static and dynamic flexion (or extension) postures and activities must be reinforced. These activities often cause peripheralization of the patient's symptoms, as well as painfully reduced ROMs. Recovery of function is considered complete when full normal repeated weight-bearing flexion produces no more than the expected strain at the end range of all movements.²¹

CRANIAL MANIPULATION

Cranial manipulation developed along parallel lines in both the osteopathic and chiropractic professions beginning in the mid-1930s. Misperceptions and misunderstandings have led to controversy over cranial manipulation. Much of the controversy comes from those who look at the cranium as a fused structure and therefore believe that manipulation has no basis. Cranial manipulation was begun in the chiropractic profession by Cottam and in the osteopathic profession by Sutherland. Both Cottam and Sutherland hypothesized that the cranial bones moved and formed the basis for most cranial techniques. The concept of cranial bone motion, and the therapeutic application of forces on the



Figure 7-13 McKenzie method (side-gliding movement). A, Clinician assisted. B, Patient exercise.



Figure 7-14 McKenzie method (flexion reduction exercise). The seventh derangement is an anterior disc derangement, requiring a flexion reduction.

cranium, is by no means universally accepted. However, the previously held belief that the cranial bones fuse in relatively early life has been shown to be faulty.²²⁻²⁴ Although the process of ossification starts in the mid-20s, sutures may remain open throughout life, and some motion flexibility may last into old age.²⁵ The cranial bones are connected and held together by connective tissue called the *sutural ligament* or *membrane*, which remains nonossified. Nerve fibers accompany the collagenous connective tissue, although their exact function has not been established.

There are three hypotheses regarding cranial motion. Sutherland suggested that there is a continuous, rhythmic, oscillating motion within the dural tube. Dysfunction in the dural tension system leads to lesions that are identifiable and correctable. A second concept is that the cranium moves in a spontaneous and unpredictable motion that is influenced by the pressures of vascular and cerebrospinal fluid (CSF) circulation. The third idea is that the cranium does not actually move, and stresses and pressures that can be influenced by cranial manipulation build up in the joints and bones.

Practitioners incorporating cranial manipulation purport to be able to palpate a rhythmic (8 to 14 oscillations per minute) cranial movement.²⁶⁻²⁸ However, there have been no interrater reliability studies published. Sutherland called the rhythmic movement within the dural system the *primary respiratory mechanism*. The pulsations within the dural tube are thought to aid in the circulation of CSF. CSF bathes and nourishes the central nervous system. The potential effects of cranial dysfunction are listed in Box 7-9.

Although the mechanism of cranial movement remains unknown, there is agreement that sutural asymmetry, jamming of the sutures, and pain and muscle tightness can be palpated.²⁵ The evaluation for cranial dysfunction includes observation for skull asymmetries; palpation of skull contours and the sutures themselves to identify widening, narrowing, or tenderness; and determination of the rate, rhythm, and amplitude of the craniosacral mechanism. The goal of cranial manipulation is to improve sutural motion, reduce membranous tension, improve circulation, and increase the vitality of the primary respiratory mechanism.

BOX 7-9

Potential Effects of Cranial Dysfunction

- Interference in CSF flow
- Blood pressure disturbances
- Irritation of sutural nerve pathways
- Cranial nerve entrapment
- Impeded vascular circulation to the brain
- Endocrine dysfunction
- Sensory disturbances
- Neurologic disorganization
- TMJ dysfunction

CSF, Cerebrospinal fluid; TMJ, temporomandibular joint.

Direct and indirect manipulative procedures have been described for the treatment of cranial dysfunction. Mild forces are used in conjunction with the patient's breathing pattern.

SAGITTAL SUTURE SPREAD (Figure 7-15)

With the patient in a seated or supine position, use digital contacts of your four fingers of both hands on either side of the sagittal suture. Use a gentle tractioning to separate the suture. This same procedure can be done on any of the sutures.

CRANIAL UNIVERSAL (Figure 7-16)

With the patient prone, grasp the mastoid processes with the thumb and middle finger of one hand. Place the fingers of the other hand over the occipital bone. Rotate both hands in the same direction, using mild pressure and repeating four or five times.

PARIETAL LIFT (Figure 7-17)

With the patient supine, apply digital contacts of the fingers of both hands on the inferior aspect of the parietal bone at the squamosal suture. Use the thumbs of both hands to hold at the parietal suture. Use the digital contacts, applying a mild lifting pressure and repeating four or five times.



Figure 7-15 Sagittal suture spread.



Figure 7-16 Cranial universal.



Figure 7-17 Parietal lift. Elevating pressure on the right parietal bone.

SOFT TISSUE MANIPULATION

Humans have been performing therapeutic procedures to the soft tissues for as long as humans have been able to touch one another. STM techniques are defined as those physical methods applied to muscles, ligaments, tendons, fascia, and other connective tissues with the goal of therapeutically affecting the body.²⁹ A common source of pain and disability is soft tissue injury with its resultant fibrosis and loss of elasticity and strength. Soft tissue injury and fibrosis may result from acute or repetitive trauma to muscular, tendinous, myofascial, or ligamentous tissue.³⁰⁻³² Although all manual techniques have some effect on the soft tissues, the justification for a separate STM classification is to draw attention to the prime importance of including techniques that have the specific purpose of improving the vascularity and extensibility of the soft tissues.¹¹ One of the signs of joint dysfunction and subluxation is the presence of muscle hypertonicity. Localized increased paraspinal muscle tone can be detected with palpation. Janda³³ recognizes five different types of increased muscle tone: limbic dysfunction, segmental spasm, reflex spasm, trigger points, and muscle tightness. Liebensohn³⁴ has discussed the treatment of these five types

using active muscle contraction and relaxation procedures. Reflex muscle spasm or splinting follows trauma or injury to any of the pain-sensitive structures of the spine. The pain-sensitive spinal tissues include the zygapophyseal joints, posterior ligaments, paravertebral muscles, dura mater, anterior and posterior longitudinal ligaments, and IVD.³⁵ Mechanical deformation or chemical irritation of any of these tissues will cause restricted motion by way of muscle spasm. Treatment directed at the tissue source of pain will reduce the reflex muscle spasm and increase the ROM; however, if the muscle spasm has been present for some time, it may require direct treatment as well.

Visceral disease can also cause reflex muscle splinting. The diagnosis of a viscerosomatic reflex is based on a history of visceral disease or current visceral disease symptomatology and objective palpation findings.³⁶ Objective palpation findings include two or more adjacent spinal segments that show evidence of fixation located within a specific autonomic reflex area; a deep paraspinal muscle splinting reaction; resistance to segmental joint motion; and skin and subcutaneous tissue changes consistent with the acuteness or chronicity of the reflex.³⁶

EFFECTS OF SOFT TISSUE MANIPULATION

When considering the use of treatment procedures directed toward the soft tissues, a working knowledge of the fundamental principles behind them is necessary and helpful. Manual procedures applied to the soft tissues are intended to enhance proper tone and extensibility or both through a number of proposed mechanisms. Although specific and singular effects are presented, invariably it will be a combination of effects that occur.

Blood Flow and Temperature Effects

STM has been purported to produce an increase in blood flow and cutaneous temperature. Deep stroking and kneading of the soft tissues in the extremities of normal patients, patients with rheumatoid arthritis, and patients with spasmodic paralysis create a consistent and clinically significant increase in blood flow and cutaneous temperature.³⁷ These findings are supported by other studies.³⁸⁻⁴⁰ However, it must be emphasized that the clinical procedure being tested in all of these reports was a deep or heavy massage application. Therefore, conclusions on the effects of light-force stimulation of the body wall cannot be drawn from these data.

Massage can induce a fall in blood viscosity, hematocrit count, and plasma viscosity. Ernst, Matrai, and Magyarosy⁴¹ found massage to equal pharmacologic agents used to treat blood flow problems. The proposed mechanism may be hemodilation resulting from either an increase in blood plasma volume or reactive hyperemia. Therefore, the effect of massage and circulation appears to be an increased perfusion of blood with plasma fluid, allowing for improved blood flow.

Metabolic Effects

Cuthbertson⁴² performed a literature review on the effects of massage on metabolic processes, including vital signs and waste products of the body. He reported that in normal subjects there was no increase in basal consumption of oxygen, pulse rate, or blood pressure, although an increase in urine output was observed. His conclusion hypothesized that to effect a change in the vital signs, a

systemic effect must be achieved and that the massage procedures used did not do it. Schneider and Havens⁴³ did find that STM produced an increase in red blood cells needed to bring oxygen to the tissues. This provides some support for soft tissue procedures being able to increase circulation and nutrition to desired areas. Again, these were vigorous massage procedures described, so caution is necessary when trying to apply these principles to other procedures.

Massage therapy has been further evaluated as a treatment for reducing blood pressure. Hernandez-Reif and colleagues⁴⁴ reported that 10 30-minute massage sessions over 5 weeks reduced diastolic blood pressure, as well as anxiety and depression, to a statistically significant level over a control group receiving progressive muscle relaxation instructions. These findings supported an earlier study also demonstrating a decrease in diastolic and systolic blood pressure.⁴⁵ In a study of light massage of preoperative patients, it was reported that there was a relaxation response from the parasympathetic nervous system producing a decrease in both blood pressure and heart rate, as well as an increase in skin temperature.⁴⁶

Reflex Muscle Spasm Effects

Changes in muscle length and tension are monitored by two stretch receptors: muscle spindles and Golgi tendon organs (GTOs). The muscle spindle has a highly sensitive filament, the annulospiral ending, that fires rapidly and with high velocity with the smallest change in length. It also has smaller filaments and slower spray receptors, which are slower in response and more likely to respond to the magnitude and speed of stretch. The GTOs are located at the junction of the muscle and its tendon and monitor the tension exerted on the contracting muscle or imposed by external forces. Massage procedures can cause an overload of the GTOs, resulting in reflex inhibition and muscle relaxation.⁴⁷ Lumbar muscle strain with concomitant hypertonicity has been treated by many means with mixed results. Yu⁴⁸ reports on 55 cases treated with massage therapy that showed good results in decreasing pain, inflammation, and hypertonicity. In a review article, Goats⁴⁹ reports on the effectiveness of massage therapy for reducing muscle spasm. Massage was shown to significantly diminish motor neuron excitability when compared with controls, forming the basis for suggesting that massage can lead to muscle relaxation.⁵⁰ The effect of massage intensity was also studied.⁵¹ Light-pressure and deep-pressure massage was applied to the triceps surae muscle. H-reflex amplitudes were significantly reduced in both massage groups as compared with the control group. Moreover, deep massage reduced the H-reflex more than the light massage, suggesting that the mechanism involved is pressure-sensitive.⁵¹

SPECIFIC TECHNIQUES

What follows is a description of manual therapies used by chiropractic physicians and other health care providers to primarily influence the soft tissues of the body. It is not intended to be a compendium of all STM and reflex techniques. The procedures presented have been selected to illustrate the fundamental concepts applicable to many of the forms of STM and to present the specific application of commonly applied procedures. One of the main functions of soft tissue therapy is to prepare the irritated

region for subsequent mobilizations or manipulations. STM can be stationary (done on one spot) or progressive (done by moving hands from one place to another). Moreover, it may be variable in the intensity of pressure exerted, surface area treated, and frequency of application.

STM includes massage (stroking or effleurage, kneading or pétrissage, vibration or tapotement, and transverse friction massage), connective tissue massage, trigger point therapy (Nimmo technique), myofascial release techniques (MRTs; muscle energy techniques [METs], postisometric relaxation [PIR], and proprioceptive neuromuscular facilitation [PNF]), and body wall reflex techniques (Chapman lymphatic reflexes, Bennett vascular reflexes, and acupressure point stimulation) (Box 7-10).

Massage Techniques

Massage therapy is older than recorded time, and rubbing was the primary form of treatment until the pharmaceutical revolution of the 1940s.⁵² Today, classical or traditional massage procedures form the basis for many other procedures. Simply defined, massage consists of hand motions applied to the surface of the body with a defined therapeutic goal.⁵³ In a more clinical or practical definition, *massage* is a term used to describe certain manipulations of the soft tissues; it is a form of manipulation most effectively performed by the hands and administered for the purpose of producing effects on the nervous, muscular, circulatory, and

BOX 7-10 Forms of Soft Tissue Manipulation

MASSAGE TECHNIQUES

- Effleurage (stroking)
- Pétrissage (kneading)
- Tapotement (vibration)
- Roulomont (rolling)
- Friction or transverse friction massage
- Connective tissue massage

FUNCTIONAL TECHNIQUES

- Strain-counterstrain
- Positional release technique
- MRT

MANUAL RESISTANCE TECHNIQUES

- MET
- PIR
- PNF
- Ischemic compression
- Trigger point therapy
- Spray and stretch
- Receptor-tonus technique (Nimmo)

BODY WALL REFLEX TECHNIQUES

- Acupressure point stimulation
- Chapman lymphatic reflexes
- Bennett vascular reflexes

MET, Muscle energy technique; *MRT*, myofascial release technique; *PIR*, postisometric relaxation; *PNF*, proprioceptive neuromuscular facilitation.

lymph systems.⁵⁴ The variations of massage movements include effleurage, pétrissage, roulomont, tapotement, and friction. With most forms of massage, some form of lubricant is useful. It is recommended that for short sessions, a water-based lotion be used, because it will be absorbed by the skin. For longer sessions, an oil-based lotion is preferred.

Effleurage. *Effleurage* is a French word that means gliding or stroking, and it is applied over a large area using broad contacts. It may be deep or very superficial, creating general relaxation and a superficial warming as a result of a mild erythema. During a single therapeutic session, it is desirable and recommended to begin and end with effleurage. This maneuver is a slow rhythmic stroking in which the hands of the clinician make light contact with the skin of the patient (Figure 7-18). A broad palmar contact is used over large surfaces, but the thumbs or fingers can be used over smaller areas. The clinician's hand should be sufficiently relaxed to mold itself to the area being treated as it passes almost insensibly from distal to proximal over the region being treated. Hand pressure is evenly dispersed, and the degree of pressure varies with the size and the region of the part being treated. The strokes are made in long, gliding sweeps. The movement should be slow, at the rate of about 15 sweeps per minute, with the returning stroke traveling a little faster than the treating stroke. Effleurage produces a soothing relaxation and mild hyperemia for the patient while decreasing pain in the superficial soft tissues and reducing muscle tension (Box 7-11).

Pétrissage. *Pétrissage* involves grasping the skin and underlying muscular tissue while applying a cross-fiber stroking or stretching action to the tissue beneath. *Pétrissage* is a French word for kneading, although it has also been called *pinching*. This technique is directed at improving the tissue-fluid exchange, vascularity, and normal texture of subcutaneous and deep soft tissue. This is accomplished through alternate traction, or taking up or squeezing and relaxing movements of a localized mass of tissue held between the thumb and fingers. The hands raise a large fold of skin and underlying muscle between the thumbs and other fingers (Figure 7-19). The tissues are rolled, squeezed, and raised by alternately tightening and loosening the grasp. As the hold is loosened, the tissues are allowed to fall back to their original position because of their elasticity. The fingers should be held close together. The hands work together, changing the direction of the torsion. If the hands slip over the surface or pinch the skin, the maneuver will be painful.

Pétrissage is thought to diminish swelling and fluid accumulations, produce hyperemia in muscle, and improve elasticity and contractility of connective tissue. Furthermore, *pétrissage* can decrease muscle tone as the lifting, rolling, and squeezing action affects the spindle cell proprioceptors in the muscle belly. As the muscle belly is squeezed, the muscle feels less stretched. The lifting action produces stretch in the tendons, causing a potential reaction by the Golgi tendon receptors. When these two phenomena occur, the sensory input can reflexively relax the muscle. *Pétrissage* also has the mechanical effect of softening and creating space around the actual muscle fibers and making the tendons more pliable.⁵⁵ Moreover, it has the potential to break cross-linkage adhesions that have formed between fascial planes, connective tissue, or muscle fibers (Box 7-12).

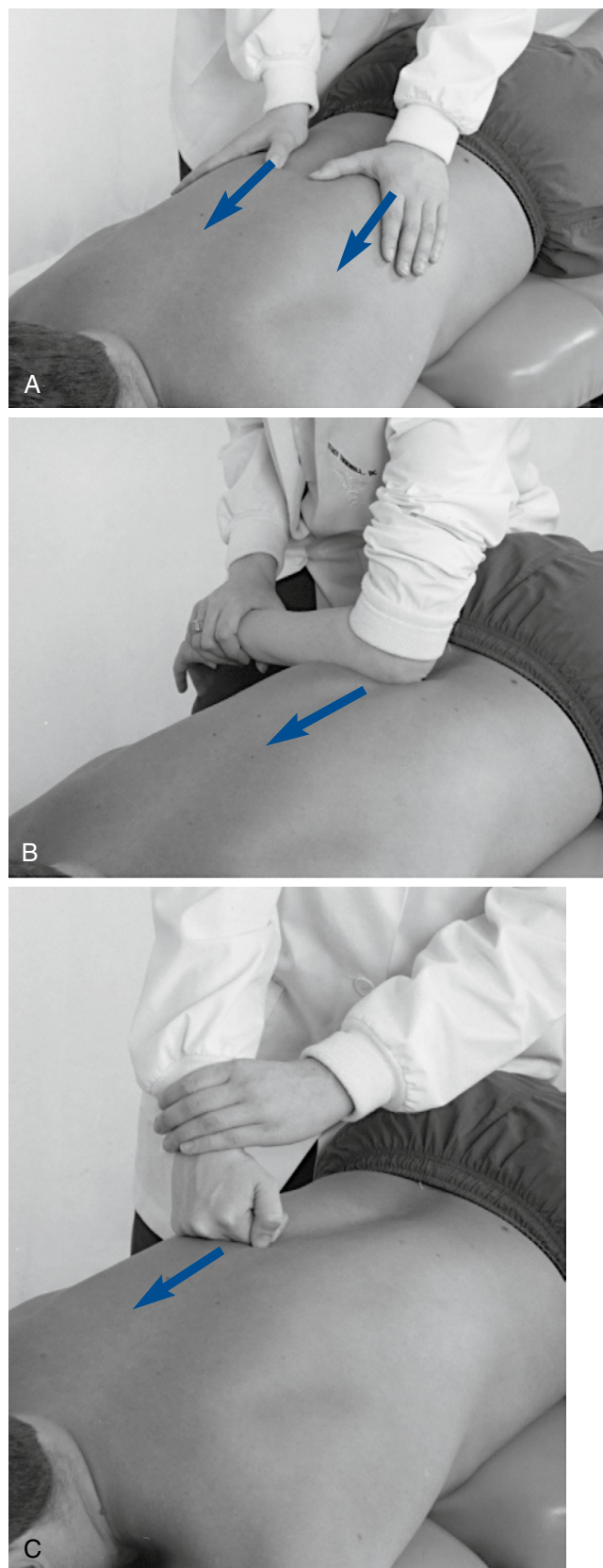


Figure 7-18 Effleurage (focus is along muscle fibers). A, Double hand contacts. B, Forearm contact. C, Loose fist contact.



Figure 7-19 *Pétrissage* (focus is across muscle fibers). **A**, Two hands grasping. **B**, Grasping and lifting tissue.



Figure 7-20 *Roulomont* (skin rolling). A quick tugging is applied to pull the skin away from fascial adhesions.

Roulomont. *Roulomont*, or skin rolling, lifts the skin away from fascial surfaces beneath; when adhesive areas are encountered, a pull is applied to the skin to allow freer movement (Figure 7-20). Skin rolling is a procedure suitable for long muscles. Skin rolling

BOX 7-11 Effects of Effleurage

Relaxation
Improved circulation
Hyperemia

BOX 7-12 Effects of Pétrissage

Reduction of pain
Release of adhesions
Reduction of edema
Increased tissue temperature

BOX 7-13 Effects of Roulomont

Release of adhesions
Reflex stimulation of cutaneous receptors
Increased tissue temperature
Softening of superficial fascia

has been considered a variation of *pétrissage*. However, with skin rolling, only the skin is lifted from the underlying muscle layer; *pétrissage* attempts to lift the muscular layer. Skin rolling has a warming and softening effect on the superficial fascia and can cause reflexive stimulation of cutaneous receptors. It can also be used directly over the spine. Areas where the skin does not easily come away from the fascia may have an underlying joint dysfunction problem (Box 7-13).

Tapotement. *Tapotement* is described as a tapping or vibratory action applied to the soft tissue in a rapid fashion creating a stimulatory effect. The tapping vibration is produced typically through a rapid series of blows by the hands that are held with the palms facing each other. The fingers and wrist remain relaxed as the elbows flex and extend. The ulnar borders of the hands and the fingers produce a rapid multiple percussive stimulation over the area being treated (Figure 7-21, *A*). In addition, the fingertips can be used to produce a compressive tapping by rapidly alternating wrist flexion and extension (Figure 7-21, *B*). This procedure is most useful in the extremities. A variation of tapping uses the cupped hand to produce a deep percussive vibration especially useful over the thorax and abdomen (Figure 7-21, *C*). The vibration is applied perpendicular to the muscle fibers, with an impulse frequency of 8 to 10 sinusoidal vibrations per second. The resulting tonal effect may not immediately be apparent; the average time needed before results can be seen is 2 to 5 minutes. Vibration must be done long enough and at a sufficient intensity to produce reflexive physiologic effects. Vibration is used to tone muscles and to produce sensory stimulation. It may be given with the entire hand or with the fingertips, depending on the area to be treated. Rapid continuous vibrations are transmitted to the skin through movements of the hands. Initially, the hand contacts



Figure 7-21 Tapotement (tapping percussion). **A**, Double loose knife-edges. **B**, Fingertips. **C**, Cupping.

BOX 7-14 Effects of Tapotement

Hyperemia
Improve muscle tonicity
Reflex stimulation of cutaneous receptors

produce tissue compression, followed by the trembling form of vibration produced by alternating contraction and relaxation of forearm muscles (Box 7-14).

Friction. Friction is the application of moderate, steady pressure, typically with the palmar aspects or edges of the thumbs in small areas. During the application of friction massage, the thumb induces rapid side-to-side or small circular movements, moving the skin over the subcutaneous tissues and muscular layer (Figure 7-22, *A*). The fingers, palm, or flat part of the elbow can be used as alternative contacts (Figure 7-22, *B* and *C*). The purpose of using friction is to move tissue under the skin; therefore, lubricant is usually avoided

to prevent sliding of the tissues. The goal is to induce rapid transverse movements through the underlying myofascial or ligamentous tissue (Box 7-15).

When applied vigorously across the fibers of the tissue treated, it is called *transverse friction massage*. The treatment goal of friction is to break up adhesions and to encourage absorption of exudates. The focus of friction is to produce a controlled inflammatory response that causes heat and redness from the release of histamine and increased circulation. Although a small amount of edema will occur as water binds with the connective tissue, there should not be any bruising. Friction can also be used in combination with compression and passive joint movement. This is considered a form of myofascial release and is discussed in that section.

Friction massage is a vigorous procedure and because of the forceful nature, the patient's tolerance must be considered. As the procedure is applied, a "friction anesthesia" will be produced, allowing the contact pressure to increase. Friction massage is generally performed for 30 seconds to 10 minutes, depending on the patient's tolerance and treatment goals. Friction is a mechanical approach best applied to areas of high connective

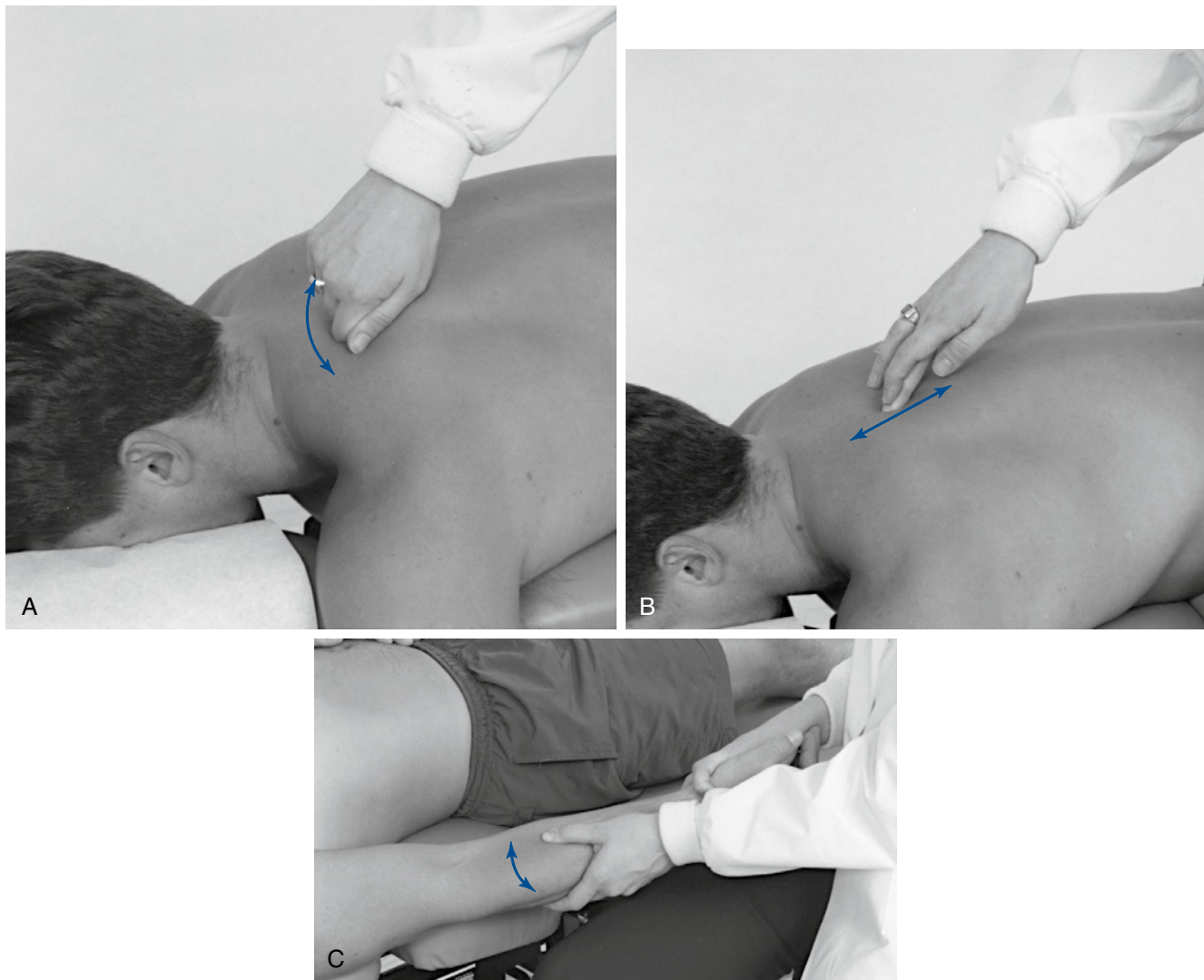


Figure 7-22 Friction. **A**, Edge of supported thumb moves rapidly across muscle fibers (levator muscle). **B**, Stabilized fingers move across muscle fibers (rhomboids). **C**, Forearm and elbow stretch along muscle fibers and move across fibers.

BOX 7-15 Effects of Friction

- Stretching or release of adhesions
- Reduction of edema
- Reduction of fibrosis
- Dispersion of pathologic deposits
- Reduction of pain
- Diminished muscle spasm

tissue concentration, such as the musculotendinous junction. Consideration can be given to whether the myofascial tissue in the area to be treated should be in a stretched or relaxed position.

Connective Tissue Massage

Elizabeth Dicke, a German physical therapist, developed connective tissue massage in 1929. Ebner⁵⁶ defines *connective tissue massage* as a form of STM carried out in the layers of connective tissue

on the body surface. This stroking technique is a form of friction that consists of a tangential pull on the skin and subcutaneous tissues away from the fascia with tips of the third and fourth fingers. It is applied to identified segmental changes in the tension of the skin and subcutaneous and other connective tissue corresponding to the location of head reflex zones. *Head zones* are hyperalgesic areas of the skin speculated to be referred from diseased internal organs. The systematic application of connective tissue massage is time-consuming to learn and apply; however, aspects of this technique can be used without applying the whole system. This technique may have both a reflex (circulation and pain) and a physical (stretching and mobilizing connective tissue) effect.

The essential feature of connective tissue massage is that a tensile strain is applied to the connective tissue to produce the desired physical and reflex effects. The patient is most commonly in a seated position with the knees and hips at right angles. However, the side-posture, prone, and supine positions may also be used. Assessment begins with observation for symmetry, followed by

palpation. The palpatory procedure moves the skin against the deep fascia by using the middle and ring fingers to produce small pushing strokes. Comparison is made from side to side, with asymmetric increased tension indicating a problem. The stroke produces a pressure that causes a bulge of tissue. Alteration in the tension of connective tissue will change the size of the bulge and the sensation produced, indicating a compromise in the fascial tissue. A combination of short and long strokes applied to specific regions of the body are done to locate fascial changes. One commonly used procedure incorporates a long vertical stroke performed by the middle and ring fingers, starting along the L5 segment and pulling up along the spine to C7.

Treatment is applied with a long or short stroking action, carried out using the second and third fingertips of the relaxed hand to draw the skin slack (Figure 7-23, *A* and *B*). A pulling stroke similar to the one used in assessment is used for treatment. Sufficient tension must be developed between the fingers in the skin; therefore, a lubricant is not used. The treatment strokes start at the sacrum and work up along the trunk and neck. Although connective tissue massage exerts an effect using the skin and subcutaneous tissue, it is primarily a superficial form of STM on the lighter end of the depth spectrum. Cantu and Grodin⁵⁷ suggest that connective tissue massage can be applied to individuals who have hypersensitivity of the autonomic nervous system, such as in the case of reflex sympathetic dystrophy. The purported effects include a marked hyperemia and sweat gland stimulation. Bruising sometimes occurs after treatment, depending on the degree of capillary fragility.

The mechanism for the effect of connective tissue massage is based on a reflex hypothesis. This procedure is purported to cause a release of a histamine-like substance that acts on the autonomic nervous system. Histamine acts as a powerful vasodilator of small blood vessels and causes an increase in vascular permeability that leads to increased fluid loss from fine blood vessels. It is thought that the mechanical stimulation of the pulling stroke on the connective tissue is an adequate stimulus to elicit the proposed reflex. Excitation of neurologic receptors in the skin produces impulses that travel from the skin either through the somatosensory spinal nerve via a posterior root ganglion to the gray matter or over the vascular plexus to the same segmental sympathetic ganglia or to the ganglia of the neighboring segment.⁵⁸

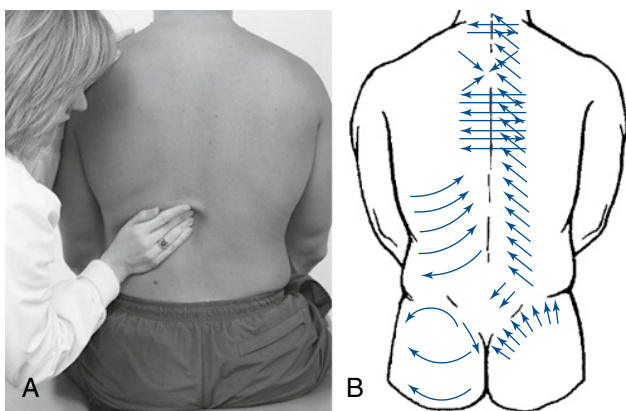


Figure 7-23 Connective tissue massage. **A**, Second and third fingertips draw skin slack. **B**, Pattern of long and short strokes.

Functional Techniques

This group of techniques focuses on the interrelationship of structure and function in the body with the tendency toward self-regulation.⁵⁹ These procedures place emphasis on motion rather than position and quality of movement rather than quantity of movement. The theory is that dysfunctional motion segments alter neural input, which in turn produce abnormal efferent signals, particularly to the NMS system.⁵⁹ Functional technique restores normal ease of movement and reduction of abnormal afferent input. These techniques have in common the goal of reducing exaggerated mechanoreceptor and nociceptor input and spindle responses from facilitated segmental muscles, thereby restoring joint mobility and muscle tone. Typically, the involved joint is passively positioned so that the facilitated muscle spindle is shortened, reducing the afferent discharge.

Strain-Counterstrain. Strain-counterstrain uses muscle positioning based on concepts developed by the osteopath, Lawrence Jones, to assist in the reduction of trigger point (tender point) pain, muscle spasm, and joint immobility.⁶⁰ Dr. Jones noted that acute joint problems were associated with tender points that he called *trigger points*, although they were not the same as Travell's or Nimmo's concepts (see pages 7-28-32). Dr. Jones also serendipitously discovered that patients' painful complaints could be permanently relieved if the patients were placed for a period in a position of comfort ("position of ease"). From this accidental discovery, Dr. Jones went on to develop a method of examination and treatment procedures used to identify offending triggers (tender points) and inhibit them by moving the offending joint or limb away from pain to a sustained position of comfort.

This procedure is based on the hypothesis that the initially strained (stretched) muscle is not necessarily the only source of the patient's continued discomfort. Rather, it is theorized that abnormal input and sustained contraction from the muscle that is antagonistic to an overstretched muscle may be the source of continued pain and stiffness. This purportedly occurs during an overstretching injury when the antagonistic muscle is suddenly and maximally shortened, producing an increase rate of gamma motor neuron firing. The initial increased rate of gamma motor neuron activity is theorized to induce sustained contraction of the muscle spindle within the affected muscle. The resultant tension in the intrafusal mechanism causes excitation of the central nervous system, stimulation of alpha motor neurons, and the maintenance of extrafusal fiber contraction. When the involved muscles return to a normal resting length, abnormal tension is maintained as a result of abnormal spindle activity. This produces a cycle of increased spasm and tension in the antagonistic muscle and abnormal pulling forces against the apposing muscles.

Counterstrain theory proposes that restoration of pain-free movement can only be established when the antagonistic muscle is coerced to return to a normal resting length. This is accomplished by placing the involved muscle in a position of maximal shortening, followed by a stretch and slow return to neutral length.⁶⁰ The position is held for 90 seconds to allow gamma motor neuron activity to decrease and the spindle to reset to normal activity status.

Positional Release Therapy. *Positional release therapy (PRT)* is a form of counterstrain developed by D'Ambrogio and Roth.⁶¹ Its major characteristics include specific body positioning, the use of tender points, and an indirect approach to treatment. Treatment using PRT is accomplished by placing the involved tissues in an ideal position of comfort. This is designed to reduce the irritability of the tender points and normalize the tissues associated with the dysfunction. This ideal position is determined subjectively by the patient's perception of tenderness and objectively by the reduction in palpable tone of the tender point. PRT theoretically addresses neuromuscular hyperirritability and muscular hypertonicity as mediated by the proprioceptive system.⁶¹

These procedures involve localization of a sensitive myofascial tender point followed by positioning of the muscle and related joint into a position of comfort or ease (Figure 7-24, A and B). Pressure is applied to the tender point while the muscle and related joint are maintained in the position of maximal relaxation. The abnormal reflex and resultant muscle spasm are inhibited by the tender point pressure and opposite counterstrain created by the positioning. The pressure and position are held for 90 seconds, followed by a slow passive return to neutral. A return to normal length of the muscle is achieved through the positional release of abnormal neural reflexive activities. This has led to this procedure also being called *positional release technique*.

Myofascial Release Technique. Although used for decades, very little has been documented or written about MRT. MRT procedures are the product of a variety of concepts originating from orthopedics, physiotherapy, osteopathy, and chiropractic. Today MRT is most frequently associated with physical therapist John F. Barnes. Barnes⁶² defines *MRT* as a whole-body, hands-on approach for the evaluation and treatment of the human structure, focusing on the fascial system. MRT evaluation of the fascial system includes analysis of the human frame by palpating the tissue texture of the fascial layers and observing the rate and rhythm of the craniosacral mechanism.⁶² Release, in the myofascial release

concept, is the tissue relaxation (including muscle relaxation) that follows adequate application of stress on the tissue.⁵⁹

The goal of myofascial release treatment is to establish functional three-dimensional whole-body symmetry and motion by removing fascial restrictions and restoring the body's equilibrium.⁶³ The indicated application of this method requires the determination of the best location of entry into the musculoskeletal system, selection of the most suitable types of stress to induce the inhibitory effect, and sensitivity in palpation to react properly to tissue response.⁶⁴

MRT is reported to reduce muscle spasm and fascial restrictions, eliminating irritation to these pain-sensitive structures and allowing increased ROM.⁶² Myofascial release is thought to soften connective tissue, rehydrate it, and remold it. This is done by spreading the tissue, pulling or stretching it, and creating motion where the tissue was adhered. The effect of myofascial release is a relaxation of tissue tension and subsequent decrease in myofascial tightness. Therefore, MRT is used to normalize myofascial activity, regain tissue extensibility, and reduce pain.

MRT is performed by applying carefully directed manual forces against soft tissue barriers (Figure 7-25). Deep, long, gliding strokes using the palms, fists, or forearms to engage various layers of the fascia are used to spread and lengthen the fascia. A lifting and twisting motion is used more often than compression.⁶⁵ When a fascial restriction is identified, gentle pressure is applied in the direction of restriction. This pressure pulls the elastic fibers, producing a springy feel. As the stretch continues, a firm barrier will be engaged, representing the collagenous connective tissue component. Sustained pressure is maintained until a "release" is felt.⁶² Connective tissue stretching occurs when the tissue is taken to a point of resistance and then stretched. This is usually done across fibers with the muscle in a specific position. Box 7-16 identifies the ways that myofascial releases can occur. MRTs are not always painless, although the initial discomfort typically subsides as the contact is maintained over time.

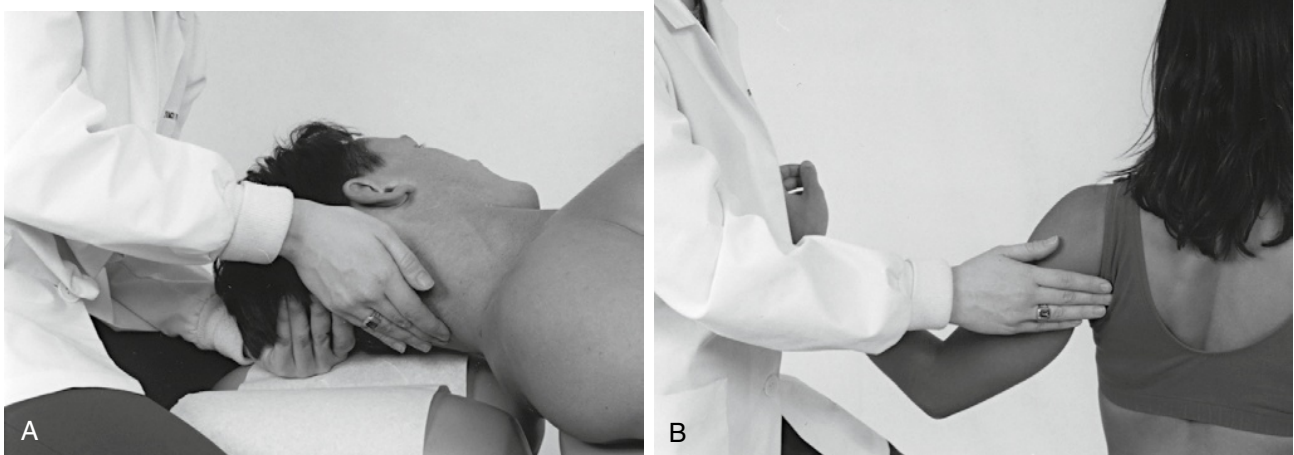


Figure 7-24 Strain and counterstrain (positional release). **A**, Tender point located on the posterior aspect of the C3 articular pillar. Patient's head is laterally flexed and rotated away. **B**, Tender point located at the upper lateral border of the scapula. Patient sits with arm at the side and elbow bent. Clinician grasps the forearm and extends, adducts, and externally rotates the shoulder.

BOX 7-16 Ways Myofascial Releases Can Occur

Forcible separation or compression of joints
 Forcible loading of asymmetrically tightened tissue
 Myotactically controlled mechanoreceptor responses
 Muscle tightening and asymmetries

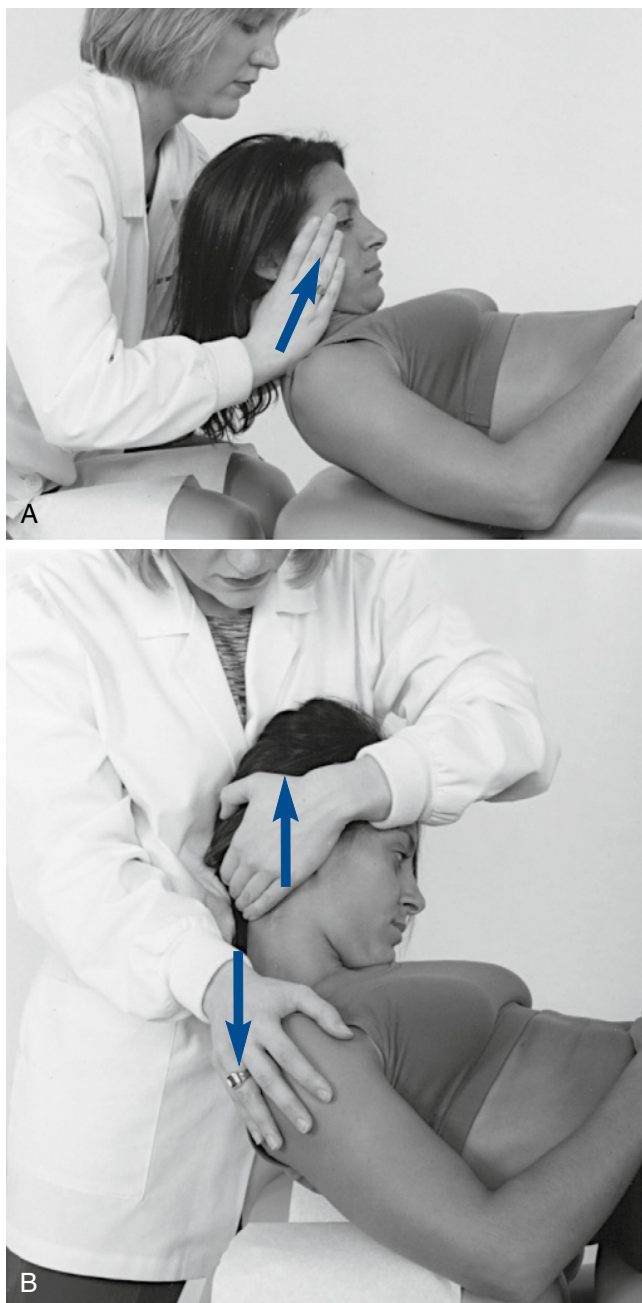


Figure 7-25 Myofascial release (Barnes method). **A**, Cervical release to relieve fascial restrictions through the neck and upper back. Patient's occiput is cradled in the clinician's hands with cephalic traction sustained without producing too much flexion. **B**, Alternate cervical release emphasizing the sternocleidomastoid and levator areas.

Active MRT (active release technique [ART]) is a modification promoted by Leahy.⁶⁶ Leahy asserts that ART is best suited for treatment of soft tissue conditions that fall under the category of cumulative injury disorders.⁶⁶ These problems result from acute injury, repetitive injury, or a constant pressure-tension injury. The soft tissues are examined for changes in texture, tension, movement, and function. When any of these factors are altered, there are changes in the mechanical function of the joints and soft tissues. The identified affected tissue is placed in a shortened position at the start of treatment. The soft tissue lesion is contacted, pressure is applied, and the tissue is stretched through slow active or passive movements (Figure 7-26).

Manual Resistance Techniques

This general classification of soft tissue manipulative techniques includes those procedures that have varying degrees of contact pressure and joint movement or positioning. They also use varying degrees of muscle contraction from none to strong



Figure 7-26 Active myofascial release (Leahy) for the left supraspinatus muscle. **A**, Starting position, with patient's arm abducted and double thumb pressure applied to muscle. **B**, Ending position, with patient actively and slowly lowering and adducting the arm while pressure is maintained.

sustained contractions. Some use light or no contact pressure over painful areas, and others use heavy pressure. This variability has the advantage for these procedures to be applied based on the clinical presentation (acute or chronic) and patient ability (tolerance). These procedures have similar characteristics, which leads to some confusion in terminology and specific intent.

Muscle Energy Technique

MET was developed by Fred Mitchell, D.O., as a procedure that incorporates the use of the patient's muscular effort in the application of the procedure. The primary effect of MET is to influence joint function, although there will certainly be effects on the muscles being used as well. The aim of MET is to restore normal joint position and function while influencing proper posture. Mitchell's basic principle is that voluntary muscle contractions are exerted against a precisely executed counterforce to loosen the specifically localized joints for passive articulation during post-contraction relaxation.⁶⁷ Patient-initiated muscle contractions are used at varying intensities from a precisely controlled position, in a specific direction, against a distinctly executed counterforce. The corrective process requires active patient participation, encouraging the patient to assume responsibility for self-care.⁶⁴ METs are applied with facilitation and gravity during the lengthening phase, classically with no passive stretching performed by the therapist. However, many modifications and variations have led to different applications of this procedure and significant confusion in nomenclature, as well as in proposed therapeutic intent.

METs involve two opposing forces, the patient's and the clinician's, producing isometric or isotonic contractions or both. Variability in the direction, type of contraction (isometric or isotonic), patterns, and amount of force produced by the patient or the clinician serves to distinguish the various forms of MET. To effectively deliver MET procedures, the restrictive barrier must be found and accurately engaged.

The *barrier* phenomenon was first described in relationship to joints, but it can be applied to nearly all structures of the locomotor system. The barrier concept provides an important distinction between massage procedures and manipulative procedures for the soft tissues. The barrier is perceived by the clinician when the slightest resistance is engaged. Slight overpressure should produce a springing movement characteristic of elastic. Characteristics of specific dysfunctional barriers are identified in Table 7-1.

The barrier phenomenon makes accuracy of diagnosis and positioning very important. Localization of the force to the affected spinal level is more important than the amount of force and counterforce (Figure 7-27). Full maximal contraction of the muscles is not required for this method of joint treatment, and often very light force is desirable. It is sometimes necessary to caution the patient not to use too much force. The patient should use only enough force to match that provided by the physician. If excessive force is used, accessory muscles will be brought in, and this detracts from the effectiveness of the treatment on the specific tissues that need to relax. The procedure for applying muscle energy for joint mobilization is detailed in Box 7-17.

Postisometric Relaxation

METs have undergone significant modification and evolution with many variations. With modification, METs can increase joint mobility and muscle flexibility. PIR is one such evolution. The focus of PIR techniques is on the muscular system and on inducing muscle lengthening, whereas MET focuses primarily on joint changes. However, both MET and PIR affect the joint and related muscles. *PIR* refers to the effect of subsequent relaxation experienced by muscle after brief periods of isometric contraction. PIR techniques tend to be simplistic and gentle and can be given as home treatment. Because PIR has evolved from MET, both procedures are being used interchangeably. Moreover, modifications to PIR techniques have also occurred.

MET techniques are applied to aid in the lengthening of shortened tissue by reducing the limiting effects of active muscle contraction and contracture. These procedures can be helpful even in instances in which muscle shortening may not be just a product of active muscle contraction but rather a product of contractures within the muscle's connective tissue. By relaxing the active elements within the muscle, the lengthening procedure may be more effective at stretching the connective tissue elements, normalizing the function of the contractile and noncontractile elements in the muscle.

These MET procedures are based on the observed phenomenon that maximal muscle relaxation often follows strong isometric muscle contraction. Sherrington⁶⁸ first noted this phenomenon in 1909. This principle is applied in the variation of MET known as *PIR*. Modified forms of MET involve three phases: contraction, relaxation, and stretch. The patient contracts into the resistance provided by the clinician. This is followed by a period of muscle relaxation and passive stretching by the therapist (Figure 7-28).

TABLE 7-1 Types and Characteristics of Dysfunctional Barriers

Barrier Type	Palpatory Finding	Cause
Neuromuscular barrier	Elastic quality	Increased myotonus
Fascial barrier	Inelastic quality	Fibrosis
Passive congestion barrier	Abrupt but fades	Edema
Osseous barrier	Abrupt but unyielding	Intraarticular joint locking
Anatomic barrier	Hard with increased ROM	Hypermobility, instability



Figure 7-27 Muscle energy technique for joint dysfunction (L4–L5). **A**, Isometric procedure for a right rotational restriction and right lateral flexion restriction with barrier engaged. Patient is instructed to left rotate and left laterally flex against the clinician's resistance. **B**, Isotonic procedure for a right rotational restriction and right lateral flexion restriction with barrier engaged. Patient is instructed to right rotate and right laterally flex against the clinician's resistance.

BOX 7-17 Muscle Energy Technique Procedure for Joint Mobilization

1. Take the joint gently to tension (engage barrier).
2. Induce a gentle isometric contraction away from the barrier for 3 to 5 seconds. The patient's force should not overpower the doctor or create any sensation of pain. An alternate method uses an isotonic contraction into the barrier.
3. Mobilize into the barrier by gently pressing or pushing the joint in the direction of correction for 5 to 15 seconds or until a release or "melting" sensation is perceived.
4. After the first release, wait about 5 to 15 seconds for subsequent release if normal motion has not been restored.
5. Repeat until normal motion is perceived (3 to 5 times).

Different models of how these procedures may produce a therapeutic effect have been presented, but the precise mechanism by which these techniques accomplish a soft tissue change remains unclear. One theory stresses the role of the GTOs. In this model, sustained isometric contraction increases tension in the already

contracted muscle, causing the Golgi receptor system to sense the increased tension and reflexively induce muscle relaxation or lengthening.

Another method of induced muscle relaxation is generated by taking advantage of the phenomenon of reciprocal inhibition (RI). When a muscle contracts isometrically, its antagonist is inhibited, and the antagonist relaxes immediately after the contraction. This principle is applied in procedures that imitate isometric muscle contraction in the direction of reduced flexibility.

PIR appears to work best in chronic cases, and RI is better in acute settings.⁶⁹ However, both may be used in either case, providing that no pain is produced or no attempt is made to force or stretch joint structures.⁶⁹ When applying PIR, the isometric contraction phase of treatment is held for approximately 7 seconds (range of 4 to 10 seconds; increasing contraction time enhances the effect of PIR) and the relaxation and passive stretching phase lasts approximately 15 seconds (Figures 7-29 and 7-30). The isometric contraction should be of minimal force, and it is important that it equal the force exerted by the patient and be unyielding. The patient should be instructed to match the resistive force of the doctor and not overpower the doctor. Failure to maintain the position and failure to reach the barrier accurately will make the treatment less effective.

The barrier concept is very important to the application of PIR techniques. During the relaxation phase, the clinician applies

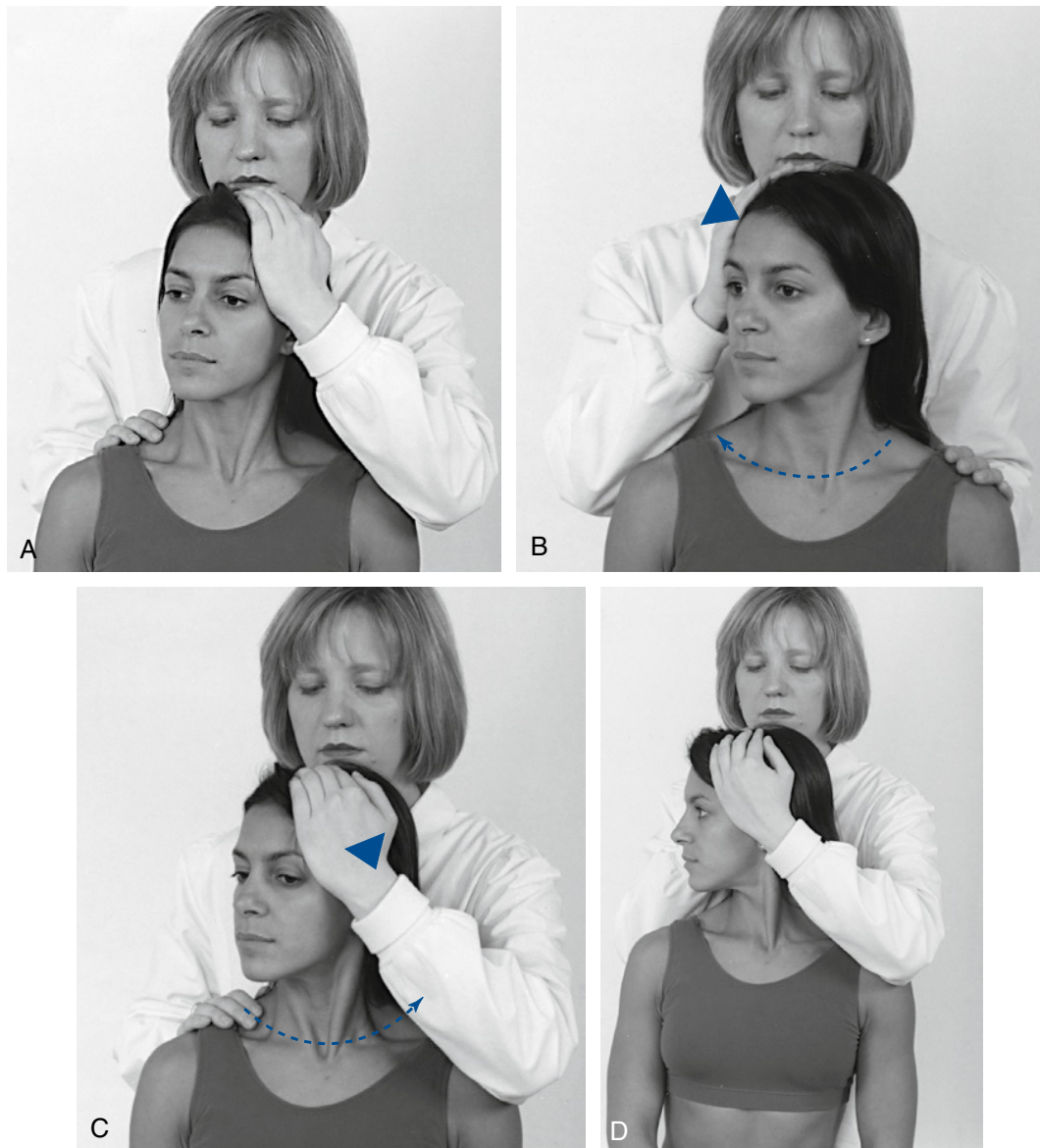


Figure 7-28 Example of modified muscle energy technique applied for decreased right cervical rotation.

1. Patient is in the seated position.
2. Clinician stands behind the patient.
3. Clinician assesses cervical rotation, moving to the point of engaging the barrier (A).
4. Patient is then instructed to contract the cervical musculature to produce either left rotation or right rotation against the clinician's resistance.
5. If right rotation is produced against resistance, postisometric relaxation is produced (B).
6. If left rotation is produced against resistance, a reciprocal inhibition effect is produced (C).
7. Clinician reassesses cervical rotation, noting increase in right cervical rotation (D).

gentle pressure and lengthens the muscle to engage a new barrier, the point at which restriction or pain is again felt. This process is repeated using the newly achieved ROM until the restriction of mobility is reduced, the spasm of the muscle is relieved, and the intensity of the pain can be alleviated (Box 7-18).

The patients themselves can also carry out many useful types of self-administered PIR. After patient education, such procedures are useful in bridging the intervals between patient visits. Although they are useful, they have limitations in their efficiency. This is particularly true in the upper cervical and occipital regions.

Evjenth and Hamberg⁷⁰ recommend modifying PIR by stimulating the antagonistic muscle or muscles at the end of the treatment sequence. This variation of PIR incorporates an additional active contraction on the part of the patient. In this method, the patient assists in producing the desired movement after the stretching phase by actively contracting the antagonist muscles. This is accomplished by having the patient actively increase movement in the direction of restriction against mild resistance applied by the clinician. This modification of PIR is referred to as the *contract, relax, antagonist contract method* or *RI*. When a muscle contracts isomet-

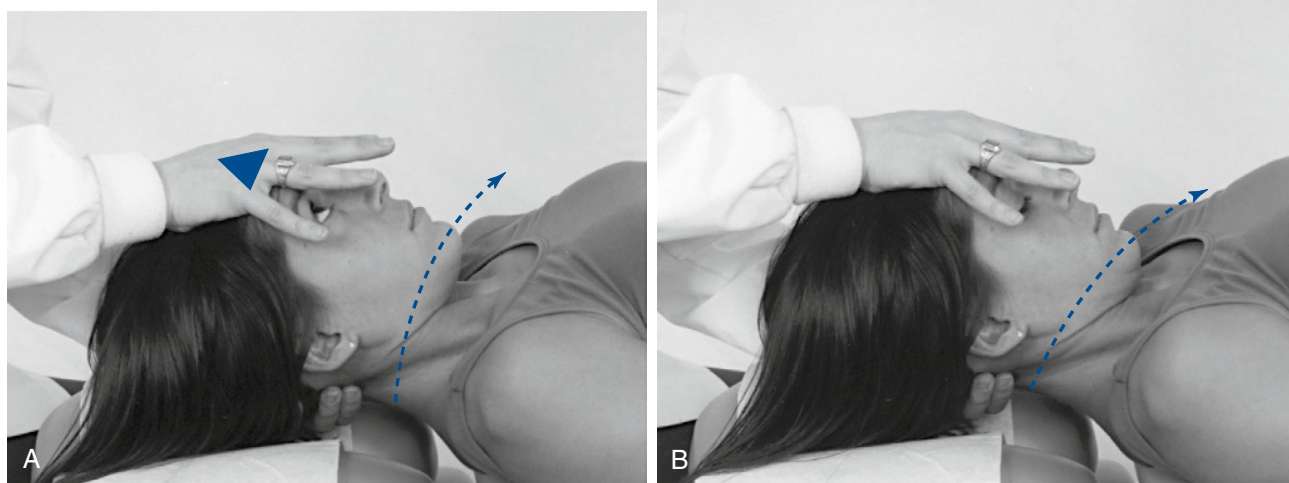


Figure 7-29 Postisometric relaxation procedure for a cervical flexion restriction.

1. Patient flexes the head to the point of restriction (barrier).
2. Patient looks up and contracts against the counterpressure applied by the clinician (**A**).
3. Patient looks down and exhales, and the clinician passively stretches the patient's head forward (**B**).

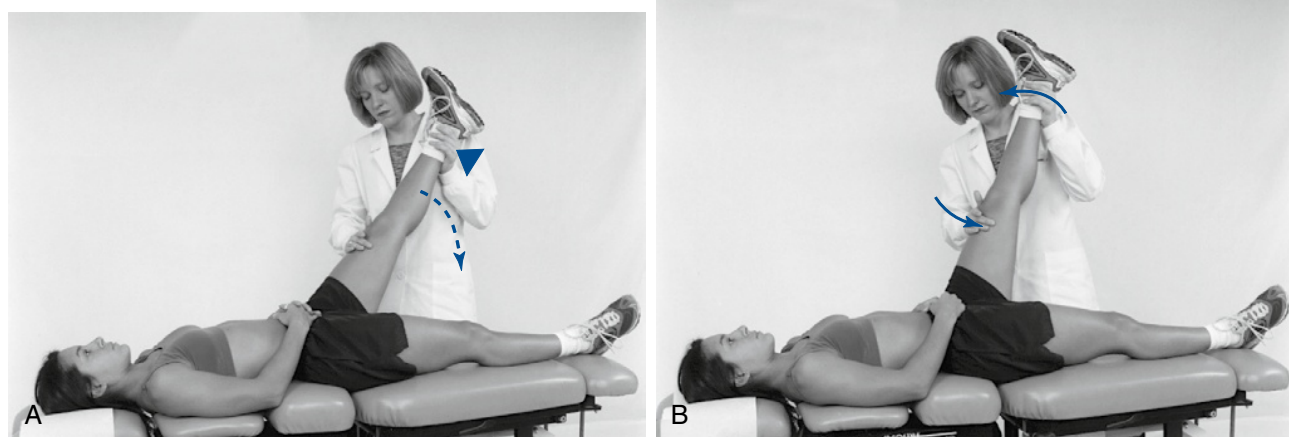


Figure 7-30 Postisometric relaxation procedure for the hamstring muscle.

1. Patient lies in a supine position.
2. Patient's hip is positioned in full flexion, with the knee extended as far as the shortened muscle will allow.
3. Doctor stands on the side of the involved leg, rests the patient's knee on his or her shoulder, and grasps the patient's leg just above the knee (**A**).
4. Patient is instructed to contract against the doctor's shoulder, attempting to extend the hip or flex the knee or both.
5. Patient maintains contraction for approximately 7 seconds.
6. Patient is instructed to relax, and the doctor gradually flexes the hip and extends the knee (**B**).

BOX 7-18

Procedure for Applying Postisometric Relaxation to a Painful Muscle Spasm

1. The muscle is lengthened (without stretching) to engage a barrier.
2. The patient provides a minimal isometric contraction against resistance for 10 seconds.
3. The patient is instructed to relax or "let go."
4. Repeat three to five times from the point at which a release to a new barrier occurred.

rically, its antagonist is inhibited from contracting. Therefore, by isometrically contracting the antagonist of a shortened muscle, the shortened muscle relaxes through RI. Contraction of antagonist muscles may be useful when the shortened (agonist) muscle is too painful to contract. There are a number of other procedures that use the antagonist contraction mechanism.

Proprioceptive Neuromuscular Facilitation

PNF was developed by Herman Kabat, M.D., and Margaret Knott, P.T., as a method to treat patients with neurologic dysfunction. Dr. Kabat developed the PNF method during the 1950s

after being introduced to the work of Sister Elisabeth Kenny, the “Kenny method,” and Sister Kenny’s treatment of polio patients. The early focus of their methods paralleled that of Sister Kenny and focused on the treatment of patients with paralysis and polio. Over the years, the procedures have evolved and techniques have been applied to a broadening array of conditions affecting motor function and the NMS system. It is now common for these procedures to be applied in a wide variety of conditions, ranging from rehabilitation of stroke and spinal cord injury patients to the management of common NMS injuries and disorders.⁷¹ Kabat’s conceptual goals for PNF were to neurologically strengthen weak muscles. Mitchell, in developing MET, adapted these concepts for use in joint mobilization, which were in turn modified to release muscle shortness (PIR). These procedures, with their proposed mechanisms and uses, continue to evolve.

PNF is defined as the use of proprioceptors to hasten or make easier the learning of a neuromuscular task.⁶⁴ Patients are directed through a series of complex movement patterns directed to effect maximal elongation of muscle. These patterns involve spiral and diagonal movements and are modeled after normal patterns of motion associated with tasks and movements of daily living. It is through the learning or relearning (facilitation) of these complex patterns that PNF is speculated to stimulate proprioceptors and hasten recovery. PNF is considered by its developers to be a philosophy of treatment rather than a technique. It purports to have a global effect on function, involving multiple muscles and multiple planes of movement.

The basic neurophysiologic principles that serve as a foundation for this procedure include the concept that a muscle response can be influenced by resistance, stretch reflex, and other proprioceptive input. Therefore, PNF uses the concepts of resistance, stretch reflex, approximation, traction, and manual contacts to facilitate efficient motor or recruitment patterns. Through applying these basic principles, the patient’s postural responses, movement pattern, strengths, and endurance can be assessed and theoretically enhanced.

Physical manipulations using proprioceptive neuromuscular technique often use hold-relax-stretch procedures to obtain an increase in ROM and normalize muscle function about a joint. The hold-relax-stretch technique and terminology is comparable with the contract-relax-stretch technique in both application and effect.⁷² The PNF approach is based on the ability to identify dysfunctions of neuromuscular control using observation, palpation, and motion evaluation. The evaluation procedures used to identify dysfunctions are listed in Box 7-19.

Definitions and descriptions for the evaluation procedures listed in Box 7-19 follow. Passive mobility assesses a PNF diagonal for the presence of an accessible passive ROM and the patient’s ability to relax. Active mobility adds active muscle contraction to move the part through the PNF diagonal that was previously tested passively. The quality of movement initiation is evaluated for sluggishness, delayed response, hyperactive response, or inappropriate recruitment.

Coordination and control are evaluated with the addition of resistance to initiated movements to identify deviations of movement or jerky and uncoordinated movements. Again through the use of resistance, the strength of muscle and speed of contraction can be evaluated for insufficiencies. A combination of isotonic

BOX 7-19

Proprioceptive Neuromuscular Facilitation Evaluative Procedures for Identifying Dysfunction

- Passive mobility
- Active mobility
- Initiation of movement
- Coordination and control of movement
- Strength and speed
- Combination of isotonic
- Isometric contraction
- Reversal of movement
- Agonist-antagonist balance
- Trunk control

BOX 7-20

Variations in the Application of Proprioceptive Neuromuscular Facilitation

- Rhythmic initiation
- Combination of isotonic
- Repeated quick stretch from elongation
- Repeated quick stretch superimposed on an existing contraction
- Reversal of antagonists
- Isotonic reversal
- Stabilizing reversal
- Contract-relax
- Hold-relax

assesses the performance of and transition between the types of isotonic contractions (concentric, eccentric, and maintained). In addition to isotonic contractions, isometric contraction is evaluated for weakness.

The ability to reverse direction is a necessary feature of the neuromuscular system, and inadequate control, speed, or strength of the reciprocal movement results in altered arthrokinematics. Identification of a functional imbalance of the agonist and antagonist muscles is important because it may result secondarily from structural dysfunction, disc herniations, or overuse syndromes. Trunk control depends on the integration of stability and mobility and is essential for efficient function and health of associated structures.

The extensive array of procedures and principles associated with PNF make it a difficult procedure to classify. Although initially perceived as a program of rehabilitative exercise, it is clear that many of the techniques applied in PNF are not easily categorized as exercise. The common incorporation of muscular effort during the application of these procedures has led to the common classification of PNF as an MET, which is likely an oversimplification. There are nine variations in the application of PNF that can be applied based on the patient’s functional needs (Box 7-20). Each of these has a specific purpose, indication, and application.

Rhythmic Initiation. Rhythmic initiation (Figures 7-31 and 7-32) is used to evaluate and treat the patient's ability to allow passive motion; actively contract in a smooth, rhythmic fashion; and perform movement at a constant rhythm against resistance. It is therefore used to treat those functions that affect the initiation, speed, direction, or quality of the contraction. To apply the procedure, the patient is positioned in a posture conducive to relaxation



Figure 7-31 PNF rhythmic initiation for acute pain. Patient is positioned in a posture conducive to relaxation, and the clinician produces the desired motion passively. Patient then minimally assists with the movement.

and the clinician performs the desired joint movement passively. Once a smooth and rhythmic passive movement is achieved, the clinician asks the patient to minimally assist with the movement. Resistance by the clinician is applied as the patient increases active participation.

Combination of Isotonics. Combination of isotonics (Figure 7-33) is used to evaluate and develop the ability to perform controlled, purposeful movements. It is indicated for the treatment of deficiencies in strength, ROM, and decreased neuromuscular coordination. It is applied to problems of concentric contraction, eccentric contraction, or efficient maintained (isometric) contraction. Concentric, eccentric, and isometric contractions of the agonist muscle pattern are combined in any sequence with relaxation to perform controlled, purposeful movements.

Repeated Quick Stretch. Repeated quick stretch incorporates the repeated use of a stretch reflex to assist with initiation of a muscular response. It is therefore indicated to reduce fatigue, improve endurance, and increase the patient's awareness of the motion. Repeated excitation of a pathway in the central nervous system promotes transmission of impulses through that pathway. It can be applied with elongation or contraction. The muscle components will be placed in either a stretched or approximated position. The patient is then instructed to initiate a reflex contraction that is resisted by the clinician.

Reversal of Antagonists. Reversal of antagonists is designed to influence the antagonistic muscle groups that fail to work in accordance with the demand of a specific activity. It is achieved by applying resisted alternating concentric contractions or by resisting alternating isometric contractions.

Contract-Relax. Contract-relax uses muscle tension developed through concentric contraction to facilitate relaxation. It is used to increase ROM of the myofascial unit by facilitating relaxation and improving extensibility of the myofascial tissues. The segment to be treated is placed at its point of limitation within the movement pattern, and resistance is given to a concentric contraction of either the restricted agonist or antagonist. After the contraction,

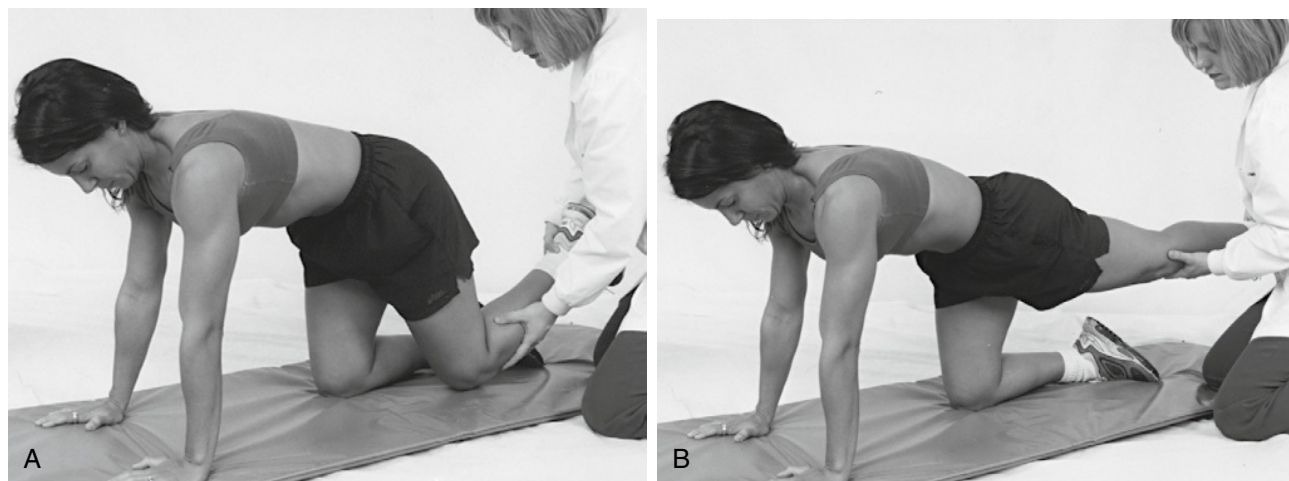


Figure 7-32 Proprioceptive neuromuscular facilitation rhythmic initiation facilitating gluteus maximus contraction (hip extension) activity for low back stability. **A**, Hip extension (crawling) begins. **B**, Hip extension is complete. This procedure is done passively, actively, and with resistance to facilitate trunk stability.



Figure 7-33 Proprioceptive neuromuscular facilitation combination of isotonics applied to the scapula patterns. **A**, Scapula is protracted, elevated, and abducted. **B**, The scapula is retracted, depressed, and adducted. The sequence of treatment is: (1) passive movements produced by the clinician, (2) some patient assistance with the movement pattern, (3) Fall active movement by the patient, and (4) resisted movement.

the patient is asked to relax completely, and the segment is passively or actively taken into a new available ROM.

Hold-Relax. Hold-relax is used to facilitate relaxation and increase ROM using an isometric rather than isotonic contraction. It is indicated when pain limits the use of isotonic contraction. It is applied by placing the joint in a pain-free position and then slowly invoking isometric contraction. The patient is told to hold the position while a resisted stress is applied.

ISCHEMIC COMPRESSION

Ischemic compression is a generic procedure, the definition of which has become confused because of the many proprietary approaches that use it. Receptor-tonus (Nimmo), trigger point therapy (Travell and Simons), neuromuscular technique (Chaitow and St. John), myotherapy (Prudden), ART (Leahy), shiatsu, and acupressure have all used this term to describe aspects of their individual therapeutic approach or mechanism of treatment.

The advocates of these procedures have different explanations and theories, but the clinical application is very similar: deep manual pressure over tender muscular nodules. Travell and Simons⁷³ were the first to use the term *ischemic compression* to describe the manual pressure applied over myofascial trigger points. Many methods of trigger point therapy, using forms of ischemic compression, including PIR and spray-and-stretch techniques, are thought to achieve their results through neurologic mediation of reflex pathways.

Several theories have been suggested to explain the mechanism of ischemic compression. Travell and Simons⁷³ believe that by holding pressure over a specific site, ischemia is induced to the sensory nerves, producing a nerve block from lack of oxygen. Travell and Simons⁷³ believe that the deep pressure produces a concentrated stretch to contracted sarcomeres associated with a trigger point, creating a separation of the actin-myosin heads. Another explanation is that after the release of pressure, a reflex vasodilation and hyperemia occur, producing a circulatory increase capable of removing chemical irritants and metabolites and bringing in oxygen and adenosine triphosphates. The last theory considers the

mechanism that deep pressure produces pain that hyperstimulates interneurons in the dorsal horn to release endorphins that block pain perception. In all likelihood, the effects of ischemic compression are the result of a combination of mechanisms.

Receptor-Tonus Technique (Nimmo)

Dr. Raymond Nimmo was one of the first chiropractors to focus on the soft tissue component of dysfunction and subluxation, advocating specific soft tissue treatment since 1950. Clinically, Nimmo observed spots on the shoulders that, when pressed, referred pain to various areas that he called *noxious generative points*.⁷⁴ In 1952, he discovered the work of individuals such as Travell and later Simons in researching myofascial pain and dysfunction. The term Travell used for the painful areas in the myofascia was *trigger points*. Nimmo called the procedure for treating trigger points the *receptor-tonus technique* to emphasize his theory that it is a reflex technique and not a form of massage therapy.⁷⁵

In the Nimmo method, evaluation and treatment emphasis is placed on posture and related muscular involvement. The soft tissues are palpated for tenderness, spasm, and trigger points with their characteristic patterns of referred pain. Nimmo devised treatment procedures that involved the application of firm manual pressure at the site of the trigger points. Nimmo technique uses deep pressure applied directly over the irritable lesion for approximately 5 to 7 seconds to produce the compression effect.⁷⁵⁻⁷⁷

The pressure is applied evenly throughout the treatment and then is quickly released. As pressure is applied, the patient will feel local or referred pain or both, which usually increases in intensity even though the manual pressure remains steady. Once the pressure is released, the initial pain intensity will slowly drop to a lower level. This process is repeated over the same trigger point once or twice more during a single office visit. Methods of applying pressure include the thumb, the index or middle finger, the elbow, a pincer grasp (thumb against the middle and index fingers), a knife-edge contact (ulnar side of the hand), and a T-bar (a mechanical hand-held device with a rubber tip) (Figure 7-34). The steps involved in Nimmo's form of ischemic compression are summarized in Box 7-21.

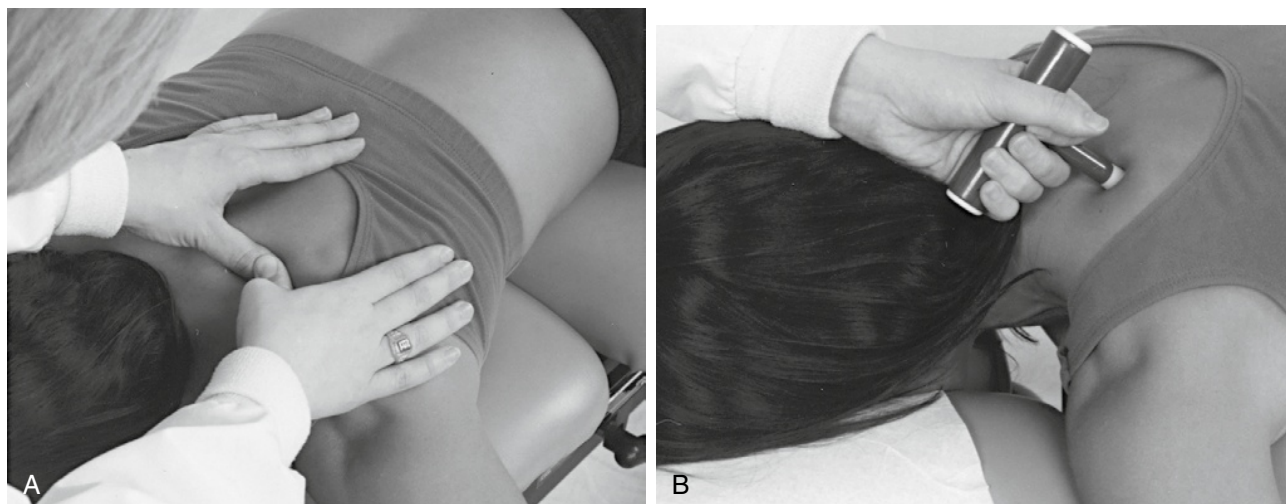


Figure 7-34 Nimmo receptor-tonus technique to disperse a trigger point in the levator scapulae muscle using thumb pressure (A) and a T-bar (B).

BOX 7-21 Steps in Receptor-Tonus Technique (Nimmo)

Tender nodules and taut bands of contracted muscle are located using scanning palpation.

Firm digital pressure is applied over the nodules while the clinician asks the patient about intensity and radiation of pain.

Pressure is held constant (no increase) over the nodule for 7 to 10 seconds.

Pressure is released and other sites are considered.

Repeat the procedure for each nodule 2 to 3 times per treatment visit.

Keep in mind that too much pressure or too many applications can lead to irritation and bruising.

Reassess the response of the nodule or taut bands with palpation and the patient's subjective response to pain.

Before treating a trigger point for a second or third time on the same visit, it is important to assess the muscle tissue for changes in tension and nodularity. Many times after the first application of pressure, the patient notes a significant reduction in the local or referred pain or both. Additional treatment of a trigger point on the same office visit should not be done if substantial decrease in tension or nodularity is noted after the first application of pressure. Moreover, if the trigger point still does not show signs of improvement after three applications of treatment, an interval of a few days is recommended before attempting further treatment. Common mistakes are using excessive pressure and treating patients too frequently. Excessive pressure, beyond the compliance of the tissues, becomes a form of trauma that may result in a hematoma or new trigger point formation.

Frail patients or those with capillary fragility, vascular disease, or a history of corticosteroid use should be treated very gently to avoid iatrogenic sequelae. Larger muscles, such as the gluteal

group, may require more pressure to achieve ischemic compression, whereas small muscles, such as the suboccipital group, require very little pressure. The clinician must always work within the pain tolerance of the patient; when in doubt, it is wiser to use too little pressure than too much.

It also appears that even patients with well-developed musculature develop some post-treatment soreness the day after a treatment and should be advised accordingly. It is recommended that a day or two be interposed between office visits and that patients get treated on a twice-weekly basis (three times weekly in acute cases). This allows the muscle tissue a chance to heal from any treatment microbruising and gives the nervous system a chance to reestablish equilibrium in the sensory-motor reflex pathways. Some chiropractors perform daily spinal manipulations with good results. However, daily treatments with Nimmo's method may lead to delayed healing from post-treatment bruising and is not recommended.

Nimmo postulated that local application of deep pressure hyperstimulates nociceptors, which in turn produce neurologic modulation, resulting in reflex inhibition of motor output and a reduction of muscle tonus. He suspected a neurologic explanation of treatment results because patients reported instantaneous pain relief after a treatment. He reasoned that only the nervous system was capable of creating changes in muscle tone within a few seconds. Clinically, release of contracted muscle has been noted to occur before the usual 5- to 7-second application time has finished. Receptor-tonus technique is therefore presented as a reflex technique that is thought to reduce muscle tonus through reflexes mediated by the central nervous system.

Trigger Point Therapy (Travell and Simons)

Janet Travell,⁷³ a medical doctor, placed a significant emphasis on the role of the myofascia in health and disease. She was the first to use the term *trigger point* to describe the tender nodules in the soft tissues. Travell and Simons⁷³ define a *trigger point* as a

hyperirritable spot, usually located in a taut band of skeletal muscle, that is painful on compression and can give rise to characteristic referred pain, tenderness, and autonomic phenomena.

Evaluation consists of postural assessment to identify gross muscle imbalances and specific palpation of the myofascia. Charts have been developed identifying the referral patterns of pain that are specific to individual muscles (Figure 7-35). Examination findings are summarized in Box 7-22. Travell⁷³ promotes the use of vapocoolant spray while the irritable tissue is being stretched, as well as the injection of an anti-inflammatory medication (e.g., procaine) into the trigger point.

Manual pressure is also advocated to inactivate a trigger point. Travell and Simons⁷³ were the first to use the term *ischemic compression* to refer to this procedure. The length of applied pressure is significantly longer in duration than the ischemic compression method described by Nimmo. Travell's method uses firm digital pressure with deep stroking massage, kneading massage, or vibratory massage to produce hypoxia, followed by a reactive hyperemia. Pressure is gradually increased over a period of 30 to 60 seconds or until the trigger point tenderness is eliminated. Massage, stretch without spray, ultrasound, heat, drug therapy, biofeedback, and transcutaneous electrical nerve stimulation (TENS) are all discussed as alternative means to inactivate a trigger point.

BODY WALL REFLEX TECHNIQUES

Body wall reflex techniques incorporate a number of different procedures. All are applied to identify a wide variety of different types of discrete soft tissue changes that are sensitive to palpation. The explanation for the presence of this palpatory sensitivity is a reflex pain mechanism. With prolonged pain impulses, a vicious cycle that spreads through an internuncial pool with connections upward, downward, and across the spinal cord, potentially reaching the thalamus occurs. Depending on the extent of these connections, it is possible to detect the presence of pain and sympathetic disturbance at a distance from the site of injury and sometimes on the other side of the body.

A factor common to the various reflex systems is that the irritable lesion appears to be situated in fascial tissue.⁷⁸ The one thing that all of these reflexes seem to have in common is that they are all responses to various types and degrees of tissue injury, including physical trauma, infection, degeneration, and chemical injury.⁷⁸

When a body wall reflex is present, there is the potential for negative effects on the skin locally, changes to blood and lymph flow, and neurologic stimulation. It is believed that intelligent and scientific manipulation of the skin and soft tissues of the body places the physiologic status of the skin at an optimum, invigorates the circulation of blood and lymph, and alerts the central and peripheral nervous systems.⁷⁹

There is much overlap of the various body wall reflexes, suggesting that each purveyor recognized a similar characteristic but explained it differently. Moreover, if all the body wall reflex points described in acupuncture and those points described by Travell (trigger points), Chapman (lymphatic reflexes), Jones (strain-counterstrain), Bennett (vascular reflexes), and others were placed together on one map of the body surface, the entire body surface

would be covered.⁷⁸ Furthermore, many of the referred pain patterns from visceral disease coincide with these points. This implies that the body wall is rich in receptors and can easily serve as a source of noxious irritation to the entire body. Although there are a number of different names with different philosophical intents, many procedures are very similar in application.

Acupressure Point Stimulation

Acupressure is a method of massage or point pressure applied to acupuncture points for the usual purpose of producing analgesia.⁶⁴ Much has been written on the clinical aspects of acupuncture, acupressure, and meridian therapy. Acupuncture points are organized along meridians that have no known neurologic or vascular pattern. There appears to be a measurable change in electrical potential in irritable points, and they can be treated by electrical stimulation, needle application, or manual pressure. Theoretically, blockage or other dysfunction in the meridian causes a departure from health.²⁹

There is a growing body of clinical research exploring the effectiveness of acupuncture. As with any treatment procedure, there are a number of contradictory studies. In a randomized single blind trial examining the effects of acupuncture on chronic myofascial neck pain, acupuncture was found to produce a modest pain reduction.⁸⁰ In a study of diabetic patients with chronic painful peripheral neuropathy treated with acupuncture analgesia, it was found that acupuncture was a safe and effective therapy for long-term management of painful diabetic neuropathy, although its mechanism of action could not be determined.⁸¹ Acupressure was studied for effectiveness in reducing nausea and vomiting of pregnancy. The results indicated that acupressure was effective in reducing symptoms of nausea but not frequency of vomiting in pregnant women.⁸² A small population study on acupuncture for neck pain revealed no significant difference between acupuncture and placebo, although both groups demonstrated moderate improvement. The conclusion was that acupuncture may have no greater effect than a placebo.⁸³ Ernst⁸⁴ reports in a systematic review of the literature that the results of studies are highly contradictory with many trials suffering from methodologic flaws. In a randomized placebo-controlled clinical trial of acupressure for weight loss, it was concluded that acupressure appeared to be safe but did not promote significantly greater weight or fat loss, nor did it promote a decline in blood pressure.⁸⁵ A particularly disturbing concern was identified in an article about acupuncture for chronic conditions in the United Kingdom. The survey⁸⁶ revealed that acupuncture was widely used in the treatment of chronic pain but that one fifth of the practitioners had not received any formal training in acupuncture. This brings to light concerns that many individuals may be practicing forms of complementary care without adequate training. The conclusions of a consensus process on acupuncture identified that there were many studies of potential usefulness, but that these studies provide equivocal results because of flaws in design and sample size.⁸⁷ However, promising results exist demonstrating efficacy of acupuncture in adult postoperative and chemotherapy nausea and vomiting.⁸⁷ Furthermore, there are conditions such as addiction, stroke rehabilitation, headache, menstrual cramps, tennis elbow, fibromyalgia, myofascial pain, osteoporosis, LBP, carpal tunnel syndrome, and asthma in which acupuncture may be

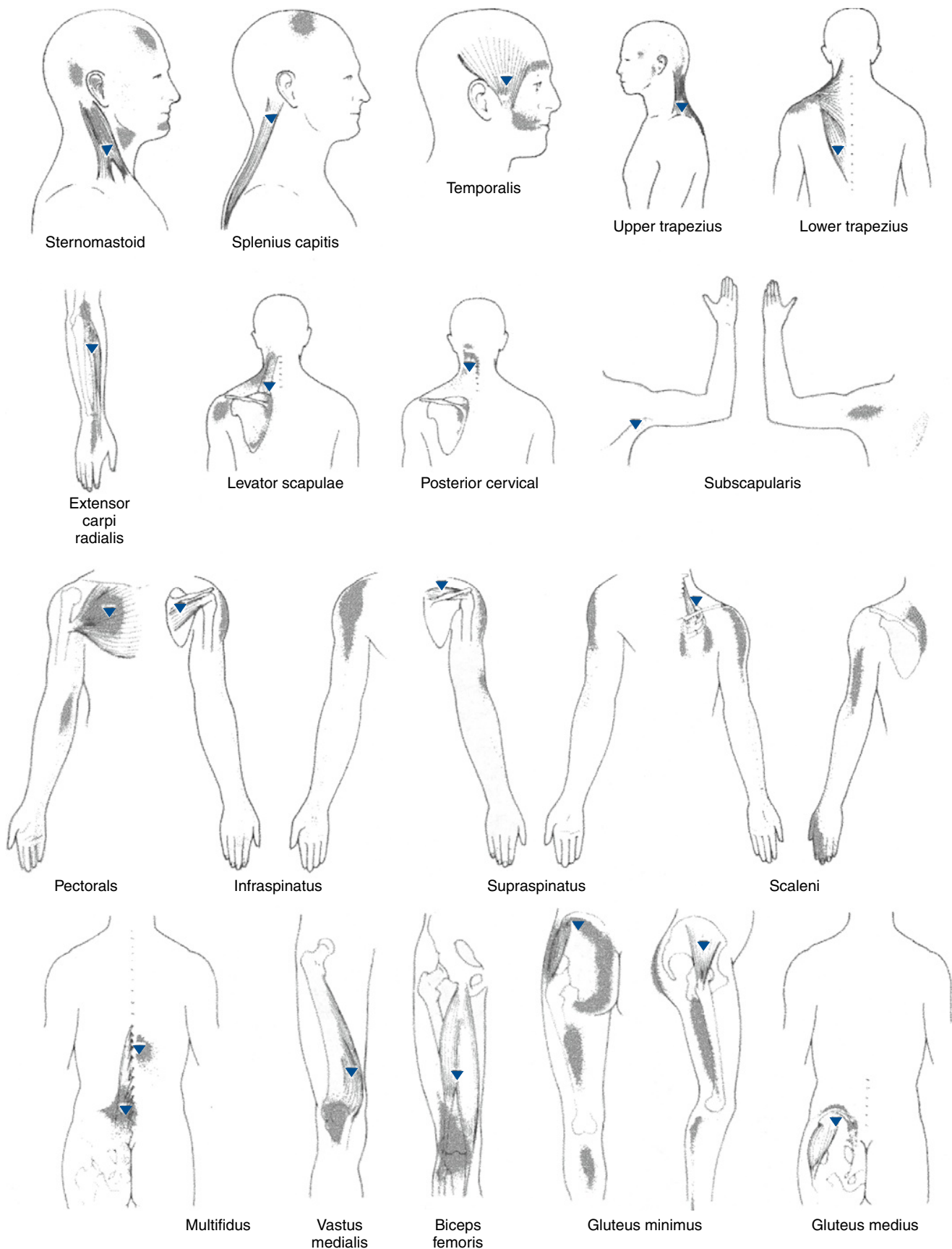


Figure 7-35 Chart of common trigger points (*triangle*) and referral patterns. (Modified from Chaitow L: *Modern neuromuscular techniques*, London, 1996, Churchill Livingstone.)

BOX 7-22 Examination Findings for Myofascial Trigger Points

When active trigger points are present, passive or active stretching of the affected muscle increases pain. The stretch range of the affected muscle is reduced. Pain is increased when the affected muscle is contracted against resistance. The maximum contractile force of the affected muscle is decreased (muscle weakness). Muscle palpation reveals tense, ropy fibers or palpable bands with a point of maximal tenderness. Pain and dysesthesia are commonly referred to specific sites that have been mapped for each muscle. Digital pressure applied to an active trigger point usually elicits a “jump sign” in which the patient pulls away suddenly or cries out. Snapping palpation of the trigger point may elicit a local twitch response in the muscle.

useful as an adjunct treatment or an acceptable alternative or may be included in a comprehensive management program.⁸⁷

Acupuncture points are usually located beneath the skin, although at times they are deep enough to require penetration of the needle to reach tendons and ligaments. It is thought that an acupuncture point becomes active and in need of treatment because of some form of reflex stimulation. They become sensitive to pressure, which therefore serves as a diagnostic finding. Acupuncture points are located in palpable depressions, with the skin over the point being somewhat thinner.⁸⁸ Beneath each acupuncture point lies a combination of tissues including fibrous tissue, fat, nerves and nerve trunks, large veins, muscle, and tendon receptors. Therefore, a number of tissues are simultaneously affected by the application of an acupuncture needle or finger pressure.

Active acupuncture points will typically be sensitive to correctly applied pressure and can often be neutralized by manual pressure or a combination of chilling, manual pressure, and stretching.⁷⁸ Acupressure is typically applied with the fingertip to produce pressure in a circular, transverse, or deep fashion (Figure 7-36). The applied pressure should produce some local tenderness that decreases during the treatment session. The pressure is usually applied for 30 to 90 seconds, although one point can be treated for as long as 3 to 4 minutes. To sedate an overactive acupuncture point, up to 5 minutes of sustained or intermittent pressure or rotary massage maybe required. To stimulate an acupuncture point, pressure is applied from 20 seconds to 2 minutes. The caveat is that pressure sustained beyond a certain point can produce the opposite effect.

Acupressure and acupuncture are theorized to have an effect on pain reduction through the modulation of endorphin levels⁶⁴ or through the stimulation of local mechanoreceptors and nociceptors (stimulus-produced analgesia) or both. Nociceptor activity may be reduced by the activation of sensory mechanoreceptors and nociceptors in the involved myofascial area.⁶⁴ The nerve reflex theory of acupuncture contends that when an abnormal condition occurs in



Figure 7-36 Acupressure applied using circular friction massage over spleen 6.

an internal organ, alterations will take place in the skin and muscles related to that organ by means of the nervous system. Reflex symptoms are classified into sensation reflexes, interlocking reflexes, and autonomic system reflexes. A sensation reflex occurs when an abnormal impulse travels to the spinal cord and the reflex action causes a hypersensitivity reaction in the skin. An interlocked reflex occurs when an abnormality in any internal organ causes a limited contraction or stiffening of muscles in an area near the part of the body that is connected neurologically to the affected organ. An autonomic system reflex occurs when abnormalities in the internal organs set up a reflex action in the sweat glands, sebaceous glands, pilomotor muscles, and blood vessels in the skin. Acupuncture points represent areas of abnormal physiologic activity that produces a continuous, low-level input to the central nervous system.⁸⁹ This might lead to a combining with noxious stimuli from other structures innervated by the same segments. Melzack, Stillwell, and Fox⁸⁹ report and that there is a 70% correlation between trigger point maps and acupuncture point charts.

Chapman (Neurolymphatic) Reflexes

The surface changes associated with Chapman reflexes are presented as palpable and are thought to be from contractions found in the deep fascia, located at specific points. The amount of tenderness is an important consideration in differentiating the contractions from subcutaneous fat globules. These palpable changes are located in specific anatomic areas on the anterior and posterior aspects of the body and are reported to be associated with specific viscera. The palpatory findings may vary in size from a pellet to a large bean. It is thought that these reflexes can be used as a diagnostic aid to locate a pathologic change without knowing its nature and to influence visceral function through nervous system input. Frank Chapman, D.O., was of the opinion that these body wall reflexes are clinically useful for diagnosis of visceral pathologic conditions; for influencing the motion of fluids, mostly lymph; and for influencing visceral function through the nervous system.

The anterior reflexes are located in the intercostal spaces near the sternum, and the posterior reflexes are found along the spine midway between the spinous processes and the tips of the transverse processes (Figure 7-37). There are also points located on the pelvis and the thighs. However, points on the surface of the body are never likely to be precisely located or identifiable by description of anatomic position, although general identification is possible. Therefore, in terms of charts and maps, the positions identified are approximate, and there is great variation from individual to individual.

Chapman reflexes are treated by light digital pressure applied to the anterior point and then the posterior point. After the pad of the middle finger has contacted the surface locus, a firm, gentle contact is maintained, and a rotary motion is imparted to the finger through the arm and hand to express the fluid content of the locus into the surrounding tissues (Figure 7-38). The actual application to a given reflex is expressed in terms of seconds, but in practice it is actually determined by a response to palpation. Therefore, treatment time may vary from 20 seconds to 2 minutes or more.⁷⁸ If the sensitivity of the reflex point has diminished, no more treatment is applied. If the sensitivity of the reflex point is unchanged, a second application is applied. The application of pressure should follow the guideline that overtreatment is counterproductive and may produce an unwanted effect.

The proposed mechanism by which these reflexes located in the intercostal spaces act is through stimulation of receptors lying between the anterior and posterior layers of the anterior intercostal fascia and acting on the intercostal nerve. The intercostal nerve innervates the internal and external intercostal muscles and through its connection with sympathetic fibers can affect the intercostal arteries, veins, and lymph nodes. Impulses from afferent and efferent vessels draining these tissues may increase or decrease because of the stimulation. With involvement of sympathetic fibers associated with specific tissue sites, lymph nodes of vital organs can be affected.⁷⁸

Regardless of the proposed mechanism, there is some research evidence to support the clinical application of Chapman reflexes. In a study determining whether stimulation of a Chapman reflex could reduce blood pressure, it was found that there was no effect on blood pressure but that there was an alteration in aldosterone levels.⁹⁰ Many hypertensive patients have high aldosterone and low renin levels affecting the renal tubules and causing sodium retention. Abnormalities in aldosterone levels have been shown in populations of essential hypertension. Stimulation of Chapman reflexes caused a consistent lowering of aldosterone levels. It was suggested that not enough time was allowed to see the blood pressure lower, because even with drugs that lower aldosterone, it takes 5 to 7 days for the blood pressure to lower.

Bennett (Neurovascular) Reflexes

Terrence Bennett, D.C., described reflex points mainly on the skull but also on other body parts that he believed were irritable reflexes reflecting the vascular condition of organs.^{91,92} These reflex points are used both diagnostically and therapeutically. Bennett developed the neurovascular dynamics technique, which

he proposed alters and restores autonomic homeostasis as a means to treat visceral and functional physiologic changes.

Bennett describes the palpable tissue changes in much the same way as Chapman: as a change in texture when the tissue is contracted or indurated. Many of the points are located on the head, although there are some on the anterior aspect of the torso as well (Figure 7-39). Treatment of Bennett reflexes consists of a slight degree of digital pressure combined with a slight stretching or tugging of the skin (Figure 7-40). As the skin is stretched, the fingertips are drawn apart slightly and a yielding sensation occurs. Also perceived within a few seconds after the contact is taken is a slight pulsation, believed to be from the microscopic capillary bed. If no pulsation is perceived, change direction of the skin stretch. The contact is maintained until a response is noted in the form of tissue alteration, relaxation, and pulsation. Treatment could take from a few seconds to a couple of minutes for these changes to emerge. As with other body wall reflex techniques, caution must be given not to overtreat.

Bennett reflexes are hypothesized as being viscerosomatic reflexes. Therefore, they are believed to have the same characteristics and neurologic mechanisms as viscerosomatic reflexes. Persistent pathologic conditions in visceral structures have the theoretic potential to induce reflexive dysfunction in somatic structures. Visceral disease or dysfunction may activate the autonomic nervous system through connections with the lateral horn cells in the cord to produce vasomotor, trophic, visceral, or metabolic changes. Viscerosomatic reflexes result from afferent stimuli arising from dysfunction of a visceral structure. The reflex is initiated by afferent impulses arising from visceral receptors that are transmitted to the dorsal horn of the spinal cord, where they synapse with interconnecting neurons. The stimuli are then conveyed to sympathetic and motor efferents, resulting in changes in the somatic tissues.

It has been suggested that the body wall manifestations of visceral disease are an integral part of the disease process, rather than just physical signs and symptoms.⁹³ Korr suggests that viscerosomatic reflex activity may be noted before any clinically identifiable symptoms of visceral change and therefore has diagnostic and prognostic value,⁹⁴ although the definitive etiologic factors and the characteristic response of the individual reflex are still unknown. There have been no clinical trials using Bennett reflexes reported in the retrievable literature. There are case reports that suggest there is benefit from treatment of these reflexes, concluding that more study is needed.^{92,95}

LOGAN BASIC TECHNIQUE

Hugh B. Logan, D.C.,⁹⁶ developed the Logan basic technique, emphasizing that the body must have normal structure to have normal function. In part, this system takes into account the effects of gravity on the spine and its related structures. Logan hypothesized that the body of the lowest freely moveable vertebra will rotate toward the side of the inferiorly displaced sacrum (or the vertebra upon which it rests), that is, that the body of that vertebra rotates toward the side of least support. This is usually the low side of the sacrum. Unequal weight-bearing support offered at the base of the spine or unequal weight transfer from above and through the

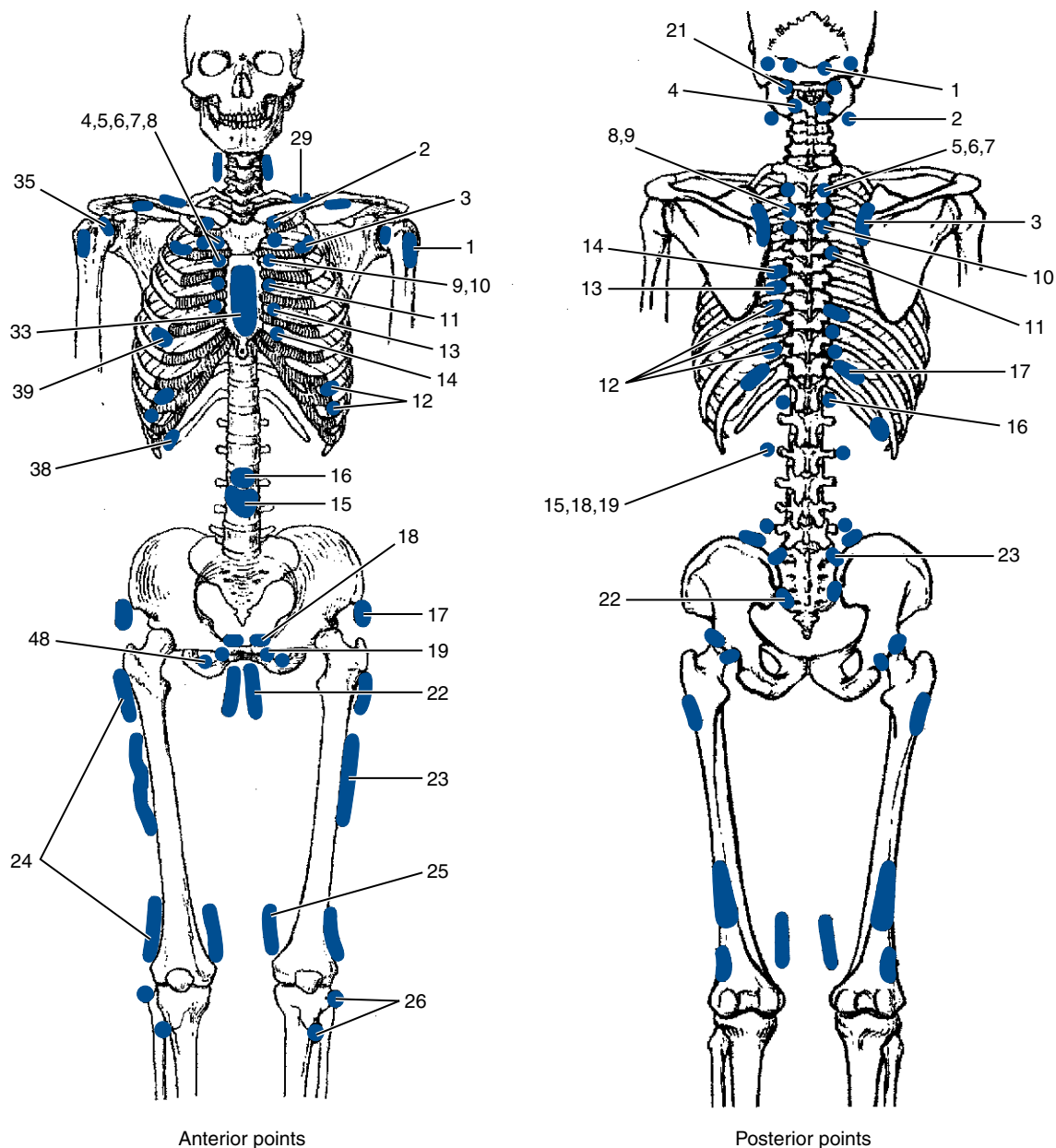


Figure 7-37 Chart for the location of Chapman reflexes (neurolymphatic).

Reflex	Symptoms or Area	Reflex	Symptoms or Area	Reflex	Symptoms or Area	Reflex	Symptoms or Area
1	Conjunctivitis	16	Kidneys	28	Cerebellar congestion (memory and concentration)	40	Liver and gallbladder congestion
2	Nasal problems	17	Atonic constipation	29	Otitis media	41	Salpingitis or vasculitis
3	Arms (circulation)	18	Abdominal tension	30	Pharyngitis	42	Ovaries
4	Tonsillitis	19	Urethra	31	Laryngitis	43	Uterus
5	Thyroid	20	Arm and shoulder pain	32	Sinusitis	44	Uterine fibroma
6	Bronchitis	21	Cerebral congestion (paralysis and paresis)	33	Pyloric stenosis	45	Rectum
7	Esophagus	22	Clitoral irritation or vaginismus	34	Neurasthenia	46	Broad ligament (uterine involvement)
8	Myocarditis	23	Prostate	35	Wry neck (torticollis)	47	Groin glands (circulation and drainage of legs and pelvic organs)
9	Upper lung	24	Spastic constipation or colitis	36	Splenitis	48	Hemorrhoids
10	Neuritis of upper limb	25	Leukorrhea	37	Adrenals (allergies, exhaustion)	49	Tongue
11	Lower lung	26	Sciatic neuritis	38	Mesoappendix		
12	Small intestines	27	Torpid liver (nausea, fullness, malaise)	39	Pancreas		
13	Gastric hypercongestion						
14	Gastric hyperacidity						
15	Cystitis						

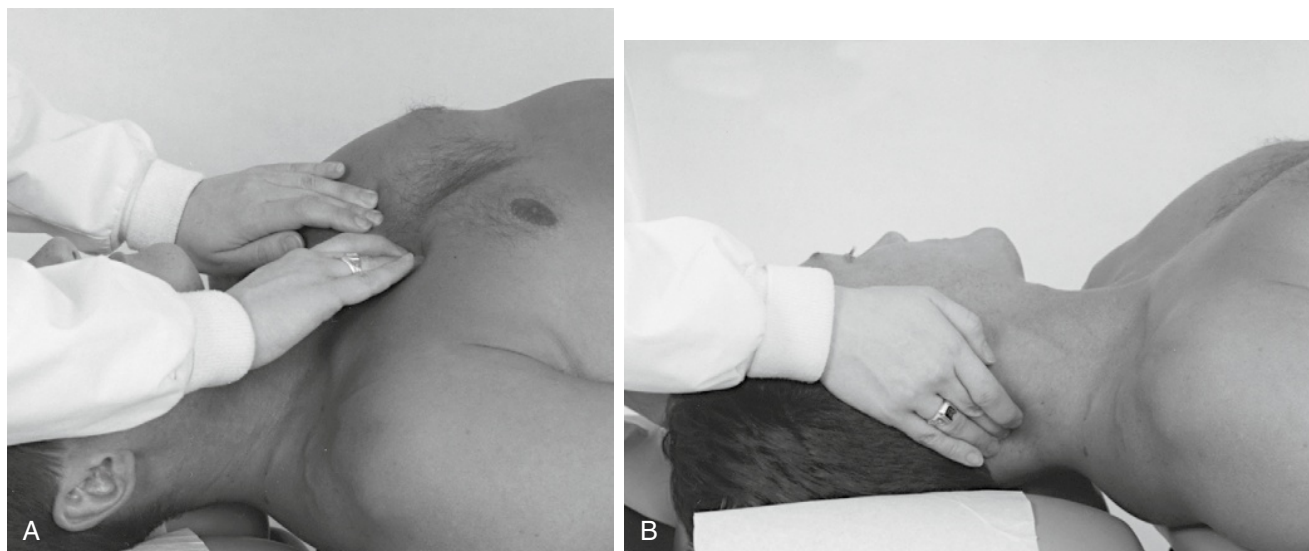


Figure 7-38 Treatment of Chapman (neurolymphatic) reflex for sinus involvement, using rapid circular massage over anterior reflex located between the second and third intercostal spaces parasternally (**A**) and posterior reflex located over the lamina of C2 (**B**).

sacrum becomes an etiologic factor of spinal distortions and segmental subluxation. Priority of treatment shifts to providing more equal weight-bearing support below the spine, sacral base leveling, and more equal weight transfer from the body above.

Logan⁹⁶ and others noted that if the spine is posturally deficient because of the effects of gravity, it places a greater energy demand on the body, which over time could lead to more serious effects on the human body. The reduction of distortions is approached from balancing weight-bearing support to the spine, balancing muscular efforts to reduce the distortion, and balancing ligamentous resistance to distortion. One major concept produced and developed by Logan was the value of the foundational support elements to the spinal column. Deviations in lower limbs, pelvic alignment, sacral position, and lower lumbar vertebrae were categorized as possible initiators of spinal distortions and subsequent segmental subluxation. Differences between the lower limbs resulting in altered spinal support included arch height; ankle, knee, and hip joint space; tibia and fibula lengths; femur length; and ankle, knee, and hip angulation. These were classified as variables in weight-bearing support of the pelvis and then of the spine.

Spinal mechanics are evaluated through postural analysis, spinal palpation and analysis (static and kinetic), full-spine x-ray mensuration, spinal posture musculature activity, lowered pain threshold points and patterns, bilateral scales, and other instrumentation. More specifically, Logan's analysis^{96,97} seeks to determine significant factors of foot, leg, pelvic, and sacral roles in altered weight-bearing support of the lumbar spine. Furthermore, these effects to the lumbar spine are evidenced by lateral bending, rotation, curvature, fragmentation (subluxation), reactive muscular activity, unequal ligamentous tensions, and others.

Logan⁹⁶ believed that the sacrum was the biomechanical keystone of the body, because it supported the spine and allowed for locomotion, and he believed that the spine would respond to changes in the sacrum. Thus, returning the sacrum to normal

relationships with its articulating bones was essential in reducing spinal involvements. He thought that this could be accomplished by applying light to moderate force to specific contacts established along the sacrotuberous and sacrospinous ligaments. The amount of pressure applied is described as the amount a person could withstand against the eyeball. Pressure is applied steadily to these contacts with no true thrust delivered. One of the most common contacts involves approximately 2 to 10 ounces of pressure applied to the junction of the sacrotuberous and sacrospinous ligaments (Figure 7-41). Somatic changes in muscle tone, skin and core body temperature, respiration, and perspiration have been reported as anecdotes.^{96,97} Basic technique uses many procedures, including balancing weight-bearing to the spine, temporary lifts, lower extremity therapy, sacroiliac therapy, balancing muscle efforts, postural education, notch and ulnar adjustment, and balancing ligamentous resistance.

Logan⁹⁶ includes all of the suggested vertebrogenic causes and involvements presented in classical chiropractic principles. The rationale at this level is simply "if the clinical entity has any possible connection to vertebral subluxation [initial or sustaining] then the reduction of same would have therapeutic value [the chiropractic adjustment of spinal segments]."⁹⁶ Logan further asserts that vertebrogenic involvement of a single segmental lesion with its neurologic elements can extend the effect to multiple neurologic functions and levels. This level of spinogenic involvement has two routes of neurologic pathophysiology: the direct route of neurologic involvement associated with spinal distortions and the indirect route of neurologic involvement associated with fragmentation of the system (vertebral subluxation).

Logan's treatment procedures⁹⁶ are aimed at the first route: the reduction of spinal distortions and associated neurologic involvement directly and indirectly reducing the segmental lesions and specific levels of neurologic involvement. The Logan approach is the correction of spinal distortions and the reduction of pathomechanical processes associated with

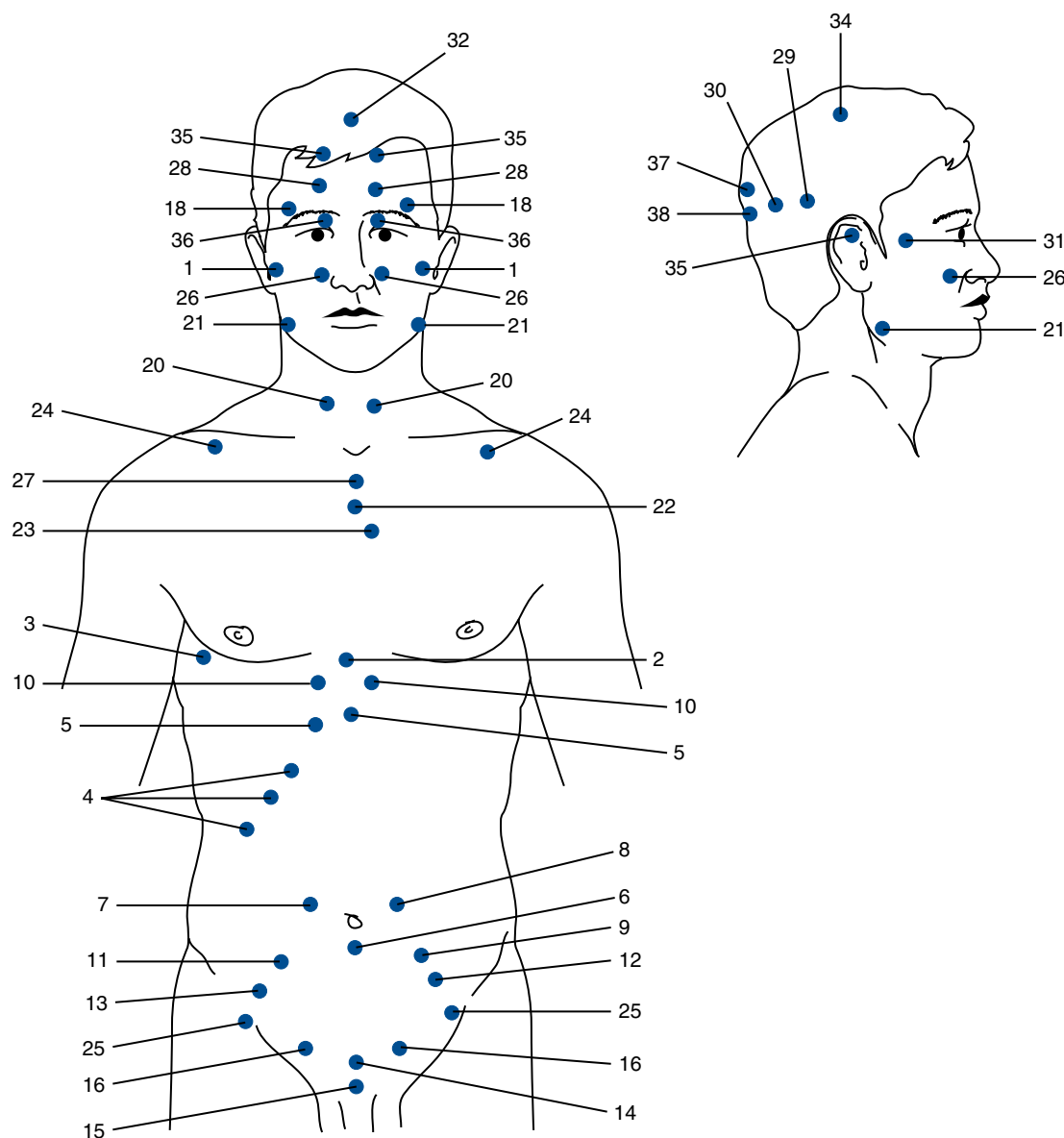


Figure 7-39 Chart for the location of Bennett reflexes (neurovascular).

Reflex	Area Affected	Reflex	Area Affected	Reflex	Area Affected
1	Parotid gland	14	Bladder	27	Bronchial region
2	Cardiac sphincter	15	Prostate or uterus	28	Frontal-emotional
3	Liver	16	Spermatic cord or ovary	29	Vagal
4	Gallbladder	17	Super-renal	30	Parietal
5	Pancreas	18	Anterior pituitary	31	Temporal-emotional
6	Pylorus	19	Posterior pituitary	32	Anterior fontanel
7	Second segment of duodenum	20	Thyroid	33	Midsylvian
8	Third segment of duodenum	21	Carotid sinus	34	Fissure of Rolando
9	Fourth segment of duodenum	22	Aortic sinus	35	Frontal eye muscles
10	Kidneys	23	Heart tone	36	Extrinsic eye muscles
11	Ileocecal valve	24	Subclavian lymphatics	37	Posterior fontanel
12	Internal rectal sphincter	25	Femoral lymphatics	38	Menopause-glandular
13	Appendix	26	Maxillary sinus		

those distortions. Spinal biomechanics of motion and support adversely affected by or as part of spinal distortions are specific targets of diagnosis and therapy directly. Specifically, Logan's treatment procedures are aimed at correction of altered supportive elements and altered motion elements of the complete system, sections, and units.

Logan's teachings⁹⁶ give clinical significance to altered weight-bearing support transfer down through the spine. These alterations are recognized as etiologically significant in spinal distortions, postural faults, spinal asymmetries, curves, sectional towering, and spinal system fragmentation (vertebral subluxation). The clinical significance of vertebral subluxation in Logan's programs are



Figure 7-40 Treatment of Bennett (neurovascular) reflex for the pancreas, holding a light tugging pressure just medial to the sixth and seventh rib heads until a pulsation is felt.



Figure 7-41 Apex contact of Logan basic technique taken over the right sacrotuberous ligament.

those established by other classical chiropractic principles and spinal adjusting techniques that recognize the value of subluxation reduction to improved homeostasis and physical well-being and the adverse potentials of subluxation presence in full-spectrum pathophysiology.

SPONDYLOTHERAPY

Spondylotherapy is defined by Janse⁹⁸ as a method of treating visceral disease through the stimulation of the related reflexes by the application of a mechanical or electrical force in a properly judged fashion on the autonomically related vertebra. The principle of interrupted percussion or vibration is usually used.

Spondylotherapy is accomplished by placing a contact over either the spinous process or both transverse processes with the middle or index fingers or both (Figure 7-42). The other hand is used over the contact in a series of rapidly rebound-

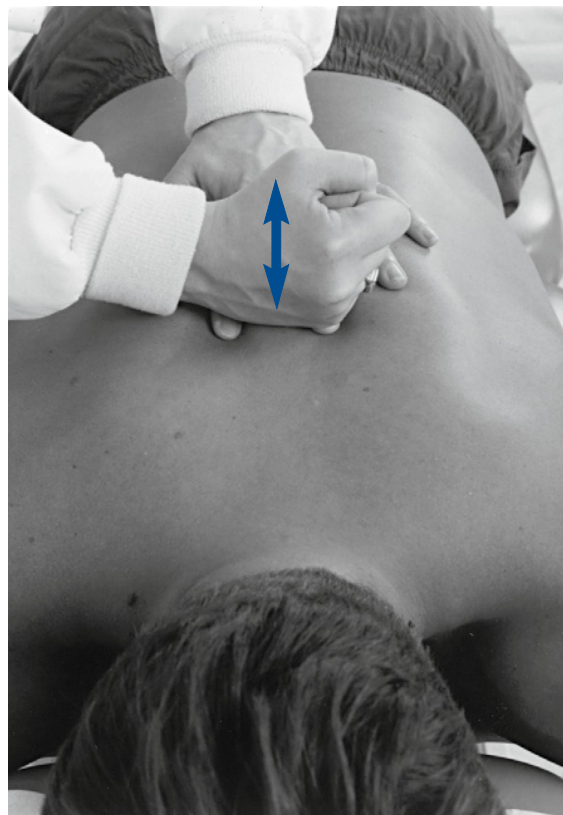


Figure 7-42 Spondylotherapy applied to the transverse processes of T5 to T9 to influence the sympathetic nerve distribution to the stomach.

ing percussions for approximately 5 seconds. About 15 percussions should be applied during this time. Treatment is typically applied to three or four adjacent vertebrae to provide neurologic input to the viscera with the same segmental innervation (Table 7-2).

Usually the segments to be treated are painful to palpation, and a mild increased sensitivity in the treated area is thought to indicate that the desired degree of stimulation has been achieved. A light, continuous contact held beyond the initial stimulation time is thought to produce relaxation through sedation, whereas a heavy continuous contact will eventually produce inhibition because of actual nerve blockage. Continuous percussion, concussion, or vibration will eventually produce inhibition as a result of fatiguing of the reflexes involved. Interrupted moderate percussion, concussion, vibration, or sinusoidal current will prolong the initial stimulation of the reflexes involved.

Direct percussion techniques have long been used by practitioners of manual therapy to influence organs through spinal pathways. Vibratory percussion is thought to invoke a somatovisceral reflex and stimulation of the segmental innervated viscera. This hypothesis suggests that persistent afferent input, driven by percussive vibration, mechanical alteration, pain, and potential local inflammation, triggers a segmental cord response, which in turn induces the development of a somatovisceral reflex. The persistent altered afferent input is then theorized to produce sensitization of local spinal neuron pools and the breakdown of abnormal somatovisceral reflexes.⁹⁹⁻¹⁰³

TABLE 7-2 Segmental Innervation Related to Viscera

Viscera	Segment
Heart	T1-T8
Lung	T3-T9
Esophagus	T5-T6
Stomach	T5-T9
Duodenum	T6-T10
Jejunum	T8-T11
Large Bowel	T9-L1
Appendix	T9-L1
Liver	T6-T11
Gallbladder	T6-T11
Spleen	T7-T10
Pancreas	T7-T9
Kidney	T9-L2
Ureter	T9-L2
Testis/Ovary	T10-T11
Prostate	T11-L1
Uterus	T12-L1

CONCLUSIONS

With the introduction of these procedures into chiropractic practice, the scope of indications for their use undoubtedly will increase. The lack of irritation, as well as the protective gentleness associated with these techniques, allows their use when thrusting forms of manual therapy might be contraindicated. A further advantage of nonthrust procedures is that they can be effective when psychologic factors influence the picture of illness.

Little evidence exists to distinguish the effectiveness or comparative effectiveness of the procedures presented in this chapter. Although a few case reports have been published, no comparative studies have been done to date. Full understanding and absolute validation may not be attainable with the technology and knowledge of today. The notion of clinical success is relative and fragile. It behooves practitioners to make every effort to substantiate the principles and procedures of clinical practice.¹⁰⁴

CHIROPRACTIC GLOSSARY OF COMMONLY USED TERMS*

GLOSSARY

Active Movement Movement accomplished without outside assistance. The patient is unassisted in moving the joint.

Adaptation The adjustment of an organism to its environment; a compensatory reaction of the body to a mechanical distortion.

Adhesion A fibrous band or structure by which parts adhere abnormally.

Adjustment A. The chiropractic adjustment is a specific form of direct articular manipulation, using either long- or short-leverage techniques with specific contacts, characterized by a dynamic thrust of controlled velocity, amplitude, and direction. B. Any chiropractic therapeutic procedure that uses controlled force, leverage, direction, amplitude, and velocity and that is directed at specific joints or anatomic regions. Chiropractors commonly use such procedures to influence joint and neurophysiologic function

Agonistic Muscles Muscles or portions of muscles attached anatomically so that when they contract, they develop forces that reinforce each other.

Alignment Arrangement of position in or along a straight line.

Amplitude Greatness of size, magnitude, breadth, or range.

Analysis Separation into component parts; the act of determining component parts.

Spinal Analysis Examination of the spinal column to determine the relationship of vertebrae to each other and adjacent structures.

Angiothlipsis Direct or indirect pressure on an artery (e.g., in the intervertebral foramen through pressure generated by a discopathy or in the transverse foramina through osteogenic reactions.)

Anomaly Congenital or developmental deviation from the normal or standard.

Antagonistic Muscles Muscles or portions of muscles attached anatomically so that when they contract, they develop forces that oppose each other.

Anterior Pelvic Tilt A position of the pelvis in which the vertical plane through the anterior superior iliac spines is anterior to the vertical plane through the symphysis pubis. It is associated with hyperextension of the lumbar spine and flexion at the hip joints.

Anterolisthesis Anterior translation of the vertebral body.

Arthrosis Degenerative joint disease of the truly movable joints of the spine or extremities.

Articular Strain The result of forces acting on a joint beyond its capacity to adapt. Refers to stretching of joint components beyond physiologic limits, causing damage.

Asymmetry Absence of symmetry of position or motion; dissimilarity in corresponding parts or organs of opposite sides of the body that are normally alike.

Axis A line around which rotatory movement takes place or along which translation occurs; the three-dimensional description of motion of an object with three axes perpendicular to one another. The right-handed Cartesian orthogonal system has three axes designated x , y , and z .

Axoplasmic Flow The flow of neuroplasm along the axon between synapses and toward and away from end organs.

Barrier The limit of impeded motion.

Anatomic Barrier The limit of anatomic integrity; the limit of motion imposed by an anatomic structure. Forcing the movement beyond this barrier would produce tissue damage.

Elastic Barrier (Physiologic) The elastic resistance that is felt at the end of passive range of movement. Further motion toward the anatomic barrier may be induced passively.

Biomechanics The study of structural, functional, and mechanical aspects of human motion. It is concerned mainly with external forces either of a static or dynamic nature dealing with human movements.

Body Mechanics The study of the static and dynamic human body to note the mechanical integration of the parts and to endeavor to restore and maintain the body in as near normal mechanical condition as possible.

Bogginess A tissue texture abnormality characterized principally by a palpable sense of sponginess in the tissue, interpreted as resulting from congestion from increased fluid content.

Bucket-Handle Rib Motion Movement of the lower ribs during respiration so that with inhalation, the lateral aspect of the rib elevates, resulting in an increase of the transverse diameter of the thorax.

Caliper Rib Movement Movement of the lower ribs during respiration so that the rib moves anteriorly in inhalation.

Center of Gravity The point in a body in which the body mass is centered.

*Modified from ACA Council on Technic: Chiropractic terminology: A report, *ACA J Chiro* 25(10): 46, Oct 1988.

Chiropractic

Chiropractic Practice A discipline of the scientific healing arts concerned with the pathogenesis, diagnostics, therapeutics, and prophylaxis of functional disturbances; pathomechanical states; pain syndromes; and neurophysiologic effects related to the statics and dynamics of the locomotor system, especially of the spine and pelvis.

Chiropractic Science A discipline concerned with the investigation of the relationship between structure (primarily the spine) and function (primarily the nervous system) of the human body that leads to the restoration and preservation of health.

Compensation Changes in structural relationships to accommodate for foundation disturbances and to maintain balance.

Contact Point The area of the adjustive hand that makes contact with the patient in the delivery of the chiropractic adjustment. There are 12 contact points: (1) pisiform, (2) hypothenar, (3) metacarpal (knife-edge), (4) digital, (5) distal interphalangeal, (6) proximal interphalangeal, (7) metacarpophalangeal or index, (8) web, (9) thumb, (10) thenar, (11) calcaneal, and (12) palmar.

Contraction A shortening or reduction in size; in connection with muscles, contraction implies shortening of muscle, development of tension in muscle, or both.

Contracture A condition of fixed high resistance to passive stretch of a muscle, resulting from fibrosis of the tissues supporting the muscle or joint.

Coupling A phenomenon of consistent association of one motion (translation or rotation) about an axis with another motion (translation or rotation) about a second axis. One motion cannot be produced without the other.

Creep Deformation of a viscoelastic material with time when it is subjected to a constant, suddenly applied load.

Curvature Deviation from a rectilinear direction; abnormal bending of the spine in any direction away from the natural contour that involves three or more vertebrae.

Curve Normal bending of the spine in the sagittal plane (e.g., primary dorsal and sacral curves, secondary cervical and lumbar curves).

Deformation A change in length or shape.

Degrees of Freedom The number of independent coordinates in a coordinate system required to completely specify the position of an object in space. One degree of freedom is rotation around or translation along one axis. The spine is considered to have six degrees of freedom because it has the capability of rotatory movement around three axes as well as translatory movement along three axes.

Diagnosis The art of distinguishing one disease from another; the use of scientific and skillful methods to establish the cause and nature of an illness.

Disc Herniation Extrusion of the nucleus pulposus into a defect in the annulus fibrosus.

Discogenic Caused by derangement of an intervertebral disc.

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

Discopathy Any pathologic changes in a disc.

Displacement State of being removed from normal position; a disrelationship of a vertebra to its relative structure.

Distortion Any mechanical departure from ideal or normal symmetry in the body framework.

Distraction The movement of two surfaces away from each other.

Dynamics A branch of mechanics that involves the study of the loads and motions of interacting bodies.

Dysarthrosis The strict meaning of joint motion restriction without the neurologic connotations; refers to kinetics.

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

Eccentric Work or Contraction Work produced by a muscle when its length is increasing.

Effleurage A form of massage using slow rhythmic stroking executed with a minimum of force and light pressure.

Elasticity The property of a material or structure to return to its original form after the removal of the deforming load.

End Play (End Feel) Discrete, short-range movements of a joint independent of the action of voluntary muscles, determined by springing each vertebra at the limit of its passive range of motion.

Equilibrium State of a body at rest in which the sums of all forces and movements are zero.

Extension The separation of two embryologically ventral surfaces; movement away from the fetal position; the return movement from flexion.

Facilitation An increase in afferent stimuli so that the synaptic threshold is more easily reached, thereby increasing the efficacy of subsequent impulses in that pathway or synapse. The consequence of increased efficacy is that continued stimulation produces hyperactive responses.

Fibrosis The formation of fibrous tissue.

Fibrositis Inflammatory hyperplasia of the white fibrous tissue of the body, especially of the muscle sheaths and fascial layers of the locomotor system.

Fixation (Dynamic Fault) The state whereby an articulation has become temporarily immobilized in a position that it may normally occupy during any phase of physiologic movement; the immobilization of an articulation in a position of movement when the joint is at rest or in a position of rest when the joint is in movement.

Flexibility The ability of a structure to deform under the application of a load.

Flexion The approximation of two embryologically ventral surfaces; movement toward the fetal position.

Foundation Any structure that supports or participates in the support of any part of the body framework.

Friction Massage A form of deep circular or transverse massage in which the skin is moved over the subcutaneous tissue.

Functional A. Of or pertaining to the function of an organ; not structural; affecting functions only. B. Of or pertaining to a function; affecting the functions but not the structure.

Gliding Movement in which the joint surfaces are flat or only slightly curved, and one articulating surface slides on the other.

Gravitational Line A vertical line through the body where body mass is centered; in theory, laterally viewed ideal posture, which starts at the external auditory canal and passes through the lateral head of the humerus at the tip of the shoulder, and across the greater trochanter and the lateral condyle of the knee, and slightly anterior to the lateral malleolus.

Health A state of optimal physical, mental, and social well-being and not merely the absence of disease and infirmity.

Homeostasis A. Maintenance of static or constant conditions in the internal environment. B. The level of well-being of an individual maintained by internal physiologic harmony.

Hyper Beyond excessive.

Hypo Under or deficient.

Impinge To press or encroach upon; to come into close contact with; to cause pressure on a nerve.

Inhibition Effect of one neuron on another, tending to prevent it from initiating impulses.

Innate Inborn; hereditary.

Innate Intelligence The intrinsic biologic ability of a healthy organism to react physiologically to the changing conditions of the external and internal environment.

Instability Quality or condition of being unstable; not firm, fixed, or constant.

Inversion A turning inward, inside-out, upside-down, or other reversal of the normal orientation of a part; often used to describe passive inverted traction.

Ischemic Compression Application of progressively stronger painful pressure on a trigger point for the purpose of eliminating the point's tenderness.

Isokinetic Exercise Exercise using a constant speed of movement of the body part.

Joint Dysfunction Joint mechanics showing area disturbances of function without structural change; subtle joint dysfunctions affecting quality and range of joint motion. It is diagnosed with the aid of motion palpation and stress and motion radiography investigation. Definition embodies disturbances in function that can be represented by decreased motion, increased motion, or aberrant motion.

Joint Hypomobility Decreased angular or linear joint movement.

Joint Hypermobility Increased angular or linear joint movement. Aberrant joint movements are typically not present.

Clinical Joint Instability Increased linear and aberrant joint movement. The instantaneous axis of rotation (centroids) and patterns of movement are disturbed.

Joint Fixation The state in which an articulation has become temporarily immobilized in a position that it may normally occupy during any phase of physiologic movement; the immobilization of an articulation in a position of movement when the joint is at rest or in a position of rest when the joint is in movement.

Joint Play Discrete, short-range movements of a joint independent of the action of voluntary muscles, determined by springing each vertebra in the neutral position.

Kinematics The division of mechanics that deals with the geometry of the motion of bodies, displacement velocity, and acceleration without taking into account the forces that produce the motion.

Kinesiology The science or study of movement and the active and passive structures involved.

Kinesthesia The sense by which movement, weight, and position are perceived; commonly used to refer specifically to the perception of changes in the angles of joints.

Kinesthetic Pertaining to kinesthesia.

Kinetic Chain A combination of several successively arranged joints, constituting a complex unit, as links in a chain.

Closed Kinetic Chain A system in which motion of one link has determinate relations to every other link in the system.

Open Kinetic Chain A combination of links in which the terminal joint is free.

Kinetics A branch of mechanics that studies the relationship between the force system acting on a body and the changes it produces in the body motion.

Klapping Tapotement (clapping or cupping).

Kneading A form of massage using forceful circular and transverse movement of a large raised fold of skin and underlying muscle.

Kyphoscoliosis Backward and lateral curvature of the spinal column.

Kyphosis Abnormally increased convexity in the curvature of the thoracic spine.

Kyphotic Affected with or pertaining to kyphosis.

Lateral Flexion Bending to the side within the coronal plane.

Laterolisthesis Lateral transitory excursion of the vertebral body.

Lesion Any pathologic or traumatic discontinuity of tissue or loss of function.

Lever A rigid bar moving on a fixed fulcrum.

Listing (Dynamic) Designation of the abnormal movement characteristic of one vertebra in relationship to subadjacent segments.

Dynamic Listing Nomenclature 1. Flexion restriction
2. Extension restriction 3. Lateral flexion restriction (right or left) 4. Rotational restriction (right or left)

Listing (Static) Designation of the spatial orientation of one vertebra in relationship to adjacent segments.

Static Listing Nomenclature 1. Flexion malposition
2. Extension malposition 3. Lateral flexion malposition (right or left) 4. Rotational malposition (right or left)
5. Anterolisthesis 6. Retrolisthesis 7. Laterolisthesis

Lordosis The anterior concavity in the curvature of the lumbar and cervical spine.

Lordotic Affected with or pertaining to lordosis.

Malposition Abnormal or anomalous position.

Static Listing Nomenclature 1. Flexion malposition
2. Extension malposition 3. Lateral flexion malposition (right or left) 4. Rotational malposition (right or left)
5. Anterolisthesis 6. Retrolisthesis 7. Laterolisthesis

Manipulation A. Therapeutic application of manual force. Spinal manipulative therapy, broadly defined, includes all procedures in which the hands are used to mobilize, adjust, manipulate, apply traction to, massage, stimulate, or otherwise influence the spine and paraspinal tissues with the aim of influencing the patient's health. B. A manual procedure that involves a directed thrust to move a joint past the physiologic range of motion without exceeding the anatomic limit (Gatterman MI, Hansen DT: Development of chiropractic nomenclature through consensus, *J Manipulative Physiol Ther* 17[5]:302, 1994).

Massage The systematic therapeutic friction, stroking, and kneading of the body; maneuvers performed by hand on and through the skin of the patient on the subcutaneous tissue. There may be variables in intensity of pressure exerted, surface area treated, and frequency of application.

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

Misalignment Arrangement of position not in or along a straight line.

Mobilization The process of making a fixed part movable; a form of manipulation applied within the physiologic passive range of joint motion characterized by nonthrust passive joint manipulation.

Motion The relative displacement with time of a body in space with respect to other bodies or some reference system.

Myofascial Syndrome Pain, autonomic phenomena, or both referred from active myofascial trigger points with associated dysfunction. The specific muscle or muscle group that causes the symptoms should be identified.

Myofascial Trigger Point A hyperirritable spot, usually within a taut band of skeletal muscle or in the muscle's fascia, that is painful on compression and that can give rise to characteristic referred pain, tenderness, and autonomic phenomena. A myofascial trigger point is to be distinguished from cutaneous, ligamentous, periosteal, and nonmuscular fascial trigger points. Types include active, latent, primary, associated, satellite, and secondary.

Myofascitis Inflammation of a muscle and its fascia, particularly of the fascial insertion of muscle to bone; pain, tenderness, other referred phenomena, and dysfunction attributed to myofascial trigger points.

Myofibrosis Replacement of muscle tissue by fibrous tissue.

Nerve Interference A chiropractic term used to refer to the interruption of normal nerve transmission (nerve energy).

Nerve Transmission The transmission of information along a nerve cell.

Impulse-Based Nerve Transmission Nerve transmission involving the generation and transfer of electrical potentials along a nerve axon.

Non-Impulsed-Based Nerve Transmission The transfer of chemical messengers along a nerve axon (e.g., axoplasmic flow).

Neurodystrophic The disease process within a nerve resulting from trauma, circulation disorders, or metabolic diseases (e.g., a neurodystrophic factor [diabetes and pernicious anemia]).

Neurogenic Originating in nerve tissue.

Neuropathogenic A disease within a tissue resulting from abnormal nerve performance (e.g., Barré-Liou syndrome resulting from neuropathogenic reflexes caused by pathomechanics of the cervical spine).

Neuropathy A general term denoting functional disturbances, pathologic changes, or both in the peripheral nervous system.

Neurophysiologic Effects Functional or aberrant disturbances of the peripheral or autonomic nervous systems. The term is used to designate nonspecific effects related to (a) motor and sensory functions of the peripheral nervous system; (b) vasomotor activity, secretomotor activity, and motor activity of smooth muscle from the autonomic nervous system (e.g., neck, shoulder, arm syndrome [the extremity becomes cool with increased sweating]); and (c) trophic activity of both the peripheral and autonomic nervous systems (e.g., muscle atrophy in neck, shoulder, and arm syndrome).

Neurothipsis Direct or indirect pressure on a nerve (e.g., in the intervertebral foramen through congestion of perineural tissues; in the carpal tunnel through direct ligamentous pressure).

Nutation Motion of the sacrum about a coronal axis in which the sacral base moves anteriorly and inferiorly and the tip of the coccyx moves posteriorly and superiorly; nodding, as of the head.

Counternutation Motion of the sacrum about a coronal axis in which the sacral base moves posteriorly and superiorly and the tip of the coccyx moves anteriorly and inferiorly; nodding, as of the head.

Osteophyte A degenerative exostosis secondary to musculotendinous stress.

Palpation The act of feeling with the hands; the application of variable manual pressure through the surface of the body for the purpose of determining the shape, size, consistency, position, inherent motility, and health of the tissues beneath.

- Motion Palpation** Palpatory diagnosis of passive and active segmental joint range of motion.
- Static Palpation** Palpatory diagnosis of somatic structures in a neutral static position.
- Palpatory Diagnosis** The process of palpating the patient to evaluate neuromusculoskeletal and visceral systems.
- Palpatory Skills** The sensory and tactile skills used in performing a physical examination.
- Palpitation** A subjective sensation of an unduly rapid or irregular heartbeat.
- Passive Movement** Movement carried through by the operator without conscious assistance or resistance by the patient.
- Pathomechanical States** Joint pathomechanics with structural changes that result from imbalanced motion and weight-bearing, trauma, and biochemical changes associated with aging and deficiency states. These tissue changes may be revealed by static radiography and biopsy and definitely diagnosed with surgical exposure. The following are three pathomechanical states: 1. arthrosis 2. spondylolisthesis 3. disc degenerations
- Pelvic Lateral Shift** A movement in the coronal plane of the pelvis in which one anterior-superior iliac spine (ASIS) moves closer to the midline while the opposite ASIS has moved farther away from the midline. It is associated with adduction and abduction of the hip joints.
- Pelvic Lateral Tilt** A position of the pelvis in which it is not level in the horizontal plane (e.g., one anterior-superior iliac spine is higher than the other). It is associated with lateral flexion of the lumbar spine and adduction and abduction of the hip joints.
- Pelvic Rotation** A position of the pelvis in which one anterior-superior iliac spine is anterior to the other. Pelvis rotation is a rotatory movement around the y or vertical axis.
- Pelvic Tilt** A deviation of the pelvis in the sagittal plane from a neutral position.
- Pétrissage** Kneading.
- Physiologic Motion** Normal changes in the position of articulating surfaces during the movement of a joint or region.
- Plane** A flat surface determined by the position of three points in space.
- Coronal Plane** A plane passing longitudinally through the body from one side to the other and dividing it into anterior and posterior portions; also called the *frontal plane*.
- Sagittal Plane** A plane passing longitudinally through the body from front to back and dividing it into right and left portions. The median or midsagittal plane divides the body into approximately equal right and left portions.
- Transverse Plane** A plane passing horizontally through the body perpendicular to the sagittal and frontal planes and dividing it into upper and lower portions.
- Plastic Deformation** A nonrecoverable deformation.
- Plasticity** The property of a material to permanently deform when it is loaded beyond its elastic range.
- Plumb Line** Weighted, true vertical line used for visual comparison with the gravitational line.
- Posture** The attitude of the body; the relative arrangement of the parts of the body. Good posture is that state of muscular and skeletal balance that protects the supporting structures of the body against injury or progressive deformity irrespective of the attitude (erect, lying, squatting, or stooping) in which these structures are working or resting.
- Prophylaxis** That branch of applied biology that seeks to reduce or eradicate disease by removing or altering the responsible causal factors; prevention of disease; preventive treatment; prevention of lesions that result from poor postural hygiene, physical fitness, and faulty body mechanics; prevention of recurrence with follow-up care (e.g., exercise). Many lesions are not curable and become quiescent with treatments; therefore, follow-up care to prevent further pathologic conditions, or at least to retard the pathomechanical process, is necessary.
- Proprioception** Sensory perception of movement or position within the body.
- Range of Motion** The range of translation and rotation of a joint for each of its six ranges of freedom.
- Reciprocal Innervation** The inhibition of antagonistic muscles when the agonist is stimulated.
- Rectilinear Motion** Motion in a straight line.
- Referred Pain** Pain felt in a part other than that in which the cause that produced it is situated.
- Reflex** The result of transforming an ingoing sensory impulse into an outgoing efferent impulse without an act of will.
- Reflex Therapy** Treatment that is aimed at stimulating afferent impulses and evoking a given response (e.g., neuromuscular).
- Relaxation** The decrease in stress in a deformed structure with time when the deformation is held constant.
- Resilience** The property of returning to a former shape, size, or state after distortion.
- Restriction** Limitation of movement; the direction of limited movement in joints that are subluxated, dysfunctional, or both.
- Retrolisthesis** Posterior translation of the vertebral body.
- Roentgenometrics** The direct measurement of structures shown in the roentgenogram or radiograph.
- Rolfing** A 10-hour cycle of deep manual intervention in the soft tissue structure of the body, formerly called *structural integration*, designed by Ida P. Rolf, PhD. Deep effleurage is used to strip tendons and to stretch myofascial tissues to achieve both postural and psychologic effects. See *Effleurage*.
- Ropiness** A tissue texture abnormality characterized by a cord-like or stringlike feeling.
- Rotation** Motion of a body around an axis.
- Sacroiliac Fixation (Sacroiliac Joint Locking)** The absence of normal motion at the sacroiliac joint, demonstrable by motion palpation in which the axis of rotation has shifted to either the superior or inferior portion of the sacroiliac joint, or rarely, a situation in which there is total joint locking with no axis of rotation.

Sacroiliac Extension Fixation (Antero-superior) A state of the sacroiliac joint in which the posterior superior iliac spine is fixed in an anterosuperior position with the innominate bone on that side fixed in extension in relationship to the sacrum. The axis of rotation then shifts inferiorly, and the superior joint remains mobile.

Sacroiliac Flexion Fixation (Posteroinferior) A state of the sacroiliac joint in which the posterior superior iliac spine is fixed in a posteroinferior position with the innominate bone on that side fixed in flexion in relationship to the sacrum. The axis of rotation then shifts superiorly, and the inferior joint remains mobile.

Scan An intermediate screening palpatory examination designed to focus the clinician on regional areas of joint dysfunction.

Scoliosis An appreciable lateral deviation in the normally straight vertical line of the spine.

Functional Scoliosis Lateral deviation of the spine, resulting from poor posture, foundation anomalies, and occupational strains, that is not yet permanently established.

Structural Scoliosis Permanent lateral deviation of the spine so that the spine cannot return to a neutral position.

Shear An applied force that tends to cause an opposite but parallel sliding motion of planes within an object.

Short Leg An anatomic, pathologic, or functional leg deficiency leading to dysfunction.

Side Bending See *Lateral Flexion*.

Somatic Dysfunction Impaired or altered function of related components of the somatic (body framework) system: skeletal, arthrodial, and myofascial structures and related vascular, lymphatic, and neural elements.

Spinography Roentgenometrics of the spine.

Spondylitis Inflammation of the vertebrae.

Spondyloarthrosis Arthrosis of the synovial joints of the spine.

Spondylolisthesis Anterior slippage of a vertebral body on its caudal fellow.

Spondylolysis A unilateral or bilateral interruption in the pars interarticularis.

Spondylophyte Degenerative spur formation arising from the vertebral endplates and usually projecting somewhat horizontally.

Spondylosis Degenerative joint disease as it affects the vertebral body endplates.

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

Sprain Joint injury in which some of the fibers of a supporting ligament are ruptured but in which the continuity of the ligament remains intact.

Spur A projecting body, as from a bone.

Statics The branch of mechanics that deals with the equilibrium of bodies at rest or in motion with zero acceleration.

Stiffness A measure of resistance offered to external loads by a specimen or structure as it deforms.

Strain An overstretching and tearing of musculotendinous tissue.

Stress The sum of the biologic reaction to any adverse stimulus—physical, mental, or emotional, internal or external—that tends to disturb the organism's homeostasis. Should these compensating reactions be inadequate or inappropriate, they may lead to disorders.

Stretching Separation of the origin and insertion of a muscle or attachments of fascia or ligaments by applying a constant pressure, lengthening the fibers of muscle or fascia.

Stringiness A palpable tissue texture abnormality characterized by fine or stringlike myofascial structures

Subacute Less than acute; between acute and chronic.

Subluxation A. An aberrant relationship between two adjacent articular structures that may have functional or pathologic sequelae, causing an alteration in the biomechanical or neurophysiologic reflections of these articular structures, their proximal structures, and body systems that may be directly or indirectly affected by them. B. The alteration of the normal dynamic, anatomic, or physiologic relationships of contiguous articular structures. C. A motion segment in which alignment, movement integrity, physiologic function, or any combination of the three are altered, although contact between the joint surfaces remains intact (Gatterman MI, Hansen DT: Development of chiropractic nomenclature through consensus, *J Manipulative Physiol Ther* 17[5]: 55, 1994).

Orthopedic Subluxation A partial or incomplete dislocation.

Subluxation Complex A theoretic model of motion segment dysfunction (subluxation) that incorporates the complex interaction of pathologic changes in nerve, muscle, ligamentous, vascular, and connective tissues (Gatterman MI, Hansen DT: Development of chiropractic nomenclature through consensus, *J Manipulative Physiol Ther* 17[5]: 55, 1994).

Subluxation Syndrome An aggregate of signs and symptoms that relate to pathophysiology or dysfunction of spinal and pelvic motion segments or to peripheral joints (Gatterman MI, Hansen DT: Development of chiropractic nomenclature through consensus, *J Manipulative Physiol Ther* 17[5]: 55, 1994).

Symmetry The similar arrangement in form and relationship of parts around a common axis or on each side of a plane of the body.

Syndesmophyte An osseous excrescence or bony outgrowth from a ligament, usually projecting vertically in the spine.

Tapotement A tapping or percussing movement in massage, including clapping, beating, and punctation.

Technique Any of a number of physical or mechanical chiropractic procedures used in the treatment of patients.

Thrust The sudden manual application of a controlled directional force on a suitable part of the patient, the delivery of which effects an adjustment.

Tonus The slight continuous contraction of muscle, which, in skeletal muscles, aids in the maintenance of posture.

Torsion A type of load that is applied by a couple of forces (parallel and directed opposite to each other about the axis of a structure).

Traction The act of drawing or exerting a pulling force.

Translation Motion of a rigid body in which a straight line in the body always remains parallel to itself.

Trigger Point See *Myofascial Trigger Point*.

Trophic Of or pertaining to nutrition.

Vertebral Motion Segment A. Two adjacent vertebral bodies and the disc between them; the two posterior joints and the ligamentous structures binding the two vertebrae to one another. B. The consideration of the anatomic and functional relationships of two vertebrae; the mechanical integration of

their articular processes and the related musculature, ligaments, and synovial membranes.

Viscoelasticity The property of a material to show sensitivity to the rate of loading or deformation. The two basic components are viscosity and elasticity.

Viscosity The property of materials to resist loads that produce shear.

X Axis A line passing horizontally from side to side; may also be referred to as the *coronal axis* or the *frontal axis*. Movement around the x axis is said to be in the sagittal plane.

Y Axis A line perpendicular to the ground; may also be referred to as the *vertical axis*. Movement around the y axis is said to be in the horizontal or transverse plane.

Z Axis A line passing horizontally from front to back; may also be referred to as the *sagittal axis*. Movement around the z axis is said to be in the coronal plane.

NAMED CHIROPRACTIC TECHNIQUES

Technique	Developer
Access Seminars	Weigant, Bloomenthal
Activator Technique	Fuhr (Lee)
Active Release Technique (ART)	Leahy
Advanced BioStructural Correction	Jutkowitz
Alberts Cerebral Meningeal Stress Syndrome Technique	Alberts
Alphabiotics (Chrane Condyle Lift)	Chrane
Alternative Chiropractic Adjustments	Wiehe
Applied Chiropractic Distortion Analysis	Kotheimer
Applied Kinesiology	Goodheart, Walters, Schmitt, Thie
Applied Spinal Biomechanical Engineering (ASBE)	Aragona
Applied Upper Cervical Biomechanics	Tiscareño, Amalu
Aquarian Age Healing	Hurley, Sanders
Arnholz Muscle Adjusting	Arnholz
Aronow Biomechanical-Soft Tissue Method	Aronow
Atlas Orthogonality Technique	Sweat
Atlas Specific	Wernsing
Bandy Seminars	Bandy
Bio Cranial System	Boyd
Bio Energetic Synchronization Technique (BEST)	Morter
Bio-Geometric Integration	Brown
Bioenergetics	Broeringmeyer
Bio Kinesiology	Barton
Bio-Kinetics Health System	Newsum
Biomagnetic Technique	Stoffels, Borham, Broeringmeyer
Bio-Polarity Technique	Cargioli
BioSET	Cutler
Blair Upper Cervical Technique	Blair
Bloodless Surgery	Lorenz, Failor, DeJarnette
Blye Cranial Technique	Blye
Body Integration	Espy
Buxton Technical Course of Painless Chiropractic	Buxton
Cerebrospinal Fluid Technique	Glassey
Charrette Extremity Adjusting Technique	Charrette
Chirodontics	Walker
Chiroenergetics	Kimmel
Chirometry	Quigly
Chiro Plus Kinesiology	Dowty
Chiropractic Concept	Prill
Chiropractic Manipulative Reflex Technique (CMRT)	DeJarnette
Chiropractic Neuro-Biochemical Analysis	Unknown
Chiropractic Spinal Biophysics (Clinical Biomechanics of Posture)	Harrison
CHOK-E System	Johnson
Clinical Kinesiology	Beardall

Technique	Developer
Collins Method of Painless Adjusting	Collins
Concept Therapy	Fleet, Dill
Contact Reflex Analysis	Versendaal
Cranial Technique	Dejarnette, Denton, Goodheart
Craniopathy	Cottam
Craniosacral Therapy	Upledger
Creed Neural Kinetic Integration Technique	Creed
Directional Nonforce Technique (DNFT)	Van Rumpt, Johns
Distraction Technique	Cox, Markey, Leander
Diversified Technique	Beatty, Bonyun, Carver, Crawford, Frank, Grecco, Janse, DeGiacomo, Logan, LeBeau, Metzinger, Reinert, States, Stonebrink, Stierwalt
Dynamic Spinal Analysis	Hochman
Endo-Nasal Technique	Gibbons, Lake, Broeringmeyer
Extremity Technique	Burns, Grecco, Gertler, Hearon, Malley, Schawn, Christenson, Charrette
Focalizer Spinal Recoil Stimulus Reflex Effector Technique	George
Freeman Chiropractic Procedure	Freeman
Functional Analysis Chiropractic Technique	Clary
Fundamental Chiropractic	Ashton
Global Energetic Matrix	Babinet
Gonstead Technique	Gonstead
Graston Instrument-Assisted Soft Tissue Mobilization	Graston
Grostick Technique	Grostick
Herring Cervical Technique	Herring
Holographic Diagnosis and Treatment	Franks, Gleason
Howard System	Howard
Keck Method of Analysis	Keck
Koren Specific Technique	Koren
King Tetrahedron Concept	King
Lemond Brain Stem Technique	Lemond
Logan Basic Technique	Logan, Coggins
Master Energy Dynamics	Bartlett
Matrix Repatterning	Roth
Mawhinney Scoliosis Technique	Mawhinney
McTimoney Technique	McTimoney
Mears Technique	Mears
Meric Technique System	Cleveland, Palmer, Loban, Forster, Riley
Micromanipulation (Micro-Chiropractic Technique)	Young
Morreim Technique	Morreim
Motion Palpation	Gillet, Faye
Muscle Palpation	Spano
Muscle Response Testing	Lepore, Fishman, Grinims
MusculoSkeletal Synchronization and Stabilization Technique	Krippenbrock
Nambudripad's Allergy Elimination Technique	Nambudripad
Nerve Signal Interference	Craton
Network Chiropractic (Network Spinal Analysis)	Epstein
Neuro Emotional Technique	Walker
Neuro Lymphatic Reflex Technique	Chapman
Neuro Organizational Technique	Ferrari
Neuro Vascular Reflex Technique	Bennett
Olesky 21st Century Technique	Olesky

Technique	Developer
Ortman Technique	Ortman
Perianal Postural Reflex Technique	Unknown
Pettibon Spinal Biomechanics Technique	Pettibon
Pierce-Stillwagon Technique	Pierce, Stillwagon
Polarity Technique	Stone
Positional Release Therapy	Roth, D'Ambrogio, Jones
Posture Imbalance Patterns	Sinclair
Pro-Adjuster Technique	Pisciottano
Pure Chiropractic Technique	Morreim
Reaver's Fifth Cervical Key	Reaver, Pierce
Receptor-Tonus Technique	Nimmo
Riddler Reflex Technique	Riddler
Sacro-Occipital Technique	DeJarnette
Soft Tissue Orthopedics	Rees
Somatic Technique	Hanna
Somatosynthesis	Ford
Spears Painless System	Spears
Specific Majors	Nemiroff
Spinal Stress (Stressology)	Ward
Spinal Touch Technique	Rosquist
Spondylotherapy	Forster, Riley
Sympathetic-Central Nerve Technique	Usselman
Tensegrity Therapy	Roth
Thompson Terminal Point Technique	Thompson, Stucky, Mitchell
Tieszen Technique	Tieszen
Toftness Technique	Toftness
Top Notch Visceral Techniques	Portelli, Marcellino
Torque Release Technique	Holder
Tortipelvis/Torticollis	Barge
Total Body Modification	Frank
Touch for Health	Thie
Trigenics Myoneural Medicine	Austin
Truscott Technique	Truscott
Ungerank Specific Low Force Chiropractic Technique	Ungerank
Upper Cervical Technique (Hole-in-One, Toggle)	Palmer, Duff, Grostic, Kale, Life, Laney, National Upper Cervical Chiropractic Association (NUCCA)
Variable Force Technique	Leighton
Von Fox Combination Technique	Von Fox
Webster Technique	Webster
Zimmerman Technique	Zimmerman
Zindler Reflex Technique	Zindler

COMPILATION OF RELIABILITY STUDIES ON JOINT EVALUATION PROCEDURES

Author, Reference	Region	Examiners, Experience	Subjects	Quality	Score Findings	Degree of Reliability
Bergström and Courtis ¹	L1–L5	2 DC, pre-trained	100 Asx	67%	% = 65 to 88	Inconclusive
Binkley et al ²	L1–S1	6 PT, at least 6 yr	18 Sx	50%	$\kappa = 0.09$ ICC = 0.25 (CI, 0–0.39)	Slight
Boline et al ³	T12–S1	1 DC (<1 yr), 1 St	50 (23 Sx, 27 Asx)	83%	$\kappa = -0.05$ to 0.33 % = 60 to 90	None to fair
Brismée et al ⁴	T5–T7	3 PT, ≥ 12 yr	41 Asx	50%	$\kappa = 0.27$ to 0.65 % = 63 to 83	Fair to substantial
Carmichael ⁵	SI	10 DC St	54 Asx	50%	$\kappa = -0.07$ to 0.19 % = 66 to 100	None to slight
Christensen et al ⁶	T1–T8	2 DC, Exp	107 (51 angina, 56 Asx)	100%	$\kappa = 0.22$ to 0.24	Fair
Comeaux et al ⁷	C2–T8	3 DO, >10 yr	54 Asx	67%	$\kappa = 0.12$ to 0.56	Slight to moderate
Deboer et al ⁸	C1–C7	3 DC, Exp	40 Asx	50%	$\kappa w = 0.03$ to 0.42	Slight to moderate
Degenhardt et al ⁹	L1–L4	3 DO, <10 yr	15 Asx	50%	$\kappa = 0.20$ % = 66	Slight
Downey et al ¹⁰	Lumbar	6 PT, 7 to 15 yr	30 Sx	33%	$\kappa = 0.23$ to 0.54	Fair to moderate
Fjellner et al ¹¹	C0–C2	2 PT, 6 & 8 yr	48 (11 Sx, 36 Asx)	67%	$\kappa w = 0.01$ to 0.18 % = 60 to 87	Slight
Fjellner et al ¹¹	C0–T5	2 PT, 6 & 8 yr	48 (11 Sx, 36 Asx)	67%	$\kappa w = -0.16$ to 0.49 % = 41 to 92	None to moderate
Flynn et al ¹²	SI	8 PT, Exp	55 Sx	33%	$\kappa = -0.08$ to 0.59	None to moderate
Gonella et al ¹³	T12–S1	5 PT, ≥ 3 yr	5 Asx	17%	Visual inspection of raw data	Inconclusive
Haas et al ¹⁴	T3–L1	2 DC, 15 yr	73, 49% Sx	67%	$\kappa = 0.08$ to 0.22	Slight to fair
Hanten et al ¹⁵	C1–C3	2 PT, Exp	40 Sx	50%	$\kappa = -0.71$ to 0.86 % = 70 to 95	None to almost perfect
Herzog et al ¹⁶	SI	10 DC, >1 yr	11 (10 Sx, 1 Asx)	33%	% = 54 to 78	Inconclusive
Hicks et al ¹⁷	L1–L5	3 PT, 1 DC/PT, 4 to 8 yr	63 Sx	33%	$\kappa w = -0.02$ to 0.26 % = 52 to 69	None to slight
Humphreys et al ¹⁸	C1–C7	20 DC St, 4th yr	Three with congenital block vertebrae	50%	$\kappa = 0.46$ to 0.76	Moderate to substantial
Inscoe et al ¹⁹	T12–S1	2 PT, ≥ 4 yr	6 Sx	17%	Scott's $\pi = 18.4\%$ % = 33.3 to 58.3	Not acceptable

TABLE 1 Motion Palpation Interexaminer Reliability Studies—Cont'd

Author, Reference	Region	Examiners, Experience	Subjects	Quality	Score Findings	Degree of Reliability
Jull and Bullock ²⁰	T12–S1	2 PT, Exp	10 Asx	0%	$r = 0.82$ to 0.94 % = 86	Inconclusive
Keating et al ²¹	T12–S1	3 DC, >2.5 yr	46 (21 Sx, 25 Asx)	67%	$\kappa = -0.18$ to 0.31	None to fair
Leboeuf ²²	L1–S1	4 DC St	45 Sx	17%	% > 90	Inconclusive
Lindsay et al ²³	L1–S1	2 PT, ≥ 6 yr	18 (Sx & Asx)	100%	$\kappa w = -0.03$ to 0.6 % = 14 to 100	None to moderate
Lindsay et al ²³	S1	2 PT, ≥ 6 yr	18 (Sx & Asx)	100%	$\kappa w = 0.2$ to 0.6 % = 50 to 100	Slight to moderate
Love and Brodeur ²⁴	T1–L5	8 DC St	32 Asx	17%	$r = 0.01$ to 0.49	Inconclusive
Lundberg and Gerdle ²⁵	T10–S1	3 PT, Exp	150 Asx	50%	$\kappa w = 0.59$ to 0.75	Moderate to substantial
Maher and Adams ²⁶	L1–L5	6 PT, ≥ 5 yr	90 Sx	67%	ICC = -0.4 to 0.73 % = 13 to 43	Poor to fair
Maher et al ²⁷	L3	5 PT, ≥ 5 yr	40 Asx	33%	ICC = 0.50 to 0.77 SEM = 0.72 to 1.58	Fair to good
Marcotte et al ²⁸	C0–C7	25 DC (1 Exp, 24 St)	3 Asx	33%	$\kappa = 0.6$ to 0.8	Moderate to substantial
Marcotte et al ²⁹	C0–C7	24 DC (1 Exp, 23 St)	3 Asx	33%	$\kappa = 0.7$ to 0.75	Moderate
McPartland and Goodridge ³⁰	C0–C3	2 DO, ≥ 10 yr	18 (7 Sx, 11 Asx)	83%	$\kappa = 0.34$ % = 66.7	Fair
Meijne ³¹	SI	2 PT St	38 (9 Sx, 29 Asx)	83%	$\kappa = -0.30$ to 0.75 % = 48 to 100	None to substantial
Mior et al ³²	C0–C2	2 DC St, 3 months training	59 Asx	50%	$\kappa = 0.15$ % = 61	Slight
Mior et al ³³	SI	3 DC, >5 yr, 74 St	15 Asx	33%	$\kappa = 0.00$ to 0.30	None to fair
Mootz et al ³⁴	L1–S1	2 DC, ≥ 7	60 Asx	33%	$\kappa = -0.17$ to 0.17	None to slight
Nansel et al ³⁵	Mid & lower C	4 DC (3 Exp, 1 St)	270 Asx	50%	$\kappa = 0.01$ % = 45.6 to 54.3	Almost none
Olson et al ³⁶	C0–C2	6 PT, ≥ 4.5 yr	10 Asx	33%	$\kappa = -0.04$ to 0.12	None to slight
Paydar et al ³⁷	SI	2 DC St	32 Asx	50%	$\kappa = 0.09$ % = 34.4	Slight
Phillips and Twomey ³⁸	L1–L5	2 PT, NI	72 (63 Sx, 9 Asx)	67%	$\kappa w = -0.15$ to 0.32 % = 55 to 99	None to fair
Rhudy et al ⁴⁰	C1–L5	3 DC, Exp	17 Sx	50%	κ values not presented	Inconclusive
Robinson et al ⁴¹	SI	2 PT, Ave 5.8 yr	61 (45 Sx, 16 Asx)	83%	$\kappa = -0.06$ % = 48	None
Smedmark et al ⁴³	C1–T1	2 PT, >25 yr	61 Sx	67%	$\kappa = 0.28$ to 0.43 % = 70 to 87	Fair to moderate
Strender et al ⁴⁴	C0–C3	2 PT, ≥ 21 yr	50 (25 Sx, 25 Asx)	83%	$\kappa = 0.06$ to 0.15 % = 26 to 44	None to slight
Strender et al ⁴⁵	L5–S1	2 MD, 2 PT, Exp	71 Sx	67%	$\kappa = -0.08$ to 0.75 % = 48 to 88	None to substantial

Continued

TABLE 1 Motion Palpation Interexaminer Reliability Studies—Cont'd

Author, Reference	Region	Examiners, Experience	Subjects	Quality	Score Findings	Degree of Reliability
Tong et al ⁴⁶	SI	4 DO, NI	24 Sx	33%	Stork test $\kappa = 0.27$ to 0.50 Flexion tests $\kappa = 0.06$ to 0.30	Fair to moderate None to fair
Vincent-Smith and Gibbons ⁴⁷	SI	9 DO, ≥ 4 yr	9 Asx	50%	$\kappa = 0.013$ to 0.09 % = 34 to 50	Slight
Wiles ⁴⁸	SI	8 DC, 2.75 yr Exp average	46 Asx	17%	$r = 0.13$ to 0.43 % = 47 to 64	Inconclusive

From Haneline M, Cooperstein R, Young M, et al: An annotated bibliography of spinal motion palpation reliability studies. *J Can Chiropr Assoc* 53(1):40-58, 2009.

Asx, Asymptomatic; Ave, average; C, cervical; CI, 95% confidence interval; DC, doctor of chiropractic; DO, doctor of osteopathic medicine; Exp, experienced; ICC, intraclass coefficient; Intra, intraexaminer reliability; Inter, interexaminer reliability; L, lumbar; MP, motion palpation; MT, manipulative therapist; NI, no information presented; PT, physical therapist; r, Pearson correlation coefficient; S, sacral; SEM, standard error of measurement; SI, sacroiliac; Sx, student; Sx, symptomatic; T, thoracic; κ , weighted.

REFERENCES

- Bergstrom E, Courtis G: An inter- and intraexaminer reliability study of motion palpation of the lumbar spine in lateral flexion in the seated position, *Eur J Chiropractic* 34:121, 1986.
- Binkley J, Stratford PW, Gill C: Interrater reliability of lumbar accessory motion mobility testing, *Phys Ther* 75:786, 1995.
- Boline P, et al: Interexaminer reliability of palpation evaluations of the lumbar spine, *Am J Chiropract Med* 1:5, 1988.
- Bismée JM, et al: Interrater reliability of a passive physiological intervertebral motion test in the mid-thoracic spine, *J Manipulative Physiol Ther* 29:368, 2006.
- Carmichael JP: Inter- and intra-examiner reliability of palpation for sacroiliac joint dysfunction, *J Manipulative Physiol Ther* 10:164, 1987.
- Christensen HW, et al: Palpation of the upper thoracic spine: an observer reliability study, *J Manipulative Physiol Ther* 25:285, 2002.
- Comeaux Z, et al: Measurement challenges in physical diagnosis: refining inter-rater palpation, perception and communication, *J Body Mov Ther* 5:245, 2001.
- Deboer KF, et al: Reliability study of detection of somatic dysfunctions in the cervical spine, *J Manipulative Physiol Ther* 8:9, 1985.
- Degenhardt BF, et al: Interobserver reliability of osteopathic palpation diagnostic tests of the lumbar spine: Improvements from consensus training, *J Am Osteopath Assoc* 105:465, 2005.
- Downey B, Taylor N, Niere K: Can manipulative physiotherapists agree on which lumbar level to treat based on palpation? *Physiotherapy* 89:74, 2003.
- Fjellner A, et al: Interexaminer reliability in physical examination of the cervical spine, *J Manipulative Physiol Ther* 22:511, 1999.
- Flynn T, et al: A clinical prediction rule for classifying patients with low back pain who demonstrate short-term improvement with spinal manipulation, *Spine* 27:2835, 2002.
- Gonella C, Paris SV, Kutner M: Reliability in evaluating passive intervertebral motion, *Phys Ther* 62:436, 1982.
- Haas M, et al: Reliability of manual end-play palpation of the thoracic spine, *Chiropr Tech* 7:120, 1995.
- Hanten WP, Olson SL, Ludwig G: Reliability of manual mobility testing of the upper cervical spine in subjects with cervicogenic headache, *J Man Manip Ther* 10:76-82, 2002.
- Herzog W, et al: Reliability of motion palpation procedures to detect sacroiliac joint fixations, *J Manipulative Physiol Ther* 12:86, 1989.
- Hicks GE, et al: Interrater reliability of clinical examination measures for identification of lumbar segmental instability, *Arch Phys Med Rehabil* 84:1858, 2003.
- Humphreys BK, Delahaye M, Peterson CK: An investigation into the validity of cervical spine motion palpation using subjects with congenital block vertebrae as a "gold standard." *BMC Musculoskeletal Disord* 5:19, 2004.
- Inscoe E, et al: Reliability in evaluating passive intervertebral motion of the lumbar spine, *J Man Manip Ther* 3:135, 1995.
- Jull G, Bullock M: A motion profile of the lumbar spine in an aging population assessed by manual examination, *Physiother Pract* 3:70, 1987.
- Keating JC Jr, et al: Interexaminer reliability of eight evaluative dimensions of lumbar segmental abnormality, *J Manipulative Physiol Ther* 13:463, 1990.
- Leboeuf C: Chiropractic examination procedures: a reliability and consistency study, *J Aust Chiropr Assoc* 19:101, 1984.
- Lindsay D, et al: Interrater reliability of manual therapy assessment techniques, *Phys Ther Can* 47:173, 1995.
- Love RM, Brodeur RR: Inter- and intra-examiner reliability of motion palpation for the thoracolumbar spine, *J Manipulative Physiol Ther* 10:1, 1987.
- Lundberg G, Gerdle B: The relationships between spinal sagittal configuration, joint mobility, general low back mobility and segmental mobility in female homecare personnel, *Scand J Rehabil Med* 31:197, 1999.
- Maher C, Adams R: Reliability of pain and stiffness assessments in clinical manual lumbar spine examination, *Phys Ther* 74:801, 1994, discussion 809.
- Maher C, Latimer J, Adams R: An investigation of the reliability and validity of posteroanterior spinal stiffness judgments made using a reference based protocol, *Phys Ther* 78:829, 1998.
- Marcotte J, Normand MC, Black P: The kinematics of motion palpation and its effect on the reliability for cervical spine rotation, *J Manipulative Physiol Ther* 25:E7, 2002.
- Marcotte J, Normand MC, Black P: Measurement of the pressure applied during motion palpation and reliability for cervical spine rotation, *J Manipulative Physiol Ther* 28:591, 2005.
- McPartland JM, Goodridge JP: Counterstrain and traditional osteopathic examination of the cervical spine compared, *J Body Mov Ther* 1:173, 1997.
- Meijne W, et al: Intraexaminer and interexaminer reliability of the Gillet test, *J Manipulative Physiol Ther* 22:4, 1999.
- Mior S, et al: Intra and interexaminer reliability of motion palpation in the cervical spine, *J Can Chiropr Assoc* 29:195, 1985.
- Mior SA, McGregor M, Schut B: The role of experience in clinical accuracy, *J Manipulative Physiol Ther* 13:68, 1990.
- Mootz RD, et al: Intra- and interobserver reliability of passive motion palpation of the lumbar spine, *J Manipulative Physiol Ther* 12:440, 1989.

35. Nansel DD, et al: Interexaminer concordance in detecting joint play asymmetries in the cervical spines of otherwise asymptomatic subjects, *J Manipulative Physiol Ther* 12:428, 1989.
36. Olson KA, et al: Radiographic assessment and reliability study of the craniovertebral sidebending, *J Manual Manipulative Ther* 6:87, 1998.
37. Paydar D, Thiel H, Gemmell H: Intra- and interexaminer reliability of certain pelvic palpatory procedures and the sitting flexion test for sacroiliac joint mobility and dysfunction, *J Neuromusculoskel Sys* 2:65, 1994.
38. Phillips DR, Twomey LT: A comparison of manual diagnosis with a diagnosis established by a unilevel lumbar spinal block procedure, *Man Ther* 1:82, 1996.
39. Potter L, McCarthy C, Oldham J: Intraexaminer reliability of identifying a dysfunctional segment in the thoracic and lumbar spine, *J Manipulative Physiol Ther* 29:203, 2006.
40. Rhudy T, Sandefur M, Burk J: Interexaminer/intertechnique reliability in spinal subluxation assessment: a multifactorial approach, *Am J Chiropract Med* 1:111, 1988.
41. Robinson HS, et al: The reliability of selected motion and pain provocation tests for the sacroiliac joint, *Man Ther* 12:72, 2007.
42. Sebastian D, Chovvath R: Reliability of palpation assessment in non-neutral dysfunctions of the lumbar spine, *Orthop Phys Ther Pract* 16:23, 2004.
43. Smedmark V, Wallin M, Arvidsson I: Inter-examiner reliability in assessing passive intervertebral motion of the cervical spine, *Man Ther* 5:97, 2000.
44. Strender LE, Lundin M, Nell K: Interexaminer reliability in physical examination of the neck, *J Manipulative Physiol Ther* 20:516, 1997.
45. Strender LE, et al: Interexaminer reliability in physical examination of patients with low back pain, *Spine* 22:814, 1997.
46. Tong HC, et al: Interexaminer reliability of three methods of combining test results to determine side of sacral restriction, sacral base position, and innominate bone position, *J Am Osteopath Assoc* 106:464, 2006.
47. Vincent-Smith B, Gibbons P: Inter-examiner and intra-examiner reliability of the standing flexion test, *Man Ther* 4:87, 1999.
48. Wiles M: Reproducibility and interexaminer correlation of motion palpation findings of the sacroiliac joints, *J Can Chiropr Assoc* 24:56, 1980.

TABLE 2 Reliability of Pain Provocation in the Spine and/or Sacroiliac Region Studies

Author	Region	Examiners, Experience	Subjects	Study Type	Qual	Findings	Degree of Reliability
Boline et al (1988) ¹	T12–S1	1 DC, 1 yr, 1 St	50 (23 Sx, 27 Asx)	Inter	83	$\kappa = -0.03$ to 0.49 % = 60 to 90	None to moderate
Boline et al (1993) ²	L1–S1	3 DC, Exp	28 Sx	Inter	50	$\kappa = 0.48$ to 0.90 % = 79 to 96	Moderate to almost perfect
Christensen et al ³	T1–T8	2 DC, Exp	107 (51 Sx angina, 56 Asx)	Inter	100	$\kappa = 0.38$ to 0.70	Fair to substantial
	T1–T8	2 DC, Exp	107 (51 Sx angina, 56 Asx)	Intra	100	$\kappa = 0.34$ to 0.77	Fair to substantial
Deboer et al ⁴	C1–C7	3 DC, Exp	40 Asx	Inter	50	$\kappa = -0.04$ to 0.48	None to moderate
	C1–C7	3 DC, Exp	40 Asx	Intra	25	$\kappa = 0.20$ to 0.56	Fair to moderate
Hubka and Phelan ⁵	C2–C7	2 DC, 1–5 yr	30 Sx	Inter	50	$\kappa = 0.68$	Substantial
Keating et al ⁶	T12–S1	3 DC, 2.5 yr	46 (21 Sx, 25 Asx)	Inter	67	$\kappa = 0.19$ to 0.48	Slight to moderate
Lundberg and Gerdle ⁷	T10–S1	2 PT, Exp	150	Inter	50	$\kappa = 0.67$ to 0.71	Substantial
McCombe et al ⁸	L1–L5, SI	3 MD, 1 PT, Exp	83 Sx	Inter	17	$\kappa = 0.28$ to 0.47	Fair to moderate
Paydar et al ⁹	SI	2 St	32 Asx	Inter	50	$\kappa = 0.73$ % = 90.6	Substantial Near perfect
	SI	2 St	32 Asx	Intra	25	$\kappa = 0.91$ % = 96.8	
Strender et al ¹⁰	C0–C3	2 PT, ≥ 21 yr	50 (25 Sx, 25 Asx)	Inter	67	$\kappa = 0.31$ to 0.52 % = 58 to 68	Fair to moderate

Continued

TABLE 2 Reliability of Pain Provocation in the Spine and/or Sacroiliac Region Studies—Cont'd

Author	Region	Examiners, Experience	Subjects	Study Type	Qual	Findings	Degree of Reliability
Strender et al ¹¹	L5–S1	2 MD, 2 PT, Exp	71 Sx	Inter	67	$\kappa = 0.06$ to 0.71 % = 73 to 88	Slight to substantial
Van Suijlekom ¹²	CO–C7	2 neuro, Exp	24 Sx	Inter	17	$\kappa = 0.14$ to 0.31	Slight to fair
Viikari-Juntura ¹³	C1–C7	1 MD, 1 PT, Exp	52 Sx	Inter	17	$\kappa = 0.47$ to 0.56	Moderate
Waddell et al ¹⁴	L1–S1	4 MD, Exp	475 Sx, 335 Asx	Inter	33	$\kappa = 1.0$ % = 100	Almost perfect

From Haneline MT, Morgan Young M: A review of intraexaminer and interexaminer reliability of static spinal palpation: A literature synthesis. *J Manipulative Physiol Ther* 32:379, 2009.

Asx, Asymptomatic; C, cervical; DC, doctor of chiropractic; Exp, experienced; Inter, interexaminer reliability; Intra, intraexaminer reliability; L, lumbar; MD, medical doctor; neuro, neurologist; PT, physical therapist; S, sacral; SI, sacroiliac; St, student; Sx, symptomatic; T, thoracic.

Percentages rounded to the nearest whole number.

REFERENCES

- Boline P, et al: Interexaminer reliability of palpatory evaluations of the lumbar spine, *Am J Chiropr Med* 1:5, 1988.
- Boline PD, et al: Interexaminer reliability of eight evaluative dimensions of lumbar segmental abnormality: Part II, *J Manipulative Physiol Ther* 16:363, 1993.
- Christensen HW, et al: Palpation of the upper thoracic spine: an observer reliability study, *J Manipulative Physiol Ther* 25:285, 2002.
- Deboer KE, et al: Reliability study of detection of somatic dysfunctions in the cervical spine, *J Manipulative Physiol Ther* 8:9, 1985.
- Hubka MJ, Phelan SP: Interexaminer reliability of palpation for cervical spine tenderness, *J Manipulative Physiol Ther* 17:591, 1994.
- Keating J, et al: Interexaminer reliability of eight evaluative dimensions of lumbar segmental abnormality, *J Manipulative Physiol Ther* 13:463, 1990.
- Lundberg G, Gerdle B: The relationships between spinal sagittal configuration, joint mobility, general low back mobility and segmental mobility in female homecare personnel, *Scand J Rehabil Med* 31:197, 1999.
- McCombe PE, et al: Volvo Award in clinical sciences. Reproducibility of physical signs in low back pain, *Spine* 14:908, 1989.
- Paydar D, Thiel H, Gemmell H: Intra- and interexaminer reliability of certain pelvic palpatory procedures and the sitting flexion test for sacroiliac joint mobility and dysfunction, *J Neuromusculoskeletal Syst* 2:65, 1994.
- Strender LE, Lundin M, Nell K: Interexaminer reliability in physical examination of the neck, *J Manipulative Physiol Ther* 20:516, 1997.
- Strender LE, et al: Interexaminer reliability in physical examination of patients with low back pain, *Spine* 22:814, 1997.
- Van Suijlekom HA, et al: Interobserver reliability in physical examination of the cervical spine in patients with headache, *Headache* 40:581, 2000.
- Viikari-Juntura E: Interexaminer reliability of observations in physical examinations of the neck, *Phys Ther* 67:1526, 1987.
- Waddell G, et al: Normality and reliability in the clinical assessment of backache, *Br Med J (Clin Res Ed)* 284:1519, 1982.

TABLE 3 Reliability of Locating Landmarks in the Spine and/or Sacroiliac Region Studies

Author	Region	Examiners, Experience	Subjects	Study Type	Qual	Findings	Degree of Reliability
Keating et al ¹	T12–S1	3 DC, 2.5 \geq yr	46 (21 Sx, 25 Asx)	Inter	67	$\kappa = -0.08$ to 0.03	None to slight
Billis et al ²	C5, T6, L5 C5, T6, L5	17 PT \geq 2 yr, 13 PT St	9 Asx	Inter	67	F = 18.43 P = 0.001	Inconclusive
		17 PT, \geq 2 yr, 13 PT St	9 Asx	Intra	50	F = 2.09 P = 0.161	Inconclusive
Binkley et al ³	L1–S1	6 PT, at least 6 yr	18 Sx	Inter	50	$\kappa_w = 0.30$ ICC = 0.69 (CI, 0.53–0.82)	Fair Fair to good
Broadbent et al ⁴	T12–S1	2 MD, NI	100 Sx	Inter	50	$\kappa_w = 0.43$ –0.63	Moderate to substantial
Byfield and Humphreys ⁵	L1, L4	2 DC, Exp	42 Asx	Inter	17	% = 55–81	Inconclusive
	L1, L4	2 DC, Exp	42 Asx	Intra	0	% = 39–62	Inconclusive

Continued

TABLE 3 Reliability of Locating Landmarks in the Spine and/or Sacroiliac Region Studies—Cont'd

Author	Region	Examiners, Experience	Subjects	Study Type	Qual	Findings	Degree of Reliability
Downey et al ⁶	L1–L5	6 PT, >7 yr	20 Sx	Inter	33	$\kappa_w = 0.44–0.98$	Moderate to almost perfect
Holmgren and Waling ⁷	L5 and SI	3 PT, ≈15 yr	25 Sx	Inter	67	$\kappa = 0.11–0.17$	Slight
McKenzie and Taylor ⁸	L1–L5	14 PT, Inexp 3 PT	5 Asx	Inter	17	$\kappa = 0.28$ % = 56	Fair
	L1–L5		5 Asx	Intra	25	$\kappa = 0.61–0.9$ % = 84–96	Substantial to almost perfect
O'Haire and Gibbons ⁹	SI	10 DO, fifth-year St 10 DO, fifth-year St	10 Asx	Inter	50	$\kappa = 0.04–0.08$	Slight
	SI		10 Asx	Intra	25	$\kappa = -0.05$ to 0.58	None to moderate
Simmonds and Kumar ¹⁰	L4, SI	20 PT, St	20 Asx	Inter	33	Coef Var = 0.48–0.65	Inconclusive
	L4, SI	20 PT, St	20 Asx	Intra	25	Coef Var = 0.28–0.78	Inconclusive

From Haneline MT, Morgan Young M: A review of intraexaminer and interexaminer reliability of static spinal palpation: A literature synthesis. *J Manipulative Physiol Ther* 32:379, 2009. Asx, Asymptomatic; C, cervical; CI, 95% confidence interval; Coef Var, coefficient of variation; DC, doctor of chiropractic; DO, doctor of osteopathic medicine; Exp, experienced; F, observed F value ICC, intraclass coefficient; Inexp, inexperienced; Inter, interexaminer reliability; Intra, intraexaminer reliability; L, lumbar; MD, doctor of medicine; NI, no information presented; P, significance level; PT, physical therapist; S, sacral; SI, sacroiliac; St, student; Sx, symptomatic; T, thoracic.

REFERENCES

- Keating J, et al: Interexaminer reliability of eight evaluative dimensions of lumbar segmental abnormality, *J Manipulative Physiol Ther* 13:463, 1990.
- Billis EV, Foster NE, Wright CC: Reproducibility and repeatability: errors of three groups of physiotherapists in locating spinal levels by palpation, *Man Ther* 8:223, 2003.
- Binkley J, Stratford PW, Gill C: Interrater reliability of lumbar accessory motion mobility testing, *Phys Ther* 75:786, 1995.
- Broadbent CR, et al: Ability of anaesthetists to identify a marked lumbar interspace, *Anaesthesia* 55:1122, 2000.
- Byfield D, Humphreys K: Intra- and inter-examiner reliability of bony landmark identification in the lumbar spine, *Eur J Chiropr* 40:13, 1992.
- Downey BJ, Taylor NE, Niere KR: Manipulative physiotherapists can reliably palpate nominated lumbar spinal levels, *Man Ther* 4:151, 1999.
- Holmgren U, Waling K: Inter-examiner reliability of four static palpation tests used for assessing pelvic dysfunction, *Man Ther* 13(1):50–56, 2008.
- McKenzie A, Taylor N: Can physiotherapists locate lumbar spinal levels by palpation? *Physiotherapy* 83:235, 1997.
- O'Haire C, Gibbons P: Inter-examiner and intra-examiner agreement for assessing sacroiliac anatomical landmarks using palpation and observation: Pilot study, *Man Ther* 5:13, 2000.
- Simmonds M, Kumar S: Health care ergonomics. Part II: location of bony structures by palpation—a reliability study, *Int J Ind Ergon* 11(2):145, 1993.

TABLE 4 Reliability of Spine and/or Sacroiliac Position or Alignment Studies

Author	Region	Examiners, Experience	Subjects	Study Type	Qual	Findings	Degree of Reliability
Collaer et al ¹	Lumbar	3 PT	30 Sx	Inter	67	$\kappa = 0.18–0.39$ % = 63–76	Slight to fair
Fryer et al	SI	10 DO St, trained and untrained	10 Asx	Inter	33	$\kappa = 0.08$	Slight
	SI	10 DO St, trained and untrained	10 Asx	Intra	25	trained, 0.15 untrained $\kappa = 0.54$	Moderate
Hart ³	C1–C2	12 DC, NI	31 Sx	Inter	33	$\kappa = -0.27$ to 0.38 % = 11–58	None to fair

Continued

TABLE 4 Reliability of Spine and/or Sacroiliac Position or Alignment Studies—Cont'd

Author	Region	Examiners, Experience	Subjects	Study Type	Qual	Findings	Degree of Reliability
Keating et al ⁴	T12–S1	3 DC	46 (21 Sx, 25 Asx)	Inter	67	$\kappa = -0.16$ to 0.22	None to fair
Potter and Rothstein ⁵	SI	8 PT	17 Sx	Intra	0	% = 44–50	Inconclusive
Spring and Tehan ⁶	L1–L5	10 DO, St	10 Asx	Inter	83	$\kappa = 0.04$	Slight
	L1–L5	10 DO, St	10 Asx	Intra	75	$\kappa = 0.04$	Slight

From Haneline MT, Morgan Young M: A review of intraexaminer and interexaminer reliability of static spinal palpation: A literature synthesis. *J Manipulative Physiol Ther* 32:379, 2009.
 Asx, Asymptomatic; C, cervical; DC, doctor of chiropractic; DO, doctor of osteopathic medicine; Inter, interexaminer reliability; Intra, intraexaminer reliability; L, lumbar; NI, no information presented; PT, physical therapist; S, sacral; SI, sacroiliac; St, student; Sx, symptomatic; T, thoracic.

REFERENCES

1. Collaer JW, McKeough DM, Boissonnault WG: Lumbar isthmic spondylolisthesis detection with palpation: Interrater reliability and concurrent criterion-related validity, *J Man Manipulative Ther* 14:22, 2006.
2. Fryer GM, McPherson H, O'Keefe P: The effect of training on the interexaminer and intra-examiner reliability of the seated flexion test and assessment of pelvic anatomical landmarks with palpation, *Int J Osteopat Med* 8:131, 2005.
3. Hart J: Palpation and X-ray of the upper cervical spine: a reliability study, *J Vertebral Subluxation Res* October 25, 2006, pp. 1–14.
4. Keating J, et al: Interexaminer reliability of eight evaluative dimensions of lumbar segmental abnormality, *J Manipulative Physiol Ther* 13:463, 1990.
5. Potter NA, Rothstein JM: Intertester reliability for selected clinical tests of the sacroiliac joint, *Phys Ther* 65:1671, 1985.
6. Spring F, Gibbons P, Tehan P: Intra-examiner and interexaminer reliability of a positional diagnostic screen for the lumbar spine, *J Osteopat Med* 4:47, 2001.

TABLE 5 Citation Synopsis for Interexaminer and Intraexaminer Reliability Studies for End Feel or Joint Play Motion Palpation Procedures (Not Including All PA Stiffness)

Citation & Year	Who	Location	Procedure	Intra	Inter	Comments/Summary of Results	Stats Used
DeBoer et al 1985 ¹	PhD DC	C-spine	Sitting	X	X	Does not describe actual procedure; used static and motion palpation as well as tenderness and muscle palpation. Poor to moderate agreement.	% Agree
Bergstrom and Countis 1986 ²	DC	L-spine	Sitting LF	X	X	High inter and higher intra.	% Agree
Love and Brodeur 1987 ³	DC	T1–L5	Sitting	X	X	Poor design—used “most hypomobile” segment (i.e., examiners had to pick one). Insignificant reliability.	Pearson
Boline et al 1988 ⁴	DC PhD	L-spine	Sitting		X	Also evaluated muscle hypertonicity and soft tissue pain. Weak support for inter on motion palpation. Stronger concordance on pain.	κ : +/-
Nansel et al 1989 ⁵	PhD DC	C-spine	Sitting LF		X	Reports joint play but actually end feel. Sitting and supine. Done on “normal” subjects. “May not be an internally valid predictor of joint dysfunction in asymptomatic individuals.” Poor agreement.	κ : +/-

Continued

TABLE 5 Citation Synopsis for Interexaminer and Intraexaminer Reliability Studies for End Feel or Joint Play Motion Palpation Procedures (Not Including All PA Stiffness)—Cont'd

Citation & Year	Who	Location	Procedure	Intra	Inter	Comments/Summary of Results	Stats Used
Mootz et al 1989 ⁶	DC PhD	L-spine	Sitting F, E, LF, Rot	X	X	Minimal support for intra and no support for inter.	κ : +/-
Haas et al 1995 ⁷	DC	T-spine	Sitting Rot	X	X	Intra: moderate; inter: poor.	κ : -/se
Binkley 1995	PT	L1–S1	PA on SP		X	18 mechanical LBP pts. 6 PTs evaluated PA accessory motion. Poor interrater in identifying marked lumbar levels and poor interrater on movement.	ICC +/- κ
Phillips and Twomey 1996 ⁸	PT	L-spine	PA, lateral transverse		X	Technique alone vs. technique with verbal pain response. Compared with anesthetic blocks. Highly accurate in determining the lumbar segment responsible for pain (sensitive and specific). High agreement in upper lumbar, less agreement lower lumbar for inter.	κ
Marcotte et al 2002 ⁹	DC	C-spine	Supine rotation		X	Used a computerized system of movement analysis. Discusses need for high level of standardization of the test. Good level of reliability following good mechanics of the test procedure.	κ : +/-se
Christensen et al 2002 ¹⁰	DC MD PhD	T-spine	Sitting	X	X	Assessed sitting end feel, prone joint play, and soft tissue palpation; 107 symptomatic and asymptomatic, κ values for strict agreement low but expanded κ indicated good intra and moderate to good inter. Rationale for using expanded κ .	Expanded κ : +/-
Piva et al 2006 ¹¹	PT	C-spine	Supine Lat Glide		X	Also evaluated AROM with an inclinometer, found to be moderate to highly reliable; passive lateral glide had moderate reliability.	κ : +/+
Brismee et al 2006 ¹²	PT	Mid T-spine	Sitting		X	43 asymptomatic, 3 Pts; results indicated fair to substantial inter. Although compared with other EP studies, this did not use over-pressure; rather it looked at the relative change in SP position during passive extension, side bending and rotation.	κ : +/+

AROM, Active range of motion; C, cervical; DC, doctor of chiropractic; E, extension; EP, end play; F, flexion; inter, interexaminer reliability; intra, intraexaminer reliability; L, lumbar; Lat, lateral; LBP, low back pain; LF, lateral flexion; MD, medical doctor; PA, posterior to anterior; PT, physical therapist; Rot, rotation; se, standard error; SP, spinous process; T, thoracic.

1. DeBoer KF, et al: Inter- and intra-examiner reliability of leg length differential measurement: a preliminary study, *J Manipulative Physiol Ther* 6(2):61, 1983.
2. Bergstrom E, Courtis G: An inter- and intra-examiner reliability study of motion palpation of the lumbar spine in lateral flexion in the seated position, *Eur J Chiropr* 34:121, 1986.
3. Love RM, Brodeur RR: Inter- and intra-examiner reliability of motion palpation for the thoracolumbar spine, *J Manipulative Physiol Ther* 10:1, 1987.
4. Boline P, et al: Interexaminer reliability of palpatory evaluations of the lumbar spine, *Am J Chiropr Med* 1(1):5, 1988.
5. Nansel DD, et al: Interexaminer concordance in detecting joint-play asymmetries in the cervical spines of otherwise asymptomatic subjects, *J Manipulative Physiol Ther* 12(6):428, 1989.
6. Mootz RD, Keating JC, Kontz HP: Intra- and inter-examiner reliability of passive motion palpation of the lumbar spine, *J Manipulative Physiol Ther* 12(6):440, 1989.
7. Haas M, et al: Reliability of manual end play palpation of the thoracic spine, *Chiropr Tech* 7:120, 1995.
8. Phillips DR, Twomey LT: A comparison of manual diagnosis with a diagnosis established by a uni-level lumbar spinal block procedure, *Man Ther* 2:82, 1996.
9. Marcotte J, Normand MC, Black P: The kinematics of motion palpation and its effect on the reliability for cervical spine rotation, *J Manipulative Physiol Ther* 25(7):E7, 2002.
10. Christensen HW, et al: Palpation of the upper thoracic spine: an observer reliability study, *J Manipulative Physiol Ther* 25(5): 285–292, 2002.
11. Piva SR, et al: Inter-tester reliability of passive intervertebral and active movements of the cervical spine, *Man Ther* 11:321, 2006.
12. Brismée JM, et al: Interrater reliability of a passive physiological intervertebral motion test in the mid-thoracic spine, *J Manipulative Physiol Ther* 29(5):368–373, 2006.

TABLE 6 Citation Synopsis for Literature Reviews on Interexaminer and Intraexaminer Reliability/Validity Studies for Motion Palpation Procedures

Citation & Year	Who	Location	Procedure	Intra	Inter	Comments/Summary of Results
Hestboek and Lebouef-Yde 2000 ¹	DC	Lumbar pelvic	Lit rev reliability validity	X	X	Looked at many evaluative tools: MP lumbar, MP SIJs, leg length, SOT, palpatory pain. Only studies focusing on pain had consistent reliability values
Huijbregts 2002 ²	PT	Full spine	Lit rev reliability validity	X	X	Looked at multiple professional approaches to spinal motion evaluation. Intra varies from less than chance to generally moderate or substantial; inter only rarely exceeds poor to fair; ratings that include presence or absence of pain yield higher agreement.
Seffinger et al 2003 ³	DO DC MD PhD	Full spine	Lit rev reliability validity	X	X	Looked at content validity and reliability of spinal palpation procedures from DC, PT, MD, and DO literature. Provides a summary of each paper included. No overall conclusions provided.
Najm et al 2003 ⁴	DO DC MD PhD	Full spine	Lit rev reliability validity			Lack of acceptable reference standards may have contributed to the weak sensitivity findings. The sensitivity of studies looking at range-of-motion tests and pain varied greatly. Poor sensitivity was reported for range-of-motion studies regardless of the examiner's experience. A slightly better sensitivity (82%) was reported in one study that examined cervical pain.

Continued

TABLE 6 Citation Synopsis for Literature Reviews on Interexaminer and Intraexaminer Reliability/Validity Studies for Motion Palpation Procedures—Cont'd

Citation & Year	Who	Location	Procedure	Intra	Inter	Comments/Summary of Results
Van Trijffela 2005 ⁵	MD	Full spine	Lit rev reliability validity		X	To determine interexaminer reliability of passive assessment of segmental intervertebral motion in the cervical and lumbar spine as well as to explore sources of heterogeneity. Assessment of motion segments C1–C2 and C2–C3 almost consistently reached at least fair reliability. Overall, interexaminer reliability was poor to fair. Most studies were found to be of poor methodologic quality.
Stochkendahl et al 2006 ⁶	DC PhD	Full spine	Lit rev reliability reprod	X	X	Looked at many evaluative tools: MP, static palpation, osseous pain, soft tissue pain, soft tissue changes, global movement FS. Acceptable (strong) for palpation for pain inter and intra, global assessment intra. No evidence or conflicting evidence for static palpation. Motion palpation: good intra, unacceptable inter.
Haneline et al 2008 ⁷	DC	Full spine	Lit rev reliability	X	X	Reviewed different forms of MP (excursion vs. end feel) to determine whether a difference in reported reliability was observed when the method of MP varied. Not statistically significant.

DC, Doctor of chiropractic; DO, doctor of osteopathic medicine; FS, full spine; *inter*, interexaminer reliability; *intra*, intraexaminer reliability; *lit rev*, literature review; MD, medical doctor; MP, metacarpophalangeal; PT, physical therapist; *reprod*, reproduction; *SJJ*, sacroiliac joint; *SOT*, sacral occipital technique.

- Hestboek L, Leboeuf-Yde C: Are chiropractic tests for the lumbo-pelvic spine reliable and valid? A systematic critical literature review, *J Manipulative Physiol Ther* 23:258, 2000.
- Huijbregts P: Spinal motion palpation: a review of reliability studies, *J Man Manip Ther* 10:24, 2002.
- Seffinger M, et al: Spinal palpatory diagnostic procedures utilized by practitioners of spinal manipulation: Annotated bibliography of reliability studies, *J Can Chiropr Assoc* 47:89, 2003.
- Najm WI, et al: Content validity of manual spinal palpatory exams—A systematic review, *BMC Complement Altern Med* 3:1, 2003.
- van Trijffela E, et al: Inter-examiner reliability of passive assessment of intervertebral motion in the cervical and lumbar spine: a systematic review, *Man Ther* 10:256, 2005.
- Stochkendahl MJ, Christensen HW, Hartvigsen J, et al: Manual examination of the spine: a systematic critical literature review of reproducibility, *J Manipulative Physiol Ther* 29:475, 2006.
- Haneline MT, et al: Spinal motion palpation: a comparison of studies that assessed intersegmental end feel vs excursion, *J Manipulative Physiol Ther* 31:616, 2008.

TABLE 7 Citation Synopsis for Interexaminer Validity for Motion Palpation Procedures

Citation & Year	Who	Location	Procedure	Intra	Inter	Comments/Summary of Results	Stats
Humphreys 2004 ¹	DC	Cervical	Sitting EP, rot, & LF		X	20 students palpated 3 subjects with congenital fusions as a “gold standard.” Substantial overall agreement; sensitivity ranged from 55% to 78%; specificity was high (91%–98%).	κ
Jull et al 1997 ²	PT	Upper cervical	Palpable pain, not explained		X	6 examiners agreed on the presence or absence of painful upper cervical joint dysfunction using their own methods on 40 symptomatic and nonsymptomatic subjects. Percent agreement was 70% for inter.	κ
King et al 2007 ³	MD	Cervical	Palpable pain		X	Palpation of painful joints compared to diagnostic blocks. High sensitivity for C z-joint pain, poor specificity. Statistically no different from previous studies but concluded that manual examination of the cervical spine lacks validity.	Counting tables, sens, spec
Childs et al 2004 ⁴	PT						
Fritz et al 2005 ⁵	PT	Lumbar	PA mobility		X	Predictive validity of PA mobility testing in a group of patients with low back pain.	κ ANOVA

ANOVA, Analysis of variance; C, cervical; DC, doctor of chiropractic; EP, end play; inter, interexaminer reliability; intra, intraexaminer reliability; LF, lateral flexion; MD, medical doctor; PA, posterior-anterior; PT, physical therapy; rot, rotation; sens, sensitivity; spec, specificity.

- Humphreys B, Delahaye M, Peterson CK: An investigation into the validity of cervical spine motion palpation using subjects with congenital block vertebrae as a “gold standard,” BMC Musculoskel Disord 5:19, 2004.
- Jull GA, et al: Interexaminer reliability to detect painful upper cervical joint dysfunction. Aust J Physiother 43(2):125–129, 1997.
- King W, Lau P, Lees R, et al: The validity of manual examination in assessing patients with neck pain, Spine J 7:22, 2007.
- Childs MJ, Fritz JM, Flynn TW: A clinical prediction rule to identify patients with low back pain most likely to benefit from spinal manipulation, Ann Intern Med 141(12):920, 2004.
- Fritz JM, et al: Lumbar spine segmental mobility assessment: an examination of validity of determining intervention strategies in patients with low back pain, Arch Phys Med Rehabil 86:1745, 2005.

TABLE 1 Citation Synopsis for Interexaminer Validity for Motion Palpation Procedures

Validity							
Citation & Year	Who	Location	Procedure	Intra	Inter	Comments/Summary of Results	Stats
Humphreys 2004 ⁴²⁰	DC	Cervical	Sitting EP ROT and LF		X	20 students palpated 3 subjects with congenital fusions as a "gold standard." Substantial overall agreement; sensitivity ranged from 55% to 78%; specificity was high (91%–98%).	K
Jull 1997*	PT	Upper cervical	Palpable pain, not explained		X	6 examiners agreed on the presence or absence of painful upper cervical joint dysfunction using their own methods on 40 symptomatic and nonsymptomatic subjects. Percent agreement was 70% for inter examiner.	K
King 2007 ²⁵⁸	MD	Cervical	Palpable pain		X	Palpation of painful joints compared with diagnostic blocks. High sensitivity for C z-joint pain, poor specificity. Statistically no different from previous studies, but concluded that manual examination of the cervical spine lacks validity.	Counting tables, sens, spec
Childs 2004 ²⁶⁰	PT	Lumbar	Multifactorial including PA mobility SP			Use of clinical prediction rule criteria (symptom duration, symptom location, fear-avoidance beliefs, lumbar mobility, and hip rotation range of motion) improved the response rate for manipulation of low back pain patients.	
Fritz 2005 ²⁶¹	PT	Lumbar	PA mobility SP		X	Predictive validity of PA mobility testing in a group of patients with low back pain	K ANOVA

ANOVA, Analysis of variance; DC, doctor of chiropractic; EP, end play; K, kappa; LF, lateral flexion; MD, medical doctor; PA, posterior-anterior; PT, physical therapist; ROT, rotation; C, cervical; Z, zygapophyseal joint.

*Jull G, Zito G, Trott P, Potter H, Shirley D. Inter-examiner reliability to detect painful upper cervical joint dysfunction. Australian Journal of Physiotherapy 43(2):125-129, 1997.

CHAPTER 1

1. Brantingham JW: Still and Palmer: the impact of the first osteopath and the first chiropractor, *Chiropr Hist* 6:20, 1986.
2. Coulter ID: Chiropractic: a philosophy for alternative health care, 1999, Butterworth Heinemann, pp 37–55.
5. Kinsinger S: Advancing the philosophy of chiropractic: advocating virtue, *J Chiropr Humanit* 11:24–28, 2004.
4. Briggance BB: A proposal regarding the identity of chiropractic: embrace the centrality of the spine, *J Chiropr Humanit* 12:8–15, 2005.
5. Miller A: Transcendentalism's inspiration to chiropractic philosophy and practice, *Today's Chiropractic* 29:2, 2000.
6. Cleveland A, Phillips R, Clum G: The chiropractic paradigm. In Redwood D, Cleveland CS, editors: *Fundamentals of chiropractic*, St. Louis, 2003, Mosby.
7. Palmer DD: *The science, art and philosophy of chiropractic*, Portland, OR, 1910, Portland Printing House Co.
8. Gibbons R: Go to jail for chiro?, *J Chiropr Humanit* 1:61, 1994.
9. Wardwell WI: Why did chiropractic survive? *J Chiropr Humanit* 8(1):2, 1998.
10. Beideman RP: Seeking the rational alternative: The National College of Chiropractic from 1906 to 1982, *Chiropr Hist* 3:17, 1983.
11. Gibbons RW: The evolution of chiropractic: medical and social protest in America. In Haldeman S, editor: *Modern developments in the principles and practice of chiropractic*, East Norwalk, CT, 1980, Appleton-Century-Crofts.
12. Donahue RJDD: Palmer and innate intelligence. Development, division, and derision, *Chiropr Hist* 6:31, 1986.
13. Palmer DD: *The chiropractor's adjuster, textbook of the science, art and philosophy of chiropractic for students and practitioners*, Portland, OR, 1910, Portland Printing House Co.
14. Palmer BJ: *History in the making* (vol. XXXV), (as reprinted with corrections by C. Jensen, Sacramento, 1985), Davenport, IA, 1957, Palmer School of Chiropractic.
15. Waagen G, Strang V: Origin and development of traditional chiropractic philosophy. In Haldeman S, editor: *Principles and practice of chiropractic*, Norwalk, CT, 1992, Appleton and Lange.
16. Northrup GW: *Osteopathic medicine: an American reformation*, ed 2, Chicago, 1970, American Osteopathic Association.
17. Jackson R: *Vis Medicatrix Naturae: vital force to innate intelligence and concepts for 2000*, *J Chiropr Humanit* 2001.
18. Winterstein JF: Is traditional "chiropractic philosophy" valid today? *Philos Constructs Chiropr Prof* 1:3–5, 1991.
19. Phillips R: Philosophy and chiropractic divisions and directions, *J Chiropr Humanit* 5:2–7, 1995.
20. Gelardi TA: The science of identifying professions as applied to chiropractic, *J Chiropr Humanit* 6:11–17, 1996.
21. Wardwell WI: Chiropractic "philosophy," *J Chiropr Humanit* 3:3–8, 1993.
22. Donahue J: Are philosophers just scientists without data? *Philos Constructs Chiropr Prof* 1:21–23, 1991.
23. Coulter ID: Uses and abuses of philosophy in chiropractic, *Philos Constructs Chiropr Prof* 2:3–7, 1992.
24. McAulay BJ: Rigor in the philosophy of chiropractic: beyond the dismissivism/authoritarian polemic, *J Chiropr Humanit* 12:16–32, 2005.
25. Brooks WH, et al: Neuroimmunomodulation: neural anatomical basis for impairment and facilitation, *Ann Neurol* 12:56, 1982.
26. Haldeman S: The clinical basis for discussion of mechanisms of manipulative therapy. In Korr IM, editor: *The neurobiologic mechanisms of manipulative therapy*, New York, 1978, Plenum.
27. Sato A: Physiological studies of the somatoautonomic reflexes. In Haldeman S, editor: *Modern developments in the principles and practice of chiropractic*, East Norwalk, CT, 1980, Appleton Century-Crofts.
28. Coote JH: Central organization of the somatosympathetic reflexes. In Haldeman S, editor: *Modern developments in the principles and practice of chiropractic*, East Norwalk, CT, 1980, Appleton-Century-Crofts.
29. Beal MC: Viscerosomatic reflexes: a review, *JAOA* 85:786, 1985.
30. The central connection: Somatovisceral/viscerosomatic interaction, 1989. In *Proceedings of International Symposium*, Cincinnati, OH, 1989.
31. Wardwell WI: *Chiropractic history and evolution of a new profession*, St Louis, 1992, Mosby.
32. Clusserath MT: A treatise on fundamental principles of the philosophy of chiropractic and related topics in the life sciences, *J Chiropr Humanit* 13:12–20, 2006.
33. Flexner A: Medical education in the United States and Canada, Carnegie Foundation Advancement Teaching Bull No 4, 1910, Carnegie Foundation.
34. Beideman RP: A short history of the chiropractic profession. In Lawrence DJ, editor: *Fundamentals of chiropractic diagnosis and management*, Baltimore, 1991, Williams & Wilkins.
35. Standards for chiropractic programs and institutions, Scottsdale, AZ, 1996, Council on Chiropractic Education.
36. Vear HJ: Quality assurance: standards of care and ethical practice, *JCCA* 35(4):215, 1991.
37. Kusserow RP: State licensure and discipline of chiropractors, DHHS Pub No OAI-01–88–00581, Washington, DC, 1989, Office of Inspector General.
38. Lamm LC, Wegner E: Chiropractic scope of practice: what the law allows, *Am J Chiropractic Med* 2(4):155, 1989.
39. Analysis of VA Health Care Utilization Among U.S. Southwest Asian War Veterans: Operation Iraqi Freedom, Operation Enduring Freedom, 2006, VHA Office of Public Health and Environmental Hazards.
40. Eisenberg DM, et al: Unconventional medicine in the United States. Prevalence, costs, and patterns of use, *N Engl J Med* 328(4):246–252, 1993.
41. Tindle HA, et al: Trends in use of complementary and alternative medicine by US adults: 1997–2002, *Altern Ther Health Med* 11(1):42–49, 2005.
42. Kreitzer MJ, Mann D: Complementary health practice review, 2008.
43. Haas M, Bronfort G, Evans R: Chiropractic clinical research: progress and recommendations, *J Manipulative Physiol Ther* 29:695–706, 2006.
44. Proceedings of the 1990 Consensus Conference on Validation of Chiropractic Technique, Seattle, *J Chiro Tech* 2(3):71, 1990.
45. Shekelle PG, et al: The appropriateness of spinal manipulation for low-back pain, Santa Monica, CA, 1991, RAND.
46. Shekelle PG, et al: Congruence between decisions to initiate chiropractic spinal manipulation for low back pain and appropriateness criteria in North America, *Ann Intern Med* 129(1):9, 1998.
47. Coulter ID, et al: The appropriateness of manipulation and mobilization of the cervical spine, Santa Monica, CA, 1996, RAND.
48. Shekelle PG, Coulter ID: Cervical spine manipulation: summary report of a systematic review of the literature and a multidisciplinary expert panel, *J Spinal Disord* 10(3):223, 1997.
49. Haldeman S, Chapman-Smith D, Peterson DM: Guidelines for chiropractic quality assurance and practice parameters, Gaithersburg, MD, 1993, Aspen.

50. Bigos S, et al: Acute low back problems in adults, Clinical Practice Guideline No 14, AHCPR Pub No 95-0642, Rockville, MD, 1994, Agency for Health Care Policy and Research, Public Health Service, US Department of Health and Human Services.
51. Manga P, et al: The effectiveness and cost-effectiveness of chiropractic management of low-back pain, Ottawa, Ontario, 1993, Pran Manga and Associates.
52. Manga P, Angus D: Enhanced chiropractic coverage under OHIP as a means of reducing health outcomes and achieving equitable access to select health services, Toronto, 2004, Ontario Chiropractic Association.
53. Legorreta AP, et al: Comparative analysis of individuals with and without chiropractic coverage, patient characteristics, utilization and costs, *Arch Intern Med* 164: 1985-1992, 2004.
54. Chapman-Smith D: WFC's world meeting on identity, *Chiropractic Report* 18(2):1-8, 2004.
55. Chapman-Smith D: Perspective on the future of chiropractic, *Cal Chiropr Assoc J* 16(2):31, 1991.
56. Phillips RB: The battle for innate: A perspective on fundamentalism in chiropractic, *J Chiropr Humanit* 11:2-10, 2004.
57. Meeker WC, Haldeman S: Chiropractic: a profession at the crossroads of mainstream and alternative medicine, *Ann Intern Med* 136(3):216-227, 2002.
58. Chapman-Smith D: WFC's consultation on the profession's identity, *Chiropractic Report* 18(1):1-8, 2004.
59. McDonald W, et al: How chiropractors think and practice: the survey of North American chiropractors, Ada, OH, Institute for social research, OH Northern University, 2003.
15. Hertling D, Kessler RM: Management of common musculoskeletal disorders, physical therapy principles and methods, ed 2, Philadelphia, 1990, JB Lippincott.
16. Holm S, Indahl A, Solomonow M: Sensorimotor control of the spine, *J Electromyogr Kinesiol* 12(3):219-234, 2002.
17. Wyke BD: Articular neurology and manipulative therapy. In Glasgow EF, et al: Aspects of manipulative therapy, Edinburgh, UK, 1985, Churchill Livingstone.
18. McLain RF: Mechanoreceptor endings in human cervical facet joints, *Spine* 19(5):495, 1994.
19. Gillette RG: A speculative argument for the coactivation of diverse somatic receptor populations by forceful chiropractic adjustments, *Manual Med* 3:1, 1987.
20. Malinsky J: The ontogenetic development of nerve terminations in the intervertebral discs of man, *Acta Anat (Basel)* 38:96, 1959.
21. Carr J, Shepherd R: Movement science, Gaithersburg, MD, 2000, Aspen Publishers Inc.
22. Shumway-Cook A, Woollacott M: Motor Control: Theory and practical applications, Baltimore, MD, 1995, Williams & Wilkins, pp 239-268.
23. Baci AM, Colebatch JG: Evidence for reflex and perceptual vestibular contributions to postural control, *Exp Brain Res* 160:22-28, 2005.
24. Kristinsdottir EK, Fransson PA, Magnusson M: Changes in postural control in healthy elderly subjects are related to vibration sensation, vision and vestibular asymmetry, *Acta Otolaryngol* 121:700-706, 2001.
25. Lord SR, Clark RD, Webster IW: Postural stability and associated physiological factors in a population of aged persons, *J Gerontol* 46:M69-M76, 1991.
26. Bogduk N: The innervation of the lumbar intervertebral discs. In Grieve G, editor: Modern manual therapy of the vertebral column, Edinburgh, UK, 1986, Churchill Livingstone.
27. Shinohara H: A study on lumbar disc lesions, *J Jap Orthop Assoc* 44:553, 1970.
28. Freemont AJ, et al: Nerve in growth into diseased intervertebral disc in chronic back pain, *Lancet* 350(9072):178, 1997.
29. Bogduk N, Windsor M, Inglis A: The innervation of the cervical intervertebral discs, *Spine* 13(1):2, 1988.
30. Mendel T, Wink CS, Zimny ML: Neural elements in human cervical intervertebral discs, *Spine* 17(2):132, 1992.
31. Roberts S, et al: Mechanoreceptors in intervertebral discs: morphology, distribution, and neuropeptides, *Spine* 20(24):2645, 1995.
32. Rabischong P, et al: The intervertebral disc, *Anat Clin* 1:55, 1978.
33. Rooft PG: Innervation of the annulus fibrosus and posterior longitudinal ligament, *Arch Neurol Psychiatry* 44:100, 1940.
34. Bogduk N, Tynan W, Wilson AS: The nerve supply to the human lumbar intervertebral discs, *J Anat* 132:39, 1981.
35. Hirsch C, Ingelmark BE, Miller M: The anatomical basis for low back pain, *Acta Orthop Scand* 33:1, 1963.
36. Jackson HC, Winkelmann RK, Bickel WH: Nerve endings in the human lumbar spinal column and related structures, *J Bone Joint Surg Am* 48:1272, 1966.
37. MacConnail MA, Basmajian JV: Muscles and movements: a basis for human kinesiology, Baltimore, 1969, Williams & Wilkins.
38. Edmond SL: Manipulation and mobilization extremity and spinal techniques, St Louis, 1993, Mosby.
39. Andersson GBJ: Biomechanics of the lumbar spine. In Kirkaldy-Willis WH, Burton CV, editors: Managing low back pain, ed 3, New York, 1992, Churchill Livingstone.
40. Garg A: Occupational biomechanics and low back pain, *Occupational Medicine: State of the Art Reviews* 7(4):609, 1992.
41. Fiorini GT, McCammond D: Forces on the lumbo-vertebral facets, *J Biomed Eng* 4:354, 1976.
42. Farfan HF, et al: Effects of torsion on the intervertebral joint: the role of torsion in the production of disc degeneration, *J Bone Joint Surg Am* 52:468, 1970.

CHAPTER 2

1. White AA, Panjabi MM: Clinical biomechanics of the spine, ed 2, Philadelphia, 1990, JB Lippincott.
2. Nordin M, Frankel VH: Basic biomechanics of the musculoskeletal system, ed 2, Philadelphia, 2001, Lippincott Williams & Wilkins.
3. Goal VK, Weinstein JN: Biomechanics of the spine: clinical and surgical perspective, Boca Raton, FL, 1990, CRC Press.
4. Kendall HO, Kendall FP, Wadsworth GE: Muscle testing and function, ed 2, Baltimore, 1971, Williams & Wilkins.
5. Weiss C, Rosenberg L, Helfet AJ: An ultrastructural study of normal young adult human articular cartilage, *J Bone Joint Surg Am* 50:663, 1968.
6. Woo SLY, Adeson WH, Jemmott GF: Measurements of nonhomogeneous directional mechanical properties of articular cartilage in tension, *J Biomech* 9:785, 1976.
7. Newman AP: Articular cartilage repair, *Am J Sports Med* 26(2):309, 1998.
8. Mankin HJ: The response of articular cartilage to mechanical injury, *J Bone Joint Surg Am* 64(3):460, 1982.
9. Moran ME, Kin HK, Salter RB: Biological resurfacing of full-thickness defects in patellar articular cartilage of the rabbit: investigation of autogenous periosteal grafts subjected to continuous passive motion, *J Bone Joint Surg Br* 74:659, 1992.
10. Maigne R: Diagnosis and treatment of pain of vertebral origin, Baltimore, 1996, Williams & Wilkins.
11. Jay GD, et al: The role of lubricin in the mechanical behavior of synovial fluid, *Proc Natl Acad Sci U S A* 104(15):6194-6199, 2007.
12. Jay GD, et al: Association between friction and wear in diarthrodial joints lacking lubricin, *Arthritis Rheum* 56(11):3662-3669, 2007.
13. Gale LR, et al: Boundary lubrication of joints: characterization of surface-active phospholipids found on retrieved implants, *Acta Orthop* 78(3):309-314, 2007.
14. Akeson WH, Amiel D, LaViolette D: The connective tissue response to immobility, *Clin Orthop* 51:183, 1967.

43. Adams MA, Hutton WC: The relevance of torsion to the mechanical derangement of the lumbar spine, *Spine* 6:241, 1981.
44. Schultz AB, et al: Mechanical properties of the human lumbar spine motion segments. I. Response in flexion, extension, lateral bending and torsion, *J Biomech Eng* 101:46, 1979.
45. Skipor AF, et al: Stiffness, properties and geometry of lumbar spine posterior elements, *J Biomech* 18:821, 1985.
46. Bogduk N, Twomey LT: *Clinical anatomy of the lumbar spine*, ed 2, Melbourne, Australia, 1991, Churchill Livingstone.
47. Threlkeld AJ: Basic structure and function of the joints. In Neuman DA, editor: *Kinesiology of the musculoskeletal system*, 2002, St. Louis Mosby, p 31.
48. Zhang G: Evaluating the viscoelastic properties of biological tissues in a new way, *J Musculoskelet Neuronal Interact* 5(1):85–90.
49. Akeson WH, Amiel D, Woo SLY: Cartilage and ligament: physiology and repair processes. In Nicholas JA, Hershman EB, editors: *The lower extremity and spine in sports medicine*, St Louis, 1986, Mosby.
50. Akeson WH, et al: Collagen cross linking alterations in joint contractures: changes in reducible cross links in periarticular connective tissue collagen after 9 weeks of immobilization, *Connect Tissue Res* 5:5, 1977.
51. Burger AA: Experimental neuromuscular models of spinal manual techniques, *J Man Med* 1:10, 1983.
52. Guyton AC: *Textbook of medical physiology*, ed 4, Philadelphia, 1971, WB Saunders.
53. Astand O, Rodahl K: *Textbook of work physiology*, New York, 1970, McGraw-Hill.
54. Kaltenborn FM: *Mobilization of the extremity joints: examination and basic treatment principles*, ed 3, Oslo, 1980, Olaf-Norlis-Bokhandel.
55. Janda V: Muscle spasm: A proposed procedure for differential diagnosis, *J Man Med* 6:136, 1991.
56. Liebenson C: Active muscular relaxation techniques. I. Basic principles and methods, *J Manipulative Physiol Ther* 12(6):446, 1989.
57. Jones VT, Garrett WE, Seaber AV: Biomechanical changes in muscle after immobilization at different lengths, *Trans Orthop Res Soc* 10:6, 1985.
58. Fink B, et al: Morphologic changes in the vastus medialis muscle in patients with osteoarthritis of the knee, *Arthritis Rheum* 56(11):3626–3633, 2007.
59. Cramer GD, Darby SA: *Basic and clinical anatomy of the spine, spinal cord, and ans*, ed 2, St. Louis, 2005, Mosby, p 35.
60. Jiang H: Identification of the location, extent, and pathway of sensory neurologic feedback after mechanical stimulation of a lateral spinal ligament in chickens, *Spine* 22(1):17–25, 1997.
61. Cramer GD: General characteristics of the spine. In Cramer GD, Darby S, editors: *Basic and clinical anatomy of the spine, spinal cord, and ans*, ed 2, St. Louis, 2005, Mosby Year Book, pp 13–65.
62. Brinkley JM, Peat M: The effects of the mobilization on the ultra-structure and mechanical properties of the medial collateral ligament of rats, *Clin Orthop* 203:301, 1986.
63. Evans EB, Eggars GWN, Butler JK: Experimental remobilization of rat knee joints, *J Bone Joint Surg Am* 42:737, 1960.
64. Woo SLY, Matthews JP, Akeson WH: Connective tissue response to immobility: correlative study of biomechanical and biochemical measurements of normal and immobilized rabbit knees, *Arthritis Rheum* 18:257, 1975.
65. Dahners LE: Ligament contractions: a correlation with cellularity end actin staining, *Trans Orthop Res Soc* 11:56, 1986.
66. Solomonow M, et al: The ligamento-muscular stabilizing system of the spine, *Spine* 1(23):2552–2562, 1998.
67. Giles LGE, Taylor JR: Innervation of lumbar zygapophyseal joint synovial folds, *Acta Orthop Scand* 58:43, 1987.
68. Little JS, Khalsa PS: Human lumbar spine creep during cyclic and static flexion: creep rate, biomechanics, and facet joint capsule strain, *Ann Biomed Eng* 33(3):391–401, 2005.
69. Bogduk N, Engel R: The menisci of the lumbar zygapophyseal joints: a review of their anatomy and clinical significance, *Spine* 9:454, 1984.
70. Zaccheo D, Reale E: Contributo alla conoscenza delle articolazioni tra i processi articolari delle vertebre dell'uomo, *Arch Anotomica* 61:1, 1956.
71. Kos J, Wolf J: Les menisques intervertebraux et leur role possible dans les blocages vertebraux, *Ann Med Phys* 15:2, 1972.
72. Bogduk N, Jull G: The theoretical pathology of acute locked back: a basis for manipulative therapy, *Man Med* 1:78, 1985.
73. Giles LGE, Taylor JR: Human zygapophyseal joint capsule and synovial fold innervation, *Br J Rheumatol* 26:93, 1987.
74. Taylor JR, Twomey LT: Age changes in the lumbar zygapophyseal joints: observations on structure and function, *Spine* 11:739, 1986.
75. Adams MA, Hutton WC: The mechanical function of the lumbar apophyseal joints, *Spine* 8:327, 1983.
76. Danbury R: Functional anatomy of the intervertebral disc, *J Man Med* 6:128, 1971.
77. Farfan HF: *Mechanical disorders of the lumbar spine*, Philadelphia, 1973, Lea & Febiger.
78. Twomey L, Taylor J: Flexion creep deformation and hysteresis in the lumbar vertebral column, *Spine* 7:116, 1982.
79. Kurowski P, Kubo A: The relationship of degeneration of the intervertebral disc to mechanical loading conditions on lumbar vertebrae, *Spine* 11:726, 1986.
80. Gracovetsky S: Function of the spine, *J Biomed Eng* 8:217, 1986.
81. Aspden RM: The spine as an arch: a new mathematical model, *Spine* 14:266, 1989.
82. Panjabi M, et al: Spinal stability and intersegmental muscle forces: a biomechanical model, *Spine* 14:194, 1989.
83. Louis R: Spinal stability as defined by the three-column spine concept, *Anat Clin* 7:33, 1985.
84. Gracovetsky S, Farfan H: The optimum spine, *Spine* 11:543, 1986.
85. Levin SM: The importance of soft tissue for structural support of the body. In Dorman TA, editor: *Prolotherapy in the lumbar spine and pelvis*, *Spine: State of the art reviews*, 9(2):357, 1995.
86. Ingber DE, Jamieson JD: Cells as tensegrity structures: architectural regulation of histo differentiation by physical forces transduced over basement membrane. In Andersson LC, Gahmberg CG, Ekblom P, editors: *Gene expression during normal and malignant differentiation*, London, 1985, Academic Press.
87. Thompson DW: *On growth and form*, New York, 1977, Cambridge University Press. As cited in Ingber DE, Jamieson JD: Cells as tensegrity structures: Architectural regulation of histo differentiation by physical forces transduced over basement membrane. In Andersson LC, Gahmberg CG, Ekblom P, editors: *Gene expression during normal and malignant differentiation*, London, 1985, Academic Press.
88. Fuller BR: *Synergetics*, New York, 1975, Mcmillan.
89. Kuchera ML: Gravitational stress, musculoligamentous strain, and postural alignment. In Dorman TA: *Prolotherapy in the lumbar spine and pelvis*, *Spine: State of the Art Reviews* 9(2):243, 1995.

CHAPTER 3

1. Palmer DD: *Textbook of the science, art and philosophy of chiropractic*, Portland, OR, 1910, Portland Printing House.
2. Palmer DD, Palmer BJ: *The science of chiropractic: its principles and adjustments*, Davenport, IA, 1906, Palmer School of Chiropractic.
3. Montgomery DP, Nelson JM: Evolution of chiropractic theories of practice and spinal adjustment: 1900–1950, *Chiropr Hist* 5:71, 1985.
4. Strasser A: The chiropractic model of health: a personal perspective, *Dig Chiropr Econ* 31(2):12, 1988.
5. Strasser A: The dynamics of human structure in the chiropractic model of health, *Dig Chiropr Econ* 32(4):14, 1990.

6. Jamison JR: Chiropractic and medical models of health care: a contemporary perspective, *J Manipulative Physiol Ther* 8(1):17, 1985.
7. Sandoz R: A perspective for the chiropractic profession, *J Can Chiropr Assoc* 21(3):107, 1977.
8. Quigley WH: Chiropractic's monocausal theory of disease, *J Am Chiropr Assoc* 8(6):52, 1981.
9. Council on Chiropractic Education: Standards for doctor of chiropractic programs and requirements for institutional status, Appendix III—Glossary. Scottsdale AZ, 2007.
10. Indexed synopsis of ACA policies on public health and related matters: 1989–1990, Des Moines, IA, 1991, American Chiropractic Association.
11. Haldeman S: Spinal manipulative therapy: a status report, *Clin Orthop* 179:62, 1983.
12. Triano J: Biomechanics of spinal manipulative therapy, *Spine J* 1(2):121, 2001.
13. Triano J: The mechanics of spinal manipulation. In Herzog W, editor: *Clinical biomechanics of spinal manipulation*, New York, 2000, Churchill Livingstone, pp 92–190.
14. Bernard TN, Kirkaldy-Willis WH: Recognizing specific characteristics of nonspecific low back pain, *Clin Orthop* 217:266, 1987.
15. Stonebrink RD: Evaluation and manipulative management of common musculoskeletal disorders, Portland, OR, 1990, Western States Chiropractic College.
16. Grieve GP: Aetiology in general terms. In Grieve GP, editor: *Common vertebral joint problems*, Edinburgh, 1988, Churchill Livingstone.
17. Grieve GP: Pathological changes: General. In *Common vertebral joint problems*, Edinburgh, UK, 1988, Churchill Livingstone.
18. Grieve GP: Manipulation in general terms. In *Common vertebral joint problems*, Edinburgh, 1988, Churchill Livingstone.
19. Kirkaldy-Willis WH: Pathology and pathogenesis. In Kirkaldy-Willis WH, editor: *Managing low back pain*, ed 3, New York, 1992, Churchill Livingstone.
20. Kirkaldy-Willis WH: The three phases of the spectrum of degenerative disease. In Kirkaldy-Willis WH, editor: *Managing low back pain*, ed 3, New York, 1992, Churchill Livingstone.
21. Cassidy JD, Kirkaldy-Willis WH: Manipulation. In Kirkaldy-Willis WH, editor: *Managing low back pain*, ed 3, New York, 1992, Churchill Livingstone.
22. Cyriax J: Diagnosis of soft tissue lesions. In *Textbook of orthopedic medicine*, ed 8, vol 1, London, 1982, Bailliere Tindall.
23. Sandoz R: Some critical reflections on subluxations and adjustments, *Ann Swiss Chiropr Assoc* 9:7, 1989.
24. Nelson C: The subluxation question, *J Chiropr Humanit* 7(1):46, 1997.
25. Mootz RD: Theoretic models of subluxation. In Gatterman MI, editor: *Fundamentals of chiropractic subluxation*, ed 2, St. Louis, 2005, Mosby.
26. Sandoz R: The natural history of a spinal degenerative lesion, *Ann Swiss Chiropr Assoc* 9:149, 1989.
27. Palmer BJ: Fight to climb, Davenport, IA, 1950, Palmer School of Chiropractic.
28. Palmer BJ: The subluxation specific—The adjustment specific: an exposition of the cause of all disease, Davenport, IA, 1934, Palmer School of Chiropractic.
29. Stephenson RW: Chiropractic textbook, Davenport, IA, 1948, Palmer School of Chiropractic.
30. Sandoz R: Classification of luxations, subluxations and fixations of the cervical spine, *Ann Swiss Chiropr Assoc* 6:219, 1976.
31. Vear HJ: An introduction to the science of chiropractic, Portland, OR, 1981, Western States Chiropractic College.
32. Hildebrandt RW: The scope of chiropractic as a clinical science and art: An introductory review of concepts, *J Manipulative Physiol Ther* 1(1):7, 1978.
33. Janse J: History of the development of chiropractic concepts, chiropractic terminology. In *The research status of spinal manipulative therapy*, NINCDS Monograph No 15, DHEW Pub No 76–988, Washington, DC, 1975, US Government Printing Office.
34. Lantz CA: The vertebral subluxation complex, *ICA Rev* 45(5):37, 1989.
33. Leach RA: The chiropractic theories: A synopsis of scientific research, ed 2, Baltimore, 1986, Williams & Wilkins.
36. Palmer BJ: Our masterpiece, Davenport, IA, 1966, Palmer College of Chiropractic.
37. Haldeman S, Hammerich K: The evolution of neurology and the concept of chiropractic, *J Am Chiropr Assoc* 7:57, 1973.
38. Homewood AE: The neurodynamics of the vertebral subluxation, St Petersburg, FL, 1979, Valkyrie Press.
39. Dishman R: Review of the literature supporting a scientific basis for chiropractic subluxation complex, *J Manipulative Physiol Ther* 8(3):163, 1985.
40. ICA policy handbook and code of ethics, Arlington, VA, 1990, International Chiropractors Association.
41. Gillet H: Vertebral fixations: An introduction to movement palpation, *Ann Swiss Chiropr Assoc* 1:30, 1960.
42. Gillet H: The anatomy and physiology of spinal fixations, *J Nat Chiropr Assoc* 1963.
43. Gillet H, Liekens M: A further study of spinal fixations, *Ann Swiss Chiropr Assoc* 4:41, 1969.
44. Gillet H: Spinal and related fixations, *Dig Chiropr Econ* 14(3):22, 1973.
45. Gillet H, Liekens M: Belgian chiropractic research notes, Huntington Beach, CA, 1984, Motion Palpation Institute.
46. Gillet H: The history of motion palpation, *Eur J Chiropr* 31:196, 1983.
47. Illi FH: The vertebral column: life-life of the body, Chicago, 1951, National College of Chiropractic.
48. Mennell J McM: Back pain diagnosis and treatment using manipulative techniques, Boston, 1960, Little, Brown.
49. Mennell J McM: Joint pain diagnosis and treatment using manipulative techniques, Boston, 1964, Little, Brown.
50. Sandoz R: Some physical mechanisms and effects of spinal adjustments, *Ann Swiss Chiropr Assoc* 6:91, 1976.
51. Sandoz R: Newer trends in the pathogenesis of spinal disorders, *Ann Swiss Chiropr Assoc* 5:93, 1971.
52. Faye LJ: Motion palpation of the spine: MPI notes and review of literature, Huntington Beach, CA, 1981, Motion Palpation Institute.
53. Schafer RC, Faye LJ: Motion palpation and chiropractic technique: principles of dynamic chiropractic, Huntington Beach, CA, 1989, Motion Palpation Institute.
54. Dishman R: Static and dynamic components of the chiropractic subluxation complex: a literature review, *J Manipulative Physiol Ther* 11(2):98, 1988.
55. Triano JJ: The subluxation complex: outcome measure of chiropractic diagnosis and treatment, *J Chiropr Tech* 2(3):114, 1990.
56. Gitelman R: The treatment of pain by spinal manipulation. In *The research status of spinal manipulative therapy*, NINCDS Monograph No 15, DHEW Pub No 76–988, Washington, DC, 1975, US Government Printing Office.
57. Howe JW: Preliminary observations from cinerentgenological studies of the spinal column, *J Am Chiropr Assoc* 4:565, 1970.
58. Brantingham JW: A survey of literature regarding the behavior, pathology, etiology, and nomenclature of the chiropractic lesion, *J Am Chiropr Assoc* 19(8):65, 1985.
59. Taber's cyclopedia medical dictionary, ed 15, Philadelphia, 1985, FA Davis.
60. Gatterman MI, Hansen DT: Development of chiropractic nomenclature through consensus, *J Manipulative Physiol Ther* 17(5):302, 1994.

61. ACA Council on Technic: Chiropractic terminology: a report, *J Am Chiropr Assoc* 25(10):46, 1988.
62. Hart AC, Hopkins CA, editors: International classification of diseases—Clinical modification (ed 9) and hospital modification (ed 6), West Valley City, UT, 2001, Ingenix.
63. Ward M: Glossary of osteopathic terminology, AOA, 1981.
64. Gatterman MI: Foundations of chiropractic subluxation, St Louis, 1995, Mosby.
65. Faye LJ: Most people who erect theories come to believe them themselves, *Dynamic Chiropr* 1984.
66. Hubka MJ: Another critical look at the subluxation hypothesis, *J Chiropr Tech* 2(1):27, 1990.
67. Lantz C: The vertebral subluxation complex. In Gatterman MI, editor: Foundations of chiropractic: subluxation, St Louis, 1994, Mosby.
68. Faye LJ: The subluxation complex, *J Chiropr Humanit* 9:1–4, 2000.
69. Leboeuf-Yde C: How real is the subluxation? A research perspective, *J Manipulative Physiol Ther* 21(7):492–494, 1998.
70. Nelson CF: The subluxation question, *J Chiropr Humanit* 7: 46–55, 1997.
71. Keating JC Jr, et al: Subluxation: dogma or science? *Chiropr Osteo* 13:17, 2005.
72. Keating JC: To hunt the subluxation: clinical research considerations, *J Manipulative Physiol Ther* 19(9):613–619, 1996.
73. Keating JC: Science and politics and the subluxation, *Am J Chiropr Med* 1(3):107–110, 1988.
74. Walker BF, Buchbinder MB: Most commonly used methods of detecting spinal subluxation and the preferred term for its description: a survey of chiropractors in Victoria, Australia, *J Manipulative Physiol Ther* 20(9):583, 1997.
75. Lantz CA: Immobilization degeneration and the fixation hypothesis of chiropractic subluxation, *Chiropr Research J* 1(1):21, 1988.
76. Rahlmann JF: Mechanisms of intervertebral joint fixation: a literature review, *J Manipulative Physiol Ther* 10(4):177, 1987.
77. Lewit K: Manipulative therapy in rehabilitation of the locomotor system, Boston, 1985, Butterworths.
78. Maigne R: Orthopedic medicine: a new approach to vertebral manipulations, Springfield, IL, 1972, Charles C Thomas.
79. Kellet J: Acute soft tissue injuries: a review of the literature, *Med Sci Sports Exerc* 18(5):489, 1986.
80. Oakes BW: Acute soft tissue injuries: nature and management, *Aust Family Physician* 10(Suppl):3, 1982.
81. Cyriax J: Treatment of pain by manipulation. In The research status of spinal manipulative therapy, NINCDS Monograph No 15, DHEW Pub No 76–988, Washington, DC, 1975, US Government Printing Office.
82. Stonebrink RD: Physiotherapy guidelines for the chiropractic profession, *J Am Chiropr Assoc* 9:65, 1975.
83. Akeson WH, Amiel D, Woo SL-Y: Immobility effects of synovial joints: the pathomechanics of joint contracture, *Biorheology* 17:9, 1980.
84. Akeson WH, et al: Collagen cross-linking alterations in joint contractures: changes in the reducible cross-links in periarticular connective tissue collagen after nine weeks of immobilization, *Connect Tissue Res* 5:15, 1977.
85. Woo S.L.-Y., Matthews JV, Akeson WH: Connective tissue response to immobility: correlative study of biomechanical and biochemical measurements of normal and immobilized rabbit knees, *Arthritis Rheum* 18(3):257, 1975.
86. Akeson WH: An experimental study of joint stiffness, *J Bone Joint Surg Am* 43(7):1022, 1961.
87. Noyes FR, et al: Biomechanics of ligament failure, *J Bone Joint Surg Am* 56(7):1406, 1974.
88. Enneking WF, Horowitz M: The intra-articular effects of immobilization of the human knee, *J Bone Joint Surg Am* 54(5):973, 1972.
89. Binkley JM, Peat M: The effects of immobilization on the ultrastructure and mechanical properties of the medial collateral ligament of rats, *Clin Orthop* 203:301, 1986.
90. Amiel D, Woo S.L.-Y., Harwood F: The effect of immobilization on collagen turnover in connective tissue: a biomechanical-biomechanical correlation, *Acta Orthop Scand* 53:325, 1982.
91. Baker W, De C: Changes in the cartilage of the posterior intervertebral joints after anterior fusion, *J Bone Joint Surg Br* 51(4):736, 1969.
92. Donatelli R: Effects of immobilization on the extensibility of periarticular connective tissue, *J Orthop Sports Phys Ther* 3(2):67, 1981.
93. Videman T: Experimental models of osteoarthritis: the role of immobilization, *Clin Biomech* 2:223, 1987.
94. Still AT: Osteopathic research and practice, Kirksville, MO, 1910, AT Still.
95. Triano JJ: Buckling: a biomedical model of subluxation. In Gatterman MI, editor: Foundations of chiropractic subluxation, ed 2, St Louis, 2005, Mosby.
96. Bourdillon JF, Day EA, Bookhout MR: Spinal manipulation, ed 5, Oxford, UK, 1992, Butterworth-Heinemann.
97. Henderson CNR: Three neurophysiological theories on the chiropractic subluxation. In Gatterman MI, editor: Foundations of chiropractic: subluxation, ed 2, St Louis, 2005, Mosby, pp 296–303.
98. Leach RA, Pickar JG: Segmental dysfunction hypothesis: joint and muscle pathology and facilitation. In Leach RA, editor: The chiropractic theories, ed 4, Philadelphia, 2005, Lippincott Williams & Wilkins, pp 137–206.
99. Bakkum BW, et al: Preliminary morphological evidence that vertebral hypomobility induces synaptic plasticity in the spinal cord, *J Manipulative Physiol Ther* 30:336–342, 2007.
100. Mabit C, et al: Study of the experimental biomechanics of tendon repair with immediate active mobilization, *Surg Radiol Anat* 8(1):29, 1986.
101. Cornwall MW, Leveau B: The effect of physical activity on ligamentous strength: an overview, *J Orthop Sports Phys Ther* 5(5):275, 1984.
102. Fitz-Ritson D: Chiropractic management and rehabilitation of cervical trauma, *J Manipulative Physiol Ther* 13(1):17, 1990.
103. Waddell G: A new clinical model for the treatment of low-back pain, *Spine* 12(7):632, 1987.
104. Mealy K, et al: Early mobilization of acute whiplash injuries, *BMJ* 292:656, 1986.
105. Deyo RA: How many days of bed rest for acute low back pain? *N Engl J Med* 315:1064, 1986.
106. Mayer T, Gatchel R: Functional restoration for spinal disorders: the sports medicine approach, Philadelphia, 1988, Lea & Febiger.
107. Salter RB: The biologic concept of continuous passive motion of synovial joints: the first 18 years of basic research and its clinical application, *Clin Orthop* 242:12, 1989.
108. Salter RB: Motion vs. rest: why immobilize joints? *J Bone Joint Surg Br* 64:251, 1982.
109. Van Royen BJ, et al: Comparison of the effects of immobilization and continuous passive motion on surgical wound healing in the rabbit, *Plast Reconstr Surg* 78:360, 1986.
110. Evans E, et al: Experimental immobilization and remobilization of rat knee joints, *J Bone Joint Surg Am* 42(5):737, 1960.
111. Allen ME: Arthritis and adaptive walking and running, *Rheum Dis Clin North Am* 16(4):887, 1990.
112. Frank C, et al: Physiology and therapeutic value of passive joint motion, *Clin Orthop* 185:113, 1984.
113. Korcok M: Motion, not immobility, advocated for healing synovial joints, *JAMA* 246:1981, 2005.
114. Liebensohn C: Guidelines for effective management of spinal pain. In Liebensohn C, editor: Rehabilitation of the spine, Baltimore, 1996, Williams & Wilkins.

115. Bigos S, et al: Acute low back problems in adults, Clinical Practice Guideline No 14, AHCPR Pub No 95-0642, Rockville, MD, 1994, Agency for Health Care Policy and Research, Public Health Service, US Department of Health and Human Services.
116. Korr IM: Proprioceptors and somatic dysfunction, *J Am Osteopath Assoc* 74:638, 1975.
117. Kirkaldy-Willis WH, editor: Managing low back pain, ed 3, New York, 1992, Churchill Livingstone.
118. Gatterman MI: Chiropractic management of spine related disorders, Baltimore, 1990, Williams & Wilkins.
119. Good AB: Spinal joint blocking, *J Manipulative Physiol Ther* 8(1):1, 1985.
120. Melzack R, Wall PD: Pain mechanisms: a new theory, *Science* 150:971, 1965.
121. Fryer G, Morris T, Gibbons P: Paraspinal muscles and intervertebral dysfunction: Part one, *J Manipulative Physiol Ther* 27:267, 2004.
122. Simons D: Myofascial pain syndromes due to trigger points. I. Principles, diagnosis and perpetuating factors, *Manipulative Med* 1:67, 1985.
123. Travell J, Simons D: Myofascial pain and dysfunction: the trigger point manual, Baltimore, 1983, Williams & Wilkins.
124. de Seze S: Les accidents de la deterioration structurale du disque, *Semin Hop Paris* 1:2267, 1955.
125. de Seze S: Les attitudes antalgique dans la sciatique discoradiculaire commune, *Semin Hop Paris* 1:2291, 1955.
126. Cyriax J: Lumbago: Mechanism of dural pain, *Lancet* 1:427, 1945.
127. Cassidy JD, Kirkaldy-Willis WH: Manipulation. In Kirkaldy-Willis WH, editor: Managing low back pain, ed 3, New York, 1992, Churchill Livingstone.
128. Herbst R: Gonstead chiropractic science and art: The chiropractic methodology of Clarence S. Gonstead, Mt Horeb, WI, 1980, DC SCI-CHI Publications.
129. Barge FH: Torticollis, Davenport, IA, 1979, Bawden Bros.
130. Schmorl G, Junghans H: The human spine in health and disease, ed 2, New York, 1971, Grune & Stratton.
131. Giles LGF: Anatomical basis of low back pain, Baltimore, 1989, Williams & Wilkins.
132. Giles LGF, Taylor JR: Intra-articular synovial protrusions in the lower lumbar apophyseal joints, *Bull Hosp Joint Dis Orthop Inst* 42:248, 1982.
133. Giles LGF, Taylor JR, Cockson A: Human zygapophyseal joint synovial folds, *Acta Anat* 126:110, 1986.
134. Giles LGF, Taylor JR: Innervation of lumbar zygapophyseal joint synovial folds, *Acta Orthop Scand* 58:43, 1987.
135. Giles LGF: Lumbar apophyseal joint arthrography, *J Manipulative Physiol Ther* 7(1):21, 1984.
136. Giles LGF: Lumbo-sacral and cervical zygapophyseal joint inclusions, *Manipulative Med* 2:89, 1986.
137. Kos J, Wolf J: Les menisques intervertebraux et leur role possible dans les blocages vertebraux, *Ann Med Phys* 15:203, 1972.
138. Kos J, Wolf J: Translation of reference 119 into English, *J Orthop Sports Phys Ther* 1:8, 1972.
139. Bogduk N, Engel R: The menisci of the lumbar zygapophyseal joints: a review of their anatomy and clinical significance, *Spine* 9(5):454, 1984.
140. Bogduk N, Jull G: The theoretical pathology of acute locked back: a basis for manipulative therapy, *J Manual Med* 1:78, 1985.
141. Engel RM, Bogduk N: The menisci of the lumbar zygapophyseal joints, *J Anat* 135:795, 1982.
142. Badgley CE: The articular facets in relation to low back pain and sciatic radiation, *J Bone Joint Surg* 23:481, 1941.
143. Hadley LA: Anatomic-roentgenographic studies of the spine, ed 5, Springfield, IL, 1964, Charles C Thomas.
144. Kraft GL, Levinthal DH: Facet synovial impingement, *Surg Gynecol Obstet* 93:439, 1951.
145. Saboe L: Possible clinical significance of intra-articular synovial protrusions: a review of the literature, *J Manipulative Physiol Ther* 3:148, 1988.
146. Jones T, et al: Lumbar zygapophyseal joint meniscoids: Evidence of their role in chronic intersegmental hypomobility, *J Manipulative Physiol Ther* 12(5):374, 1989.
147. Bogduk N, Twomey LT: Clinical anatomy of the lumbar spine, ed 2, Melbourne, Australia, 1991, Churchill Livingstone.
148. Tillmann K: Pathological aspects of osteoarthritis related to surgery, *Inflammation* 8(Suppl):557, 1984.
149. Howell D: Pathogenesis of osteoarthritis, *Am J Med* 80(Suppl 4B):24, 1986.
150. Hochberg MC: Osteoarthritis: pathophysiology, clinical features, management, *Hosp Pract* 19(12):41, 1984.
151. Murray RO, Duncan C: Athletic activity in adolescence as an etiological factor in degenerative hip disease, *J Bone Joint Surg Br* 53(3):407, 1971.
152. Kos J, Wolf J: Translation of reference 94 into English, *J Orthop Sports Phys Ther* 1:8, 1972.
153. Farfan MF: Torsion and compression. In Farfan MF, editor: Mechanical disorders of the low back, Philadelphia, 1973, Lea & Febiger.
154. McGill S: Functional anatomy of the lumbar spine. In Low back disorders, ed 2, 2007, Human Kinetics, Ontario, Canada.
155. Cyriax J: Textbook of orthopedic medicine, ed 9, vol 2, London, 1974, Baillier Tindall.
156. Vanharanta H, et al: The relationship of pain provocation to lumbar disc deterioration as seen by CT discography, *Spine* 12:295, 1987.
157. Preuss R, Fung J: Can acute low back pain result from segmental spinal buckling during sub-maximal activities? A review of the current literature, *Man Ther* (10):14, 2005.
158. Kirkaldy-Willis WH, Hill RJ: A more precise diagnosis for low-back pain, *Spine* 4(2):102, 1979.
159. Yong-Hing K, Kirkaldy-Willis WH: The pathophysiology of degenerative disease of the lumbar spine, *Orthop Clin North Am* 14(3):491, 1983.
160. McGill S, Cholewicki J: Biomechanical basis for stability: an explanation to enhance clinical utility, *J Orthop Sports Phys Ther* 31:96, 2001.
161. McGill S: Low back disorders: evidence-based practice and rehabilitation, Windsor, Ontario, 2002, Human Kinetics.
162. Pope M, Frymoyer JW, Krag MH: Diagnosing instability, *Clin Orthop* 279:60, 1992.
163. Nachemson A: Lumbar spine instability: A critical update and symposium summary, *Spine* 10:290, 1985.
164. Dupuis PR, et al: Radiologic diagnosis of degenerative lumbar spinal instability, *Spine* 10(3):262, 1985.
165. Schneider G: The unstable lumbar segment definition and detection, *J Man Manipulative Ther* 1:67, 1993.
166. Paris S: Physical signs of instability, *Spine* 10:277, 1985.
167. Barr KP, Griggs M, Cadby M: Lumbar stabilization: a review of core concepts and current literature, part 2, *Am J Phys Med Rehab* 86:72, 2007.
168. Abbott JH, et al: Lumbar segmental instability: a criterion-related validity study of manual therapy assessment, *BMC Musculoskelet Disord* 6:56, 2005.
169. Kirkaldy-Willis WH, editor: Pathology and pathogenesis of low back pain; the three phases of the spectrum of degenerative disease. In Kirkaldy-Willis WH, editor: Managing low back pain, ed 3, New York, 1992, Churchill Livingstone.
170. Kirkaldy-Willis WH, et al: Pathology and pathogenesis of lumbar spondylosis and stenosis, *Spine* 3(4):319, 1978.
171. Crelin ES: A scientific test of the chiropractic theory, *Am Sci* 61:574, 1973.
172. Rydevik B, Brown M, Lundborg G: Pathoanatomy and pathophysiology of nerve root compression, *Spine* 9(1):7, 1984.
173. Young S, Sharpless SK: Mechanisms protecting nerve against compression block. In Suh CH, editor: Proceedings of the 9th Annual Biomechanics Conference on the Spine, Boulder, CO, 1978, International Chiropractors Association.

174. Giles LGF: A histological investigation of human lower lumbar intervertebral canal (foramen) dimensions, *J Manipulative Physiol Ther* 17:4, 1994.
175. Golub BS, Silverman B: Transforaminal ligaments of the lumbar spine, *J Bone Joint Surg* 51:947-956, 1969.
176. Howe JF, Loeser JD, Calvin WH: Mechanosensitivity of dorsal root ganglia and chronically injured axons: a physiological basis for the radicular pain of nerve root compression, *Pain* 17:321, 1983.
177. Wall JF, Devor M: Sensory afferent impulses originate from dorsal root ganglia as well as from the periphery in normal and nerve injured rats, *Pain* 17:321, 1983.
178. Drum DC: The vertebral motor unit and intervertebral foramen. In *The research status of spinal manipulative therapy*, NINCDS Monograph No 15, DHEW Pub No 76-988, Washington, DC, 1975, US Government Printing Office.
179. Luttges MW, Kelly PT, Gerren RA: Degenerative changes in mouse sciatic nerves: electrophoretic and electrophysiologic characterizations, *Exp Neurol* 50:706-733, 1976.
180. Luttges MW, Stodiek LS, Beel JA: Post injury changes in the biomechanics of nerves and roots in mice, *J Manipulative Physiol Ther* 9(2):89, 1986.
181. Triano JJ, Luttges MW: Nerve irritation: a possible model for sciatic neuritis, *Spine* 7:129-136, 1982.
182. Macgregor RI, Sharpless SK, Luttges MW: A pressure vessel model for nerve compression, *J Neurol Sci* 24:299, 1975.
183. Devor M, Obermayer M: Membrane differentiation in rat dorsal root ganglia and possible consequences for back pain, *Neurosci Letters* 51:341, 1984.
184. Rydevik BL: The effects of compression on the physiology of nerve roots, *J Manipulative Physiol Ther* 15:62, 1992.
185. Howe JF, Loeser JD, Calvin WH: Mechanosensitivity of dorsal root ganglia and chronically injured axons: a physiological basis for the radicular pain of nerve root compression, *Pain* 3:27, 1977.
186. Pickar JG: Neurophysiological effects of spinal manipulation, *Spine* J 2:357, 2002.
187. McCarron RF, et al: The inflammatory effect of nucleus pulposus. A possible element in the pathogenesis of low-back pain, *Spine* 12:760, 1987.
188. Kawakami M, et al: Mechanical compression of the lumbar nerve root alters pain-related behaviors induced by the nucleus pulposus in the rat, *J Orthop Res* 18:257, 2000.
189. Yabuki S, Igarashi T, Kikuchi S: Application of nucleus pulposus to the nerve root simultaneously reduces blood flow in dorsal root ganglion and corresponding hindpaw in the rat, *Spine* 25:1471, 2000.
190. Nygaard OP, Mellgren SI, Osterud B: The inflammatory properties of contained and noncontained lumbar disc herniation, *Spine* 22:2484, 1997.
191. Ozaktay AC, Kallakuri S, Cavanaugh JM: Phospholipase A 2 sensitivity of the dorsal root and dorsal root ganglion, *Spine* 23:1297, 1998.
192. Chen C, et al: Effects of phospholipase A2 on lumbar nerve root structure and function, *Spine* 22:1057, 1997.
193. Korr IM, editor: *The neurobiologic mechanisms in manipulative therapy*, New York, 1978, Plenum.
194. Sato A, Budgell B: Somatoautonomic reflexes. In Haldeman S, editor: *Principles and practice of chiropractic*, ed 3, New York, 2005, McGraw-Hill.
195. Sato A: The somatosympathetic reflexes: their physiologic and clinical significance. In Goldstein M, editor: *The research status of spinal manipulative therapy*, Washington, DC, 1975, US Government Printing Office.
196. Gillette RG: A speculative argument for the coactivation of diverse somatic receptor populations by forceful chiropractic adjustments, *Manipulative Med* 3:1, 1987.
197. Sato A: Spinal reflex physiology. In Haldeman S, editor: *Principles and practice of chiropractic*, ed 2, Norwalk, CT, 1992, Appleton & Lange.
198. Korr IM, Wright HM, Thomas PE: Effects of experimental myofascial insults on cutaneous patterns of sympathetic activity in man, *J Neural Transm* 23(22):330, 1962.
199. Korr IM, Wright HM, Chace JA: Cutaneous patterns of sympathetic activity in clinical abnormalities of the musculoskeletal system, *Acta Neuroveg* 25:589, 1964.
200. Hulse M: Disequilibrium caused by a functional disturbance of the upper cervical spine, *Manual Med* 1:18, 1983.
201. Mooney B, Robertson J: The facet syndrome, *Clin Orthop* 115:149, 1976.
202. Thabe H: Electromyography as a tool to document diagnostic findings and therapeutic results associated with somatic dysfunctions in upper cervical spinal joints and sacroiliac joints, *Manual Med* 2:53, 1986.
203. Grainger HG: The somatic component in visceral disease. In *Academy of Applied Osteopathy 1958 yearbook*, Newark, OH, 1958, American Academy of Osteopathy.
204. Beal MC: Viscerosomatic reflexes: a review, *J Am Osteopath Assoc* 85(12):786, 1985.
205. Larson NJ: Summary of site and occurrence of paraspinal soft tissue changes of patients in the intensive care unit, *J Am Osteopath Assoc* 75:840, 1976.
206. Kelso AF: A double blind clinical study of osteopathic findings in hospital patients, *J Am Osteopath Assoc* 70:570, 1971.
207. Beal MC: Palpatory findings for somatic dysfunction in patients with cardiovascular disease, *J Am Osteopath Assoc* 82:822, 1983.
208. Beal MC, Dvorak J: Palpatory examination of the spine: a comparison of the results of two methods and their relationship to visceral disease, *Manual Med* 1:25, 1984.
209. Kimura A, et al: A- and C-reflexes elicited in cardiac sympathetic nerves by single shock to a somatic afferent nerve include spinal and supraspinal components in anesthetized rats, *Neurosci Res* 25:91, 1996.
210. Budgell B, Igarashi Y: Response to arrhythmia to spinal manipulation: monitoring by ECG with analysis of heart-rate variability, *J Neuromusculoskel Syst* 9:97, 2001.
211. Delaney JP, et al: The short-term effects of myofascial trigger point massage therapy on cardiac autonomic tone in healthy subjects, *J Adv Nurs* 37:364, 2002.
212. Pollard H: The somatovisceral reflex: how important for the "type O" condition? *Chiropr J Aust* 34:93, 2004.
213. Pollard H: Reflections on the "type O" disorder, *J Manipulative Ther* 28:547.e1, 2005.
214. Toda H, et al: Responses of dorsal spinal cord blood flow to noxious mechanical stimulation of the skin in anesthetized rats, *J Physiol Sci* 58(4):263, 2008.
215. Kurosawa M, et al: Contribution of supraspinal and spinal structures to the responses of dorsal spinal cord blood flow to innocuous cutaneous brushing in rats, *Auton Neurosci* 136(1-2):96, 2007.
216. Cramer G, et al: Basic science research related to chiropractic spinal adjusting: the state of the art and recommendations revisited, *J Manipulative Physiol Ther* 29(9):726, 2006.
217. Budgell B, Polus B: The effects of thoracic manipulation on heart rate variability: a controlled crossover trial, *J Manipulative Physiol Ther* 29(8):603, 2006.
218. Budgell B, Hirano F: Innocuous mechanical stimulation of the neck and alterations in heart-rate variability in healthy young adults, *Auton Neurosci* 91(1-2):96, 2001.
219. Budgell B, Suzuki A: Inhibition of gastric motility by noxious chemical stimulation of interspinous tissues in the rat, *J Auton Nerv Syst* 80(3):162, 2000.
220. Bolton PS, et al: Influences of neck afferents on sympathetic and respiratory nerve activity, *Brain Res Bull* 47:413, 1998.
221. Lantz CA: The vertebral subluxation complex. In Gatterman MI, editor: *Foundations of chiropractic subluxation*, St Louis, 1995, Mosby.

222. Wright V, Johns JJ: Physical factors concerned with the stiffness of normal and diseased joints, *Bull Johns Hopkins Hosp* 106:216, 1960.
223. Fields HL: Pain, San Francisco, 1987, McGraw-Hill.
224. Janda V: Muscles, central nervous motor regulation and back problems. In Korr IM, editor: *The neurobiologic mechanisms in manipulative therapy*, New York, 1978, Plenum Press.
225. Grigg P, Schaible HG, Schmidt RF: Mechanical sensitivity of group III and IV afferents from posterior articular nerve in normal and inflamed cat knee, *J Neurophysiol* 55:635, 1986.
226. Schaible HG, Schmidt RF: Time course of mechanosensitivity changes in articular afferents during a developing experimental arthritis, *J Neurophysiol* 60:2180, 1988.
227. Schaible HG, Schmidt RF: Effects of an experimental arthritis on the sensory properties of fine articular afferent units, *J Neurophysiol* 54:1109, 1985.
228. Salter RB: *Textbook of disorders and injuries of the musculoskeletal system*, ed 2, Baltimore, 1983, Williams & Wilkins.
229. Ritchie AC: *Boyd's textbook of pathology*, ed 9, vol 2, Philadelphia, 1990, Lea & Febiger.
230. Kirkaldy-Willis WH: Pathology and pathogenesis of low back pain. In Kirkaldy-Willis WH, editor: *Managing low back pain*, ed 2, New York, 1988, Churchill Livingstone.
231. Gatterman MI, Hansen DT: Development of chiropractic nomenclature through consensus, *J Manipulative Physiol Ther* 17:302, 1994.
232. Gatterman MI, editor: *Foundations of chiropractic: subluxation*, St Louis, 1995, Mosby, pp 306–469.
233. Cassidy DJ, Potter GE: Motion examination of the lumbar spine, *J Manipulative Physiol Ther* 2:151, 1979.
234. Faucet B, et al: Determination of body subluxations by clinical, neurological and chiropractic procedures, *J Manipulative Physiol Ther* 3:165, 1980.
235. Russell R: Diagnostic palpation of the spine: a review of procedures and assessment of their reliability, *J Manipulative Physiol Ther* 6(4):181, 1983.
236. Sandoz R: The choice of appropriate clinical criteria for assessing the progress of a chiropractic case, *Ann Swiss Chiropr Assoc* 8:53, 1985.
237. Bryner P: A survey of indications: knee manipulation, *Chiropr Tech* 1(4):140, 1989.
238. Bryner P, Bruin J: Extremity joint technique: Survey of the status of technique in chiropractic colleges, *Chiropr Tech* 3(1):30, 1991.
239. Scaringe JG, Faye LJ: Palpation: the art of manual assessment. In Redwood D, Cleveland, III CS, editors: *Fundamentals of chiropractic*, St Louis, 2003, Mosby.
240. Bergmann TF: The chiropractic spinal examination. In Ferezy JS, editor: *The chiropractic neurological examination*, Gaithersburg, MD, 1992, Aspen.
241. Bourdillon JF, Day EA: *Spinal manipulation*, ed 4, London, 1987, William Heinemann Medical Books.
242. Hestboek L, Leboeuf-Yde C: Are chiropractic tests for the lumbo-pelvic spine reliable and valid? A systematic critical literature review, *J Manipulative Physiol Ther* 23:258, 2000.
243. Stockendahl MJ, et al: Manual examination of the spine: a systematic critical literature review of reproducibility, *J Manipulative Physiol Ther* 29:475, 2006.
244. Piva SR, et al: Inter-tester reliability of passive intervertebral and active movements of the cervical spine, *Man Ther* 11:321, 2006.
245. Schneider M, et al: Interexaminer reliability of the prone leg length analysis procedure, *J Manipulative Physiol Ther* 30:514, 2007.
246. Herzog W, et al: Reliability of motion palpation procedures to detect sacroiliac joint fixations, *J Manipulative Physiol Ther* 12:86, 1989.
247. Haas M: Interexaminer reliability for multiple diagnostic test regimens, *J Manipulative Physiol Ther* 14:95, 1991.
248. Breen A: The reliability of palpation and other diagnostic methods, *J Manipulative Physiol Ther* 15:54, 1992.
249. Smedmark V, Wallin M, Arvidsson I: Inter-examiner reliability in assessing passive intervertebral motion of the cervical spine, *Manual Ther* 5:97, 2000.
250. Pool JJ, et al: The interexaminer reproducibility of physical examination of the cervical spine, *J Manipulative Physiol Ther* 27:84, 2004.
251. Medicare carriers manual, Rev. 1565, Section 2251.2, Coverage of Chiropractic Services, www.hcfa.gov.
252. Keating JC, et al: Interexaminer reliability of eight evaluative dimensions of lumbar segmental abnormality, *J Manipulative Physiol Ther* 13(8):463, 1990.
253. Boline P, et al: Interexaminer reliability of a multi-dimensional index of lumbar segmental abnormality, II, *J Manipulative Physiol Ther* 16(6):363, 1993.
254. Hubka MJ, Phelan SP: Interexaminer reliability of palpation for cervical spine tenderness, *J Manipulative Physiol Ther* 17:591, 1994.
255. Gemmell H, Miller P: Interexaminer reliability of multidimensional examination regimens used for detecting spinal manipulable lesions: a systematic review, *Clin Chiropractic* 8(4):199–204, 2005.
256. Jull G, Bogduk N, Marsland A: The accuracy of manual diagnosis for cervical zygapophysial joint pain syndromes, *Med J Aust* 148:233, 1988.
257. King W, et al: The validity of manual examination in assessing patients with neck pain, *Spine J* 7:22, 2007.
258. Leboeuf-Yde C, Kyvik K: Is it possible to differentiate people with low back pain on the basis of tests of lumbopelvic dysfunction? *J Manipulative Physiol Ther* 23(3):160, 2000.
259. Childs MJ, Fritz JM, Flynn TW: A clinical prediction rule to identify patients with low back pain most likely to benefit from spinal manipulation, *Ann Intern Med* 141(12):920, 2004.
260. Fritz JM, et al: Lumbar spine segmental mobility assessment: an examination of validity of determining intervention strategies in patients with low back pain, *Arch Phys Med Rehab* 86:1745, 2005.
261. De Hertogh WJ, et al: The clinical examination of neck pain patients: The validity of a group of tests, *Man Ther* 11:51, 2006.
262. Flynn T, Fritz J, Whitman J: A clinical prediction rule for classifying patients with low back pain who demonstrate short-term improvement with spinal manipulation, *Spine* 27(24):2835, 2002.
263. Fritz J, Dellito A, Erhard R: Comparison of classification-based physical therapy with therapy based on clinical practice guidelines for patients with acute low back pain: a randomized clinical trial, *Spine* 28(13):1363, 2003.
264. Haas M: The reliability of reliability, *J Manipulative Physiol Ther* 14(3):199, 1991.
265. Keating J: Several strategies for evaluating the objectivity of measurements in clinical research and practice, *J Can Chiropr Assoc* 32(3):133, 1988.
266. Keating J: Inter-examiner reliability of motion palpation of the lumbar spine: a review of quantitative literature, *Am J Chiropr Med* 2(3):107, 1989.
267. Boline P, et al: Interexaminer reliability of palpatory evaluations of the lumbar spine, *Am J Chiropr Med* 1(1):5, 1988.
268. Panzer DM: The reliability of lumbar motion palpation, *J Manipulative Physiol Ther* 15:518, 1992.
269. Breen A: The reliability of palpation and other diagnostic methods, *J Manipulative Physiol Ther* 15:54, 1992.
270. Haas M, Panzer DM: Palpatory diagnosis of subluxation. In Gatterman MI, editor: *Foundation of chiropractic subluxation*, St Louis, 1995, Mosby.
271. Keating JC: Traditional barriers to standards of knowledge production in chiropractic: proceedings of the Consensus Conference on Validation of Chiropractic Methods, *Chiropr Tech* 2(3):78–85, 1990.

272. Panzer DM: Lumbar motion palpation: A literature review. In *Proceedings of the Sixth Annual Conference on Research and Education*, Monterey, CA, 1991, CORE.
273. Koran LM: The reliability of clinical methods, data and judgments, *N Engl J Med* 293:642, 1975.
274. Nelson MA, et al: Reliability and reproducibility of clinical findings in low back pain, *Spine* 4:97, 1979.
275. Alley RJ: The clinical value of motion palpation as a diagnostic tool, *J Can Chiropr Assoc* 27:91, 1983.
276. Waddell G, et al: Normality and reliability in the clinical assessment of backache, *BMJ* 284:1519, 1982.
277. Shekelle PG: Current status of standards of care, *J Chiropr Tech* 2(3):86, 1990.
278. Walker BF: The reliability of chiropractic methods used for the detection of spinal subluxation, *Australas Chiropr Osteopathy* 5:12, 1996.
279. Lewit K, Liebenson C: Palpation—problems and implications, *J Manipulative Physiol Ther* 16:586, 1993.
280. Huijbregts P: Spinal motion palpation: a review of reliability studies, *J Man Manip Ther* 10:24, 2002.
281. Seffinger M, et al: Spinal palpation diagnostic procedures utilized by practitioners of spinal manipulation: Annotated bibliography of reliability studies, *J Can Chiropr Assoc* 47:89, 2003.
282. Najm WI, et al: Content validity of manual spinal palpation exams—A systematic review, *BMC Complement Altern Med* 3:1, 2003.
283. van Trijffela E, et al: Inter-examiner reliability of passive assessment of intervertebral motion in the cervical and lumbar spine: A systematic review, *Man Ther* 10:256, 2005.
284. Haas M: Interexaminer reliability for multiple diagnostic test regimens, *J Manipulative Physiol Ther* 14(2):95, 1991.
285. Hawk C, et al: Preliminary study of the reliability of assessment procedures for indications for chiropractic adjustments of the lumbar spine, *J Manipulative Physiol Ther* 22:382, 1999.
286. De Hertogh WJ, Vaesa PH, Vijverman V, et al: The clinical examination of neck pain patients: the validity of a group of tests, *Manual Ther* 12:50, 2007.
287. Tuchin PJ, et al: Interexaminer reliability of chiropractic evaluation for cervical spine problems—a pilot study, *Australas Chiropr Osteopathy* 5:23, 1996.
288. Boline PD, et al: Interexaminer reliability of eight evaluative dimensions of lumbar segmental abnormality: part II, *J Manipulative Physiol Ther* 16:363, 1993.
289. Keating JC, et al: Interexaminer reliability of eight evaluative dimensions of lumbar segmental abnormality, *J Manipulative Physiol Ther* 13:463, 1990.
290. French SD, Green S, Forbes A: Reliability of chiropractic methods commonly used to detect manipulable lesions in patients with chronic low back pain, *J Manipulative Physiol Ther* 23:231, 2000.
291. Abbott JH, et al: Manual physical assessment of spinal segmental motion: intent and validity, *Manual Ther* 14:36, 2009.
292. Haas M, et al: Short-term responsiveness of manual thoracic end-play assessment to spinal manipulation: a randomized controlled trial of construct validity, *J Manipulative Physiol Ther* 18:582, 1995.
293. Roberts AR, Yeager K: *Evidence-based practice manual: research and outcome measures in health and human services*, New York, 2004, Oxford University Press USA.
294. Bergmann TF: Introduction and opening statement, *Consensus Conference on Validation of Chiropractic Methods*, *Chiropr Tech* 2(3):71, 1990.
295. Liebenson C: *Rehabilitation of the spine: a practitioner's manual*, Baltimore, MD, 1996, Williams & Wilkins.
296. Yeomans SG: *The clinical application of outcomes assessment*, Stamford, CT, 2000, Appleton & Lange.
297. Lewis T: *Pain*, New York, 1987, McGraw-Hill.
298. Kellgren JH: The anatomical source of back pain, *Rheumatol Rehabil* 16(3):3, 1977.
299. Deyo RA: Measuring the functional status of patients with low back pain, *J Chiropr Tech* 2(3):127, 1990.
300. Vernon H: The neck disability index: a study of reliability and validity, *J Manipulative Physiol Ther* 14(7):409, 1991.
301. Love A, LeBoeuf C, Crisp T: Chiropractic chronic low back pain sufferers and self-report assessment methods. I. A reliability study of the visual analogue scale, the pain drawing and the McGill Pain Questionnaire, *J Manipulative Physiol Ther* 12(2):21, 1989.
302. Price DD, et al: The validation of visual analogue scales as ratio scale measures for chronic and experimental pain, *Pain* 17:45, 1983.
303. Price DD, Harkins SW: The combined use of visual analogue scales and experimental pain in proving standardized assessment of clinical pain, *Clin J Pain* 3:1, 1987.
304. Nyiendo J: A comparison of low back pain profiles of chiropractic teaching clinic patients with patients attending private clinicians, *J Manipulative Physiol Ther* 13(8):437, 1990.
305. Finch L, Melzack R: Objective pain measurement: a case for increased clinical usage, *Physiother Can* 34(6):1, 1982.
306. Pel JJM, et al: Biomechanical analysis of reducing sacroiliac joint shear load by optimization of pelvic muscle and ligament forces, *Ann Biomed Eng* 36(3):415, 2008.
307. Mayer TG, et al: Use of noninvasive techniques for quantification of spinal range-of-motion in normal subjects and chronic low-back dysfunction patients, *Spine* 9(6):588, 1984.
308. Klausen K: The shape of the spine in young males with and without back complaints, *Clin Biomech* 1:81, 1986.
309. Brunarski DJ: Chiropractic biomechanical evaluations: validity in myofascial low back pain, *J Manipulative Physiol Ther* 5(4):155, 1982.
310. Wietz EM: The lateral bending sign, *Spine* 6(4):119, 1981.
311. Peters RE: The facet syndrome, *J Aust Chiropr Assoc* 13(3):15, 1983.
312. Giles LGF, Taylor JR: Low-back pain associated with leg length inequality, *Spine* 6(5):510, 1981.
313. Enwemeka CS, et al: Postural correction in persons with neck pain. I. A survey of neck positions recommended by physical therapists, *J Orthop Sports Phys Ther* 8(5):235, 1986.
314. Enwemeka CS, et al: Postural correction in persons with neck pain. II. Integrated electromyography of the upper trapezius in three simulated neck positions, *J Orthop Sport Phys Ther* 8(5):240, 1986.
315. Pope MH, Bevins T, Wilder DG: The relationship between anthropometric, postural, muscular, and mobility characteristics of males ages 18–55, *Spine* 10(7):644, 1983.
316. Burton AK: Variation in lumbar sagittal mobility with low-back trouble, *Spine* 14(6):584, 1989.
317. Triano JJ, Schultz A: Correlation of objective measure of trunk motion and muscle function with low-back disability ratings, *Spine* 12(6):561, 1987.
318. Percy M, Portek I, Shepard J: The effect of low-back pain on lumbar spinal movement measured by three-dimensional x-ray analysis, *Spine* 10(2):150, 1985.
319. Fairbank J, et al: Influence of anthropometric factors and joint laxity in the incidence of adolescent back pain, *Spine* 9(5):461, 1984.
320. Mellin G: Correlations of spinal mobility with degree of chronic low back pain after correction for age and anthropometric factors, *Spine* 12(5):464, 1987.
321. Giles LGF, Taylor JR: Lumbar spine structural changes associated with leg length inequality, *Spine* 7(2):159, 1982.
322. Giles LGF: Lumbosacral facetal “joint angles” associated with leg length inequality, *Rheumatol Rehabil* 20(4):233, 1981.
323. Sandoz R: Principles underlying the prescription of shoe lifts, *Ann Swiss Chiropr Assoc* 9:49, 1989.
324. Papaioannou T, Stokes I, Kenwright J: Scoliosis associated with limb-length inequality, *J Bone Joint Surg Am* 64:59, 1982.
325. Illi C, Sandoz R: Spinal equilibrium: further developments of the concepts of Fred Illi, *Ann Swiss Chiropr Assoc* 8:81, 1985.

326. Dieck G, et al: An epidemiologic study of the relationship between postural asymmetry in the teen years and subsequent back and neck pain, *Spine* 10(10):872, 1985.
327. Hansson T, et al: The lumbar lordosis in acute and chronic low-back pain, *Spine* 10(2):154, 1985.
328. Phillips R, et al: Stress x-rays and the low back pain patient, *J Manipulative Physiol Ther* 13(3):127, 1990.
329. Bigos SJ, et al: A prospective study of work perceptions and psychosocial factors affecting the report of back injury, *Spine* 16(1):1, 1991.
330. Battie MC, et al: The role of spinal flexibility in back pain complaints within industry: a prospective study, *Spine* 15(8):768, 1990.
331. Haas M, et al: Lumbar motion trends and correlation with low back pain. I. A roentgenological evaluation of coupled lumbar motion in lateral bending, *J Manipulative Physiol Ther* 15(3):145, 1992.
332. Haas M, Nyiendo J: Lumbar motion trends and correlation with low back pain. II. A roentgenological evaluation of quantitative segmental motion in lateral bending, *J Manipulative Physiol Ther* 15(4):224, 1992.
333. Nansel D, et al: Time course considerations for the effects of unilateral lower cervical adjustments with respect to the amelioration of cervical lateral-flexion passive end-range asymmetry, *J Manipulative Physiol Ther* 13(6):297, 1990.
334. Phillips R, et al: Low back pain: a radiographic enigma, *J Manipulative Physiol Ther* 9(3):183, 1986.
335. Daniels L, Worthingham C: Muscle testing techniques of manual examination, ed 3, Philadelphia, 1972, Saunders.
336. Greenman PE: Principles of manual medicine, Baltimore, 1989, Williams & Wilkins.
337. Vernon H: An assessment of the intra- and inter-reliability of the posturometer, *J Manipulative Physiol Ther* 6(2):57, 1983.
338. Adams AA: Intra- and inter-examiner reliability of plumb line posture analysis measurements using a three dimensional electrogoniometer, *Res Forum* 4(3):60, 1988.
339. D'Angelo MD, Grieve DW: A description of normal relaxed standing postures, *Clin Biomech* 2:140, 1987.
340. Dunk NM, et al: The reliability of quantifying upright standing postures as a baseline diagnostic clinical tool, *J Manipulative Physiol Ther* 27(2):91, 2004.
341. Fedorak C, Ashworth N, Marshall J, et al: Reliability of the visual assessment of cervical and lumbar lordosis: how good are we? *Spine* 28:1857, 2003.
342. Arnold CM, Beatty B, Harrison EL: Investigation of the validity of postural evaluation skills in assessing lumbar lordosis using photographs of clothed subjects, *J Orthop Sports Phys Ther* 52:286, 2000.
343. Cooperstein R: The Derefild pelvic leg check: a kinesiological interpretation, *Chiropr Tech* 3:60, 1991.
344. Mannello DM: Leg length inequality, *J Manipulative Physiol Ther* 15:576, 1992.
345. Haas M, et al: Reactivity of leg alignment to articular pressure testing: evaluation of a diagnostic test using a randomized crossover clinical trial approach, *J Manipulative Physiol Ther* 16:220, 1993.
346. Nguyen HT, et al: Interexaminer reliability of activator methods relative leg-length evaluation in the prone extended position, *J Manipulative Physiol Ther* 22:565, 1999.
347. Thompson JC: Thompson technique reference manual, Elgin, IL, 1984, Thompson Educational Workshops SM and Williams Manufacturing.
348. Fuhr A: Activator methods chiropractic technique seminars, Wilmar, MN, 1985.
349. Lawrence DJ: Chiropractic concepts of the short leg: A critical review, *J Manipulative Physiol Ther* 8:157, 1985.
350. DeBoer KF, et al: Inter- and intra-examiner reliability of leg length differential measurement: A preliminary study, *J Manipulative Physiol Ther* 6(2):61, 1983.
351. Fuhr AW, Osterbauer PJ: Interexaminer reliability of relative leg-length evaluation in the prone, extended position, *J Chiropr Tech* 1(1):13, 1989.
352. Venn EK, Wakefield KA, Thompson PR: A comparative study of leg-length checks, *Eur J Chiropr* 31:68, 1983.
353. Shambaugh MS, Sclafani L, Fanselow D: Reliability of the Derefild-Thomas test for leg length inequality, and use of the test to determine cervical adjusting efficacy, *J Manipulative Physiol Ther* 11(5):396, 1988.
354. Falltrick D, Pierson SD: Precise measurement of functional leg length inequality and changes due to cervical spine rotation in pain-free students, *J Manipulative Physiol Ther* 12(5):364, 1989.
355. Rhudy TR, Burk JM: Inter-examiner reliability of functional leg-length assessment, *Am J Chiropr Med* 3(2):63, 1990.
356. Cooperstein R, et al: Validity of compressive checking in measuring artificial leg-length inequality, *J Manipulative Physiol Ther* 26(9):557, 2003.
357. Haas M, et al: Responsiveness of leg alignment changes associated with articular pressure testing to spinal manipulation: the use of a randomized clinical trial design to evaluate a diagnostic test with a dichotomous outcome, *J Manipulative Physiol Ther* 16:306, 1993.
358. Cocchiarella L, Andersson GBJ, editors: Guides to the evaluation of permanent impairment, Chicago, 2001, American Medical Association.
359. Marras WS, et al: The quantification of low back disorder using motion measures, *Spine* 24:2091, 1999.
360. Wilson L, et al: Interrater reliability of a low back pain classification system, *Spine* 24:248, 1999.
361. Donelson R, Aprill C, Grant W: A prospective study of centralization of lumbar and referred pain: a predictor of symptomatic discs and anular competence, *Spine* 22:115, 1997.
362. Delany PM, Hubka HJ: The diagnostic utility of McKenzie clinical assessment for lower back pain, *J Manipulative Physiol Ther* 22:628, 1999.
363. Kilby J, Stignant M, Roberts A: The reliability of back pain assessment by physiotherapists using a McKenzie algorithm, *Physiotherapy* 76:579, 1990.
364. Fiddle DL, Rothstein JM: Inter-tester reliability of McKenzie's classifications of the type of syndrome present in patients with low back pain, *Spine* 18:1333, 1993.
365. Kilpikoski S, et al: Interexaminer reliability of low back pain assessment using the McKenzie method, *Spine* 27(8):E207, 2002.
366. Clare HA, Adams R, Maher CG: Reliability of detection of lumbar lateral shift, *J Manipulative Physiol Ther* 26(8):476, 2003.
367. Clare HA, Adams R, Maher CG: Reliability of McKenzie classification of patients with cervical or lumbar pain, *J Manipulative Physiol Ther* 28(2):123, 2005.
368. Johnston W, et al: Interexaminer study of palpation in detecting location of spinal segmental dysfunction, *J Am Osteopath Assoc* 82(11):839, 1983.
369. Gill K, et al: Repeatability of four clinical methods for assessment of lumbar spinal motion, *Spine* 13:50, 1988.
370. Merritt IF, et al: Measurement of trunk flexibility in normal subjects: reproducibility of three clinical methods, *Mayo Clin Proc* 61:192, 1986.
371. Liebenson C, Phillips RB: The reliability of range of motion measurements for lumbar spine flexion: a review, *Chiropr Tech* 1:69, 1989.
372. Nitschke JE, et al: Reliability of the American Medical Association guides' model for measuring spinal range of motion: Its implication for whole person impairment rating, *Spine* 24(3):262, 1999.
373. Kelly J, et al: Quantification of lumbar function. V. Reliability of range of motion measures in the sagittal plane and in vivo torso rotation measurement technique, *Spine* 11:31, 1986.
374. Newton M, Waddell G: Reliability and validity of clinical measurement of the lumbar spine in patients with chronic low back pain, *Physiotherapy* 77:796, 1991.

375. Petra M, et al: Lumbar range of motion: reliability and validity of the inclinometer technique in the clinical measurement of trunk flexibility, *Spine* 11:1332, 1996.
376. Johnston W, et al: Palpatory findings in the cervicothoracic region: variations in normotensive and hypertensive subjects—a preliminary report, *J Am Osteopath Assoc* 79(5):300, 1980.
377. Deboer KF, et al: Reliability study of detection of somatic dysfunctions in the cervical spine, *J Manipulative Physiol Ther* 8(1):9, 1985.
378. Viikari-Juntura E: Interexaminer reliability of observations in physical examinations of the neck, *Phys Ther* 67:1526, 1987.
379. Boline PD, et al: Interexaminer reliability of eight evaluative dimensions of lumbar segmental abnormality. II, *J Manipulative Physiol Ther* 16:363, 1993.
380. Paydar D, Thiel H, Gemmell H: Intra- and inter-examiner reliability of certain pelvic palpatory procedures and the sitting flexion test for sacroiliac mobility and dysfunction, *J Neuromusculoskeletal System* 2:65, 1994.
381. Nilsson N: Measuring cervical muscle tenderness: a study of reliability, *J Manipulative Physiol Ther* 18:88, 1995.
382. Streder L, Lumdin M, Nell K: Interexaminer reliability in physical examination of the neck, *J Manipulative Physiol Ther* 20:526, 1997.
383. Streder L, et al: Interexaminer reliability in physical examination of patients with low back pain, *Spine* 22:814, 1997.
384. Huijbregts PA: Spinal motion palpation: a review of reliability studies, *J Manual Manipulative Ther* 10:24–39, 2002.
385. Schneider M, Erhard R, Brach J, et al: Spinal palpation for lumbar segmental mobility and pain provocation: an interexaminer reliability study, *J Manipulative Physiol Ther* 31:465, 2008.
386. Van Trijffel E, et al: Perceptions and use of passive intervertebral motion assessment of the spine: A survey among Dutch physiotherapists specializing in manual therapy, *Man Ther* 14(3):243–251, 2008.
387. Jull B, Bullock M: A motion profile of the lumbar spine in an aging population assessed by manual examination, *Physiotherapy* 3:70, 1987.
388. Jull G, Bogduk N, Marsland A: The accuracy of manual diagnosis for cervical zygapophysial joint pain syndromes, *Med J Aust* 148:233, 1988.
389. Leboeuf C, et al: Chiropractic examination procedures: a reliability and consistency study, *J Aust Chiropr Assoc* 19(3):101, 1989.
390. Haneline MT, et al: Spinal motion palpation: A comparison of studies that assessed intersegmental end feel vs excursion, *J Manipulative Physiol Ther* 31:616, 2008.
391. Wiles MR: Reproducibility and interexaminer correlation of motion palpation findings of the sacroiliac joints, *J Can Chiropr Assoc* 24:59, 1980.
392. Gonnella C, Paris SV, Kutner M: Reliability in evaluating passive intervertebral motion, *Phys Ther* 62(4):436, 1982.
393. Matyas T, Bach T: The reliability of selected techniques in clinical arthrometrics, *Aust J Physiother* 31:175, 1985.
394. Potter NA, Rothstein JM: Intertester reliability for selected clinical tests of the sacroiliac joint, *Phys Ther* 65:1671, 1995.
395. Mior SA, et al: Intra- and inter-examiner reliability of motion palpation in the cervical spine, *J Can Chiropr Assoc* 29:195, 1985.
396. Bergstrom E, Courtis G: An inter- and intra-examiner reliability study of motion palpation of the lumbar spine in lateral flexion in the seated position, *Eur J Chiropr* 34:121, 1986.
397. Love RM, Brodeur RR: Inter- and intra-examiner reliability of motion palpation for the thoracolumbar spine, *J Manipulative Physiol Ther* 10:1, 1987.
398. Carmichael JP: Inter- and intra-examiner reliability of palpation for sacroiliac joint dysfunction, *J Manipulative Physiol Ther* 10:164, 1987.
399. Rhudy TR, Sandefur MR, Burk JM: Interexaminer/intertechnique reliability in spinal subluxation assessment: a multifactorial approach, *Am J Chiropr Med* 1:111, 1988.
400. Nansel DD, et al: Interexaminer concordance in detecting joint-play asymmetries in the cervical spines of otherwise asymptomatic subjects, *J Manipulative Physiol Ther* 12(6):428, 1989.
401. Herzog W, et al: Reliability of motion palpation to detect sacroiliac joint fixations, *J Manipulative Physiol Ther* 12:86, 1989.
402. Mootz RD, Keating JC, Kontz HP: Intra- and inter-examiner reliability of passive motion palpation of the lumbar spine, *J Manipulative Physiol Ther* 12(6):440, 1989.
403. Mior SA, McGregor M, Schut AB: The role of experience in clinical accuracy, *J Manipulative Physiol Ther* 13:68, 1990.
404. Haas M, et al: Reliability of manual end play palpation of the thoracic spine, *Chiropr Tech* 7:120, 1995.
405. Meijne W, et al: Intraexaminer and interexaminer reliability of the Gillet test, *J Manipulative Physiol Ther* 22:4, 1999.
406. Mior SA, McGregor M, Schut B: The role of experiment in clinical accuracy, *J Manipulative Physiol Ther* 13:68, 1990.
407. Mior SA, et al: Intra- and interexaminer reliability of motion palpation in the cervical spine, *J Can Chiropr Assoc* 29:195–198, 1985.
408. Nansel RD, et al: Intra- and interobserver reliability of passive motion palpation of the lumbar spine, *J Manipulative Physiol Ther* 12:440, 1989.
409. Nansel DD, Peneff AL, Jansen RD, et al: Interexaminer agreement in detecting cervical joint play asymmetries in the spine of otherwise asymptomatic subjects, *J Manipulative Physiol Ther* 12:428, 1989.
410. Lewit K, Liebenson C: Palpation-problems and implications, *J Manipulative Physiol Ther* 16:586, 1993.
411. Robinson R, et al: Reliability and validity of a palpation technique for identifying the spinous processes of C7 and L5, *Man Ther* 14(4):409–414, 2009.
412. Ross JK, Bereznick DE: Determining cavitation location during lumbar and thoracic spinal manipulation: is spinal manipulation accurate and specific, *Spine* 29(13):1451, 2004.
413. Bereznick DE, Ross KJ, McGill S: The frictional properties at the thoracic skin–fascia interface: implications in spine manipulation, *Clin Biomech* 17:297, 2002.
414. Harvey D, Byfield D: Preliminary studies with a mechanical model for the evaluation of spinal motion palpation, *Clin Biomech* 6:79, 1991.
415. Hides JA, et al: Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain, *Spine* 19:165, 1994.
416. Schwarzer AC, et al: The false positive rate of uncontrolled diagnostic blocks of the lumbar zygapophyseal joints, *Pain* 58:195, 1994.
417. Maher C, Latimer J: Pain or resistance: the manual therapists' dilemma, *Aust J Physiother* 38:257, 1992.
418. Jull G, Treleaven J, Versace G: Manual examination: is pain provocation a major cue for spinal dysfunction? *Aust J Physiother* 40:159, 1994.
419. Humphreys B, Delahaye M, Peterson CK: An investigation into the validity of cervical spine motion palpation using subjects with congenital block vertebrae as a “gold standard,” *BMC Musculoskelet Disord* 5:19, 2004.
420. Leboeuf-Yde C, et al: Motion palpation findings and self-reported low back pain in a population-based study sample, *J Manipulative Physiol Ther* 25:80, 2002.
421. Fernández-de-las-Peñas C, Downey C, Miangolarra-Page JC: Validity of the lateral gliding test as tool for the diagnosis of intervertebral joint dysfunction in the lower cervical spine, *J Manipulative Physiol Ther* 28:610, 2005.
422. Haas M, et al: Efficacy of cervical endplay assessment as an indicator for spinal manipulation, *Spine* 28:1091, 2003.
423. Haas M, et al: Dose response for chiropractic care of chronic cervicogenic headache and associated neck pain: a randomized pilot study, *J Manipulative Physiol Ther* 27(9):547, 2004.

424. Troyanovich SJ, Harrison DD, Harrison DE: Motion palpation: it's time to accept the evidence, *J Manipulative Physiol Ther* 21:568, 1998.
425. Saal JS: General principles of diagnostic testing as related to painful lumbar spine disorders: a critical appraisal of current diagnostic techniques, *Spine* 27(22):2538, 2002.
426. Lok CE, Morgan CD, Ranganathan N: The accuracy and interobserver agreement in detecting the gallop sounds by cardiac auscultation, *Chest* 114:1283, 1998.
427. Riddle DL, Freburger JK: North American Orthopaedic Rehabilitation Research Network: evaluation of the presence of sacroiliac joint region dysfunction using a combination of tests: a multicenter intertester reliability study, *Phys Ther* 82:772, 2002.
428. Dreyfuss P, et al: Positive sacroiliac screening tests in asymptomatic adults, *Spine* 19:1138–1143, 1994.
429. Fortin JD, et al: Sacroiliac joint: pain referral maps upon applying a new injection/arthrography technique, part II: Clinical evaluation, *Spine* 19:1483, 1994.
430. Stoddard A: *Manual of osteopathic technique*, London, 1969, Hutchinson Medical Publishing Ltd.
431. Greenman P, Riddle DL, Freburger JK: North American Orthopaedic Rehabilitation Research Network: evaluation of the presence of sacroiliac joint region dysfunction using a combination of tests: a multicenter intertester reliability study, *Phys Ther* 82:772, 2002.
432. Greenman PH: *Principles of manual medicine*, Baltimore, 1989, Lippincott Williams & Wilkins.
433. Haldeman S: *Modern developments in the principles and practice of chiropractic*, New York, NY, 1980, Appleton-Century-Crofts.
434. Lee DG: *The pelvic girdle*, ed 3, Edinburgh, 2004, Elsevier.
435. Laslett M, Williams M: The reliability of selected pain provocation tests for sacroiliac joint pathology, *Spine* 19(11):1243, 1994.
436. Van der Wurff P, Hagmeijer RHM, Meyne W: Clinical tests of the sacroiliac joint. A systematic methodological review. Part 1: reliability, *Manual Ther* 5(1):30, 2000.
437. Hungerford BA, et al: Evaluation of the ability of physical therapists to palpate intrapelvic motion with the Stork Test on the support side, *Phys Ther* 87:879, 2007.
438. Broadhurst NA, Bond MJ: Pain provocation tests for the assessment of sacroiliac joint dysfunction, *J Spinal Disord* 11:341, 1998.
439. Laslett M: Pain provocation sacroiliac joint tests: reliability and prevalence. In Vleeming A, et al, editors: *Movement, stability and low back pain: the essential role of the pelvis*, ed 1, New York, 1997, Churchill Livingstone.
440. Laslett M, et al: Diagnosis of sacroiliac joint pain: validity of individual provocation tests and composites of tests, *Man Ther* 10:207, 2005.
441. Arab AM, et al: Inter- and intra-examiner reliability of single and composites of selected motion palpation and pain provocation tests for sacroiliac joint, *Man Ther* 14(2):2009.
442. Laslett M: Evidence-based diagnosis and treatment of the painful sacroiliac joint, *J Manual Manipulative Ther* 16(3):142, 2008.
443. Boline PD, et al: Interexaminer reliability of eight evaluative dimensions of lumbar segmental abnormality: part 2, *J Manipulative Physiol Ther* 16:363, 1993.
444. Hubka MJ, Phelan SP: Interexaminer reliability of palpation for cervical spine tenderness, *J Manipulative Physiol Ther* 17:591, 1994.
445. Tunks E, et al: The reliability of examination for tenderness in patients with myofascial pain, chronic fibromyalgia and controls, *J Rheumatol* 22:944, 1995.
446. Travell J, Rinzler SH: The myofascial genesis of pain, *Postgrad Med* 11:425, 1952.
447. Kuchera ML, et al: Musculoskeletal examination for somatic dysfunction. In Ward RC, editor: *Foundations for osteopathic medicine*, Baltimore, 1997, William & Wilkins, pp 486–500.
448. Grieve GP: *Pathological changes: general*. In *Common vertebral joint problems*, ed 2, Edinburgh, UK, 1988, Churchill Livingstone.
449. Christensen HW, et al: Palpation of the upper thoracic spine: an observer reliability study, *J Manipulative Physiol Ther* 25:285, 2002.
450. Brodeur R: The audible release associated with joint manipulation, *J Manipulative Physiol Ther* 18:155, 1995.
451. Haas M, Peterson D: A roentgenological evaluation of the relationship between segmental motion and mal-alignment in lateral bending, *J Manipulative Physiol Ther* 15(6):350, 1992.
452. Hubka MJ: Palpation for spinal tenderness: a reliable and accurate method for identifying the target of spinal manipulation, *Chiropr Tech* 6:5, 1994.
453. Phillips DR, Twomey LT: A comparison of manual diagnosis with a diagnosis established by a uni-level lumbar spinal block procedure, *Man Ther* 2:82, 1996.
454. Kendall HO, Kendall FP, Wadsworth GE: *Muscles testing and function*, ed 2, Baltimore, 1971, Williams & Wilkins.
455. Walther DS: *Applied kinesiology: The advanced approach in chiropractic systems*, DC, 1976, Pueblo, CO.
456. Haas M, et al: The reliability of muscle testing response to a provocative vertebral challenge, *Chiropr Tech* 5:95, 1993.
457. Haas M, et al: Muscle testing response to provocative vertebral challenge and spinal manipulation: a randomized controlled trial of construct validity, *J Manipulative Physiol Ther* 17:141, 1994.
458. Evans RC: *Illustrated orthopedic physical assessment*, ed 3, St. Louis, 2009, Mosby.
459. Hildebrandt R: Chiropractic spinography and postural roentgenology. I. History of development, *J Manipulative Physiol Ther* 3(2):87, 1980.
460. Howe J: Some considerations in spinal x-ray interpretations, *J Clin Chiropr Arch* 4:75, 1971.
461. Sherman R: Chiropractic x-ray rationale, *J Can Chiropr Assoc* 30:33, 1986.
462. Howe JW: Facts and fallacies, myths and misconceptions in spinography, *J Clin Chiropr Arch* 3:34, 1972.
463. Plaughner G, Hendricks A: The inter- and intra-examiner reliability of the Gonstead pelvic marking system, *J Manipulative Physiol Ther* 14(9):503, 1991.
464. Logan HB: *Textbook of Logan basic methods*, St Louis, 1950, Logan Chiropractic College.
465. Gregory RR: *Manual for upper cervical x-ray analysis*, Monroe, MI, 1971, National Upper Cervical Chiropractic Association.
466. Blair WG: Blair Clinic of Lubbock, TX, *Dig Chiropr Econ* 14(1):10, 1971.
467. Weinert DJ: Influence of axial rotation on chiropractic pelvic radiographic analysis, *J Manipulative Physiol Ther* 30(1):78, 2007.
468. Haas M, Taylor JAM, Gillette RG: The routine use of radiographic spinal displacement analysis: a dissent, *J Manipulative Physiol Ther* 22(4):254, 1999.
469. Sandoz R: Some reflections on subluxations and adjustments, *Ann Swiss Chiropr Assoc* 9:7, 1989.
470. Bussi eres AE, Peterson C, Taylor JA: Diagnostic imaging practice guidelines for musculoskeletal complaints in adults—an evidence-based approach: Introduction, *J Manipulative Physiol Ther* 30(9):617, 2007.
471. Bussi eres AE, Taylor JA, Peterson C: Diagnostic imaging practice guidelines for musculoskeletal complaints in adults—an evidence-based approach. Part 1: Lower extremity disorders, *J Manipulative Physiol Ther* 30(9):684, 2007.
472. Bussi eres AE, Peterson C, Taylor JA: Diagnostic imaging guideline for musculoskeletal complaints in adults—an evidence-based approach—Part 2: upper extremity disorders, *J Manipulative Physiol Ther* 31(1):2, 2008.
473. Bussi eres AE, Taylor JA, Peterson C: Diagnostic imaging practice guidelines for musculoskeletal complaints in adults—an evidence-based approach—Part 3: spinal disorders, *J Manipulative Physiol Ther* 31(1):33, 2008.

474. Ammendolia C, et al: Adherence to radiography guidelines for low back pain: a survey of chiropractic schools worldwide, *J Manipulative Physiol Ther* 31(6):412, 2008.
475. Plaugher G: Textbook of clinical chiropractic, Baltimore, MD, 1993, Williams & Wilkins.
476. Deyo RA, Diehl AK: Lumbar spine films in primary care: Current use and effects of selective ordering criteria, *J Gen Intern Med* 1:20, 1986.
477. Taylor JAM: The role of radiograph in evaluating subluxation. In Gatterman MI, editor: Foundations of chiropractic subluxation, St Louis, 1995, Mosby.
478. Hildebrandt RW: Full spine radiography: a matter of clinical justification, *J Chiropr* 23(8):56, 1986.
479. Sellers T: Diagnostic or non-diagnostic, *J Am Chiropr Assoc* 22(8):71, 1988.
480. Peterson C, Gatterman MI, Wei T: Chiropractic radiology. In Gatterman MI, editor: Chiropractic management of spine related disorders, Baltimore, 1990, Williams & Wilkins.
481. Howe JW: The chiropractic concept of subluxation and its roentgenological manifestations, *J Clin Chiropr Arch Sept/Oct*: 64-70, 1973.
482. Harrison DE, Harrison DD, Troyanovich SJ: Reliability of spinal displacement analysis on plain x-rays: a review of commonly accepted facts and fallacies with implications for chiropractic education and technique, *J Manipulative Physiol Ther* 21:252, 1998.
483. Troyanovich SJ, et al: Intra- and inter-examiner reliability of chiropractic biophysics lateral lumbar radiographic menstruation procedure, *J Manipulative Physiol Ther* 18:519, 1995.
484. Jackson BL, et al: Chiropractic biophysics lateral cervical film analysis reliability, *J Manipulative Physiol Ther* 16:384, 1993.
485. Sigler DC, Howe JW: Inter- and intra-examiner reliability of the upper cervical x-ray marking system, *J Manipulative Physiol Ther* 8:75, 1985.
486. Schram SB, Hosek R, Silverman HL: Spinographic positioning errors in Gonstead pelvic x-ray analysis, *J Manipulative Physiol Ther* 4(4):179, 1981.
487. Schram SB, Hosek RS: Error limitations in x-ray kinematics of the spine, *J Manipulative Physiol Ther* 5(1):5, 1982.
488. Plaugher G, Cremata E, Phillips RB: A retrospective consecutive case analysis of pretreatment and comparative static radiological parameters following chiropractic adjustments, *J Manipulative Physiol Ther* 13(9):498, 1990.
489. Phillips RB: An evaluation of the graphic analysis of the pelvis on the A-P full spine radiograph, *J Am Chiropr Assoc* 9:S139, 1975.
490. Frymoyer JW, et al: A comparative analysis of the interpretations of lumbar spinal radiographs by chiropractors and medical doctors, *Spine* 11:1020, 1986.
491. Peterson CK, Haas M, Harger BL: A radiographic study of sacral base, sacrovertebral, and lumbosacral disc angles in persons with and without defects in the pars interarticularis, *J Manipulative Physiol Ther* 13:491, 1990.
492. Howe JW: The role of x-ray findings in structural diagnosis. In The research status of spinal manipulative therapy, NINCDS Monograph No 15, DHEW Pub No 76-988, Washington, DC, 1975, US Government Printing Office.
493. Banks SD: The use of spinographic parameters in the differential diagnosis of lumbar facet and disc syndromes, *J Manipulative Physiol Ther* 6:113, 1983.
494. Reinert OC: An analytical survey of structural aberrations observed in static radiographic examinations among acute low back cases, *J Manipulative Physiol Ther* 11:24, 1988.
495. Grostic JD, DeBoer KF: Roentgenographic measurement of atlas laterality and rotation: a retrospective pre- and post-manipulation study, *J Manipulative Physiol Ther* 5:63, 1982.
496. Anderson RT: A radiographic test of upper cervical chiropractic theory, *J Manipulative Physiol Ther* 4:129, 1981.
497. Jackson BL, et al: Inter- and intra-examiner reliability of the upper cervical x-ray marking system: a second look, *J Manipulative Physiol Ther* 10:157, 1987.
498. Jackson BL, Barker WF, Gamble AG: Reliability of the upper cervical x-ray marking system: a replication study, *Chiropractic Res J* 1(1):10, 1988.
499. Keating JC, Boline PD: The precision and reliability of an upper cervical marking system: lessons from the literature, *Chiropractic* 1:43, 1988.
500. Owens EF: Line drawing analysis of static cervical x-rays used in chiropractic, *J Manipulative Physiol Ther* 15:442, 1992.
501. Plaugher G, et al: The reliability of patient positioning for evaluating static radiographic parameters of human pelvis, *J Manipulative Physiol Ther* 16:517, 1993.
502. Burk JM, Thomas RR, Ratliff CR: Intra- and inter-examiner agreement of the Gonstead line marking method, *Am J Chiropr Med* 3:114, 1990.
503. Yi-Kai L, Yun-Kun Z, Shi-Zhen Z: Diagnostic value on signs of subluxation of cervical vertebrae with radiological examination, *J Manipulative Physiol Ther* 21:617, 1998.
504. Hass M, Peterson D: A roentgenological evaluation of the relationship between segmental motion and segmental malalignment in lateral bending, *J Manipulative Physiol Ther* 15:350, 1992.
505. Sandoz R: Technique and interpretation of functional radiography of the lumbar spine, *Ann Swiss Chiropr Assoc* 3:66, 1965.
506. Cassidy JD: Roentgenological examination of the functional mechanics of the lumbar spine in lateral flexion, *J Can Chiropr Assoc* 20(2):13, 1976.
507. Grice A: Radiographic, biomechanical and clinical factors in lumbar lateral flexion. I, *J Manipulative Physiol Ther* 2(1):26, 1979.
508. Vernon H: Static and dynamic roentgenography in the diagnosis of degenerative disc disease: a review and comparative assessment, *J Manipulative Physiol Ther* 5(4):163, 1982.
509. Hviid H: Functional radiography of the cervical spine, *Ann Swiss Chiropr Assoc* 3:37, 1963.
510. Prantl K: X-ray examination and functional analysis of the cervical spine, *Manual Med* 2:5, 1985.
511. Penning L: Normal movements of the cervical spine, *Am J Roentgenol* 130:317, 1978.
512. Pennal GF, et al: Motion studies of the lumbar spine, *J Bone Joint Surg Br* 54(3):442, 1972.
513. Pitkanen M, et al: Limited usefulness of traction compression films in the radiographic diagnosis of lumbar instability, *Spine* 22:193, 1997.
514. Van Akerveeken PF, O'Brien JP, Park WM: Experimentally induced hypermobility in the lumbar spine, *Spine* 4:236, 1979.
515. Muggleton JM, Allen R: Insights into the measurement of vertebral translation in the sagittal plane, *Med Eng Phys* 20(1):21, 1998.
516. Dvorak J, et al: Functional radiographic diagnosis of the lumbar spine: flexion-extension and lateral bending, *Spine* 16(5):562, 1991.
517. Dvorak J, et al: Clinical validation of functional flexion-extension roentgenograms of the lumbar spine, *Spine* 16(8):943, 1991.
518. Dvorak J, et al: Clinical validation of functional flexion-extension radiographs of the cervical spine, *Spine* 18(1):120, 1993.
519. Hasner E, Schalimtzek M, Snorrason E: Roentgenological examination of the function of the lumbar spine, *Acta Radiol* 37:141, 1952.
520. Fielding JW: Cinerontgenography (CR) of the normal cervical spine, *J Bone Joint Surg Br* 39A:1280, 1957.
521. Howe JW: Cineradiographic evaluation of normal and abnormal cervical spinal function, *J Clin Chiropr* 1:42, 1976.
522. Cholewicki J, et al: Method for measuring vertebral kinematics from videofluoroscopy, *Clin Biomech* 6:73, 1991.
523. Breen A: Integrated spinal motion: a study of two cases, *J Can Chiropr Assoc* 35:25, 1991.

524. Breen A, Allen R, Morris A: An image processing method for spine kinematics: preliminary studies, *Clin Biomech* 3:5, 1988.
525. Humphreys K, Breen A, Saxton D: Incremental lumbar spine motion in the coronal plane: an observer variation study using digital videofluoroscopy, *Eur J Chiropr* 38:56, 1990.
526. Antos JC, et al: Interrater reliability of fluoroscopic detection of fixation in the mid-cervical spine, *J Chiropr Tech* 2(2):53, 1990.
527. Gatterman B: Protocol for the use of spinal videofluoroscopy, ACCR Guidelines 1990.
528. Boden SD, et al: Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects, *J Bone Joint Surg Am* 72(3):403, 1990.
529. Fischer AA: Pressure tolerance over muscles and bones in normal subjects, *Arch Phys Med Rehabil* 67:406, 1986.
530. Fischer AA: Pressure threshold meter: its use for quantification of tender spots, *Arch Phys Med Rehabil* 67:836, 1986.
531. Fischer AA: Pressure algometry over normal muscles: standard values, validity and reproducibility of pressure threshold, *Pain* 30:115, 1987.
532. Reeves JL, Jaeger B, Graff-Radford SB: Reliability of the pressure algometer as a measure of myofascial trigger point sensitivity, *Pain* 24:313, 1986.
533. Fischer AA: Documentation of myofascial trigger points, *Arch Phys Med Rehabil* 69:286, 1988.
534. Takala E-P: Pressure pain threshold on upper trapezius and levator scapulae muscles, *Scand J Rehabil Med* 22:63, 1990.
535. Ohrback R, Gale EN: Pressure pain thresholds, clinical assessment and differential diagnosis: reliability and validity in patients with myogenic pain, *Pain* 39:157, 1989.
536. Fischer AA: Application of pressure algometry in manual medicine, *Manual Med* 5:145, 1990.
537. List T, Helkimo M, Falk G: Reliability and validity of a pressure threshold meter in recoding tenderness in the masseter muscle and the anterior temporalis muscle, *Cranio* 7:223, 1989.
538. Langemark M, et al: Pressure pain thresholds and thermal nociceptive thresholds in chronic tension-type headache, *Pain* 38:203, 1989.
539. Gerez-Simon EM, et al: Measurement of pain threshold in patients with rheumatoid arthritis, osteoarthritis, ankylosing spondylitis, and health controls, *Clin Rheumatol* 8:467, 1989.
540. Vernon HT, et al: Pressure pain threshold evaluation of the effect of spinal manipulation in the treatment of chronic neck pain: a pilot study, *J Manipulative Physiol Ther* 13:13, 1990.
541. Hsieh J, Hong C: Effect of chiropractic manipulation on the pain threshold of myofascial trigger point: a pilot study. In *Proceedings of the 1990 International Conference on Spinal Manipulation*, Washington, DC, May 11-12, 1990.
542. Uemtsu S: Symmetry of skin temperature comparing one side of the body to the other, *Thermology* 1:4, 1985.
543. Silverstein EB, Bahr GJM, Katan B: Thermographically measured normal skin temperature asymmetry in the human male, *Cancer* 36:1506, 1975.
544. Christiansen J: Thermographic anatomy and physiology. In Christiansen J, Gerow G, editors: *Thermography*, Baltimore, 1990, Williams & Wilkins.
545. Meeker WC, Gahlinger PM: Neuromusculoskeletal thermography: A valuable diagnostic tool? *J Manipulative Physiol Ther* 9:257, 1986.
546. Swenson RS: Clinical investigations of reflex function. In Haldeman S, editor: *Principles and practice of chiropractic*, ed 2, Norwalk, CT, 1992, Appleton & Lange.
547. Chusid JG: *Correlative neuroanatomy and functional neurology*, ed 17, Los Altos, CA, 1979, Lange Medical Publications.
548. Peterson AR, editor: *Segmental neuropathy*, Toronto, Canadian Memorial Chiropractic College.
549. Jones CH: Physical aspects of thermography in relation to clinical techniques, *Bibl Radiol* 6:1, 1974.
550. Judavich B, Bates W: *Pain syndromes: diagnosis and treatment*, ed 4, Philadelphia, 1954, FA Davis.
551. Plaugher G: Skin temperature assessment for neuromusculoskeletal abnormalities of the spinal column, *J Manipulative Physiol Ther* 15:365, 1992.
552. Triano JJ, Skogsbergh DR, Kowalski MH: The use of instrumentation and laboratory examination procedures by the chiropractor. In Haldeman S, editor: *Principles and practice of chiropractic*, ed 2, Norwalk, CT, 1992, Appleton & Lange.
553. Nansel D, Jansen R: Concordance between galvanic skin response and spinal palpation findings in pain free males, *J Manipulative Physiol Ther* 11:2267, 1988.
554. Plaugher G, et al: The interexaminer reliability of a galvanic skin response instrument, *J Manipulative Physiol Ther* 16:453, 1993.
555. Riley LH, Richter CP: Uses of the electrical skin resistance method in the study of patients with neck and upper extremity pain, *Johns Hopkins Med J* 137:69, 1975.
556. Yamagata S, et al: A diagnostic reevaluation of electrical skin resistance, skin temperature and deeper tenderness in patients with abdominal pain, *Tohoku J Exp Med* 118:183, 1976.
557. Turker KS: Electromyography: some methodological problems and issues, *Physical Ther* 73(10):698, 1993.
558. Kent C: Surface electromyography in the assessment of changes in paraspinal muscle activity associated with vertebral subluxation: a review, *J Vertebral Subluxation Res* 1(3):15, 1997.
559. Specter B: Surface electromyography as a model for the development of standardized procedures and reliability testing, *J Manipulative Physiol Ther* 2(4):214, 1979.
560. Komi P, Buskirk E: Reproducibility of electromyographic measurements with inserted wire electrodes and surface electrodes, *Electromyography* 10:357, 1970.
561. Giroux B, Lamontague M: Comparisons between the surface electrode and intramuscular wire electrodes in isometric and dynamic conditions, *Electromyogr Clin Neurophysiol* 30:397, 1990.
562. Anderson G, Johnson B, Ortengren R: Myoelectric activity in individual lumbar erector spinae muscles in sitting: a study with surface and wire electrodes, *J Rehabil Med* 3:91, 1974.
563. Thompson J, Erickson R, Offord K: EMG muscle scanning: stability of hand-held electrodes, *Biofeedback Self Regul* 14(1):55, 1989.
564. Cram JR, Lloyd J, Cahn TS: The reliability of EMG muscle scanning, *Int J Psychosom* 41:41, 1994.
565. Sihvonen T, Partanen J, Hanninen O: Averaged (RMS) surface EMG in testing back function, *Electromyogr Clin Neurophysiol* 28:335, 1988.
566. Cram J: Surface EMG recordings and pain related disorders: a diagnostic framework, *Biofeedback Self Regul* 13(2):123, 1988.
567. Ng LKY: New approaches to treatment of chronic pain: Review of multidisciplinary pain centers, National Institute of Drug Abuse Research Monograph Series No 36, Rockville, MD, 1983, Department of Health and Human Services.
568. Cram J, Lloyd J, Cahn T: The reliability of EMG muscle scanning, *Int J Psychosom* 37:68, 1990.
569. Lehman GJ: Clinical considerations in the use of surface electromyography: three experimental studies, *J Manipulative Physiol Ther* 25:293, 2002.
570. Ritvanen T, et al: Dynamic surface electromyographic responses in chronic low back pain treated by traditional bone setting and conventional physical therapy, *J Manipulative Physiol Ther* 30:31, 2007.
571. Myerowitz M: Scanning paraspinal surface EMG: A method for corroborated post treatment spinal and related neuromusculoskeletal symptom improvement, *J Occup Rehabil* 4(3):171, 1994.
572. Meyer JJ: The validity of thoracolumbar paraspinal scanning EMG as a diagnostic test: an examination of the current literature, *J Manipulative Physiol Ther* 17(8):539, 1994.

573. Meeker W, Matheson D, Wong A: Lack of evidence for a relationship between low back pain and asymmetrical muscle activity using scanning electromyography: analysis of pilot data. In *Proceedings of the International Conferences on Spinal Manipulation*, Washington, DC, 1990.
574. Triano JJ: Surface electrode EMG/lumbar spine—static paraspinal EMG scanning: clinical utility and validity issues. In *Proceedings of the Consortium for Chiropractic Research*, 1993.

CHAPTER 4

1. Lamm LC, Wegner E, Collard D: Chiropractic scope of practice: what the law allows. II, *J Manipulative Physiol Ther* 18:16, 1995.
2. Hawk C, et al: Use of complementary healthcare practices among chiropractors in the United States: a survey, *Altern Ther Health Med* 5(1):56, 1999.
3. Department of Statistics: 1985 survey, *J Am Chiro Assoc* 23(2):68, 1986.
4. Commission of Inquiry into Chiropractic: Chiropractic in New Zealand, reprinted by Davenport, IA, 1979, Palmer College of Chiropractic.
5. Vear HJ: Standards of chiropractic practice, *J Manipulative Physiol Ther* 8(1):33, 1985.
6. Nyiendo J, Haldeman S: A prospective study of 2,000 patients attending a chiropractic college teaching clinic, *Med Care* 25(6):516, 1987.
7. Kelner M, Hall O, Coulter I: Chiropractors: do they help?, Toronto, 1980, Fitzhenry & Whiteside.
8. Shekelle PG, Brook RH: A community-based study of the use of chiropractic services, *Am J Public Health* 81(4):439, 1991.
9. ACA Council on Technic: Chiropractic terminology: a report, *J Am Chiro Assoc* 25(10):46, 1988.
10. Maigne R: Personal method: The rule of no pain and free movement. In *Orthopedic medicine: a new approach to vertebral manipulations*, Springfield, IL, 1972, Bannerstone House.
11. Wood KW: Acute torticollis: chiropractic therapy and management, *Chiro Tech* 3:3, 1991.
12. Hammond B: Torticollis, *Eur J Chiro* 31(3):162, 1983.
13. Bergmann TF: The chiropractic spinal examination. In Ferezy JS, editor: *The chiropractic neurological examination*, Rockville, MD, 1992, Aspen.
14. Vear HJ: Introduction. In Vear HJ, editor: *Chiropractic standards of practice and quality of care*, Rockville, MD, 1992, Aspen.
15. Bartol KM: A model for the categorization of chiropractic treatment procedures, *J Chiro Tech* 3(2):78, 1991.
16. Gatterman MI, Hansen DT: Development of chiropractic nomenclature through consensus, *J Manipulative Physiol Ther* 17(5):302, 1994.
17. Palmer DD: *Textbook of the science, art and philosophy of chiropractic*, Portland, OR, 1910, Portland Printing House.
18. Stephenson RW: *The art of chiropractic*, Davenport, IA, 1947, Palmer School of Chiropractic.
19. Gatterman MI, Hansen DT: Development of chiropractic nomenclature through consensus, *J Manipulative Physiol Ther* 17(5):302, 1994.
20. Levine M: *The structural approach to chiropractic*, New York, 1964, Comet Press.
21. Haldeman S: Spinal manipulative therapy and sports medicine, *Clin Sports Med* 5(2):277, 1986.
22. Haldeman S: Spinal manipulative therapy: A status report, *Clin Orthop Relat Res* 179:62, 1993.
23. Sandoz R: Some physical mechanisms and affects of spinal adjustments, *Ann Swiss Chiropr Assoc* 6:91, 1976.
24. Senstad O, Leboeuf-Yde C, Borchgrevink C: Frequency and characteristics of side effects of spinal manipulative therapy, *Spine* 15(4):435, 1997.
25. Herzog W, Kawchuk GN, Conway PJ: Relationship between preload and peak forces during spinal manipulative treatments, *J Manipulative Physiol Ther* 1(2):52, 1993.
26. Bartol KM: Algorithm for the categorization of chiropractic technic procedures, *J Chiro Tech* 4(1):8, 1992.
27. Bartol KM: The categorization of chiropractic procedures. In *Proceedings of the sixth annual conference on research and education*, Monterey, CA, 1991.
28. Nwuga VC: *Manipulation of the spine*, Baltimore, 1976, Williams & Wilkins.
29. Cyriax J: *Textbook of orthopedic medicine*, ed 8, vol 1, London, 1982, Bailliere Tindall.
30. Grieve GP: *Common vertebral joint problems*, ed 2, New York, 1988, Churchill Livingstone.
31. Nyberg R: Role of physical therapists in spinal manipulation. In Basmajian JV, editor: *Manipulation, traction and massage*, ed 3, Baltimore, 1985, Williams & Wilkins.
32. Grice A, Vernon H: Basic principles in the performance of chiropractic adjusting: historical review, classification, and objectives. In Haldeman S, editor: *Principles and practice of chiropractic*, ed 2, San Mateo, CA, 1992, Appleton & Lange.
33. Hass M, et al: Efficacy of cervical endplay assessment as an indicator for spinal manipulation, *Spine* 28:1091, 2003.
34. Bereznick D, Kim Ross KJ, McGill S: The frictional properties at the thoracic skin–fascia interface: implications in spine manipulation, *Clin Biomech* 17:297, 2002.
35. Lawrence D, et al: Chiropractic management of low back and low back-related complaints: a literature synthesis, *J Manipulative Physiol Ther* 31:659, 2008.
36. Bronforth G, et al: Efficacy of spinal manipulative therapy for low back and neck pain: a systematic review and best evidence synthesis, *Spine J* 4(3):335, 2004.
37. Bronforth G, Asendelft WJJ, Evans RL, et al: Efficacy of spinal manipulation for chronic headache: a systematic review, *J Manipulative Physiol Ther* 24(7):457, 2001.
38. Greenman PE: Principles of soft tissue and articular (mobilization with impulse) technique. In *Principles of manual medicine*, Baltimore, 1989, Williams & Wilkins.
39. Sheriff J: A flexible approach to traction. In *Grieve's modern manual of therapy*, ed 2, Edinburgh, UK, 1994, Churchill Livingstone.
40. Mennell JM: *The musculoskeletal system differential diagnosis from symptoms and physical signs*, Gaithersburg, MD, 1992, Aspen.
41. Kaltenborn FM: *Mobilization of the extremity joints*, ed 3, Oslo, Norway, 1980, Olaf Norlis Bokhandel.
42. Manek NJ, MacGregor A: Epidemiology of back disorders: prevalence, risk factors and prognosis, *Curr Opin Rheumatol* 17(2):134, 2005.
43. Waddell G: 1987 Volvo award in clinical sciences. a new clinical model for the treatment of low-back pain, *Spine* 12(7):632, 1987.
44. Deyo RA, Weinstein JN: Low back pain, *N Engl J Med* 344(5):364, 2001.
45. Chou R, et al: Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society, *Ann Intern Med* 147(7):478, 2007.
46. Van Tulder M, Becker A, Breen A: European guidelines for the management of acute nonspecific low back pain in primary care, *Eur Spine J* 15s:169, 2006.
47. McCarthy CJ, et al: The reliability of the clinical tests and questions recommended in international guidelines for low back pain, *Spine* 32(8):921, 2007.
48. Herbert J, Kippenhaver S, Fritz J: Clinical prediction for success of interventions for managing low back pain, *Clin Sports Med* 27:463, 2008.
49. Murphy DR, Hurwitz E: A theoretical model for the development of a diagnosis-based clinical decision rule for the management of patients with spinal pain, *BMC Musculoskelet Disord* 8:75, 2007.

50. Childs JD, Fritz JM, Flynn TW: A clinical prediction rule to identify patients with low back pain most likely to benefit from spinal manipulation: A validation study, *Ann Intern Med* 141(12):920, 2004.
51. Deyo R: Treatments for back pain: can we get past trivial effects, *Ann Intern Med* 141(12):957, 2004.
52. Herbert J, Kippenhaver S, Fritz J: Clinical prediction for success of interventions for managing low back pain, *Clin Sports Med* 27:463, 2008.
53. Grainger HG: The somatic component in visceral disease. In *Academy of Applied Osteopathy 1958 Yearbook*, Newark, OH, 1958, American Academy of Osteopathy.
54. Triano JJ: The subluxation complex: outcome measure of chiropractic diagnosis and treatment, *J Chiro Tech* 2(3):114, 1990.
55. Kirkaldy-Willis WH: The pathology and pathogenesis of low back pain. In Kirkaldy-Willis WH, editor: *Managing low back pain*, ed 3, New York, 1992, Churchill Livingstone.
56. Kirkaldy-Willis WH: The three phases of the spectrum of degenerative disease. In Kirkaldy-Willis WH, editor: *Managing low back pain*, ed 3, New York, 1992, Churchill Livingstone.
57. Murphy DR, Hurwitz E: A diagnosis-based clinical decision rule for spinal pain part 2: review of the literature, *BMC Musculoskeletal Disord* 16:7, 2008.
58. Phillips RB, et al: Stress x-rays and the low back pain patient, *J Manipulative Physiol Ther* 13:127, 1990.
59. Haas M, et al: Lumbar motion trends and correlation with low back pain. I. A roentgenological evaluation of coupled lumbar motion in lateral bending, *J Manipulative Physiol Ther* 15:145, 1992.
60. Haas M, Nyiendo J: Lumbar motion trends and correlation with low back pain. II. A roentgenological evaluation of quantitative segmental motion in lateral bending, *J Manipulative Physiol Ther* 15:224, 1992.
61. Haas M, Peterson D: A roentgenological evaluation of the relationship between segmental mal-alignment and motion in lateral bending, *J Manipulative Physiol Ther* 15:350, 1992.
62. Peters RE: The facet syndrome, *J Aust Chiro Assoc* 13(3):15, 1983.
63. Yeomans SG, Liebenson C: Applying outcomes management to clinical practice, *J Neuromusculoskeletal Syst* 5:1, 1997.
64. Deyo RA, et al: Outcome measures for low back pain research: a proposal for standardized use, *Spine* 23:1998, 2003.
65. Liebenson C: *Rehabilitation of the spine: A practitioners manual*, Baltimore, 1996, Williams & Wilkins.
66. Fairbank JCT, et al: The Oswestry low back pain disability questionnaire, *Physiotherapy* 66:271, 1980.
67. Roland M, Morris R: A study of the natural history of low back pain. II, *Spine* 8:141, 1983.
68. Vernon H: The neck disability index: a study of reliability and validity, *J Manipulative Physiol Ther* 14(7):409, 1991.
69. Alaranta H, et al: Reliability and normative data base, *Scand J Rehab Med* 26:211, 1994.
70. Yeomans S, Liebenson C: Quantitative functional capacity evaluation: The missing link to outcomes assessment, *Top Clin Chiro* 3(1):32, 1996.
71. Gatterman MI: Standards for contraindications to spinal manipulative therapy. In Vear HJ, editor: *Chiropractic standards of practice and quality of care*, Baltimore, 1992, Aspen.
72. Gatterman MI: Contraindications and complications of spinal manipulative therapy, *J Am Chiro Assoc* 15:75, 1981.
73. Shekelle PG, Adams AH: The appropriateness of spinal manipulation for low-back pain: Project overview and literature review, Santa Monica, CA, 1991, RAND.
74. Ladermann JP: Accidents of spinal manipulations, *Ann Swiss Chiro Assoc* 7:161, 1981.
75. Brewerton DA: Conservative treatment of painful neck, *Proc R Soc Med* 57:16, 1964.
76. Kleynhans AM: Complications of and contraindications to spinal manipulative therapy. In Haldeman S, editor: *Modern developments in the principles and practice of chiropractic*, East Norwalk, CT, 1980, Appleton-Century-Crofts.
77. Shekelle PG, et al: Spinal manipulation for back pain, *Ann Intern Med* 117:590, 1992.
78. Hurwitz EL, et al: Manipulation and mobilization of the cervical spine: a systematic review of the literature, *Spine* 21:1746, 1996.
79. Klougart N, Leboueuf-Yde C, Rasmussen LR: Safety in chiropractic practice. In *The occurrence of cerebrovascular accidents after manipulation to the neck in Denmark from 1978–1988*, *J Manipulative Physiol Ther* 19:371, 1996.
80. Assendelft WJ, Bouter LM, Knipschild PG: Complications of spinal manipulation: a comprehensive review of the literature, *J Fam Pract* 42(5):475, 1996.
81. Terrett AGJ, Kleynhans AM: Complications from manipulation of the low back, *Chiro J of Australia* 4:129, 1992.
82. Dabbs V, Lauretti WJ: A risk of assessment of cervical manipulation vs. NSAIDs for the treatment of neck pain, *J Manipulative Physiol Ther* 18:530, 1995.
83. Dvorak J, et al: Musculoskeletal complications. In Haldeman S, editor: *Principles and practice of chiropractic*, Norwalk, CT, 1992, Appleton & Lange.
84. Oliphant D: Safety of spinal manipulation in the treatment of lumbar disk herniations: a systematic review and risk assessment, *J Manipulative Physiol Ther* 27:197, 2004.
85. Cassidy JD, Boyle E, Cote P: Risk of vertebrobasilar stroke and chiropractic care: results of a population-based case-control and case crossover study, *Spine* 33(48):176, 2008.
86. Oppenheim JS, Spitzer DE, Segal DH: Nonvascular complications following spinal manipulation, *Spine J* 5:660, 2005.
87. Ernst E: Adverse effects of spinal manipulation: a systematic review, *J R Soc Med* 100:330, 2007.
88. Rubinstein SM, et al: The benefits outweigh the risks for patients undergoing chiropractic care for neck pain: A prospective, multicenter, cohort study, *J Manipulative Physiol Ther* 30(6):408, 2007.
89. Rubinstein SM: Adverse events following chiropractic care for subjects with neck or low-back pain: do the benefits outweigh the risks? *J Manipulative Physiol Ther* 31:461, 2008.
90. Chapman-Smith D: The chiropractic report 8(3):1994.
91. Henderson DJ, et al, editors: *Clinical guidelines for chiropractic practice in Canada*, Toronto, 1994, Canadian Chiropractic Association.
92. Terrett AGJ: Vascular accidents from cervical spinal manipulation: a report of 107 cases, *J Aust Chiro Assoc* 17:15, 1987.
93. Murphy DR, Beres JL: Cervical myelopathy: a case report of a "near miss" complication to cervical manipulation, *J Manipulative Physiol Ther* 31(7):553, 2008.
94. Haldeman S: Nonvascular complications following spinal manipulation (letter), *Spine J* 5:660, 2005.
95. Chapman-Smith D: Cervical adjustment, *The chiropractic report* 13(4):1999.
96. Haldeman S, Kohlbeck F, McGregor M: Risk factors and precipitating neck movements causing vertebrobasilar artery dissection after cervical trauma and spinal manipulation, *Spine* 24:785, 1999.
97. Triano J, Kawchuk G: *Current concepts in spinal manipulation and cervical arterial incidents*, Clive, IA, 2006, NCMIC Chiropractic Solutions.
98. Haneline MT, Croft AC, Frishberg BM: Association of internal carotid artery dissection and chiropractic manipulation, *Neurologist* 9:35, 2003.
99. Terrett AGJ: Vascular accidents from cervical spine manipulation: The mechanisms, *J Aust Chiropractor's Assoc* 17:131, 1987.
100. Bland JH: *Disorders of the cervical spine*, Philadelphia, 1987, Saunders.
101. Farris AA, et al: Radiographic visualization of neck vessels in healthy men, *Neurol* 13:386, 1961/1963.
102. Patten J: *Neurological differential diagnosis*, London, 1977, Harold Starke.

103. Haynes MJ, et al: Vertebral arteries and cervical rotation: modeling and magnetic resonance angiography studies, *J Manipulative Physiol Ther* 25(6):370, 2002.
104. Toole JF, Tucker SH: Influence of head position upon cerebral circulation: studies on blood flow in cadavers, *Arch Neurol* 2:616, 1960.
105. Hardesty WH, et al: Studies on vertebral artery blood flow in man, *Surg Gynecol Obstet* 116:662, 1963.
106. Licht PB, et al: Triplex ultrasound of vertebral artery flow during cervical rotation, *J Manipulative Physiol Ther* 21:27, 1998.
107. Licht PB, et al: Vertebral artery flow and spinal manipulation: a randomized, controlled and observer blinded study, *J Manipulative Physiol Ther* 21:141, 1998.
108. Licht PB, Christensen HW, Houlund-Carsen PF: Vertebral artery volume flow in human beings, *J Manipulative Physiol Ther* 22:363, 1999.
109. Yi-Kai L: Changes and implications of blood flow velocity of the vertebral artery during rotation and extension of the head, *J Manipulative Physiol Ther* 22:91, 1999.
110. Symons BP, Leonard T, Herzog W: Internal forces sustained by the vertebral artery during spinal manipulative therapy, *J Manipulative Physiol Ther* 25(8):504, 2002.
111. Rubinstein SM, Haldeman S, van Tulder MW: An etiologic model to help explain the pathogenesis of cervical artery dissection: implications for cervical manipulation, *J Manipulative Physiol Ther* 29(4):336, 2006.
112. Terrett AGJ: Vertebrobasilar stroke following manipulation, West Des Moines, IA, 1996, National Chiropractic Mutual Insurance Company.
113. Terrett AGJ: It is more important to know when not to adjust, *Chiro Tech* 2(1):1, 1990.
114. Terrett AGJ, Kleynhans AM: Cerebrovascular complications of manipulation. In Haldeman S, editor: *Modern developments in the principles and practice of chiropractic*, ed 2, East Norwalk, CT, 1902, Appleton-Century-Crofts.
115. Bassi P, Lattuada P, Gomitoni A: Cervical cerebral artery dissection: A multicenter prospective study (preliminary report), *Neurol Sci* 24(Suppl 1):S4, 2003.
116. Ernst E: Manipulation of the cervical spine: a systematic review of case reports of serious adverse events, 1995–2001, *Med J Aust* 176:376, 2002.
117. Miley ML, et al: Does cervical manipulative therapy cause vertebral artery dissection and stroke? *Neurologist* 14:66, 2008.
118. Terrett AGJ: Current concepts in vertebrobasilar complications following spinal manipulation, West Des Moines, IA, 2001, National Chiropractic Mutual Insurance Company.
119. Gutman GG: Injuries to the vertebral artery caused by manual therapy, *Manuelle Medizin* 21:2, 1983 (English abstract).
120. Dvorak J, Orelli F: How dangerous is manipulation to the cervical spine? *Manual Medicine* 2:1, 1985.
121. Powell FC, Hanigan WC, Olivero WC: A risk/benefit analysis of spinal manipulation therapy for relief of lumbar or cervical pain, *Neurosurgery* 33:73, 1993.
122. Haldeman S, Chapman-Smith D, Peterson DM: Guidelines for chiropractic quality assurance and practice parameters, Gaithersburg, MD, 1993, Aspen.
123. Haynes MJ: Stroke following cervical manipulation in Perth, *Chiro J Aust* 23:42, 1994.
124. Terrett AGJ, Kleynhans AM: Cerebrovascular complications of manipulation. In Haldeman S, editor: *Principles and practice of chiropractic*, Norwalk, CT, 1992, Appleton & Lange.
125. Fries JF: Assessing and understanding patient risk, *Scand J Rheumatol* 92(Suppl):21, 1992.
126. Terrett AGJ: Misuse of the literature by medical authors in discussing spinal manipulative therapy injury, *J Manipulative Physiol Ther* 18:203, 1995.
127. Leboeuf-Yde C, Rasmussen LR, Klougart N: The risk of over-reporting spinal manipulative therapy-induced injuries: a description of some cases that failed to burden the statistics, *J Manipulative Physiol Ther* 19:5, 1996.
128. Haldeman S, et al: Clinical perceptions of the risk of vertebral artery dissection after cervical manipulation: the effect of referral bias, *Spine J* 2:334, 2002.
129. Rothwell DM, Bondy SJ, Williams JI: Chiropractic manipulation and stroke: a population-based case-control study, *Stroke* 32:1054, 2001.
130. Smith WS, et al: Spinal manipulative therapy is an independent risk factor for vertebral artery dissection, *Neurology* 67:1809, 2003.
131. Cote P, Cassidy JD, Haldeman S: Spinal manipulative therapy is an independent risk factor for vertebral artery dissection, *Neurology* 61:1314, 2003.
132. Lauretti B: Risk factors for vertebral artery dissection and stroke, *ACA News* 4(7):9, 2008.
133. Rubinstein SM, et al: A systematic review of the risk factors for cervical artery dissection, *Stroke* 36:1575, 2005.
134. Cote P, et al: The validity of the extension rotation test as a clinical screening procedure before neck manipulation: a secondary analysis, *J Manipulative Physiol Ther* 19:159, 1996.
135. Terrett AGJ: Importance and interpretation of tests designed to predict susceptibility to neurocirculatory accidents from manipulation, *J Am Chiro Assoc* 13(2):2, 1983.
136. Hulse M: Disequilibrium, caused by a functional disturbance of the upper cervical spine, *Manual Med* 1:18, 1983.
137. Bracher E, et al: A combined approach for the treatment of cervical vertigo, *J Manipulative Physiol Ther* 23:96, 2000.
138. Terrett AGJ: Importance and interpretation of tests designed to predict susceptibility to neurocirculatory accidents from manipulation, *J Aust Chiro Assoc* 13(2):29, 1983.
139. Ivancic JJ, Bryce D, Bolton PS: Use of provocative tests by clinicians to predict vulnerability of patients to vertebrobasilar insufficiency, *Chiropr J Aust* 23(2):59, 1993.
140. Bolton PS, Stick PE, Lord RSA: Failure of clinical tests to predict ischemia before neck manipulation, *J Manipulative Physiol Ther* 12(4):403, 1989.
141. George PE: Identification of the high risk pre-stroke patient, *J Chiropr* 15:S26, 1981.
142. Savoie SSM: The George's test: a review and update, *ICA Int Rev Chiropr* 42(3):18, 1986.
143. Ziegler DK, et al: Correlation of bruits over the carotid artery with angiographically demonstrated lesions, *Neurology* 21:860, 1971.
144. Nelson C, et al: The efficacy of spinal manipulation, amitriptyline and the combination of both therapies for the treatment of the prophylaxis of migraine, *J Manipulative Physiol Ther* 21:511, 1998.
145. Cassidy JD, Lopes AA, Yong-Hing K: The immediate effect of manipulation versus mobilization on pain and range of motion in the cervical spine: A randomized controlled trial, *J Manipulative Physiol Ther* 15:570, 1992.
146. Cassidy ID, et al: The effect of manipulation on pain and range of motion in the cervical spine: a pilot study, *J Manipulative Physiol Ther* 15:495, 1992.
147. Hviid H: The influence of the chiropractic treatment on the rotary mobility of the cervical spine: a kinesiometric and statistical study, *Ann Swiss Chiropractors' Association* 5:1, 1971.
148. Koes BW, et al: Spinal manipulation and mobilization for back and neck pain: a blinded review, *Br Med J* 303:1298, 1991.
149. Koes BW, et al: A randomized clinical trial of manual therapy and physiotherapy for persistent back and neck complaints: subgroup analysis and relationship between outcome measures, *J Manipulative Physiol Ther* 16:211, 1993.
150. Koes BW, et al: A blinded randomized clinical trial of manual therapy and physiotherapy for chronic back and neck complaints: physical outcome measures, *J Manipulative Physiol Ther* 15:16, 1992.

151. Koes BW, et al: Randomized clinical trial of manipulative therapy and physiotherapy for persistent back and neck complaints: Results of one year follow up, *BMJ* 304:601, 1992.
152. Koes BW, et al: The effectiveness of manual therapy, physiotherapy, and treatment by the general practitioner for nonspecific back and neck complaints: a randomized clinical trial, *Spine* 17:28, 1992.
153. Vernon H: Chiropractic manipulative therapy in the treatment of headaches: a retrospective and prospective study, *J Manipulative Physiol Ther* 5:109, 1982.
154. Vernon H: Spinal manipulation and headaches of cervical origin: a review of literature and presentation of cases, *J Manual Med* 6:73, 1991.
155. Vernon HT: Spinal manipulation and headaches of cervical origin, *J Manipulative Physiol Ther* 12:455, 1989.
156. Ferezy JS: Neural ischemia and cervical spinal manipulation: the chiropractic neurological examination, Rockville, MD, 1992, Aspen.
157. Bogduk N, Lambert G, Duckworth JW: The anatomy and physiology of the vertebral nerve in relation to cervical migraine, *Cephalgia* 1, 1981.
158. Senstad L, Leboeuf-Yde C, Borchgrevink C: Predictors of side effects of spinal manipulative therapy, *J Manipulative Physiol Ther* 19:441, 1996.
159. Darbert O, Freeman DG, Weis AJ: Spinal meningeal hematoma, warfarin therapy and chiropractic adjustment, *JAMA* 214:2058, 1970.
160. Triano J, Schultz AB: Loads transmitted during lumbosacral spinal manipulative therapy, *Spine* 22(17):1955, 1997.
161. Haldeman S, Rubenstein SM: Cauda equina syndrome in patients undergoing manipulation of the lumbar spine, *Spine* 17(12):1469, 1992.
162. Richard J: Disc rupture with cauda equina syndrome after chiropractic adjustment, *NY State J Med* 67:249, 1967.
163. Farfan HF, et al: Effects of torsion on the intervertebral joint: the roll of torsion in the production of disc degeneration, *J Bone Joint Surg* 52A:468, 1970.
164. Adams MA, Hutton WC: The relevance of torsion to the mechanical derangement of the lumbar spine, *Spine* 6(3):241, 1981.
165. Schultz AB, et al: Mechanical properties of the human lumbar spine motion segments. I. Response in flexion, extension, lateral bending and torsion, *J Biomech Eng* 101:46, 1979.
166. Skipor AF, et al: Stiffness, properties and geometry of lumbar spine posterior elements, *J Biomech* 18:821, 1985.
167. Bogduk N: Clinical anatomy of the lumbar spine and sacrum, ed 3, New York, 1997, Churchill Livingstone.
168. Adams MA, Hutton WC: The role of the apophyseal joints in resisting intervertebral compressive force, *J Bone Joint Surg Br* 62:358, 1980.
169. Adams MA, Hutton WC: 1981 Volvo Award in Basic Science. Prolapsed intervertebral disc: a hyperflexion injury, *Spine* 7(3):184, 1982.
170. Broberg KB: On the mechanical behavior of intervertebral discs, *Spine* 8(2):151, 1983.
171. Perry O: Fracture of the vertebral endplate in the lumbar spine, *Acta Orthop Scand (suppl)* 25, 1957.
172. Jagbandhansingh MP: Most common causes of chiropractic malpractice lawsuits, *J Manipulative Physiol Ther* 20:60, 1997.
173. Garg A: Occupational biomechanics and low back pain, *Occup Med* 7(4):609, 1992.
174. Fiorini GT, McCammond D: Forces on the lumbo-vertebral facets, *J Biomed Eng* 4:354, 1976.
175. Christensen M, Morgan D: Job analysis of chiropractic, Greeley, CO, 1993, National Board of Chiropractic Examiners.
176. Kane RL, et al: Manipulating the patient: A comparison of the effectiveness of physician and chiropractor care, *Lancet* 1:1333, 1974.
177. Cherkin DC, MacCornack FA: Health care delivery: patient evaluations of low back pain care from family physicians and chiropractors, *West J Med* 150(3):351, 1989.
178. Cherkin DC, MacCornack FA, Berg AO: The management of low back pain: a comparison of the beliefs and behaviors of family physicians and chiropractors, *West J Med* 149:475, 1988.
179. Manga P, et al: The effectiveness and cost-effectiveness of chiropractic management of low-back pain, Ottawa, Ontario, 1993, Pran Manga and Associates.
180. Carey TS, et al: The outcomes and costs of care for acute low back pain among patients seen by primary care practitioners, chiropractors, and orthopedic surgeons, *N Engl J Med* 333(14):913, 1995.
181. Carey TS, et al: Acute severe low back pain: a population-based study of prevalence and care-seeking, *Spine* 21:339, 1996.
182. Verhoef MJ, Page SA, Waddell SC: The chiropractic outcome study: pain, functional ability and satisfaction with care, *J Manipulative Physiol Ther* 20:235, 1997.
183. Stano M, Smith M: Chiropractic and medical costs of low back care, *Med Care* 34:191, 1996.
184. American Chiropractic Association: Comparison of chiropractic and medical treatment of nonoperative back and neck injuries, 1976-77, Des Moines, IA, 1978, American Chiropractic Association.
185. Hertzman-Miller RP, et al: Comparing the satisfaction of low back pain patients randomized to receive medical or chiropractic care: results from the UCLA low back pain study, *Am J Public Health* 92(10):1628, 2002.
186. Brunarski DJ: Clinical trials of spinal manipulation: A critical appraisal and review of the literature, *J Manipulative Physiol Ther* 7(4):253, 1984.
187. Nyiendo J: Chiropractic effectiveness: Series no 1, Oregon Chiropractic Physicians Association Legislative Newsletter, April 1991. Portland, OR.
188. Anderson R, et al: A meta-analysis of clinical trials of spinal manipulation, *J Manipulative Physiol Ther* 15:181, 1992.
189. Coulter ID, et al: The appropriateness of manipulation and mobilization of the cervical spine, Santa Monica, CA, 1996, RAND.
190. Shekelle PG, Coulter ID: Cervical spine manipulation: summary report of a systematic review of the literature and a multidisciplinary expert panel, *J Spinal Disord* 10(3):223, 1997.
191. Bigos S, et al: Acute low back problems in adults, Clinical Practice Guideline No 14, AHCPR Pub No 95-0642, Rockville, MD, 1994, Agency for Health Care Policy and Research, Public Health Service, US Department of Health and Human Services.
192. Waagen GN, Haldeman S, Cook G: Short term trial of chiropractic adjustments for the relief of chronic low back pain, *Manipulative Med* 2:63, 1986.
193. Meade TW, Dyer SD, Brown W: Low back pain of mechanical origin: Randomized comparison of chiropractic and hospital outpatient treatment, *BMJ* 300:1431, 1990.
194. Vernon H, McDermaid CS, Hagino C: Systematic review of randomized clinical trials of complementary/alternative therapies in the treatment of tension-type and cervicogenic headache, *Complement Ther Med* 7:142-155, 1999.
195. Bronfort G, et al: Noninvasive physical treatments for chronic headache, *Cochrane Database Syst Rev* (3):CD001878, 2004.
196. Nilsson N, Christensen HW, Hartvigsen J: The effect of spinal manipulation in the treatment of cervicogenic headache, *J Manipulative Physiol Ther* 20:326-330, 1997.
197. Cherkin DC, et al: A review of the evidence for the effectiveness, safety, and cost of acupuncture, massage therapy, and spinal manipulation for back pain, *Ann Intern Med* 38:898-906, 2003.
198. Waddell G, et al: Clinical guidelines for the management of acute low back pain: 1996 low back pain evidence review, London, 1996, Royal College of General Practitioners.

199. Van Tulder M, et al: European guidelines for the management of acute nonspecific low back pain in primary care, *Eur Spine J* 15(S2):S169, 2006.
200. Airaksinen O, Brox JI, Cedraschi C: European guidelines for the management of chronic nonspecific low back pain, *Eur Spine J* 15(S2):S1192, 2006.
201. Chou R, Huffman L: Nonpharmacologic therapies for acute and chronic low back pain: a review of the evidence for an American Pain Society/American College of Physicians clinical practice guideline, *Ann Intern Med* 147(7):492, 2007.
202. Nyiendo J: Disabling low back Oregon workers' compensation claims. III. Diagnosing and treatment procedures and associated costs, *J Manipulative Physiol Ther* 14(5):287, 1991.
203. Greenwood JG: Work-related back and neck injury cases in West Virginia, *Orthop Rev* 14(2):51, 1985.
204. Stano M: The economic role of chiropractic: an episode analysis of relative insurance costs for low back care, *J Neuromusculoskel Syst* 1(2):64, 1993.
205. Stano M: A comparison of health care costs for chiropractic and medical patients, *J Manipulative Physiol Ther* 16(5):291, 1993.
206. Stano M: Further analysis of health care costs for chiropractic and medical patients, *J Manipulative Physiol Ther* 17(7):442, 1994.
207. Legorreta AP, et al: Comparative analysis of individuals with and without chiropractic coverage: patient characteristics, utilization, and costs, *Arch Intern Med* 1164(18):1985, 2004.
208. UK BEAM Trial Team: United Kingdom back pain exercise and manipulation (UK BEAM) randomized trial: effectiveness of physical treatments for back pain in primary care, *BMJ* 329:1377, 2004.
209. UK BEAM Trial Team: United Kingdom back pain exercise and manipulation (UK BEAM) randomized trial: Cost effectiveness of physical treatments for back pain in primary care, *BMJ* 329:1381, 2004.
210. Shekelle PG, Markovich M, Louie R: Comparing the costs between provider types of episodes of back pain care, *Spine* 20(2):221, 1995.
211. Montgomery DP, Nelson JM: Evolution of chiropractic theories of practice and spinal adjustment, 1900–1950, *Chiropr Hist* 5:71, 1985.
212. Strasser A: The chiropractic model of health: A personal perspective, *Dig Chiro Econ* 13(2):12, 1988.
213. Nansel D, Szlazak M: Somatic dysfunction and the phenomenon of visceral disease simulation: A probable explanation for the apparent effectiveness of somatic therapy in patients presumed to be suffering from true visceral disease, *J Manipulative Physiol Ther* 18(6):379, 1995.
214. Balon J, et al: A comparison of active and simulated chiropractic manipulation as adjunctive treatment for childhood asthma, *N Engl J Med* 339(15):1013, 1998.
215. Wilson PT: Experimental work in asthma at the Peter Bent Brigham Hospital, *J Am Osteopath Assoc* 25:212, 1925.
216. Miller WD: Treatment of visceral disorders by manipulative therapy. In Goldstein M, editor: *The research status of spinal manipulative therapy*, Washington, DC, 1975, US Government Printing Office.
217. Hviid C: A comparison of the effect of chiropractic treatment on respiratory function in patients with respiratory distress symptoms and patients without, *Bull Eur Chiropractic Union* 26:17, 1978.
218. Jamison JR: Asthma in a chiropractic clinic: A pilot study, *J Aust Chiropractic Assoc* 16:137, 1986.
219. Nilsson N, Christiansen B: Prognostic factors in bronchial asthma in chiropractic practice, *J Aust Chiropractic Assoc* 18:85, 1988.
220. Wilson PT: Osteopathic cardiology. In *Academy of Applied Osteopathy 1956 Year Book*, Newark, OH, 1956, American Academy of Osteopathy.
221. Koch RS: A somatic component in heart disease, *J Am Osteopath Assoc* 60:735, 1961.
222. Fischera AP, Celandier DR: Effect of osteopathic manipulative therapy on autonomic tone as evidenced by blood pressure changes and activity of the fibrinolytic system, *J Am Osteopath Assoc* 68:1036, 1969.
223. McKnight ME, DeBoer KF: Preliminary study of blood pressure changes in normotensive subjects undergoing chiropractic care, *J Manipulative Physiol Ther* 11:261, 1988.
224. Yates RG, et al: Effects of chiropractic treatment on blood pressure and anxiety: a randomized, controlled trial, *J Manipulative Physiol Ther* 11:484, 1988.
225. Wagnon RJ, et al: Serum aldosterone changes after specific chiropractic manipulation, *Am J Chiropr Med* 1:66, 1988.
226. Morgan JP, et al: A controlled trial of spinal manipulation in the management of hypertension, *J Am Osteopath Assoc* 85:308, 1985.
227. Downing WJ: Osteopathic manipulative treatment of non-surgical gallbladder, *J Am Osteopath Assoc* 39:104, 1939.
228. Denslow JS: Acute cholecystitis and colitis: report of a case, *J Am Osteopath Assoc* 32:285, 1933.
229. Mattern AV: Gastro-duodenal ulcer and its non-surgical treatment, *J Am Osteopath Assoc* 33:188, 1934.
230. Meyers TJ: Gastric ulcer: case histories, *J Am Osteopath Assoc* 30:474, 1931.
231. Wilson PT: Case history and discussion. III, *J Am Osteopath Assoc* 33:348, 1934.
232. Klougart N, et al: Infantile colic treated by chiropractors: a prospective study of 316 cases, *J Manipulative Physiol Ther* 12:281, 1989.
233. LeBoeuf C, et al: Chiropractic care of children with nocturnal enuresis: a prospective outcome study, *J Manipulative Physiol Ther* 14:110, 1991.
234. Olafsdottir E, et al: Randomized controlled trial of infantile colic treated with chiropractic spinal manipulation, *Arch Dis Child* 84:138, 2001.
235. Herzog W: Mechanical, physiologic, and neuromuscular considerations of chiropractic treatments, *Adv Chiropr* 3:269, 1996.
236. Stonebrink RD: Evaluation and manipulative management of common musculoskeletal disorders, Portland, OR, 1990, Western States Chiropractic College.
237. Grieve GP: Aetiology in general terms. In *Common vertebral joint problems*, Edinburgh, UK, 1988, Churchill Livingstone.
238. Kirkaldy-Willis WH: The pathology and pathogenesis of low back pain. In Kirkaldy-Willis WH, editor: *Managing low back pain*, ed 3, New York, 1992, Churchill Livingstone.
239. Salter RB: The biologic concept of continuous passive motion of synovial joints: The first 18 years of basic research and its clinical application, *Clin Orthop* 242:12, 1989.
240. Salter RB: Motion vs. rest: Why immobilize joints? *J Bone Joint Surg Br* 64:251, 1989.
241. Waddell G: A new clinical model for the treatment of low-back pain, *Spine* 12(7):632, 1987.
242. Gelberman R, Manske P, Akeson W: Kappa Delta award paper: flexor tendon repair, *J Orthop Res* 4:119, 1986.
243. Kellert J: Acute soft tissue injuries: A review of the literature, *Med Sci Sports Exerc* 18(5):489, 1986.
244. Fitz-Ritson D: Chiropractic management and rehabilitation of cervical trauma, *J Manipulative Physiol Ther* 13(1):17, 1990.
245. Mealy K, et al: Early mobilization of acute whiplash injuries, *BMJ* 292:656, 1986.
246. Akeson W, Amiel D, Woo S: Immobility effects on synovial joints: The pathomechanics of joint contracture, *Biorheology* 17:95, 1980.
247. Van Royen BJ, O'Driscoll SW, Wouter JAD: Comparison of the effects of immobilization and continuous passive motion on surgical wound healing in the rabbit, *Plast Reconstr Surg* 78:360, 1986.
248. Amiel D, Woo SL-Y, Harwood F: The effect of immobilization on collagen turnover in connective tissue: A biomechanical-biochemical correlation, *Acta Orthop Scand* 53:325, 1982.

249. Rubak JM, Poussa M, Ritsila V: Effects of joint motion on the repair of articular cartilage with free periosteal grafts, *Acta Orthop Scand* 53:187, 1982.
250. McDonough AL: Effects of immobilization and exercise on articular cartilage: A review of literature, *J Orthop Sports Phys Ther* 3:2, 1981.
251. Akeson WH, Amiel D, Mechanic GL: Collagen cross-linking alterations in joint contractures: Changes in the reducible cross-links in periarticular connective tissue collagen after nine weeks of immobilization, *Connect Tissue Res* 5:15, 1977.
252. Woo S.L.-Y., Matthews JV, Akeson WH: Connective tissue response to immobility: Correlative study of biomechanical and biochemical measurements of normal and immobilized rabbit knees, *Arthritis Rheum* 18(3):257, 1975.
253. Enneking WF, Horowitz M: The intra-articular effects of immobilization of the human knee, *J Bone Joint Surg Am* 54:973, 1972.
254. Mayer T, Gatchel R: Functional restoration for spinal disorders: The sports medicine approach, Philadelphia, 1988, Lea & Febiger.
255. Fordyce W, Roberts A, Sternbach R: The behavioral management of chronic pain: A response to critics, *Pain* 22:112, 1985.
256. Herzog W: Mechanical and physiological responses to spinal manipulative treatments, *J Neuromusculoskel Syst* 3:1, 1995.
257. Kirstukas SJ, Backman JA: Physician-applied contact pressure and table force response during unilateral thoracic manipulation, *J Manipulative Physiol Ther* 22:269, 1999.
258. Herzog W, Kawchuk GN, Conway PJ: Relationship between preload and peak forces during spinal manipulative treatments, *J Neuromusculoskel Syst* 1:53, 1993.
259. Herzog W, Kats M, Symons B: The effective forces transmitted by high-speed, low amplitude thoracic manipulation, *Spine* 26(19):2105, 2002.
260. Cramer G, et al: Basic science research related to chiropractic spinal adjusting: The state of the art and recommendations revisited, *J Manipulative Physiol Ther* 29(9):726, 2006.
261. Cohen E, et al: Biomechanical performance of spinal manipulation therapy by newly trained vs. practicing providers: Does experience transfer to unfamiliar procedures, *J Manipulative Physiol Ther* 18(6):347, 1995.
262. Gal JM, et al: Biomechanical studies of spinal manipulative therapy: Quantifying the movements of vertebral bodies during SMT, *J Can Chiropr Assoc* 38:11, 1994.
263. Ianuzzi A, Khalsa PS: Comparison of human lumbar facet joint capsule strains during simulated high-velocity, low-amplitude spinal manipulation versus physiological motions, *Spine* 5(3):277, 2005.
264. Cramer D, Tuck N, Knudsen JT, et al: Effects of side-posture positioning and side-posture adjusting on the lumbar zygapophyseal joints as evaluated by magnetic resonance imaging: A before and after study with randomization, *J Manipulative Physiol Ther* 23(6):380, 2000.
265. Cramer GD, et al: The effects of side-posture positioning and spinal adjusting on the lumbar Z joints: A randomized controlled trial with sixty-four subjects, *Spine* 27(2):2459, 2002.
266. Reggars JW, Pollard HP: Analysis of zygapophyseal joint cracking during chiropractic manipulation, *J Manipulative Physiol Ther* 18:65, 1995.
267. Beffa R, Mathews R: Does the adjustment cavitate the targeted joint? An investigation into the location of cavitation sounds, *J Manipulative Physiol Ther* 27(2): 118–122 2004.
268. Ross JK, Bereznick D, McGill S: Determining cavitation location during lumbar and thoracic spinal manipulation, *Spine* 29(13):1452, 2004.
269. Haas M, et al: Efficacy of cervical endplay assessment as an indicator for spinal manipulation, *Spine* 28:1091, 2003.
270. Haas M, et al: Dose response for chiropractic care of chronic cervicogenic headache and associated neck pain: A randomized pilot study, *J Manipulative Physiol Ther* 27(9):547, 2004.
271. Brodeur R: The audible release associated with joint manipulation, *J Manipulative Physiol Ther* 18:155, 1995.
272. Herzog W: On sounds and reflexes, *J Manipulative Physiol Ther* 19:216, 1996.
273. Harvey EN, McElroy WD, Whiteley AH: On cavity formation in water, *J Appl Physics* 18:162, 1947.
274. Gal JM, et al: Forces and relative vertebral movements during SMT to unembalmed post-rigor human cadavers: Peculiarities associated with joint cavitation, *J Manipulative Physiol Ther* 18(1):4, 1995.
275. Roston JB, Wheeler Haines R: Cracking in the metacarpophalangeal joint, *J Anat* 81:165, 1947.
276. Sandoz R: The significance of the manipulative crack and of other articular noises, *Ann Swiss Chiropr Assoc* 4:47, 1969.
277. Unsworth A, Dowson D, Wright V: Cracking joints, *Ann Rheum Dis* 30:348, 1971.
278. Meal GM, Scott RA: Analysis of the joint crack by simultaneous recording of sound and tension, *J Manipulative Physiol Ther* 9(3):189, 1986.
279. Herzog W: Biomechanical studies of spinal manipulative therapy, *J Can Chiropr Assoc* 35(3):156, 1991.
280. Conway PJW, et al: Forces required to cause cavitation during spinal manipulation of the thoracic spine, *Clin Biomech* 8:210, 1993.
281. Herzog W, et al: Cavitation sounds during spinal manipulative treatments, *J Manipulative Physiol Ther* 16:523, 1993.
282. Chen YL, Israelachvili J: New mechanism of cavitation damage, *Science* 252:1157, 1991.
283. Mireau D, et al: Manipulation and mobilization of the third metacarpophalangeal joint: A quantitative radiographic and range of motion study, *Manual Med* 3:135, 1998.
284. Cramer G, et al: Basic science research related to chiropractic spinal adjusting: The state of the art and recommendations revisited, *J Manipulative Physiol Ther* 29(9):726, 2006.
285. Pritsch M, et al: Adhesions of distal tibiofibular syndesmosis: A cause of chronic ankle pain after fracture, *Clin Orthop* (289):220, 1993.
286. Hagberg L, Wik O, Gerdin B: Determination of biomechanical characteristics of restrictive adhesions and of functional impairment after flexor tendon surgery: A methodological study of rabbits, *J Biomech* 24(10):935, 1991.
287. Schollmeier G, et al: Effects of immobilization on the capsule of the canine glenohumeral joint: A structural functional study, *Clin Orthop* 304:37, 1994.
288. Gillet H: The anatomy and physiology of spinal fixations, *J Nat Chiro Assoc* 33(12):22, 1963.
289. Schmorl G, Junghans H: The human spine in health and disease, ed 2, Orlando, FL, 1971, Grune & Stratton.
290. Maigne R: Orthopedic medicine: A new approach to vertebral manipulations, Springfield, IL, 1972, Charles C Thomas.
291. Giles LGF, Taylor JR: Innervation of lumbar zygapophyseal joint synovial folds, *Acta Orthop Scand* 58:43, 1987.
292. Giles LGF: Lumbar apophyseal joint arthrography, *J Manipulative Physiol Ther* 7(1):21, 1984.
293. Giles LGF: Lumbo-sacral and cervical zygapophyseal joint inclusions, *Manipulative Med* 2:89, 1986.
294. Kos J, Wolf J: Les ménisques intervertébraux et leur rôle possible dans les blocages vertébraux, *Ann Med Phys* 15:203, 1972.
295. Kos J, Wolf J: Intervertebral meniscoids and their possible role in vertebral blockage (English translation of Kos J, Wolf J: Les ménisques intervertébraux et leur rôle possible dans les blocages vertébraux, *Ann Med Phys* 15:203, 1972), *J Orthop Sports Phys Ther* 1:8, 1972.
296. Bogduk N, Engel R: The menisci of the lumbar zygapophyseal joints: A review of their anatomy and clinical significance, *Spine* 9:454, 1984.
297. Bogduk N, Jull G: The theoretical pathology of acute locked back: A basis for manipulative therapy, *Man Med* 1:78, 1985.

298. Bogduk N, Twomey LT: Clinical anatomy of the lumbar spine, ed 2, Melbourne, Australia, 1991, Churchill Livingstone.
299. Badgley CE: The articular facets in relation to low back pain and sciatic radiation, *J Bone Joint Surg* 23:481, 1941.
300. Hadley LA: Anatomico-roentgenographic studies of the spine, ed 5, Springfield, IL, 1964, Charles C Thomas.
301. Kraft GL, Levinthal DH: Facet synovial impingement, *Surg Gynecol Obstet* 93:439, 1951.
302. Farfan MF: Torsion and compression. In *Mechanical disorders of the low back*, Philadelphia, 1973, Lea & Febiger.
303. Cassidy DJ, Thiel HW, Kirkaldy-Willis WH: Side posture manipulation for lumbar disk intervertebral herniation, *J Manipulative Physiol Ther* 16(2):96, 1993.
304. Naylor A: Intervertebral disc prolapse and degeneration: The biomechanical and biophysical approach, *Spine* 1(2):108, 1976.
305. De Seze S: Les accidents de la deterioration structurale du disque, *Semin Hop Paris* 1:2267, 1955.
306. De Seze S: Les attitudes antalgique dans la sciaticque discoradiculaire commune, *Semin Hop Paris* 1:2291, 1955.
307. Cyriax JH: Lumbago: Mechanism of dural pain, *Lancet* 1:427, 1945.
308. Bogduk N: Clinical anatomy of the lumbar spine and sacrum, ed 2, New York, 1991, Churchill Livingstone.
309. Herbst R: Gonstead chiropractic science and art: The chiropractic methodology of Clarence S. Gonstead, Mt Horeb, WI, 1968, SCI-CHI.
310. Hancock MJ, et al: Systematic review of tests to identify the disc, SIJ, or facet joint as the source of low back pain, *Euro Spine J* 2007 (doi:10.1007/s00586-007-0391-1).
311. Lavernieux J: Les tractions vertebrales, Paris, 1960, L'Expansion.
312. Matthews JA, Yates DAH: Reduction of lumbar disc prolapse by manipulation, *BMJ* 3:696, 1969.
313. Christman OD, Mittnacht A, Snook GA: A study of the results following rotatory manipulation in the lumbar intervertebral-disc syndrome, *J Bone Joint Surg Am* 46:517, 1964.
314. Mensor MC: Non-operative treatment of, including manipulation for, lumbar intervertebral disc syndrome, *J Bone Joint Surg* 37A:925, 1995.
315. Nwuga VCB: Relative therapeutic efficacy of vertebral manipulation and conventional treatment in back pain management, *Am J Phys Med* 61:273, 1982.
316. Kuo PPF, Loh ZCL: Treatment of lumbar intervertebral disc protrusions by manipulation, *Clin Orthop* 215:47, 1987.
317. Cox JM: Mechanism, diagnosis and treatment of lumbar disc protrusion and prolapse, *J Am Chiro Assoc* 8:181, 1974.
318. Hubka MJ, et al: Lumbar intervertebral disc herniation: Chiropractic management using flexion, extension and rotational manipulative therapy, *Chiropr Tech* 3(1):5, 1991.
319. Beneliyahu DJ: Chiropractic management and manipulative therapy for MRI documented disc herniation, *J Manipulative Physiol Ther* 17:17785, 1994.
320. Stern PJ, Cote P, Cassidy DJ: A series of consecutive cases of low back pain with radiating leg pain treated by chiropractors, *J Manipulative Physiol Ther* 18:335, 1995.
321. Bergmann T, Jongeward BV: Manipulative therapy in low back pain with leg pain and neurological deficit: A case report and literature review, *J Manipulative Physiol Ther* 21(4):288, 1998.
322. Burton K, et al: A comparative trial of chemonucleolysis and manipulation in the treatment of symptomatic lumbar disc herniations. Paper presented at the Third International Forum for Primary Care Research on Low Back Pain, Manchester, UK, 1998.
323. Lisi AJ, Holmes EJ, Ammendolia C: High-velocity low-amplitude spinal manipulative for symptomatic lumbar disc disease: a systematic review of the literature, *J Manipulative Physiol Ther* 28(6):429, 2005.
324. Cassidy JD, Haymo WT, Kirkaldy-Willis WH: Manipulation. In *Kirkaldy-Willis WH, editor: Managing low back pain*, ed 3, New York, 1992, Churchill Livingstone.
325. Gatterman MI: Chiropractic management of spine related disorders, Baltimore, 1990, Williams & Wilkins.
326. McKenzie RA: The lumbar spine: Mechanical diagnosis and therapy, Waikanae, New Zealand, 1981, Spinal Publications.
327. Donelson R, et al: A prospective study of centralization of lumbar and referred pain, *Spine* 22(10):1115, 1997.
328. White AA, Panjabi MM: Clinical biomechanics of the spine, ed 2, Philadelphia, 1990, JB Lippincott.
329. Muhlemann D: Hypermobility as a common cause for chronic back pain, *Ann Swiss Chiro Assoc* (in press).
330. Paris SV: Physical signs of instability, *Spine* 10(3):277, 1985.
331. Gatterman MI: Chiropractic management of spine related disorders, Baltimore, 1990, Williams & Wilkins.
332. Barr KP, Grigs MCadby T: Lumbar stabilization: A review of core concepts and current literature, part 2, *Am J Phys Med Rehabil* 86:72, 2007.
333. Gitelman R: Spinal manipulation in the relief of pain. In Goldstein M, editor: The research status of spinal manipulative therapy, NINDCS Monograph No 15, DHEW Pub No 76-998, Washington, DC, 1975, US Government Printing Office.
334. Kirkaldy-Willis WH, Cassidy JD: Spinal manipulation in the treatment of low-back pain, *Can Fam Physician* 31:535, 1985.
335. Bernard TN, Kirkaldy-Willis WH: Recognizing specific characteristics of nonspecific low back pain, *Clin Orthop* 217:266, 1987.
336. Vernon HT, et al: Spinal manipulation and beta-endorphin: A controlled study of the effect of a spinal manipulation on plasma beta-endorphin levels in normal males, *J Manipulative Physiol Ther* 9(2):115, 1986.
337. Terrett A, Vernon H: Manipulation and pain tolerance: A controlled study of the effect of spinal manipulation on paraspinal cutaneous pain tolerance levels, *Am J Phys Med* 63(5):217, 1984.
338. Vernon HT, et al: Pressure pain threshold evaluation of the effects of spinal manipulation in the treatment of chronic neck pain: A pilot study, *J Manipulative Physiol Ther* 13:13, 1990.
339. Vicenzino B, et al: An investigation of the interrelationship between manipulative therapy-induced hypoalgesia and sympathoexcitation, *J Manipulative Physiol Ther* 21:448, 1998.
340. Roberts WJ, Gillette RG, Kramis RC: Somatosensory input from lumbar paraspinal tissues: Anatomical terminations and neuronal responses to mechanical and sympathetic stimuli, *Soc Neurosci Abst* 15:755, 1989.
341. Gillette RG: A speculative argument for the coactivation of diverse somatic receptor populations by forceful chiropractic adjustments, *Manipulative Med* 3:1, 1987.
342. Gillette RG, Kramis RC, Roberts WJ: Spinal neurons likely to mediate low back and referred leg pain, *Soc Neurosci Abstr* 16:1704, 1990.
343. Gillette RG, Kramis RC, Roberts WJ: Convergent input onto spinal neurons likely to mediate low back pain, 0000, 1991, 3rd IBRO World Congress of Neuroscience Abstracts Montreal, Canada.
344. Boal RW, Gillette RG: Central neuronal plasticity, low back pain and spinal manipulative therapy, *J Manipulative Physiol Ther* 27(5):314, 2004.
345. Korr IM: Proprioceptors and somatic dysfunction, *J Am Osteopath Assoc* 74:638, 1975.
346. Sandoz RW: Some reflex phenomena associated with spinal derangements and adjustments, *Ann Swiss Chiropr Assoc* 6:60, 1981.
347. Grice AS: Muscle tonus change following manipulation, *J Can Chiro Assoc* 18(4):29, 1974.
348. Watts DG, et al: Analysis of muscle receptor connection by spike-triggered averaging, spindle primary, and tendon organ afferents, *J Neurophysiol* 39(6):1378, 1976.
349. Zhu Y, Haldeman S, Hsieh C-YJ, et al: Do cerebral potentials to magnetic stimulation of paraspinal muscles reflect changes in palpable muscle spasm, low back pain, and activity scores? *J Manipulative Physiol Ther* 23:458, 2000.

350. Herzog W, et al: Reflex responses associated with manipulative treatments on the thoracic spine, *J Manipulative Physiol Ther* 18:223, 1995.
351. Suter E, et al: Reflex response associated with manipulative treatment of the thoracic spine, *J Neuromusculoskel Syst* 2:124, 1994.
352. Shambaugh P: Changes in electrical activity in muscles resulting from chiropractic adjustment: A pilot study, *J Manipulative Physiol Ther* 10:300, 1987.
353. Palmer BJ: The science of chiropractic: Its principles and philosophies, vol 1, Davenport, IA, 1906/1910, Palmer School of Chiropractic.
354. Stephenson RW: Chiropractic textbook, Davenport, IA, 1948, Palmer School of Chiropractic.
355. Haldeman S, Hammerich K: The evolution of neurology and the concept of chiropractic, *ACA J Chiropr* 7:S57, 1973.
356. Janse J: History of the development of chiropractic concepts: Chiropractic terminology. In Goldstein M, editor: The research status of spinal manipulative therapy, NINCDS Monograph No 15, DHEW Pub No 76-988, Washington, DC, 1975, US Government Printing Office.
357. Homewood AE: The neurodynamics of the vertebral subluxation, St Petersburg, FL, 1979, Valkyrie Press.
358. Leach RA, editor: In The chiropractic theories: A synopsis of scientific research, ed 2, Baltimore, 1986, Williams & Wilkins.
359. Crelin ES: A scientific test of the chiropractic theory, *Am Sci* 61:574, 1973.
360. Giles LG: A histological investigation of human lower lumbar intervertebral canal (foramen) dimensions, *J Manipulative Physiol Ther* 17(1):4, 1994.
361. Leach RA: Nerve compression hypothesis. In The chiropractic theories: A synopsis of scientific research, ed 2, Baltimore, 1986, Williams & Wilkins.
362. Coote JH: Somatic sources of afferent input as factors in aberrant autonomic, sensory and motor function. In Korr IM, editor: The neurobiologic mechanisms in manipulative therapy, New York, 1978, Plenum.
363. Kiyomi K: Autonomic system reactions caused by excitation of somatic afferents: Study of cutaneo-intestinal reflex. In Korr IM, editor: The neurobiologic mechanisms in manipulative therapy, New York, 1978, Plenum.
364. Sato A: The somatosympathetic reflexes: Their physiological and clinical significance, NINCDS Monograph No 15, DHEW Pub No 76-988, Washington, DC, 1975, US Government Printing Office.
365. Appenzeller O: Somatoautonomic reflexology: Normal and abnormal. In Korr IM, editor: The neurobiologic mechanisms in manipulative therapy, New York, 1978, Plenum.
366. Sato A, Swenson R: Sympathetic nervous system response to mechanical stress of the spinal column in rats, *J Manipulative Physiol Ther* 7(3):141, 1984.
367. Kunert W: Functional disorders of internal organs due to vertebral lesions, *Ciba Symp* 13:85, 1965.
368. Greenman PE: Principles of manual medicine, Baltimore, 1989, Williams & Wilkins.
369. Kellgren JH: The anatomical source of back pain, *Rheumatol Rehabil* 16:3, 1977.
370. Selye H: Stress and disease, New York, 1956, McGraw-Hill.
371. Selye H: The stress of life, New York, 1956, McGraw-Hill.
372. Selye H: Stress without distress, Philadelphia, 1974, JB Lippincott.
373. Thornton LM, Andersen BL: Psychoneuroimmunology examined: The role of subjective stress, *J Cell Sci* 2(4):66, 2006.
374. Cohen S, Herbert TB: Health psychology: Psychological factors and physical disease from the perspective of human psychoneuroimmunology, *Ann Rev Psychol* 47:113, 1996.
375. Chrousos GP, Gold PW: The concepts of stress and stress system disorders. Overview of physical and behavioral homeostasis, *J Am Med Assoc* 267:1244, 1992.
376. Glaser R, Kiecolt-Glaser JK: Handbook of human stress and immunity, San Diego, 1994, Academic Press.
377. McEwen BS, et al: The role of adrenocorticoids as modulators of immune function in health and disease: Neural, endocrine and immune interactions, *Brain Res Rev* 23(1-2):79, 1997.
378. Olff M: Stress, depression and immunity: The role of defense and coping styles, *Psychiatry Res* 85:7, 1999.
379. Rabin B: Stress, immune function, and health: The connection, New York, 1999, Wiley-Liss.
380. Reiche EMV, Nunes SOV, Morimoto HK: Stress, depression, the immune system, and cancer, *Lancet Oncol* 5(10):617, 2004.
381. Padgett DA, Glaser R: How stress influences the immune response, *Trends Immunol* 24(8):444, 2003.
382. Bellinger DL, et al: Psychoimmunology today: Mechanisms mediating the effects of psychological status on the immune function. In Lewis CE, O'Brien RM, Barraclough J, editors: The psychoimmunology of cancer, New York, 2002, Oxford University Press, pp 3-99.
383. Segerstrom SC, Miller GE: Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry, *Psychol Bull* 130(4):601, 2004.
384. Mason JW: A re-evaluation of the concept of non-specificity in stress theory, *Psychol Res* 8:323, 1971.
385. Stein M, Schiavi RC, Camerino M: Influence of brain and behavior in the immune system, *Science* 191:435, 1976.
386. Hess WR: Functional organization of the diencephalon, Orlando, FL, 1957, Grune & Stratton.
387. Pert C: Molecules of emotion, New York, 1997, Scribner's.
388. Leach RA: The chiropractic theories: A synopsis of scientific research, ed 3, Baltimore, 1994, Williams & Wilkins.
389. Fidelibus JC: An overview of neuroimmunomodulation and a possible correlation with musculoskeletal system manipulative function, *J Manipulative Physiol Ther* 12:289, 1989.
390. Wiberg JMM, Nordsteen J, Nilsson N: The short-term effect of spinal manipulation in the treatment of infantile colic: A randomized controlled clinical trial with a blinded observer, *J Manipulative Physiol Ther* 22(1):13, 1999.
391. Sanders GE, et al: Chiropractic adjustive manipulation on subjects with acute low back pain: Visual analog pain scores and plasma B-endorphin levels, *J Manipulative Physiol Ther* 13(7):391, 1990.
392. Christian GF, Stanton GJ, Sissons D: Immunoreactive ACTH, B-endorphin, and cortisol levels in plasma following spinal manipulative therapy, *Spine* 3(12):1411, 1998.
393. Whelan TL, et al: The effect of chiropractic manipulation on salivary cortisol levels, *J Manipulative Physiol Ther* 25(3):149, 2002.
394. Teodorezyk-Injeyan JA, Injeyan HS, Ruegg R: Spinal manipulative therapy reduces inflammatory cytokines but not substance P production in normal subjects, *J Manipulative Physiol Ther* 29:14, 2006.
395. Brennan PC, Kokjohn K, Kaltinger CJ, et al: Enhanced phagocytic cell respiratory burst induced by spinal manipulation: Potential role of substance P, *J Manipulative Physiol Ther* 14(7):399, 1991.
396. Brennan PC, et al: Immunologic correlates of reduced spinal mobility: Preliminary observations in a dog model. In Proceedings of the International Conference on Spinal Manipulation, Apr 12-13, 1991, Arlington, VA, 1991, Foundation for Chiropractic Education and Research, p 118.
397. Brennan PC, et al: Enhanced neutrophil respiratory burst as a biological marker for manipulation forces: duration of the effect and association with substance P and tumor necrosis factor, *J Manipulative Physiol Ther* 15(2):83, 1992.
398. Brennan PC, et al: Lymphocyte profiles in patients with chronic low back pain enrolled in a clinical trial, *J Manipulative Physiol Ther* 17(4):219, 1994.
399. Lohr GE, O'Brien JC, Nodine DL, et al: Natural killer cells as an outcome measure of chiropractic treatment efficacy. In Proceedings

- of the International Conference on Spinal Manipulation, 1990 May 11–12, Washington, DC, Arlington, VA, 1990, Foundation for Chiropractic Education and Research, p 109.
400. Graham MA, Brennan PC: Functional ability of natural killer cells as an outcome measure for chiropractic treatment efficacy. In Proceedings of the International Conference On Spinal Manipulation, 1991 Apr 12–13, Arlington, VA, 1991, Foundation for Chiropractic Education and Research, p 84.
 401. Kokjohn K, et al: The effect of spinal manipulation on pain and prostaglandin levels in women with primary dysmenorrheal, *J Manipulative Physiol Ther* 15(5):279, 1992.
 402. Triano JJ, et al: Respiratory burst (RB) activity as a function of manipulation site. In Proceedings of the International Conference on Spinal Manipulation, 1994 Jun 10–11, Palm Springs, Calif, Arlington, Va, 1994, Foundation for Chiropractic Education and Research, p 117.
 403. Kokjohn K, et al: Plasma substance P following spinal manipulation. In Proceedings of the 1990 International Conference on Spinal Manipulation, Arlington, VA, 1990, Foundation for Chiropractic Education and Research.
 404. Hoag JM, Cole WV, Bradford SG: Osteopathic medicine, New York, 1969, McGraw-Hill.
 405. Breeman NE: Decrease in blood volume after prolonged hyperactivity of the sympathetic nervous system, *Am J Physiol* 103:185, 1933.
 406. Greenman PE: Principles of craniosacral (inherent force) technique. In Principles of manual medicine, Baltimore, 1989, Williams & Wilkins.
 407. Gal J, et al: Movements of vertebrae during manipulative thrusts to unembalmed human cadavers, *J Manipulative Physiol Ther* 20(1):30, 1997.
 408. Kaltenborn FM: The spine: Basic evaluation and mobilization techniques, ed 2, Minneapolis, 1993, OPTP.
 409. Haas M: The physics of spinal manipulation. III. Some characteristics of adjusting that facilitate joint distractions, *J Manipulative Physiol Ther* 13(6):305, 1990.
 410. Haas M: The physics of spinal manipulation. IV. A theoretical consideration of the physician impact force and energy requirements to produce synovial joint cavitation, *J Manipulative Physiol Ther* 13(7):378, 1990.
 411. Maigne R: Localization of manipulations of the spine. In Orthopedic medicine, ed 3, Springfield, IL, 1979, Charles C Thomas.
 412. Good J: An analysis of diversified (leg artis) type adjustments based upon assisted-resisted model of intervertebral motion unit prestress, *Chiropr Tech* 4:117, 1992.
 413. Wells D: From workbench to high tech: The evaluation of the adjustment table, *Chiropr Hist* 7(2):35, 1987.
 414. Bergmann TF, Davis PT: Mechanically assisted manual techniques: Distraction procedures, St Louis, 1998, Mosby.
 415. Plaughner G, Lopes MA: The knee-chest table: Indications and contraindications, *J Chiro Tech* 2(4):163, 1990.
 416. Holm SM, Rose KA: Work-related injuries of doctors of chiropractic in the United States, *J Manipulative Physiol Ther* 29:518, 2006.
 417. Haas M: The physics of spinal manipulation. I. The myth of $F = ma$, *J Manipulative Physiol Ther* 13(4):204, 1990.
 418. Haas M: The physics of spinal manipulation. II. A theoretical consideration of the adjustive force, *J Manipulative Physiol Ther* 13:253, 1990.
 419. Bereznick DE, Kim Ross KJ, McGill SM: Where should forces be applied to produce cavitation. In Proceedings of the Association of Chiropractic Colleges and the Research Agenda Conference, Phoenix, 2006, 0000.
 420. Schafer RC, Faye LJ: Motion palpation and chiropractic technic: Principles of dynamic chiropractic, ed 1, Huntington Beach, CA, 1989, Motion Palpation Institute.
 421. Jackson RD: Thompson terminal point technique, *Today's Chiropr* 16(3):73, 1987.
 422. Bergmann TF: Manual force, mechanically assisted articular chiropractic technique using long and/or short levers: A literature review, *J Manipulative Physiol Ther* 16:33, 1993.
 423. Thompson JC: Thompson technique reference manual, Elgin, IL, 1984, Williams Manufacturing.
 424. Taylor H: The McManis table: Professional papers, *ACA J Chiropr* 12:87, 1978.
 425. Mennell JMcM: Joint pain, Boston, 1964, Little, Brown and Company.

CHAPTER 5

1. Ross JK, Bereznick D, McGill S: Determining cavitation location during lumbar and thoracic spinal manipulation, *Spine* 29(13):1452–1457, 2004.
2. Ianuzzi A, Khalsa PS: Comparison of human lumbar facet joint capsule strains during simulated high-velocity, low-amplitude spinal manipulation versus physiological motions, *Spine* 5(3):277, 2005.
3. Reggars JW, Pollard HP: Analysis of zygapophyseal joint cracking during chiropractic manipulation, *J Manipulative Physiol Ther* 18:65, 1995.
4. Beffa R, Mathews R: Does the adjustment cavitate the targeted joint? An investigation into the location of cavitation sounds, *J Manipulative Physiol Ther* 27(2):e2, 2004.
5. White AA, Panjabi MM: Clinical biomechanics of the spine, ed 2, Philadelphia, 1990, JB Lippincott.
6. Harrison DD: Ideal normal upright static spine. In Harrison DD, editor: Spinal Biomechanics: a chiropractic perspective, Evanston, WY, 1992, Harrison CBP Seminars, pp 33–42.
7. Panjabi M, et al: Three-dimensional movements of the upper cervical spine, *Spine* 13:726, 1988.
8. Harrison DL, Harrison DD: Chiropractic: Spinal mechanics and human biophysics, Sunnyvale, CA, 1980, Harrison Chiropractic Seminars.
9. Jackson R: The cervical syndrome, Springfield, IL, 1977, Charles C Thomas.
10. Pal GP, Sherk HH: The vertical stability of the cervical spine, *Spine* 13:447, 1988.
11. Pierce VW: Results, Dravosburg, PA, 1979, Chirp.
12. Jochumsen OH: The curve of the cervical spine, *J Am Chiropr Assoc* 7:549, 1970.
13. Suh CH: Computer model of the spine. In Haldeman S, editor: Modern developments in the principles and practice of chiropractic, East Norwalk, CT, 1980, Appleton-Century-Crofts.
14. Lysell E: Motion in the cervical spine, *Acta Orthop Scand Suppl* 126:1, 1969.
15. Rasch PJ, Burke RK: Kinesiology and applied anatomy, ed 5, Philadelphia, 1974, Lea & Febiger.
16. Krag MH, et al: Internal displacement: Distribution from in vitro loading of human thoracic and lumbar spinal motion segments—Experimental results and theoretical predictions, *Spine* 12:1001, 1987.
17. White AA, et al: Biomechanical analysis of clinical stability in the cervical spine, *Clin Orthop* 109:85, 1975.
18. MacRae JE: Roentgenometrics in chiropractic, Toronto, 1974, Canadian Memorial Chiropractic College.
19. Bernhardt M, Bridwell KH: Segmental analysis of the sagittal plane alignment of the normal thoracic and lumbar spines and the thoracolumbar junction, *Spine* 14:717, 1989.
20. Bradford S: Juvenile kyphosis. In Bradford DS, et al, editors: Moe's textbook of scoliosis and other spinal deformities, Philadelphia, 1987, WB Saunders.
21. Pratt NE: Clinical musculoskeletal anatomy, Philadelphia, 1991, JB Lippincott.
22. Kendall HO, Kendall FP, Boyton DA: Posture and pain, Huntington, NY, 1952.

23. Jahn WT, Griffiths JH, Hacker RA: Conservative management of Scheuermann's juvenile kyphosis, *J Manipulative Physiol Ther* 1:228, 1978.
24. Gatterman MI, Panzer DM: Disorders of the thoracic spine. In Gatterman MI, editor: *Chiropractic management of spine related disorders*, Baltimore, 1990, William's & Wilkins.
25. Panjabi MM, et al: Thoracic spine centers of rotation in the sagittal plane, *J Orthop Res* 1:387, 1984.
26. Panjabi MM, Brand RA, White AA: Three dimensional flexibility and stiffness properties of the human thoracic spine, *J Biomech* 9:185, 1976.
27. Miles M, Sullivan WE: Lateral bending at the lumbar and lumbosacral joints, *Anat Rec* 139:387, 1961.
28. Punjabi MM, et al: How does posture affect the coupling? *Spine* 14:1002, 1989.
29. Hollinsted WH, Cornelius R: *Textbook of anatomy*, ed 4, Philadelphia, 1985, Harper & Row.
30. Davis PR: The thoracolumbar mortise joint, *J Anat* 89:370, 1955.
31. Maigne R: Low back pain from thoracolumbar origin, *Arch Phys Med Rehabil* 61:389, 1980.
32. Bereznic D, Kim Ross KJ, McGill S: The frictional properties at the thoracic skin-fascia interface: Implications in spine manipulation, *Clin Biomech* 17:297, 2002.
33. King AI, Prasad P, Ewing CL: Mechanism of spinal injury due to caudocephalad acceleration, *Orthop Clin North Am* 6:19, 1975.
34. Adams MA, Hutton WC: The effects of posture on the role of the apophyseal joints in resisting intervertebral compression forces, *J Bone Joint Surg Br* 62:358, 1980.
35. Bernhardt M, Bridwell KH: Segmental analysis of the sagittal plane alignment of the normal thoracic and lumbar spines and the thoracolumbar junction, *Spine* 14:717, 1989.
36. Moe JH, Bradford DS: *Kyphosis-lordosis: General principles. Scoliosis and other spinal deformities*, Philadelphia, 1978, Saunders.
37. DeSmet AA: Radiographic evaluation. In DeSmet AA, editor: *Radiology of spinal curvature*, St Louis, 1985, Mosby.
38. Propst-Proctor SL, Bleck EE: Radiographic determination of lordosis and kyphosis in normal and scoliotic children, *J Pediatr Orthop* 3:344, 1983.
39. Percy M, Portek I, Shepard J: Three dimensional x-ray analysis of normal movement in the lumbar spine, *Spine* 9:294, 1984.
40. Percy MJ: Stereo radiography of normal lumbar spine motion, *Acta Orthop Scand* 56(Suppl):212, 1985.
41. Posner I, et al: A biomechanical analysis of the clinical stability of the lumbar and lumbosacral spine, *Spine* 7:374, 1982.
42. Miles M, Sullivan WE: Lateral bending at the lumbar and lumbosacral joints, *Anat Rec* 139:387, 1961.
43. Grice A: Radiographic, biomechanical and clinical factors in lumbar lateral flexion. I, *J Manipulative Physiol Ther* 2:26, 1979.
44. Cassidy JD: Roentgenological examination of the functional mechanics of the lumbar spine in lateral flexion, *J Can Chiropr Assoc* 20:13, 1976.
45. Bronfort G, Jochumsen OH: The functional radiographic examination of patients with low-back pain: A study of different forms of variations, *J Manipulative Physiol Ther* 7:89, 1984.
46. Dimnet J, et al: Radiographic studies of lateral flexion in the lumbar spine, *J Biomech* 11:143, 1978.
47. Dupuis PR, et al: Radiologic diagnosis of degenerative lumbar spinal instability, *Spine* 10:262, 1985.
48. Dvorak J, et al: Functional radiographic diagnosis of the lumbar spine, *Spine* 16:562, 1991.
49. Dvorak J, et al: Clinical validation of functional flexion-extension roentgenograms of the lumbar spine, *Spine* 16:943, 1991.
50. Frymoyer JW, et al: The mechanical and kinematic analysis of the lumbar spine in normal living human subjects in vivo, *J Biomech* 12:165, 1979.
51. Hanley EN, Matteri RE, Frymoyer JW: Accurate roentgenographic determination of lumbar flexion-extension, *Clin Orthop Rel Res* 115:145, 1976.
52. Korpi J, Poussa M, Heliovaara M: Radiographic mobility of the lumbar spine and its relation to clinical back motion, *Scand J Rehabil Med* 20:71, 1988.
53. Phillips RB, et al: Stress x-rays and the low back pain patient, *J Manipulative Physiol Ther* 13:127, 1990.
54. Sandoz RW: Technique and interpretation of functional radiography of the lumbar spine, *Ann Swiss Chiro Assoc* 3:66, 1965.
55. Shaffer WO, et al: The consistency and accuracy of roentgenograms for measuring sagittal translation in the lumbar vertebral motion segment: An experimental model, *Spine* 15:741, 1990.
56. Soini J, et al: Disc degeneration and angular movement of the lumbar spine: Comparative study using plain and flexion-extension radiography and discography, *J Spinal Disord* 4:183, 1991.
57. Stokes IAF, et al: Assessment of patients with low-back pain by biplanar radiographic measurement of intervertebral motion, *Spine* 6:233, 1981.
58. Tanz SS: Motion of the lumbar spine, *Am J Roentgenol* 69:399, 1953.
59. Van Akkerveeken PF, O'Brien JP, Park WM: Experimentally induced hypermobility in the lumbar spine, *Spine* 4:236, 1979.
60. Vernon H: Static and dynamic roentgenography in the diagnosis of degenerative disc disease: A review and comparison assessment, *J Manipulative Physiol Ther* 5:163, 1982.
61. Weitz EM: The lateral bending sign, *Spine* 6:388, 1981.
62. Haas M, et al: Lumbar motion trends and correlation with low back pain. I. A roentgenological evaluation of coupled lumbar motion in lateral bending, *J Manipulative Physiol Ther* 15:145, 1992.
63. Percy MJ, Tibrewal SB: Axial rotation and lateral bending in the normal lumbar spine measured by three-dimensional radiography, *Spine* 9:582, 1984.
64. Bereznic DE, Kim Ross KJ, McGill SM: Where should forces be applied to produce cavitation? In *Proceedings of the Association of Chiropractic Colleges and the Research Agenda Conference*, Phoenix, 2006.
65. Cramer D, et al: Effects of side-posture positioning and side-posture adjusting on the lumbar zygapophyseal joints as evaluated by magnetic resonance imaging: A before and after study with randomization, *J Manipulative Physiol Ther* 23(6):380, 2000.
66. Cramer GD, et al: The effects of side-posture positioning and spinal adjusting on the lumbar Z joints: A randomized controlled trial with sixty-four subjects, *Spine* 27(2):2459, 2002.
67. Grieve GP: *Common vertebral joint problems*, ed 2, Edinburgh, UK, 1988, Churchill Livingstone.
68. Cox HH: Sacroiliac subluxations as a cause of backache, *Surg Gynecol Obstet* 45:637, 1927.
69. Jessen AR: The sacroiliac subluxation, *ACA J Chiro* 7(Suppl):65, 1973.
70. Cyriax E: Minor subluxations of the sacroiliac joints, *Br J Phys Med* 9:191, 1934.
71. Dontigney RL: A review, *Phys Ther* 65:35, 1985.
72. Solonen KA: The sacroiliac joint in the light of anatomical, roentgenological and clinical studies, *Acta Orthop Scand Suppl* 26:9, 1957.
73. Bowen V, Cassidy JD: Macroscopic and microscopic anatomy of the sacroiliac joint from embryonic life until the eighth decade, *Spine* 6:620, 1986.
74. Otter R: Review study of differing opinions expressed in the literature about the anatomy of the sacroiliac joint, *Eur J Chiro* 33:221, 1985.
75. McGregor M, Cassidy JD: Post-surgical sacroiliac joint syndrome, *J Manipulative Physiol Ther* 6:1, 1983.
76. Grieve GP: The sacroiliac joint, *Physiotherapy* 62:384, 1976.
77. Frigerio NA, Stowe RR, Howe JW: Movement of the sacroiliac joint, *Clin Orthop* 100:370, 1974.

78. Grice AS, Fligg DB: Biomechanics of the pelvis: Denver conference monograph, Des Moines, IA, 1980, ACA Council of Technic.
79. Grice AS: Mechanics of walking, development and clinical significance, *J Can Chiropr Assoc* 16:15, 1972.
80. Schafer RC, Faye LJ: Motion palpation and chiropractic technic: Principles of dynamic chiropractic, Huntington Beach, CA, 1989, Motion Palpation Institute.
81. Hungerford BA, et al: Evaluation of the ability of Physical therapist to palpate intrapelvic motion with the stork test on the support side, *Phys Ther* 87(7):879, 2007.
82. Illi F: The vertebral column: Lifeline of the body, Chicago, 1951, National College of Chiropractic.
83. Greenman P: Principles of manual medicine, Baltimore, 1989, Williams & Wilkins.
84. Stureson B, Selvik G, Uden A: Movements of the sacroiliac joints: A roentgen stereophotogrammetric analysis, *Spine* 14:162, 1989.
85. Gatterman MI: Chiropractic management of spine related disorders, Baltimore, 1990, Williams & Wilkins.
86. Dupuis PR, Kirkaldy-Willis WH: The spine: Integrated function and pathophysiology. In Cruess RL, Rennie WRJ, editors: Adult orthopaedics, New York, 1984, Churchill Livingstone.
19. Hoppenfeld S: Physical examination of the spine and extremities, Norwalk, CT, 1976, Appleton-Century-Crofts.
20. Upton ARM, McComas AJ: The double crush hypothesis in nerve entrapment syndromes, *Lancet* 2:359, 1973.
21. Kapandji IA: The physiology of the joints, vol 2, Edinburgh, UK, 1970, Churchill Livingstone.
22. Ferezy JS: Chiropractic management of meralgia paresthetica: A case report, *J Chiro Tech* 1(2):52, 1989.
23. Neumann DA: In Kinesiology of the Musculoskeletal System, St Louis, 2002, Mosby, p 435.
24. Kotwicz JA: Biomechanics of the foot and ankle, *Clin Sports Med* 1:19, 1982.

CHAPTER 6

1. Palmer DD: The chiropractor's adjuster, Davenport, IA, 1910, Palmer School of Chiropractic.
2. Broome RT: The relevance of peripheral joints in clinical practice: An overview. In Broome RT, editor: Chiropractic peripheral joint technique, Oxford, UK, 2000, Butterworth-Heinemann.
3. Hertling D, Kessler RM: Management of common musculoskeletal disorders: Physical therapy principles and methods, ed 2, Philadelphia, 1990, JB Lippincott.
4. Pinto OF: A new structure related to temporomandibular joint and the middle ear, *J Prosthet Dent* 12:95, 1962.
5. Ermshar CB: Anatomy and neurology. In Morgan DH, et al, editors: Diseases of the temporomandibular apparatus, ed 2, St Louis, 1985, Mosby.
6. Farrar WB, McCarty WL: A clinical outline of the temporomandibular joint: Diagnosis and treatment, Montgomery, AL, 1983, Walter.
7. Curl D: Acute closed lock of the temporomandibular joint: Manipulative paradigm and protocol, *J Chiro Tech* 3(1):13, 1991.
8. Kraus SL, editor: TMJ disorders: Management of the craniomandibular complex, New York, 1987, Churchill Livingstone.
9. Schoenholtz F: Conservative management of temporomandibular joint dysfunction, *J Am Chiro Assoc* 12(Suppl):57, 1978.
10. Türp JC, Minagi S: Palpation of the lateral pterygoid region in TMD—Where is the evidence?, *J Dent* 29(7):475, 2001.
11. Stratmann U, et al: Clinical anatomy and palpability of the inferior lateral pterygoid muscle, *J Prosthet Dent* 83(5):548, 2000.
12. Long JH Jr: Occlusal adjustment as treatment for tenderness in the muscles of mastication in category patients, *J Prosthet Dent* 67(4):519, 1992.
13. Thomas CA, Okeson JP: Evaluation of lateral pterygoid muscle symptoms using a common palpation technique and a method of functional manipulation, *Cranio* 5(2):125, 1987.
14. Stelzenmüller W, et al: Is the lateral pterygoid muscle palpable? A pilot study for determining the possibilities of palpating the lateral pterygoid muscle, *J Dent Oral Med* 8(1): 325, 2006.
15. Wadsworth CT: Manual examination and treatment of the spine and extremities, Baltimore, 1988, Williams & Wilkins.
16. Kapandji IA: The physiology of the joints, ed 2, vol 1, Edinburgh, UK, 1970, Churchill Livingstone.
17. Nordin M, Frankel VH: Basic biomechanics of the musculoskeletal system, ed 2, Philadelphia, 1989, Lea & Febiger.
18. Donnatelli R, Wooden MJ, editors: Orthopedic physical therapy, New York, 1989, Churchill Livingstone.
1. Cassidy JD, Lopes AA, Yong-Hing K: The immediate effect of manipulation versus mobilization on pain and range of motion in the cervical spine: A randomized controlled trial, *J Manipulative Physiol Ther* 15:570, 1992.
2. Kenna C, Murtagh J: Back pain and spinal manipulation, Sydney, Australia, 1989, Butterworths.
3. Grieve G: Mobilization of the spine, ed 4, Edinburgh, UK, 1984, Churchill Livingstone.
4. Bergmann TF: Various forms of chiropractic technique, *Chiro Tech* 5(2):53, 1993.
5. Haldeman S: Spinal manipulative therapy and sports medicine, *Clin Sports Med* 5(2):277, 1986.
6. Tuchin C, Mootz RD: Mobilization concepts and their application to mechanical dysfunction in the lower extremity, *Chiro Tech* 7(3):82, 1995.
7. Maitland GD: Vertebral manipulation, ed 3, London, 1973, Butterworths.
8. Paris SV: Mobilization of the spine, *Phys Ther* 59:988, 1979.
9. Eder M, Tilscher H: Chiropractic therapy, diagnosis and treatment, Rockville, MD, 1990, Aspen.
10. Kaltenborn FM: Mobilization of the extremity joints, ed 3, Oslo, 1980, Olaf Norlis Bokhandel.
11. Grieve GP: Common vertebral joint problems, New York, 1988, Churchill Livingstone.
12. Sheriff J: A flexible approach to traction. In Grieve GP, editor: Modern manual therapy, ed 2, Edinburgh, UK, 1994, Churchill Livingstone.
13. Taylor H: The McManis table: Professional papers, *ACA J Chiropr* 12:100, 1978.
14. McManis JV: A treating table innovation, *J Am Osteopath Assoc* 565, 1910 July.
15. Cox JM: Low back pain: Mechanism, diagnosis, and treatment, Baltimore, 1985, Williams & Wilkins.
16. Bergmann TF, Davis PT: Mechanically assisted manual techniques: Distraction procedures, St Louis, 1997, Mosby.
17. Donelson R: A prospective study of centralization of lumbar and referred pain, *Spine* 22(10):1115, 1997.
18. Donelson R, Silva B, Murphy K: Centralization phenomenon: Its usefulness in evaluating and treating referred pain, *Spine* 15(3):211, 1990.
19. McKenzie RA: A perspective on manipulative therapy, *Physiotherapy* 75:440, 1989.
20. Moss JM: Cervical and lumbar pain syndromes. In Boyling JD, Palastanga N, editors: Grieve's modern manual therapy, ed 2, New York, 1994, Churchill Livingstone.
21. Robinson MG: The McKenzie method of spinal pain management. In Boyling JD, Palastanga N, editors: Grieve's modern manual therapy, ed 2, New York, 1994, Churchill Livingstone.
22. Retzlaff E, Mitchell F: The cranium and its sutures, New York, 1987, Springer-Verlag.
23. Pritchard JJ: The structure and development of cranial and facial sutures, *J Anat* 90:73, 1956.
24. Frymann VM: A study of the rhythmic motions of the living cranium, *J Am Osteopath Assoc* 70:1, 1971.

25. Ebner JA: An overview of cranial manipulation. In Curl DD, editor: *Chiropractic approach to head pain*, Baltimore, 1994, Williams & Wilkins.
26. Sutherland W: *The cranial bowl*, Mankato, MN, 1939, Free Press.
27. Upledger J, Vredevoogd J: *Craniosacral therapy*, Seattle, 1983, Eastland Press.
28. DeJarnette M: *Cranial technique*, Nebraska City, NE, 1973, Author.
29. Meeker WC: Soft tissue and non-force techniques. In Haldeman S, editor: *Principles and practice of chiropractic*, Norwalk, CT, 1992, Appleton & Lange.
30. Lantz CA: Immobilization degeneration and the fixation hypothesis of chiropractic subluxation, *Chiro Res J* 1(1):21, 1988.
31. Rahlmann JF: Mechanisms of intervertebral joint fixation: A literature review, *J Manipulative Physiol Ther* 10(4):177, 1987.
32. Lewit K: *Manipulative therapy in rehabilitation of the locomotor system*, Boston, 1985, Butterworths.
33. Janda V: Muscle spasm: A proposed procedure for differential diagnosis, *J Manual Med* 6:136, 1991.
34. Liebensohn C: Active muscular relaxation techniques. I. Basic principles and methods, *J Manipulative Physiol Ther* 12(6):446, 1989.
35. Bogduk N, Twomey LT: *Clinical anatomy of the lumbar spine*, ed 2, Melbourne, Australia, 1991, Churchill Livingstone.
36. Beal MC: Viscerosomatic reflexes: A review, *J Amer Osteopath Assoc* 85(12):53, 1985.
37. Wakim KG: The effects of massage on the circulation in normal and paralyzed extremities, *Arch Phys Med* 30:135, 1949.
38. Wolfson H: Studies on the effect of physical therapeutic procedures on function and structure, *JAMA* 96:2020, 1931.
39. Carrier EB: Studies on physiology of capillaries: Reaction of human skin capillaries to drugs and other stimuli, *Am J Physiol* 61:528, 1922.
40. Martin GM, Roth GM: Cutaneous temperature of the extremities of normal subjects and patients with rheumatoid arthritis, *Arch Phys Med Rehab* 27:665, 1946.
41. Ernst E, Matrai A, Magyarosy IE: Massage causes changes in blood fluidity, *Physiotherapy* 73(1):43, 1987.
42. Cuthbertson DP: Effect of massage on metabolism: A survey, *Glasgow Med J* 2:200, 1933.
43. Schneider EC, Havens LC: Changes in the contents of hemoglobin and red corpuscles in the blood of men at high altitudes, *Am J Physiol* 36:360, 1915.
44. Hernandez-Reif M, et al: High blood pressure and associated symptoms were reduced by massage therapy, *J Body Mov Ther* 4(1):31, 2000.
45. Barr JS, Taslitz N: The influence of back massage on autonomic functions, *J Phys Ther* 50(12):1679, 1970.
46. Tovar MK, Cassmere VL: Touch: The beneficial effects for the surgical patient, *AORN J* 49:1356, 1989.
47. Cassar M: *Handbook of massage therapy*, Oxford, UK, 1999, Butterworth-Heinemann.
48. Yu C: 55 cases of lumbar muscle strain treated by massage, *Int J Clin Acupunct* 10(2):189, 1999.
49. Goats GC: Massage: The scientific basis of an ancient art. I. The techniques, *Br J Sports Med* 28(3):149, 1994.
50. Morelli M, Sullivan SJ, Chapman CE: Inhibitory influence of soleus massage onto the medial gastrocnemius H-reflex, *Electromyogr Clin Neurophysiol* 38(2):87, 1998.
51. Goldberg J, Sullivan SJ, Seaborne DE: The effect of two intensities of massage on H-reflex amplitude, *Phys Ther* 72(6):449, 1992.
52. Field TM: Massage therapy effects, *Am Psychol* 53(12):1270, 1998.
53. Hofkosh JM: Classical massage. In Basmajian JV, editor: *Manipulation, traction and massage*, ed 3, Baltimore, 1985, Williams & Wilkins.
54. Beard G, Wood EC: *Massage: Principles and techniques*, Philadelphia, 1964, Saunders.
55. Fritz S: *Fundamentals of therapeutic massage*, St Louis, 1995, Mosby.
56. Ebner M: Connective tissue massage, *Physiotherapy* 64(7):208, 1978.
57. Cantu RI, Grodin AJ: *Myofascial manipulation theory and clinical application*, Gaithersburg, MD, 1992, Aspen.
58. Bischof I, Elmiger G: Connective tissue massage. In Licht S, editor: *Massage manipulation and traction*, Huntington, NY, 1976, Robert E Krieger.
59. Greenman PE: *Principles of manual medicine*, Baltimore, 1989, Williams & Wilkins.
60. Jones LH, Kusunose R, Goering E: *Jones' Strain-Counterstrain*, Boise, ID, 1995, Authors.
61. D'Ambrogio KJ, Roth GB: *Positional release therapy*, St Louis, 1997, Mosby.
62. Barnes JF: Myofascial release. In Hammer WI, editor: *Functional soft tissue examination and treatment by manual methods*, ed 2, Gaithersburg, MD, 1999, Aspen.
63. Barnes JF: Why myofascial release is unique, *Clin Bull Myofascial Ther* 2(1):43, 1997.
64. Basmajian JV, Nyberg R: *Rational manual therapies*, Baltimore, 1993, Williams & Wilkins.
65. Loving JE: *Massage therapy theory and practice*, Stamford, CT, 1999, Appleton & Lange.
66. Leahy PM: Active release techniques: Logical soft tissue treatment. In Hammer WI, editor: *Functional soft tissue examination and treatment by manual methods*, ed 2, Gaithersburg, MD, 1999, Aspen.
67. Mitchell FL: Elements of muscle energy technique. In Basmajian JV, Nyberg R, editors: *Rational manual therapies*, Baltimore, 1993, Williams & Wilkins.
68. Sherrington CS: On plastic tonus and proprioceptive reflexes, *Quart J Exp Physiol* 109, 1909.
69. Chaitow L: *Muscle energy techniques*, New York, 1996, Churchill Livingstone.
70. Evjenth O, Hamberg J: *Muscle stretching in manual therapy: A clinical manual*, Alfta, Sweden, 1984, Alfta Rehab.
71. Voss DE, Ionta MK, Meyers BJ: *Proprioceptive neuromuscular facilitation*, ed 3, Philadelphia, 1984, Harper & Row.
72. Basmajian JV: *Manipulation traction and massage*, ed 3, Baltimore, 1985, Williams & Wilkins.
73. Travell J, Simons DG: *Myofascial pain and dysfunction: The trigger point manual*, Baltimore, 1983, Williams & Wilkins.
74. Cohen JC, Gibbons RW: Raymond Nimmo and the evolution of trigger point therapy, 1929–1986, *J Manipulative Physiol Ther* 21:167, 1998.
75. Schneider MJ, Cohen JH: Nimmo receptor tonus technique: A chiropractic approach to trigger point therapy. In Sweere JJ, editor: *Chiropractic family practice*, Gaithersburg, MD, 1992, Aspen.
76. Nimmo RL: The receptor and tonus control method defined, *Receptor* 1:1, 1957.
77. Cohen JH, Schneider MJ: Receptor-tonus technique: An overview, *Chiro Tech* 2(1):13, 1990.
78. Chaitow L: *Soft-tissue manipulation*, Rochester, VT, 1988, Healing Arts Press.
79. Wakim KG: Physiologic effects of massage. In Basmajian JV, editor: *Manipulation, traction and massage*, ed 3, Baltimore, 1985, Williams & Wilkins.
80. Birch S, Jamison RN: Controlled trial of Japanese acupuncture for chronic myofascial neck pain: Assessment of specific and nonspecific effects of treatment, *Clin J Pain* 14(3):248, 1998.
81. Abuaisha BB, Costanzi JB, Boulton AJ: Acupuncture for the treatment of chronic painful peripheral diabetic neuropathy: A long-term study, *Diabetes Res Clin Pract* 39(2):115, 1998.
82. Belluomini J, et al: Acupressure for nausea and vomiting of pregnancy: A randomized, blinded study, *Obstet Gynecol* 84(2):245, 1994.
83. Petrie JP, Hazleman BL: A controlled study of acupuncture in neck pain, *Br J Rheumatol* 25(3):271, 1986.
84. Ernst E: Acupuncture as a symptomatic treatment of osteoarthritis: A systematic review, *Scand J Rheumatol* 26(6):444, 1997.
85. Allison DB, et al: The randomized placebo controlled clinical trial of an acupressure device for weight loss, *Int J Obs Relat Metab Disord* 19(9):653, 1995.

86. Woolham CH, Jackson AO: Acupuncture in the management of chronic pain, *Anesthesia* 53(6):593, 1998.
87. Acupuncture, NIH Consens Statement 15(5):1-34, 1997.
88. Boley J: Acupuncture and electro-therapeutic research, *Acupunct Electrother Res* 9(2):79, 1984.
89. Melzack R, Stillwell DM, Fox EJ: Trigger points and acupuncture points of pain, *Pain* 3:3, 1977.
90. Mannino R: The application of neurological reflexes to the treatment of hypertension, *J Am Osteopath Assoc* 79(4):225, 1979.
91. Bennett TJ: Dynamics of correction of abnormal function, Sierra Madre, CA, 1977, Ralph J Martin.
92. Nelson WA: Diabetes mellitus: Two case reports, *Chiro Tech* 1(2):37, 1989.
93. Grainger HG: The somatic component in visceral disease. In *Academy of Applied Osteopathy 1958 Yearbook*, Newark, OH, 1958, American Academy of Osteopathy.
94. Korr I: Spinal cord as organizer of disease process. In *Academy of Applied Osteopathy 1976 Yearbook*, Newark, OH, 1976, American Academy of Osteopathy.
95. Nelson WA: Rheumatoid arthritis: A case report, *Chiro Tech* 2(1):17, 1990.
96. Logan HB: *Textbook of Logan basic methods*, St Louis, 1950, Author.
97. Lawson DA: Logan basic technique: Short and long lever, mechanical assisted. In *Proceedings of the 6th Annual CORE*, Monterey, CA, 1991, CORE.
98. Janse JJ: *Principles and practice of chiropractic*, Lombard, IL, 1947, National College of Chiropractic.
99. Sato A: The somatosympathetic reflexes: Their physiologic and clinical significance. In Goldstein M, editor: *The research status of spinal manipulative therapy*, Washington, DC, 1975, US Government Printing Office.
100. Homewood AE: *The neurodynamics of the vertebral subluxation*, St Petersburg, FL, 1979, Valkyrie Press.
101. Gitelman R: The treatment of pain by spinal manipulation. In *The research status of spinal manipulative therapy*, NINCDS Monograph No 15, DHEW Pub No 76-988, Washington, DC, 1975, US Government Printing Office.
102. Gillette RG: A speculative argument for the coactivation of diverse somatic receptor populations by forceful chiropractic adjustments, *Manipulative Med* 3:1, 1987.
103. Sato A: Spinal reflex physiology. In Haldeman S, editor: *Principles and practice of chiropractic*, ed 2, Norwalk, CT, 1992, Appleton & Lange.
104. Zucker A: Chapman's reflexes: Medicine or metaphysics? *J Am Osteopath Assoc* 93(3):346, 1993.

This page intentionally left blank

- A**
- Abbreviations
 - spinal, 174b
 - and symbols for recording joint dysfunction, 82f
 - Abduction
 - body planes and axes associated with, 12–13, 13f, 13t
 - definition of, 14
 - in hip movements, 341–343, 343f, 343t
 - Acceleration
 - of muscles, 27–28
 - and Newton's laws of motion, 25–26
 - Accessory joint movements
 - of ankle and foot, 370t
 - description and illustration of, 69–71, 69f, 70b
 - of elbow, 320b
 - of hip, 345b
 - of knee, 356t
 - of wrist and hand, 332t
 - Accreditation
 - and licensure of chiropractors, 6
 - Acetabulum, 337–339, 338f
 - Acromioclavicular joint
 - sitting adjustments, 305b, 310, 311f
 - supine adjustments, 305b, 310, 310f, 311f
 - Active mobility
 - and proprioceptive neuromuscular facilitation (PNF), 405–408, 406b, 407f, 408f
 - Active range-of-motion (AROM)
 - description and illustration of, 67–72, 68f
 - Active release technique (ART)
 - of myofascial release, 394b, 400–401, 401b, 401f
 - Acupressure point stimulation, 394b, 410–412, 411f, 412f
 - Acute locked low back, 30
 - Adams test, 56, 57f, 57t
 - Adduction
 - body planes and axes associated with, 12–13, 13f, 13t
 - definition of, 14
 - in hip movements, 341–343, 343f, 343t
 - Adjustive localization, 122b, 123–128
 - physiological *vs.* unphysiological movement, 123–124, 124f
 - reduction of articular slack, 124–127
 - Adjustive procedures; *See also* adjustive therapy; adjustments (specific techniques)
 - ankle and foot
 - intermetatarsal, 372b, 378, 378f
 - intertarsal joint, 372b, 377, 377f
 - Adjustive procedures (*Continued*)
 - metatarsophalangeal joint, 372b, 378, 378f
 - subtalar joint, 372b, 374, 375f
 - tarsometatarsal joint, 372b, 375–376, 376f
 - tibiotalar joint, 371, 372b, 373f, 374f
 - cervical spine
 - lower, 162f, 180, 180b, 181f, 182f, 184f, 185f, 186f, 187f
 - overview of, 170–174, 170f, 171f, 172f, 173f, 174b, 174f
 - upper, 174–188, 174b, 175b, 175f, 176f, 177f, 178f, 179f
 - elbow
 - sitting, 322–324, 322f, 323f, 324f
 - supine, 325, 325f
 - hand and wrist, 311, 334b, 334f, 335f, 336f, 337, 337f
 - hip, 346–349, 346b, 346f, 347f, 348f, 349f
 - knee, 358–364, 360b
 - femorotibial joint, 358–362, 360b, 361f
 - patellofemoral joint, 360b, 362, 363f
 - tibiofibular joint, 362–364, 363f, 365f
 - lumbar spine
 - flexion and extension, 252–253, 253f
 - knee-chest, 247, 253b, 260–261, 260f, 261f
 - lateral flexion, 251–252, 251f, 252f
 - prone, 247, 248f, 253b, 258–260, 259f, 260f
 - rotational, 248–251, 248f, 250f, 251f
 - side-posture, 245, 245f, 246f, 247f, 253–262, 253b, 254f, 256f, 257f, 258f
 - sitting, 248, 248f, 253b, 261–262, 261f
 - shoulder
 - acromioclavicular joint (sitting), 305b, 310, 311f
 - acromioclavicular joint (supine), 305b, 310, 310f, 311f
 - glenohumeral joint (prone), 304–307, 305b, 305f, 306f, 307f, 308f
 - glenohumeral joint (sitting), 304–307, 305b, 309f
 - glenohumeral joint (standing), 304–307, 305b, 309f
 - sternoclavicular joint (sitting), 305b, 311–313, 314f
 - sternoclavicular joint (supine), 305b, 311–313, 312f, 313f
 - temporomandibular joint (TMJ), 291–294, 291b, 292f, 293f, 294f
 - Adjustive procedures (*Continued*)
 - thoracic spine
 - flexion and extension, 209–210, 209f, 210f, 211f
 - lateral flexion dysfunction, 205f, 207f, 208–209, 208f, 209f
 - ribs, 211, 211f, 227f, 230f, 231f
 - rotational dysfunction, 204–208, 205f, 206f, 207f
 - Adjustive specificity, 127–128, 128f
 - Adjustive therapy
 - adjustive localization, 122b, 123–128
 - adjustive specificity, 127–128
 - and arthrokinematics, 121–123, 122b, 122f, 123f
 - assisted and resisted positioning, 124–126, 125f, 128f, 136t
 - basic rules for effective, 143b
 - benefits/effects of
 - cavitation, 107–111, 109f, 110f, 112f
 - circulatory system hypotheses, 120
 - joint fixation, 112–115, 112f, 113f, 114f
 - mechanical hypotheses, 106–111
 - muscle spasm relief, 116–120, 117f
 - for musculoskeletal dysfunction, 105
 - myofascial cycle disruption, 116–120, 117f
 - nerve root compression relief, 117–118
 - neurobiologic hypotheses, 115–120, 116f, 117f, 118f, 119f, 119t
 - neuroimmunology, 118–120, 119f, 119t
 - for neuromusculoskeletal (NMS) conditions, 105
 - pain relief, 115–120, 116f, 117f, 118f, 119f, 119t
 - reflex dysfunction, 118, 118f
 - classification of manipulative procedures, 85b, 85f
 - contact point, 133–134, 135f
 - contraindications and complications, 92–105, 93t
 - in cervical spine region, 94–102
 - in lumbar spine region, 103–105, 103b
 - side effects, 94b
 - side-posture IVD debate, 103–105, 104f
 - stroke, 95, 95f, 96–100, 96f, 97f, 101–102, 101b, 102b
 - in thoracic spine region, 102–103
 - vertebral artery injury, 95, 95f, 96–100, 96f, 97f, 101–102, 101b, 102b
 - vertebrobasilar ischemia (VBI), 100–101, 101b

Note: Page numbers followed by “f” refer to illustrations; page numbers followed by “t” refer to tables; page numbers followed by “b” refer to boxes.

Adjustive therapy (*Continued*)

- definitions and categorizations discussion, 84–88
 - doctor's positioning and skills, 128–142, 133f, 134f
 - equipment preferences, 129–132, 129f
 - factors to consider before beginning, 120–144, 121b, 121f, 122b, 122f
 - history of, 1–3
 - indications for, 89
 - indifferent hand, 134
 - injuries associated with, 92–105, 93t
 - joint manipulative procedures, 84–88, 85b
 - adjustments, 84–88, 85b
 - chiropractic technique, 87–88, 426–428
 - joint mobilization, 88
 - manipulation, 88
 - manual traction-distraction, 88
 - specific *vs.* general, 87
 - for joint subluxation/dysfunction syndrome (JSDS)
 - assessment of, 90–92
 - clinical features of, 90–92, 90b
 - outcome measures, 91–92, 91b
 - pain and hypersensitivity, 90–91, 91b
 - spinal physical capacity tests, 91–92, 92b
 - listing of named adjustments, 85–86, 426–428
 - manipulative procedures
 - classification of, 85b, 85f
 - for mechanical spine pain, 89–90
 - motion-assisted, 142–144
 - and patient positioning, 124–127, 125f, 126f
 - principles of, 84–144
 - psychomotor skills needed, 128–142
 - segmental contact points, 134–136, 135f, 136t
 - selection factors, 121–123, 122b, 122f
 - soft tissue procedures, 88–89, 89b
 - thrust techniques, 137–142, 138f, 139f, 140f, 141f
 - tissue pull, 136–137
- Adjustments; *See also* adjustments (specific techniques)
- basic rules for effective, 143b
 - categorizing, 86, 87b
 - cervical spine
 - flexion and extension dysfunction, 172–174, 174f
 - lateral flexion dysfunction, 171–172, 172f, 173f
 - positioning for, 175b
 - rotational dysfunction, 171, 171f
 - definition of, 84–88, 85b
 - factors governing selection of, 122b
 - for flexion and extension dysfunction, 172–174, 174f
 - high velocity-low amplitude (HVLA), 86
 - history of, 1–3
 - for lateral flexion dysfunction, 171–172, 172f, 173f
 - listing of named chiropractic, 85–86, 426–428

Adjustments; *See also* adjustments (specific techniques) (*Continued*)

- lumbar spine
 - flexion and extension, 252–253, 253f
 - knee-chest, 247, 253b, 260–261, 260f, 261f
 - lateral flexion, 251–252, 251f, 252f
 - prone, 247, 248f, 253b, 258–260, 259f, 260f
 - rotational, 248–251, 248f, 250f, 251f
 - side-posture, 245, 245f, 246f, 247f, 253–262, 253b, 254f, 256f, 257f, 258f
 - sitting, 248, 248f, 253b, 261–262, 261f
 - overall techniques and procedures, 84–144
 - for rotational dysfunction, 171, 171f
 - tables, 129f, 130f, 131f, 132f
 - techniques and illustrations; (*See* adjustments (specific techniques))
 - thoracic spine
 - knee-chest, 202, 202f, 220–221, 220f
 - side, 215, 215f
 - sitting, 191f, 202, 202f, 211, 212b, 214–215, 223–225, 224f, 231–232, 232f
 - standing, 204, 204f, 225–226, 225f, 226f
 - supine, 202–204, 203f, 204f, 211, 221–223, 221f, 223f, 224f, 226–232, 280–281
- Adjustments (specific techniques)
- ankle and foot first metatarsophalangeal joint
 - web metatarsal/finger grasp phalanx, 379, 379f
 - ankle and foot intermetatarsal joint
 - bimanual thenar/metatarsal grasp shear, 378, 378f
 - ankle and foot interphalangeal joint
 - thumb index grasp/phalanx, 379, 379f
 - ankle and foot intertarsal joint
 - bimanual web/tarsals, 377, 377f
 - ankle and foot metatarsophalangeal joint
 - thumb metatarsal/thumb phalanx shear, 378, 378f
 - thumb/index grasp/phalanx, 378, 379f
 - ankle and foot subtalar joint
 - interlaced bimanual grasp/calcaneus, 374, 375f
 - reinforced web/calcaneus, 374, 375f
 - ankle and foot tarsometatarsal joint
 - hypothenar/cuboid with forefoot distraction, 375
 - hypothenar/navicular with forefoot distraction, 376, 376f
 - reinforced hypothenar/navicular, 376, 377f
 - reinforced middle interphalangeal/cuneiform pull, 377, 377f
 - reinforced thumbs/cuneiform with forefoot distraction, 376, 376f
 - ankle and foot tibiotalar joint
 - bimanual reinforced interphalangeal/anterior talus pull, 371, 373f

Adjustments (specific techniques) (*Continued*)

- reinforced middle interphalangeal/talus pull, 373, 373f
- reinforced webs/anterior talus push, 373, 373f
- reinforced webs/talus push, 374, 374f
- web/talus, mid-hypothenar calcaneus, 374, 374f
- cervical spine (lower)
 - bilateral index/pillar push, 187, 187f
 - digit/pillar pull, 184, 184f
 - digit/pillar push, 184, 185f
 - hypothenar/pillar push, 184, 184f, 185, 186f
 - hypothenar/spinous push, 186, 187f
 - index/pillar push, 180, 181f, 184, 185f, 186, 187f
 - index/spinous push, 180, 181f
 - thumb/pillar pull, 182, 183f
 - thumb/pillar push, 182, 182f
- cervical spine (upper)
 - calcaneal/zygomatic pull, 176, 177f
 - calcaneal/zygomatic push, 176, 176f
 - digit/atlas pull, 179, 179f
 - hypothenar/ occiput lift, 174, 175f
 - hypothenar/ occiput push, 175, 175f
 - index/atlas push, 176, 177f, 178, 178f
 - index/occipital lift, 177, 177f
 - index/occipital push, 178, 178f
 - thenar/occiput push: distraction, 179, 179f
 - thenar/occiput push: extension, 180, 180f
- elbow, 322–325, 322b
 - bimanual grasp/distal humerus, 325, 325f
 - calcaneal/proximal radius forearm stabilization, 323, 323f
 - calcaneal/proximal ulna forearm stabilization, 322, 322f
 - hypothenar/radius push ulnar stabilization, 324, 324f
 - mid-hypothenar (knife-edge)/proximal ulna elbow flexion, 324, 324f
 - reinforced hypothenar/proximal radius pull, 323, 323f
 - thumb index/olecranon push, 324, 324f
 - thumb/radius push, distal forearm grasp, 323, 323f
 - web/distal humerus, forearm grasp pull, 322, 322f
 - web/proximal radius push, 325, 325f
 - web/proximal ulna push, 325, 325f
- extraspinal types of (*See* extraspinal adjustments)
- hand
 - bimanual thumbs digits/metacarpals, 337, 337f
 - thumb index grasp/metacarpophalangeal with hand stabilization, 337, 337f
- hip, 346–349, 346b
 - bimanual grasp/distal tibia pull, 347, 347f
 - bimanual grasp/proximal femur, 347, 348, 348f
 - hypothenar/proximal femur, 347, 348f

- Adjustments (specific techniques) (*Continued*)
- hypothernar/proximal femur, palmar distal femur grasp, 349, 349f
 - hypothernar/trochanter push, 349, 349f
 - knee femorotibial joint
 - bimanual grasp/distal tibia with knee thigh stabilization, 362, 362f
 - bimanual grasp/proximal tibia, 358, 360, 360f
 - hypothernar/proximal lateral tibia with leg stabilization, 361, 361f
 - hypothernar/proximal medial tibia with leg stabilization, 361, 361f
 - reinforced mid-hypothernar/proximal tibia pull, 361, 362f
 - reinforced web/proximal tibia push, 359, 360f
 - knee patellofemoral joint
 - bimanual web/patella, 362, 363f
 - knee tibiofibular joint
 - index/proximal fibula, palmar ankle push, 362, 363f
 - reinforced mid-hypothernar/proximal fibula pull, 364, 364f
 - reinforced mid-hypothernar/proximal fibula push, 364, 364f, 365f
 - reinforced thumbs/proximal fibula, 363, 363f
 - lumbar spine
 - bilateral thenar/mammillary push, 258, 259f, 260, 261f
 - digit/spinous push-pull, 258, 258f
 - hypothernar/mammillary or spinous push, 261, 261f
 - hypothernar/mammillary push, 253, 254f, 256f, 259, 259f
 - hypothernar/maximillary, 261, 261f
 - hypothernar/spinous pull, 257, 257f
 - hypothernar/spinous push, 255, 256f, 259, 260, 260f
 - thoracic spine at thoracocervical junction, 211–232, 212b
 - bilateral/thenar and hypothernar/transverse push, 213, 214f
 - hypothernar/transverse push, 212, 213f
 - thumb/spinous push, 211, 212f, 214, 214f, 215, 215f
 - thoracic spine in costosternal region
 - covered-thumb/costosternal push, 232, 232f
 - hypothernar/costosternal pull, 232, 233f
 - thoracic spine in knee-chest position
 - hypothernar/spinous push, 220, 220f
 - hypothernar/transverse and bilateral hypothernar/transverse push, 220, 220f
 - thoracic spine in prone positions
 - bilateral hypothernar/transverse push, 216, 217f
 - bilateral thenar/transverse push, 216, 216f
 - hypothernar spinous crossed thenar/transverse push, 219, 219f
 - unilateral hypothernar/spinous push, 217, 218f
 - unilateral hypothernar/transverse push, 139f, 140f, 141f, 218, 219f
 - thoracic spine in ribs area
- Adjustments (specific techniques) (*Continued*)
- covered-thumb/costal push, 230, 231f
 - hypothernar/costal push, 228, 228f, 229, 230f, 232, 232f
 - ilial hypothernar/costal push, 230, 230f
 - index/costal push, 228, 228f, 229, 229f, 231, 231f
 - modified hypothernar/costal push, 229, 229f
 - thenar/costal drop, 226, 227f
 - web/costal push, 231, 231f
 - thoracic spine in sitting position
 - hypothernar/transverse pull, 223, 224f
 - thoracic spine in standing position
 - thenar/transverse push, 225, 225f
 - thoracic long-axis distraction, 226, 226f
 - thoracic spine in supine position
 - opposite-side thenar/transverse drop, 221, 221f
 - same-side thenar/transverse drop, crossed arm, 223, 223f
 - thenar/transverse drop, pump handle, 223, 224f
 - wrist, 327–329, 334b
 - bimanual grasp/distal forearm hand, 336, 336f
 - bimanual palmar grasp/hand with arm axillary stabilization, 335, 335f
 - bimanual thumb-index radius and ulna shear, 334, 334f
 - hand grasp pull with forearm stabilization, 335, 335f
 - reinforced hypothernar/radius, 334, 335f
 - reinforced thumbs/carpal, 336f, 337
- Agency for Health Care Policy and Research (AHCPR)
- Manga report, 8–9
- Algometry, 79–80, 80f
- Alzheimer's disease, 93t
- American Medical Association (AMA), 7
- Anatomic barriers, 402, 402t
- Anatomic joints, 20, 20f, 21f
- Aneurysms, 93t
- Ankle and foot, 364–380
- adjustments
 - intermetatarsal, 372b, 378, 378f
 - intertarsal joint, 372b, 377, 377f
 - metatarsophalangeal joint, 372b, 378, 378f
 - subtalar joint, 372b, 374, 375f
 - tarsometatarsal joint, 372b, 375–376, 376f
 - tibiotalar joint, 371, 372b, 373f, 374f
 - biomechanics of, 367–368, 367f, 367t, 368f, 368t, 369f, 370t, 371f
 - capsular patterns, 71t
 - close-packed positions for, 23t
 - evaluation of, 288t, 368–371, 369f, 370f
 - functional anatomy, 295f, 365–367, 365f, 367t
 - ligaments of, 365–366, 366f
 - osseous structures, 365, 365f
- Antagonist contract method, 393–417, 394b, 405b, 405f
- Anterior cruciate ligament (ACL), 351–352, 351f, 354–357
- Anterior longitudinal ligament (ALL), 17, 17f, 145, 146f, 354
- Anterior root
- in lumbar motion segment, 43–44, 44f
- Arthrokinematics
- ankle and foot, 367–368, 367t
 - description and illustration of movements, 20, 21f
 - elbow, 318t
 - evaluation before adjustments, 121–123, 122b, 122f, 123f
 - of hip, 341–343, 343t
 - and joint anatomy/movements, 121–123, 122b, 122f, 123f
 - knee, 354t
 - and proprioceptive neuromuscular facilitation (PNF), 405–408, 406b, 407f, 408f
 - wrist and hand, 329t
- Articular capsule
- description and illustration of, 29–30, 30f
- Articular cartilage
- anatomy of, 146f
 - and joint anatomy, 16, 16f, 17f
 - microscopic anatomy of, 16f
 - zones of, 16
- Articular facets
- anatomy of, 233–234, 233f, 234f
- Articular landmarks
- bony palpation of, 65–66, 65f, 66f
- Articular neurology
- and joint anatomy, 18–20, 19f
- Articular processes
- anatomy of, 146f, 233–234, 233f, 234f
- Articulated tables, 130, 131f
- Assisted adjustable methods
- description of, 140–142
 - illustrations of, 128f, 140f, 141f
 - versus* resisted positioning, 124–126, 125f, 128f, 136t
- Association of Chiropractic Colleges
- “Paradigm of Chiropractic”, 9
- Asymmetry
- alignment scans, 151
 - evaluation of, 51, 51b
- Atherosclerosis, 93t
- Atlanto-odontoid articulation
- description and illustration of, 152–157, 153f
 - evaluation of, 165, 166f
- Atlas
- anatomical structure of, 152–157, 153f
- Autonomic nervous system
- stressors affecting, 118–120, 119f
- Autonomic reflexes, 45–46, 45b
- Axial forces
- definition of, 13
 - on intervertebral discs (IVDs), 30–33, 32f
- Axial rotation
- body planes and axes associated with, 12–13, 13f, 13t
 - stressing intervertebral discs (IVDs), 30–33, 32f
- Axis
- anatomical structure of, 152–157, 153f
 - of movement and joint anatomy, 13, 13f, 14f

B

Barnes method
 of myofascial release, 394b, 400–401, 401b, 401f

Barriers
 and postisometric relaxation, 402–405
 types and characteristics of dysfunctional, 402, 402t

Bennett (neurovascular) reflexes, 413, 416f, 417f

Biomechanics
 definition of, 11

Bipennate muscles, 26–33, 27f

Blood disorders, 93t

Blood flow
 effects of soft tissue manipulation on, 393–394

Blood vessels
 soft tissue palpation of, 66–67, 66f, 67b, 67f

Body planes of movement
 and joint anatomy, 12–13, 13f, 13t

Body wall reflex techniques, 410–413
 acupressure point stimulation, 394b, 410–412, 411f, 412f
 Bennett (neurovascular) reflexes, 413, 416f, 417f
 Chapman reflexes, 412–413, 414f, 415f

Bony elements
 and joint anatomy, 15–16, 16f

Bony end feel, 70b

Bony landmarks, 65–66, 65f, 66f, 134–136, 135f

Bony palpation, 50, 65–66, 65f, 66f

Boundary lubrication model, 18, 18f

Bursae
 elbow, 317, 318f
 hip, 340
 soft tissue palpation of, 66–67, 66f, 67b, 67f

C

Capsular end feel, 70b

Capsular patterns, 71t

Carpal tunnel syndrome, 330, 330f

Carrying angle, 316, 317f, 318–319

Cartilaginous joints
 structure and examples of, 15t

Carver, Willard, 2

Cavitation
 benefits/effects of, 107–111, 109f, 110f, 112f
 effects on joint movement, 68, 68f
 forces producing, 23, 107–111, 109f

Center of gravity
 and posture, 56, 57f, 57t
 of skull, 158f

Central nervous system
 stressors affecting, 118–120, 119f

Centralization
 of symptoms, 388

Cerebrovascular accident (CVA)
 precautions in adjustive therapy, 95, 95f, 96–100, 96f, 97f, 101–102, 101b, 102b

Cervical artery injury, 95–102, 100b

Cervical chair, 132, 132f

Cervical curve, 158–159, 158f, 159f

Cervical flexion restriction, 405f

Cervical kinetics, 161–162

Cervical spine
 abbreviations, 174b
 adjustment contraindications and complications, 94–102
 adjustment types for lower
 bilateral index/pillar push, 187, 187f
 digit/pillar pull, 184, 184f
 digit/pillar push, 184, 185f
 hypothenar/pillar push, 184, 184f, 185, 186f
 hypothenar/spinous push, 186, 187f
 index/pillar push, 180, 181f, 184, 185f, 186, 187f
 index/spinous push, 180, 181f
 thumb/pillar pull, 182, 183f
 thumb/pillar push, 182, 182f
 adjustment types for upper
 calcaneal/zygomatic pull, 176, 177f
 calcaneal/zygomatic push, 176, 176f
 digit/atlas pull, 179, 179f
 hypothenar/ occiput lift, 174, 175f
 hypothenar/ occiput push, 175, 175f
 index/atlas push, 176, 177f, 178, 178f
 index/occipital lift, 177, 177f
 index/occipital push, 178, 178f
 thenar/occiput push: distraction, 179, 179f
 thenar/occiput push: extension, 180, 180f
 bony palpation of, 65–66, 65f, 66f
 cervical curve, 158–159, 158f, 159f
 dysfunction
 flexion and extension dysfunction, 172–174, 174f
 lateral flexion dysfunction, 171–172, 172f, 173f
 rotational dysfunction, 171, 171f
 evaluation of
 flexion and extension, 165–169, 166f, 168f
 global range-of-motion ranges, 163f, 163t
 joint play, 165, 165f
 lower, 163f, 163t, 164, 164f
 motion palpation, 165–169, 165f, 166f, 167f, 168f, 169f, 170f, 174b, 174f
 observation, 162
 segmental range of motion and end play, 165, 165f, 167, 168–169
 static palpation, 162–164
 upper, 162–169, 163f, 163t
 flexion and extension
 C0–C1, 154–155, 155f, 155t, 156f, 165–169
 C1–C2, 155–157, 155t, 156f, 157f, 165–169
 C3–C7, 157–162, 157f, 158f, 160t, 165–169
 description of, 154
 evaluating, 165–169, 166f, 168f
 functional anatomy of
 cervical curve, 158–159, 158f, 159f
 lower, 157–162, 157f, 158f, 159f, 160f, 160t, 161f
 upper, 152–157, 153f, 155f, 155t, 158–159, 159f
 identifying joint subluxation/dysfunction syndrome (JSDS), 151–152, 152b

Cervical spine (*Continued*)

lower cervical spine
 evaluation of, 163f, 163t, 164, 164f
 functional anatomy of, 157–162, 157f, 158f, 159f, 160f, 160t, 161f
 manual traction-distraction techniques, 384–387, 387b
 mobilization techniques, 381–384, 382b
 overview of adjustment techniques
 lower, 162f, 180, 180b, 181f, 182f, 184f, 185f, 186f, 187f
 overview of, 170–174, 170f, 171f, 172f, 173f, 174b, 174f
 upper, 174–188, 174b, 175b, 175f, 176f, 177f, 178f, 179f

PARTS isolation of dysfunction, 151–152, 152b

range-of-motion assessment, 59–60, 60f, 61f

scanning, 146–151, 147b, 147f, 149f

segmental range-of-motion and end play
 C0–C1, 154–155, 155f, 155t, 156f, 165–169
 C1–C2, 155–157, 155t, 156f, 157f, 165–169
 C3–C7, 157–162, 157f, 158f, 160t, 165–169
 evaluating, 165–169, 166f, 168f
 structure of, 145–146, 146f
 traction, 387, 387f, 388b, 388f
 upper cervical spine
 evaluation of, 162–169, 163f, 163t
 functional anatomy of, 152–157, 153f, 155f, 155t, 158–159, 159f
 vertebrae structure, 145–146, 146f, 152–162, 153f, 156f, 157f, 158f, 160f, 161f, 172f

Cervical traction
 manual, 387, 388f
 motorized, 387, 387f, 388b, 388f

Chapman reflexes, 412–413, 414f, 415f

Chiropractic; *See also* adjustive therapy
 challenges for future, 9
 common additional services, 4
 defining primary care chiropractic physician, 35
 defining techniques, 87–88, 426–428
 doctor of chiropractic (DC)
 defining, 35
 degree, 5–6
 responsibilities of primary, 35
 and joint assessment, 35–83
 listing of named techniques, 6, 85–86, 426–428
 nonthrust procedures (*See* nonthrust procedures)
 overview of profession, 1–10, 35
 accreditation and licensure, 6
 basic principles of, 3–5, 3t
 education, 5–6
 future of, 9
 history of, 1–3
 philosophic roots of, 3
 research, 7
 responsibilities of, 35
 scope of practice, 6, 35
 standards of care guidelines, 8–9
 statistics, 6–7
 utilization statistics, 6–7

- Chiropractic physician; *See* Doctor of Chiropractic (DC)
- Chondroitin, 16, 17f
- Circulatory system
benefits/effects of adjustive therapy, 120
- Clinical joint instability
definition of, 37b
- Close-packed positions
for ankle and foot, 368t
of elbow joints, 319t
for knee joint, 353b
and muscle/ joint injuries, 28
for specific joints, 22–23, 23t
for wrist and hand joints, 328t
- Clotting disorders, 93t
- Collagen, 26–33
- Complementary and alternative medicine (CAM)
and chiropractic education, 6
chiropractic utilization statistics, 6–7
- Complications
and contraindications, 92–105, 93t
in cervical spine region, 94–102
in lumbar spine region, 103–105, 103b
side effects, 94b
side-posture IVD debate, 103–105, 104f
stroke, 95, 95f, 96–100, 96f, 97f, 101–102, 101b, 102b
in thoracic spine region, 102–103
vertebral artery injury, 95, 95f, 96–100, 96f, 97f, 101–102, 101b, 102b
vertebrobasilar ischemia (VBI), 100–101, 101b
defining, 92
red flag disorders, 92
ruling out, 121f
- Control
and proprioceptive neuromuscular facilitation (PNF), 405–408, 406b, 407f, 408f
- Coordination
and proprioceptive neuromuscular facilitation (PNF), 405–408, 406b, 407f, 408f
- Coronal plane, 12–13, 13f, 13t, 14f, 125f
- Council on Chiropractic Education (CCE)
defining primary care chiropractic physician, 35
roles of, 5–6
- Council on Chiropractic Guidelines and Practice Parameters (CCGPP), 8
- Counterthrusts, 126, 127f, 134–136, 136t, 139f, 141
- Coupled movements
hypermobility *versus* instability affecting, 42t
- Cox method, 386, 386b, 386f
- “Cracks”, 23, 68f, 86, 107–111, 109f
- Cranial dysfunction
and cranial manipulation, 392b
- Cranial manipulation, 391–392
controversy and hypotheses regarding, 391–392, 392b
and cranial dysfunction, 392b
cranial universal, 392, 393f
parietal lift, 392, 393f
sagittal suture spread, 392, 392f
- Cranial universal, 392, 393f
- Creep, 30, 32
- Cubital tunnel syndrome, 319
- Curvilinear movements, 13, 13f, 14f
- Cylindrical joint capsule
of hip, 339f
- D**
- Degenerative cycle
of vertebral subluxation complex (VSC), 42–43, 43f
- Degenerative joint disease
pathological sequence leading to, 43, 43f
- Derangement syndrome
of pain, 389, 389b, 391f, 392f
- Diabetes, 93t
- Diagnosis parameters
of joint subluxation/dysfunction syndrome (JSDS), 47, 50–51
- Contract-relax-stretch technique, 405–408, 406b, 407f, 408f
- Contraindications
and complications, 92–105, 93t
in cervical spine region, 94–102
in lumbar spine region, 103–105, 103b
side effects, 94b
side-posture IVD debate, 103–105, 104f
stroke, 95, 95f, 96–100, 96f, 97f, 101–102, 101b, 102b
in thoracic spine region, 102–103
vertebral artery injury, 95, 95f, 96–100, 96f, 97f, 101–102, 101b, 102b
vertebrobasilar ischemia (VBI), 100–101, 101b
defining, 92
red flag disorders, 92
ruling out, 121f
- Control
and proprioceptive neuromuscular facilitation (PNF), 405–408, 406b, 407f, 408f
- Coordination
and proprioceptive neuromuscular facilitation (PNF), 405–408, 406b, 407f, 408f
- Coronal plane, 12–13, 13f, 13t, 14f, 125f
- Council on Chiropractic Education (CCE)
defining primary care chiropractic physician, 35
roles of, 5–6
- Council on Chiropractic Guidelines and Practice Parameters (CCGPP), 8
- Counterthrusts, 126, 127f, 134–136, 136t, 139f, 141
- Coupled movements
hypermobility *versus* instability affecting, 42t
- Cox method, 386, 386b, 386f
- “Cracks”, 23, 68f, 86, 107–111, 109f
- Cranial dysfunction
and cranial manipulation, 392b
- Cranial manipulation, 391–392
controversy and hypotheses regarding, 391–392, 392b
and cranial dysfunction, 392b
cranial universal, 392, 393f
parietal lift, 392, 393f
sagittal suture spread, 392, 392f
- Cranial universal, 392, 393f
- Creep, 30, 32
- Cubital tunnel syndrome, 319
- Curvilinear movements, 13, 13f, 14f
- Cylindrical joint capsule
of hip, 339f
- D**
- Degenerative cycle
of vertebral subluxation complex (VSC), 42–43, 43f
- Degenerative joint disease
pathological sequence leading to, 43, 43f
- Derangement syndrome
of pain, 389, 389b, 391f, 392f
- Diabetes, 93t
- Diagnosis parameters
of joint subluxation/dysfunction syndrome (JSDS), 47, 50–51
- Diagnostic procedures
for joint subluxation/dysfunction syndrome (JSDS)
reliability of, 52
responsiveness of, 53
utility of, 53
validity of, 52
radiographic analysis, 76b
for subluxation syndromes, 36
- Diarthrotic joints
structure and examples of, 15t
- Diet
as adjunct chiropractic service, 4
- Direct thrusts
definition of, 85b
- Disc herniation
in lumbar spine, 30–33
- Discs; *See* intervertebral discs (IVDs)
- Disease; *See also* complications
body energy theories, 3–9, 3t
concept of, 3–9
and selecting adjustive methods, 122b
- Displacements, 2
- Distal radioulnar joint
capsular patterns, 71t
- Distraction
assessing long-axis, 345f
and compression, 23, 23t
flexion-distraction, 132, 132f, 386–387, 387b
manual traction-distraction techniques, 84, 85f, 88
motorized lumbar, 387, 387f, 388f
techniques producing, 112–113, 112f
- Distraction tables, 132, 132f
- Doctor of chiropractic (DC)
defined by Council on Chiropractic Education (CCE), 35
degree, 5–6
responsibilities of primary, 35
- Documentation
diagram to aid in, 83f
ICD-9CM codes for subluxation, 83t
symbols for recording joint dysfunction, 82f
- Dominant eye determination, 55, 56b
- Drop tables, 131–132, 131f, 143b
- Dura mater (D), 43–44, 44f
- Dysfunctional syndrome
of pain, 389, 389b, 391f
- E**
- Eccentric muscle activity, 27–28, 161–162
- Education
chiropractic, 5–6
- Effleurage massage, 394b, 395, 395f, 396b
- Elastic barriers, 68, 68f
- Elasticity
defining connective tissue, 26–33
effects of soft tissue manipulation on, 393–417
and joint movement, 67–72, 68f
model of, 24, 24f, 26–33
skin assessment technique, 66f
- Elastohydrodynamic model
of joint lubrication, 18, 18f
- Elbow
adjustment procedures, 322–325, 322b
sitting, 322–324, 322f, 323f, 324f
supine, 325, 325f

Elbow (*Continued*)

- biomechanics of, 317–319, 318f, 318t, 319t
 - capsular patterns, 71t
 - close-packed positions for, 23t
 - evaluation of, 319–322, 320b, 320f, 321f, 322f
 - functional anatomy, 316–317, 316f, 317f, 317t
 - ligaments, 316, 317f
 - osseous structures, 316, 316f
 - summary of adjustive techniques, 322b
 - three joints of, 316, 316f
- Electromyography, 81–82
- Empty end feel, 70b, 71
- End feel
- bony, 70b
 - capsular, 70b
 - goals of, 72b
 - normal *vs.* abnormal, 70b
- End play (EP)
- assessment during JSDS evaluation, 50, 51, 51b
 - and capsular patterns, 71t
 - clinical features of, 90–92, 90b
 - end-play zone (EPZ), 68, 68f
 - evaluation of
 - in cervical spine, 165, 165f, 167, 168–169
 - in lumbar region, 242–245, 243f, 244f
 - in thoracic spine, 197–199, 198f, 199f
 - and motion palpation, 69–71, 70b, 70f
 - motion scans, 147b, 149f, 150f
 - normal *vs.* abnormal, 70b
- Endocrine system
- chiropractic principles regarding, 3–9
 - stressors affecting, 118–120, 119f
- Endomysium, 27, 27f
- End-play zone (EPZ), 68, 68f
- Entrapment
- theory of meniscoid, 40, 40f
- Epimysium, 27, 27f
- Equilibrium, 25–26
- Equipment
- preferences by doctors, 129–132, 129f
- Ergotropic responses
- characteristics of, 119t
- Evidence-based practices (EBPs)
- in chiropractic education, 6
- Excessive anteversion, 338–339, 340–341
- Exercises
- as adjunct chiropractic service, 4
 - during spinal physical capacity test, 92b
- Extraspinal adjustments
- shoulder acromioclavicular joint
 - covered thumb/distal clavicle, 310, 310f
 - digital/distal clavicle with distraction, 310, 311f
 - hypothener/distal clavicle with distraction, 310, 311f
 - index/distal clavicle, 310, 310f
 - web/distal clavicles, 311, 311f
 - shoulder glenohumeral joint
 - bimanual thumb thenar grasp/proximal humerus, 302f, 304, 305f, 306, 307, 307f, 308, 308f
 - index/proximal humerus, 306, 306f

Extraspinal adjustments (*Continued*)

- interlaced digital/proximal humerus, 306, 306f, 308, 309, 309f
 - reinforced palmar/olecranon, 309, 309f
 - thumb web/axilla distraction, 304, 305f
- shoulder scapulocostal articulation
- bilateral digital thenar grasp/scapula, 315, 315f
 - bilateral thumb thenar/lateral scapula, 314, 314f
 - bimanual digital thenar grasp/scapula, 315, 315f
 - crossed bilateral mid-hypothenar/medial scapula, 314, 315f
- shoulder sternoclavicular joint
- covered thumb/proximal clavicle, 312, 312f
 - digital proximal/clavicle, thenar/manubrium, 314, 314f
 - digital/proximal clavicle with distraction, 313, 313f
 - hypothener/proximal clavicle with distraction, 311, 312f
 - reinforced thenar/proximal clavicle, 313, 314f
 - thenar/distal clavicle, thenar/manubrium, 313, 313f
- temporomandibular joint (TMJ)
- bilateral thumb/lower molar plica entrapment reduction, 292, 293f
 - bilateral thumb/mandible distraction, 291, 292f
 - distraction techniques, 291–293
 - reinforced palmar/distal mandible, 293, 293f
 - reinforced thumb/proximal mandible, 294, 294f
 - thenar/proximal mandible, 294, 294f
 - translation techniques, 293–294, 293f, 294f
- Extraspinal techniques
- elbow, 315–325
 - hip, 337–349
 - knee, 349–364
 - peripheral joints, 283
 - shoulder, 294–315
 - temporomandibular joint (TMJ), 283–294
 - wrist and hand, 326–337
- Extremities
- mobilization examples, 383–384, 383f, 385f
- Eye dominance, 55, 56b
- F**
- Facet joints
- characteristics of, 29–30
 - degenerative pathological sequence, 43, 43f
 - description and illustration of, 29–30, 29f, 30f
 - and joint biomechanics, 27f, 29–30, 30f
 - planes in spinal region, 27f, 29f
- Facet planes, 29f

Facets

- anatomy of, 233–234, 233f, 234f
 - plane of, 153f
 - and spine function models, 33–34
- Fascia
- effects of soft tissue manipulation on, 393–417
 - soft tissue palpation of, 66–67, 66f, 67b, 67f
- Fascial barriers, 402, 402t
- Femorotibial joint
- adjustments, 358–362, 360b, 361f
- Femur, 337–339, 338f
- Fibroadipose meniscoid, 29, 30f
- Fibrocartilage
- and joint biomechanics, 16–17
- Fibrous joints
- structure and examples of, 15t
- Fingers
- close-packed positions for, 23t
 - ligaments of, 327f
 - movements of, 328t
- First verbal report of pain, 79–80, 80f
- Flexion and extension
- body planes and axes associated with, 12–13, 13f, 13t
 - deformation zones, 235f
 - description of, 154
 - effects on hip ligaments, 342f
 - evaluation of
 - cervical spine, 165–169, 166f, 168f
 - lumbar spine, 243, 243f, 244f
 - thoracic spine, 189–190, 190f
 - functional anatomy
 - C0–C1, 154–155, 155f, 155t, 156f, 165–169
 - C1–C2, 155–157, 155t, 156f, 157f, 165–169
 - C3–C7, 157–162, 157f, 158f, 160t, 165–169
 - of knee joint, 353–354, 354f
 - range for each joint, 23t
- Flexion-distraction
- essential steps of, 387b
 - illustration of, 132, 132f
 - types of, 386–387
- Foot and ankle, 364–380
- adjustments
- intermetatarsal, 372b, 378, 378f
 - intertarsal joint, 372b, 377, 377f
 - metatarsophalangeal joint, 372b, 378, 378f
 - subtalar joint, 372b, 374, 375f
 - tarsometatarsal joint, 372b, 375–376, 376f
 - tibiotalar joint, 371, 372b, 373f, 374f
- biomechanics of, 367–368, 367f, 367t, 368f, 368t, 369f, 370t, 371f
 - close-packed positions for, 23t
 - evaluation of, 288t, 368–371, 369f, 370f
 - functional anatomy, 295f, 365–367, 365f, 367t
 - ligaments of, 365–366, 366f
 - muscles, 366–367, 367f, 367t
 - osseous structures, 365, 365f
- Force displacement curves, 109f, 110f

Forces

- on connective tissue, 24f, 26–33, 26f
- generated during adjustive therapy, 106–107
- on intervertebral discs (IVDs), 30–33, 32f
- and joint biomechanics, 11, 12f
- and loads on spine, 33–34
- and Newton's laws of motion, 25–26
- and tensegrity, 32f, 33–34, 34f
- thrust, 137–138, 138f

Forearm

- close-packed positions for, 23t

Fovea capitis, 338

Fractures, 25, 25f, 28, 93t

Friction massage, 394b, 397–398, 398b, 398f

Frontal plane, 12–13, 13f, 13t, 14f

Fulcrum, 11, 12f

Functional capacity questionnaire, 53

Functional techniques

- for soft tissue manipulation, 394b, 399–401, 400f, 401b

Functional x-ray exams, 77–78, 77f

G

Gait

- disorders that alter, 56b
- evaluation
 - during joint subluxation/dysfunction syndrome (JSDS) examination, 50, 55, 55b, 55f, 56b
- and foot and ankle movements, 367–368, 369f
- and hip movements, 338–339, 340–341
- phases of, 55f

Galvanic skin resistance (GSR), 80–81

Ganglion, 330

General spinal adjustments

- vs.* specific, 87

Glenohumeral joint

- prone adjustments, 304–307, 305b, 305f, 306f, 307f, 308f
- sitting adjustments, 304–307, 305b, 309f
- standing adjustments, 304–307, 305b, 309f

Glide

- definition of, 13–15

Gliding

- effleurage massage, 394b, 395, 395f, 396b

Gliding zones

- of cartilage, 16, 16f

Global range-of-motion (GROM)

- assessing and measuring, 50, 51b, 59–60
- to identify joint dysfunction, 147–148, 147b, 148f

ranges

- in cervical spine, 163f, 163t
- in lumbar region, 240t
- in thoracic region, 195t

Golfer's elbow, 319

Golgi tendon organs (GTOs), 394, 402–405

Graded oscillation technique

- of joint mobilization, 382, 382b

Gravity

- center in skull, 158f
- forces on spine, 33–34

Ground substance, 26–33

H

Hallux valgus, 369

Hamstring muscle

- postisometric relaxation procedure on, 405f

Hands and wrists

- adjustive procedures, 337, 337f
- biomechanics of, 327–329, 328f, 328t, 329f, 329t
- close-packed positions for, 23t
- evaluation of, 329–333, 330f, 331f, 332f, 332t, 333f
- functional anatomy, 326, 326f, 327f
- functional position of, 329, 329f
- ligaments, 326, 327f, 328f
- muscles, 326, 328f, 328t
- osseous structures, 326, 326f, 331f
- three physiologic arches of, 329f

Head zones, 398–399

Health

- body energy theories, 3–9, 3t
- promotion as adjunct chiropractic service, 4

Heat

- thermography, 80, 80f

Helical axis of motion (HAM), 21, 22f

Herniated discs

- pain caused by, 89–90

High velocity-low amplitude (HVLA)

- adjustments, 86, 107f; *See also* adjustments

Hip, 337–349

- adjustive procedures, 346–349, 346b, 346f, 347f, 348f, 349f
- biomechanics of, 341–343, 341f, 341t, 342b, 342f, 343f, 343t, 344f
- capsular patterns, 71t
- close-packed positions for, 23t
- evaluation of, 344–346, 344f, 345b, 345f
- functional anatomy, 337–341, 338f, 339f, 340f, 341f, 341t
- ligaments, 339–340, 340f
- osseous structures, 337–339, 338f
- referred pain, 344–346, 344f

History-taking

- during JSDS evaluations, 50

Hoffman ligaments, 234, 234f

Hold-relax

- and proprioceptive neuromuscular facilitation (PNF), 405–408, 406b, 407f, 408f

Hold-relax stretch, 405–408, 406b, 407f, 408f

Homeostasis

- definition of, 3, 4

Howard, John, 2

Hydraulic tables, 130, 131f

Hydrodynamic model

- of joint lubrication, 18, 18f

Hyperlordosis, 158–159, 159f

Hypermobility

- definition of, 37b
- versus* instability, 37b
- joint instability and, 41–43

Hypertonicity

- effects of soft tissue manipulation on, 393–417

Hypochondriasis, 93t

Hypolordosis, 158–159, 159f

Hypomobility

- definition of, 37b
- joint fixation, 39–41

Hysteresis, 32

Hysteria, 93t

I

ICD-9CM codes

- for subluxation, 83t

Iliofemoral ligaments, 339–340, 340f, 342f

Immune system

- benefits of chiropractic on, 118–120, 119f, 119t

Impulse thrusts, 138–139, 138f

Inclinometers, 59–60, 60f

Indirect thrusts

- definition of, 85b

Inflammation

- within vertebral subluxation complex (VSC), 46–47

Injuries; *See also* complications

- associated with adjustive therapy, 92–105, 93t

- to muscles and joints, 28

Innate intelligence, 3–9

Innominate, 337–339, 338f

Instantaneous axis of rotation (IAR), 21, 22f, 32f

Instrumentation

- for joint assessment, 79–82
- algometry, 79–80, 80f
- galvanic skin resistance (GSR), 80–81
- surface electromyography, 81–82
- thermography, 80, 80f

Interarticular block, 40, 40f

Interarticular derangements, 40–41

Interarticular end feel, 70b

Interdiscal blocks, 40–41, 41f

Internal derangements

- of intervertebral disc (IVD), 40–41

Interpedicular zone

- in lumbar motion segment, 43–44, 44f

Interphalangeal joint

- capsular patterns, 71t

Intervertebral discs (IVDs)

- anatomy of, 233–234, 233f, 234f
- characteristics of, 30–33, 31f, 32f
- degenerative pathological sequence, 43, 43f
- description and illustration of, 30–33, 31f
- and joint biomechanics, 30–33, 31f, 32f, side-posture IVD debate, 103–105, 104f
- sizes and illustrations of, 145, 146f

Intervertebral encroachment

- and nerve root compression, 43–44, 43f

Intervertebral foramen (IVF)

- illustration of dysfunction, 43–46, 43f

Intradiscal block, 40–41

Ischemic compression, 408–409

- Travell and Simons trigger points, 409–410, 411f, 412b, 412f

Ischiofemoral ligaments, 339–340, 340f, 342f

Isolation of motion segment dysfunction (PARTS), 151–152, 152b

- Isometric contractions
 - defining muscle, 27
 - and proprioceptive neuromuscular facilitation (PNF), 405–408, 406b, 407f, 408f
- Isotonic contractions
 - and proprioceptive neuromuscular facilitation (PNF), 405–408, 406b, 407f, 408f

J

- Jochumsen's measuring procedure, 159, 159f
- Joint assessment
 - bony palpation, 65, 65f
 - of joint subluxation/dysfunction syndrome (JSDS)
 - clinical evaluation of, 47–82
 - diagnosis parameters, 47, 50–51
 - diagnostic procedures, 52, 53
 - documentation of, 82f, 83f, 83t
 - examination procedures, 50–51, 51b, 54–59
 - five diagnostic categories, 50
 - gait evaluation, 55, 55b, 55f, 56b
 - leg length evaluation, 56–59, 58f
 - outcomes and measures, 53
 - pain, 50–51, 51b, 53, 54f
 - patient observation, 54–59
 - physical examination findings to support, 51b, 54–59
 - postural evaluation, 55–56, 57f, 57t
 - spinal listings and variations, 47, 48f
 - symptoms of, 53
 - terminology and nomenclature confusion, 47
 - of manipulable lesions, 36
 - manual therapy based on, 36
 - and models of spinal dysfunction and degeneration, 42–43
 - motion palpation, 61–63, 67–72
 - accessory joint motion, 68f, 69–71, 69f, 70f
 - and end play, 69–71, 70b, 70f
 - goals of, 72b
 - and joint play, 68f, 69–71, 69f, 70f
 - principles of, 67–72, 72b
 - summary of reliability studies regarding, 62, 429t, 432t, 433t, 434t, 435t, 437t
 - tools, instructions and tips, 67b, 72b
 - muscle testing, 73–74, 73f, 74b
 - orthopedic tests, 74
 - palpation, 60–65, 72b
 - bony palpation, 65, 65f
 - motion palpation, 67–72
 - reliability, 61–63
 - sacroiliac articulation, 65
 - soft tissue palpation, 66–67, 66f
 - validity, 63–65
 - percussion, 72, 73f
 - principles and procedures, 35–83
 - provocative tests, 74
 - radiographic analysis, 74–79, 75f, 76b, 79b
 - range-of-motion assessment, 59–60, 72b
 - McKenzie method, 59

- Joint assessment (*Continued*)
 - measurement procedures, 59–60, 60f, 61f, 62t
 - of subluxations, 36–37
 - symbols and diagrams for documenting, 82f, 83f
 - of vertebral subluxation complex (VSC)
 - degenerative cycle, 42–43, 43f
 - inflammatory and vascular components, 46–47
 - joint instability and hypermobility, 41–43
 - mechanical components of, 37b, 38–43
 - models of spinal dysfunction and degeneration, 42–43
 - neurobiologic components, 43–46
 - terminology, 37b
 - versus* vertebral subluxation/dysfunction syndrome, 37–47, 37b
- Joint capsules
 - anatomy of, 146f
 - capsular patterns, 71, 71t
 - definition of, 17
 - and joint biomechanics, 17, 17f
- Joint cavitation; *See* cavitation
- Joint challenge; *See* joint provocation
- Joint dysfunction
 - anatomic location and adjustment selection, 122b
 - clinical features of, 90–92, 90b
 - definition of, 37b
 - five diagnostic categories of, 50
 - motion scans, 148–151, 149f, 150f, 151f
 - symbols for documenting, 82f
- Joint fixation
 - benefits/effects of, 112–115, 112f, 113f, 114f
 - definition of, 37b
 - hypomobility, 39–41
 - interarticular adhesions, 112
 - interarticular blocks, 112–113, 112f
 - interdiscal blocks, 113–114, 113f
 - joint instability, 115
 - periarticular fibrosis and adhesions, 114–115
- Joint hypermobility
 - definition of, 37b
 - versus* instability, 42t
- Joint hypomobility
 - definition of, 37b
- Joint instability
 - definition of, 37b, 41
 - and hypermobility, 41–43, 42t
- Joint malposition, 38–39, 47
- Joint manipulation, 38–39
 - classification of procedures, 84, 85f
 - definition of, 85b
 - procedures, 84–88, 85b
 - adjustments, 84–88, 85b
 - chiropractic technique, 87–88, 426–428
 - joint mobilization, 88
 - manipulation, 88
 - manual traction-distraction, 88
 - specific *vs.* general, 87

- Joint mobilization
 - classification of, 84, 85f
 - definition of, 85b, 88, 381–384
 - examples of, 383–384, 383f, 384f
 - primary goal of, 382
 - techniques for cervical spine, 381–384, 382b
 - types of, 382–383, 382b
 - continuous stretch technique, 382
 - graded oscillation technique, 382, 382b
 - progressive stretch mobilization, 382
- Joint pain
 - and articular neurology, 18–20, 19f
 - assessment during JSDS examination, 50–51, 51b, 53, 54f
- Joint play (JP)
 - and adjustive localization, 124–127
 - assessment during JSDS evaluation, 50, 51, 51b
 - clinical features of, 90–92, 90b
 - evaluation of
 - in cervical spine, 165, 165f
 - in lumbar region, 240–241, 241f, 242f
 - in lumbar spine, 240–241, 241f, 242f
 - goals of, 72b
 - and motion palpation, 68f, 69–71, 69f, 70f
 - motion scans, 147b, 148–151, 149f, 150f
 - summary of reliability studies regarding, 62, 432t, 433t, 434t, 435t, 437t
- Joint provocation, 151–152, 152b
 - description of, 71–72
 - summary of reliability studies regarding, 62, 432t, 433t, 434t, 435t, 437t
- Joint restrictions, 47
- Joint subluxation
 - defining, 36–37
- Joint subluxation/dysfunction syndrome (JSDS)
 - assessment of, 90–92
 - clinical evaluation of, 47–82
 - clinical features of, 90–92, 90b
 - diagnosis parameters, 47, 50–51
 - diagnostic procedures, 52, 53
 - reliability of, 52
 - responsiveness of, 53
 - utility of, 53
 - validity of, 52
 - documentation of, 82f, 83f, 83t
 - examination procedures, 50–51, 51b, 54–59
 - five diagnostic categories, 50
 - gait evaluation, 55, 55b, 55f, 56b
 - identification of, 151–152, 152b
 - leg length evaluation, 56–59, 58f
 - outcome measures, 91–92, 91b
 - outcomes and measures, 53
 - pain, 50–51, 51b, 53, 54f
 - pain and hypersensitivity, 90–91, 91b
 - PARTS isolation of dysfunction, 151–152, 152b
 - patient observation, 54–59
 - physical examination findings to support, 51b, 54–59
 - postural evaluation, 55–56, 57f, 57t
 - and somatic and visceral reflexes, 45, 45f
 - spinal listings and variations, 47, 48f
 - spinal physical capacity tests, 91–92, 92b
 - symptoms of, 53
 - terminology and nomenclature confusion, 47

Joints

- anatomy and biomechanics of
 - anatomical diagrams of, 19f, 20f
 - articular cartilage, 16, 16f, 17f
 - articular neurology and pain, 18–20, 19f
 - axes of movement, 13, 13f, 14f
 - basic definitions, 11–20, 12f
 - biomechanics of, 11–20, 12f
 - and body planes of movement, 12–13, 13f, 13t
 - bony elements, 15–16, 16f
 - classification of, 15, 15t
 - close-packed positions for specific, 23t
 - compression forces, 24, 24f
 - connective tissue properties, 26–33, 26f, 27f
 - facet joints, 27f, 29–30, 30f
 - fibrocartilage, 16–17
 - functions of, 20–23, 21f, 22f, 24f
 - intervertebral discs (IVDs), 30–33, 31f, 32f
 - joint capsules, 17, 17f
 - as levers, 11–12, 12f
 - ligaments, 17, 17f, 28
 - models of spine function, 33–34, 34f
 - motion of, 13–15, 14f, 15t
 - muscles, 27–28, 27f
 - and Newton's laws of motion, 25–26
 - shear forces, 13, 24, 24f
 - structural characteristics, 15, 15t
 - synovial, 15
 - synovial fluid, 17–18, 18f
 - tension forces, 24, 24f, 25f
 - torque forces, 13, 24f, 25, 26f
 - trabecular patterns, 15–16, 16f
- arthrokinematics (*See* arthrokinematics)
- assessment of (*See* joint assessment)
- evaluating function of, 146–151
- motion palpation
 - summary of reliability studies regarding, 62, 429t, 432t, 433t, 434t, 435t, 437t
- stability and connective tissues, 26–33, 26f
- terminology of disorders, 37b
- types and classification of, 15t

K

Kenny method, 405–406

Keratin, 16, 17f

Kinematics

- chains and joint function, 20–23, 21f
- definition of, 11

Kinetics

- cervical, 161–162
- definition of, 11

Knee, 349–364

- adjustive procedures, 358–364, 360b
- femorotibial joint, 358–362, 360b, 361f
- patellofemoral joint, 360b, 362, 363f
- tibiofibular joint, 362–364, 363f, 365f
- biomechanics of, 352t, 353–354, 353b, 354f, 354t, 355f
- capsular patterns, 71t
- close-packed positions for, 23t
- evaluation of, 354–357, 356f, 356t, 357f, 358f, 359f

Knee (*Continued*)

- functional anatomy, 350–353, 350f, 351f, 352f, 352t, 353f
 - ligaments, 351–352, 351f
 - osseous structures, 350, 350f
 - referred pain, 356f
 - surface anatomy, 356f
- Knee-chest tables, 130–131, 131f

L

Lateral flexion

- body planes and axes associated with, 12–13, 13f, 13t
- cervical spine
 - description and illustration of, 161, 161f
- evaluation of, 148–151, 150f
- lumbar spine
 - adjustment techniques/illustrations, 251–252, 251f, 252f
 - evaluation of, 242, 243, 243f, 244f
- thoracic spine, 198–199, 199f

Lateral-flexion radiographs, 78, 78f

Law of acceleration, 25

Law of action-reaction, 25

Law of inertia, 25

Leader method, 387, 387f, 388f

Leahy method

- of myofascial release, 394b, 400–401, 401b, 401f

Leg length evaluation

- during joint subluxation/dysfunction syndrome (JSDS) exam, 51, 56–59, 58f

Lesions, 93t

Leverage

- as criteria for categorizing adjustments, 86

Levers

- D.D. Palmer's work on, 2
- and joint biomechanics, 11–12, 12f
- short and long, 85b
- and spine function models, 33–34

Licensure

- of chiropractors, 6

Ligamentous end feel, 70b

Ligaments

- articulations, 145, 146f
 - connective tissues, 28
 - description and illustration of, 17, 17f, 28
 - effects of soft tissue manipulation on, 393–417
 - elbow, 316, 317f
 - foot and ankle, 365–366, 366f
 - function and anatomy of, 145, 146f
 - hip, 339–340, 340f
 - and joint biomechanics, 17, 17f, 28
 - knee, 351–352, 351f
 - soft tissue palpation of, 66–67, 66f, 67b, 67f
 - and spine function models, 33–34
 - structural, 391–392
 - upper cervical spinal, 154, 154f
- Ligamentum teres, 340, 341f
- Lines of drive, 135f, 137
- Literature reviews
- on reliability/ validity studies, 437t

Loads

- on bones, 23–26, 24f
- and forces on spine, 33–34
- on intervertebral discs (IVDs), 30–33, 32f
- on muscles and ligaments, 27–28
- tensile, 24, 26–33

Localization

- of osseous structures of wrist, 331f

Logan Basic technique, 413–417, 417f

Long-lever thrusts

- and adjustment selection, 122b
- definition of, 85b
- illustration of prone, 87f
- segmental contact points, 134–136, 135f

Loose-packed positions

- for ankle and foot, 368t
- definition of, 22–23, 23t
- of elbow joints, 319t
- for knee joint, 353b
- for wrist and hand joints, 328t

Lordosis, 158–159, 158f

lumbar, 234–235, 235f

Lordotic curve, 158–159, 158f, 234–235, 235f

Low back pain (LBP)

- diagnostic triage, 89–90
- McKenzie method for, 387–391
- standards of care, 8–9

Lower cervical spine; *See* cervical spine

Lubrication models

- for synovial joints, 17–18, 18f

Lumbar distraction, 387, 387f, 388f

motorized, 387, 387f, 388f

Lumbar extension subluxation, 43–46, 43f

Lumbar flexion-distraction technique, 386, 386b, 386f, 387b

Lumbar motion segment

- interpedicular zone, 43–44, 44f

Lumbar spine

- adjustment techniques/illustrations
 - flexion and extension, 252–253, 253f
 - knee-chest, 247, 253b, 260–261, 260f, 261f
 - lateral flexion, 251–252, 251f, 252f
 - prone, 247, 248f, 253b, 258–260, 259f, 260f
 - rotational, 248–251, 248f, 250f, 251f
 - side-posture, 245, 245f, 246f, 247f, 253–262, 253b, 254f, 256f, 257f, 258f
 - sitting, 248, 248f, 253b, 261–262, 261f
- bony palpation of, 65–66, 65f, 66f
- contraindications and complications of
 - adjustive therapy, 103–105, 103b
- evaluation of, 238–245
 - flexion and extension, 243, 243f, 244f
 - global ROM ranges, 240t
 - joint play, 240–241, 241f, 242f
 - lateral flexion, 242, 243, 243f, 244f
 - motion palpation, 240–245, 241f
 - observation, 238–240, 238f, 239f
 - rotation, 237, 242, 243–245, 243f, 245f
- segmental motion palpation and end play, 242–245, 243f, 244f
- static palpation, 240, 241f

Lumbar spine (*Continued*)

- functional anatomy of
 - description and illustrations of, 233–235, 233f, 234f
 - lumbar curve, 234–235, 235f
- identifying joint subluxation/dysfunction syndrome (JSDS), 151–152, 152b
- lordosis, 234–235, 235f
- manual traction-distraction techniques, 384–387, 387b
- mobilization techniques, 381–384, 382b
- PARTS isolation of dysfunction, 151–152, 152b
- range-of-motion (ROM), 59–60, 60f, 61f, 235–237, 235t
 - flexion and extension, 235–236, 235t, 236f
 - lateral flexion, 236–237, 236f, 237f
 - rotation, 234f, 236f, 237
- spinal joint function and scanning, 146–151, 147b, 147f, 149f
- structure of, 145–146, 146f
- vertebrae structure, 145–146, 146f

M

- MacConnail and Basmajian's model, 20, 21f
- Machine, 11, 12f
- Malingering, 93t
- Malpositions
 - of joints, 47
- Mamillary processes
 - anatomy of, 233–234, 233f, 234f
- Manga report, 8
- Manipulable lesions
 - joint assessment, 36
- Manipulation
 - defining joint, 88
 - model of periarticular changes, 110f
- Manipulative procedures
 - classification of, 85b, 85f
- Manual cervical traction, 387, 388f
- Manual lumbar flexion-distraction
 - technique, 386, 386b, 386f, 387b
- Manual resistance techniques; *See also* manual therapies
 - ischemic compression, 408–409
 - muscle energy technique (MET), 402, 402t, 403b, 403f
 - Nimmo techniques, 408–409, 409b, 409f
 - postisometric relaxation technique (PIR), 402–405, 404f, 405b, 405f
 - proprioceptive neuromuscular facilitation, 405–408, 406b, 407f, 408f
 - receptor-tonus techniques, 408–409, 409b, 409f
 - for soft tissue manipulation, 394b, 401–413
 - trigger point therapy, 409–410, 411f
- Manual therapies
 - classification of, 84, 85f
 - definition of, 85b
- Manual traction-distraction
 - classification of, 84, 85f
 - definition of, 88, 384–387
 - joint manipulative procedures, 88
 - techniques

Manual traction-distraction (*Continued*)

- Cox method, 386, 386b, 386f
- definition of, 384–387
- effects of traction, 385b
- Leader method, 387, 387f, 388f
- manual cervical traction, 387, 388f
- manual lumbar flexion-distraction
 - technique, 386, 386b, 386f, 387b
- motorized cervical traction, 387, 387f, 388b, 388f
- motorized lumbar distraction, 387, 387f, 388f
- treatment aims and benefits, 384–386, 385b, 386b

Manually assisted instrument, 138f

Mass

- and Newton's laws of motion, 25–26

Massage

- classification of, 84, 85f
- connective tissue, 394b, 398–399, 399f
- definition of, 394–398, 394b
- effleurage, 394b, 395, 395f, 396b
- friction, 394b, 397–398, 398b, 398f
- pétrissage, 394b, 395, 396b, 396f
- roulomont, 394b, 396, 396b, 396f
- tapotement, 394b, 396–397, 397b, 397f

McKenzie method, 387–391

- description and benefits of, 387–391
- principles of treatment, 388
- of range-of-motion assessment, 59
- syndromes of pain, 389–391, 389b, 390f, 391f, 392f

Mechanical axis of joints, 20, 21f

Mechanical spine pain

- adjustive therapy to relieve, 89–90

Mechanics, 11

Mechanoreceptors, 18–20, 19f

Median sagittal plane, 12–13, 13f, 13t, 14f

Menisci

- of knee, 352, 352f

Meniscoid entrapment

- illustration of, 112f, 113f
- theory of, 40, 40f

Meniscoids

- illustration of, 29–30, 30f, 112f

Mercy Conference, 8

Metabolism

- effects of soft tissue manipulation on, 393–394

Metacarpophalangeal joints

- adjustive procedures, 337, 337f
- capsular patterns, 71t

Metatarsophalangeal joints

- adjustments, 372b, 378, 378f
- capsular patterns, 71t

Midcarpal joint

- capsular patterns, 71t

Midsagittal plane, 12–13, 13f, 13t, 14f

Mobilization

- classification of, 84, 85f
- of joints (*See* joint mobilization)

Models of spine function

- and joint biomechanics, 33–34, 34f

Morphologic muscles, 26–33, 27f

Motility

- skin assessment technique, 66f

Motion

- definition and axes of, 13–15, 13f, 14f
- and joint biomechanics, 13–15, 14f, 15t, 25–26

Motion palpation, 61–63, 67–72

- accessory joint motion, 68f, 69–71, 69f, 70f
- cervical spine, 165–169, 165f, 166f, 167f, 168f, 169f, 170f, 174b, 174f
- and end play, 69–71, 70b, 70f
- goals of, 72b
- and joint play, 68f, 69–71, 69f, 70f
- lumbar spine, 240–245, 241f
- principles of, 67–72, 72b
- summary of reliability studies regarding, 62, 429t, 432t, 433t, 434t, 435t, 437t

thoracic spine, 195–200, 197f, 198f

- tools, instructions and tips, 67b, 72b

Motion scans

- of end play, 147b, 149f, 150f
- to identify joint dysfunction, 148–151, 149f, 150f, 151f
- of joint play, 147b, 148–151, 149f, 150f
- of passive range-of-motion, 147b, 150f

Motion-assisted thrusts

- drop-section mechanical assistance, 143
- motorized, 143–144
- techniques, 142–144

Motorized cervical traction, 387, 387f, 388b, 388f

Motorized lumbar distraction, 387, 387f, 388f

Movements

- axes of, 13, 13f, 14f
- hypermobility *versus* instability affecting, 42t
- and joint anatomy, 12–13, 13f, 13t
- and proprioceptive neuromuscular facilitation (PNF), 405–408, 406b, 407f, 408f

Multipennate muscles, 26–33, 27f

Muscle energy technique (MET), 402, 402t, 403b, 403f

Muscle hypertonicity

- effects of soft tissue manipulation on, 393–417

Muscle relaxation

- and massage, 394–398, 394b
- postisometric relaxation technique (PIR), 402–405, 404f, 405b, 405f

Muscle spasms

- end feel, 70b
- postisometric relaxation procedure on, 405f
- relief, 116–120, 117f

Muscle tests

- and joint assessment, 73–74, 73f, 74b
- during JSDS evaluation, 51, 51b

Muscle tone

- effects of soft tissue manipulation on, 393–394

Muscles

- biomechanics of, 27–28
- as connective tissues, 26f, 27–28, 27f
- effects of soft tissue manipulation on, 393–417
- elastic and viscous stretch, 26–33, 26f

- Muscles (*Continued*)
 elbow, 316–317, 317t, 318f
 five-point grading system for, 74b
 foot and ankle, 366–367, 367f, 367t
 hip, 340–341, 341t
 and joint biomechanics, 27–28, 27f
 knee, 352–353, 352t, 353f
 manipulation of (*See* manual resistance techniques)
 roles and types of, 27–28, 27f
 soft tissue palpation of, 66–67, 66f, 67b, 67f
 and spine function models, 33–34
 suboccipital, 154, 154f
 surface electromyography to evaluate, 81–82
 wrist and hand, 326, 328f, 328t
- Muscular end feel, 70b
- Musculoskeletal system
 dysfunction
 benefits/effects of chiropractic, 105
 focus of chiropractic on, 4
 stressors affecting, 118–120, 119f
- Myofascial cycle
 benefits/effects of chiropractic on, 116–120, 117f
 diagram illustrating conditions of, 39–40, 39f
 trigger points, 409–410, 411f, 412b, 412f
- Myofascial release technique (MRT), 394b, 400–401, 401b, 401f
- Myofascial trigger points, 409–410, 411f, 412b, 412f
- N**
- National Center for Complementary and Alternative Medicine (NCCAM), 6, 7
- Nerve root compression
 benefits of adjustments on, 117–118
 and intervertebral encroachment, 43–44, 43f
 with vertebral subluxation complex (VSC), 43–44
- Nerve roots
 lumbar, 233–234, 234f
 pain, 89–90
- Nerves
 segmental innervation related to viscera, 418t
 soft tissue palpation of, 66–67, 66f, 67b, 67f
 spinal, 18–20, 19f, 20f
- Nervous system
 chiropractic principles regarding, 3–9
 segmental innervation related to viscera, 418t
 tone, 4
 trophotropic and ergotropic responses, 119t
- Neurobiologic hypotheses
 analgesic hypothesis, 115, 116f
 on myofascial cycle, 116–120, 117f
 on nerve root compression, 117–118
 on neuroimmunology, 118–120, 119f, 119t
 on reflex paradigm, 118
 regarding muscle spasms, 116
 regarding pain, 115, 116f
- Neurodystrophic hypothesis, 118–120
- Neuroimmunology
 benefits/effects of chiropractic, 118–120, 119f, 119t
- Neurolymphatic reflexes, 412–413, 414f, 415f
- Neuromuscular barriers, 402, 402t
- Neuromusculoskeletal (NMS) system
 benefits/effects of chiropractic on, 105
 as core focus area, 35
 past and present theories concerning, 3–9
 range-of-motion assessment, 59–60, 60f, 61f
 scope of practice, 6
- Neurovascular reflexes, 413, 416f, 417f
- Newton's laws of motion
 and joint biomechanics, 25–26
- Nimmo techniques, 408–409, 409b, 409f
- Nociceptors
 function of, 19
- Nomenclature
 of spinal listings, 47, 48f
- Nonpause thrusts, 139
- Nonspecific spine pain, 89–90
- Nonthrust procedures
 body wall reflex techniques, 410–413
 acupressure point stimulation, 394b, 410–412, 411f, 412f
 Bennett (neurovascular) reflexes, 413, 416f, 417f
 Chapman reflexes, 412–413, 414f, 415f
 cranial manipulation, 391–392
 controversy and hypotheses regarding, 391–392, 392b
 and cranial dysfunction, 392b
 cranial universal, 392, 393f
 parietal lift, 392, 393f
 sagittal suture spread, 392, 392f
 joint mobilization, 381–384
 definition of, 381–382
 examples of, 383–384, 383f, 384f
 primary goal of, 382
 types of, 382–383, 382b
 Logan basic technique, 413–417, 417f
 manual resistance techniques
 ischemic compression, 408–409
 muscle energy technique (MET), 402, 402t, 403b, 403f
 Nimmo techniques, 408–409, 409b, 409f
 postisometric relaxation technique (PIR), 402–405, 404f, 405b, 405f
 proprioceptive neuromuscular facilitation, 405–408, 406b, 407f, 408f
 receptor-tonus techniques, 408–409, 409b, 409f
 trigger point therapy, 409–410, 411f
 manual traction-distraction techniques
 Cox method, 386, 386b, 386f
 definition of, 384–387
 effects of traction, 385b
 Leader method, 387, 387f, 388f
 manual cervical traction, 387, 388f
 manual lumbar flexion-distraction technique, 386, 386b, 386f, 387b
 motorized cervical traction, 387, 387f, 388b, 388f
 motorized lumbar distraction, 387, 387f, 388f
 treatment aims and benefits, 384–386, 385b, 386b
- Nonthrust procedures (*Continued*)
 McKenzie method, 387–391
 description and benefits of, 387–391
 principles of treatment, 388
 syndromes of pain, 389–391, 389b, 390f, 391f, 392f
 soft tissue manipulation
 effects of, 393–394
 functional techniques, 394b, 399–401, 400f, 401b
 manual resistance techniques, 394b, 401–413
 massage techniques, 394–398, 394b
 spondylotherapy, 417, 417f, 418t
- Noxious generative points, 408–409
- Nursemaid's elbow, 319
- Nutritional supplements
 as adjunct chiropractic service, 4
- O**
- Observation
 to evaluate cervical spine, 162
 to evaluate lumbar spine, 238–240, 238f, 239f
 to evaluate thoracic spine, 195
- Orthopedic subluxation
 definition of, 37b
- Orthopedic tests
 for joint assessment, 74
- Oscillatory atlas lateral glide, 383, 383f
- Oscillatory cervical lateral flexion, 383, 383f
- Oscillatory posterior-to-anterior glide, 383, 384f
- Oscillatory techniques
 of joint mobilization, 382–383, 382b, 383f, 384f
- Osgood-Schlatter's disease, 355
- Osseous barriers, 402, 402t
- Osseous structures
 of elbow, 316, 316f
 foot and ankle, 365, 365f
 hand and wrist, 326, 326f, 331f
 hip, 337–339, 338f
 of knee, 350, 350f
- Osteoarthritis, 93t
- Osteokinematics
 ankle and foot, 367–368, 367t
 description and illustration of, 20–23, 21f
 elbow, 318t
 hip, 341–343, 343t
 knee, 354t
 wrist and hand, 329t
- Osteopathic lesions
 definition of, 37b
- Osteopathy
 philosophic roots of, 3
- Osteoporosis, 93t
- Outcomes
 measures for JSDS, 91–92, 91b
 regarding pain, 53
- P**
- Pain; *See also* referred pain
 adjustive therapy to relieve, 89–90
 articular neurology, 18–20, 19f
 assessment during JSDS examination, 50–51, 51b, 53, 54f

Pain; *See also* referred pain (*Continued*)

- derangement syndrome, 389, 389b, 391f, 392f
 - dysfunctional syndrome, 389, 389b, 391f
 - effects of soft tissue manipulation on, 393–417
 - and goals of motion palpation, 72b
 - location, quality, and intensity, 50–51, 53, 54f
 - mechanical spine, 89–90
 - nerve root, 89–90
 - neurobiologic hypotheses, 115, 116f
 - outcomes, 53
 - and PARTS acronym, 50–51, 51b
 - postural syndrome, 389, 389b, 390f
 - relief
 - benefits/effects of chiropractic on, 115–120, 116f, 117f, 118f, 119f, 119t
 - scans, 147b, 148f, 149f
 - and spinal nerves, 18–20, 19f
 - syndromes and McKenzie method, 389–391, 389b, 390f, 391f, 392f
 - visual analog severity scales, 53, 54f
- Pain assessment
- algometry, 79–80
- Pain provocation
- description of, 71–72
 - summary of reliability studies regarding, 62, 432t, 433t, 434t, 435t, 437t
- Pain reaction point (PRP), 79–80, 80f
- Pain scans
- to identify joint dysfunction, 147b, 148f, 149f
- Palmer, B.J., 2
- Palmer, Daniel David, 1–3
- Palpation, 60–65
- bony palpation, 65, 65f
 - to determine pain location, quality, and intensity, 50–51, 54f
 - motion palpation, 67–72
 - reliability, 61–63
 - sacroiliac articulation, 65
 - soft tissue palpation, 66–67, 66f
 - summary of reliability studies regarding, 62, 429t, 432t, 433t, 434t, 435t, 437t
 - validity of procedures, 63–65, 440t
- “Paradigm of Chiropractic”, 9
- Paraspinal muscle dysfunction
- surface electromyography to measure, 81–82
- Paraspinal tissue texture, 50, 51b
- Parietal lift, 392, 393f
- PARTS
- acronym, 50, 51, 51b
 - isolation of dysfunction, 151–152, 152b
- Passive congestion barriers, 402, 402t
- Passive intervertebral motion tests, 50
- Passive mobility
- and proprioceptive neuromuscular facilitation (PNF), 405–408, 406b, 407f, 408f
- Passive range-of-motion (PROM)
- description and illustration of, 67–72, 68f
- Patella, 350, 350f, 353–354, 354f, 355f, 356f

Patellofemoral joints

- accessory movements of, 354–357, 356t, 358f
 - adjustments, 360b, 362, 363f
- Patient positioning
- for adjustive therapy, 124–127, 125f, 126f
 - as criteria for categorizing adjustments, 86
- Patients
- outcome measures, 91–92, 91b
 - positioning, 86, 124–127, 125f, 126f
 - size, age, and flexibility variables, 122b
- Pause thrusts, 139–140
- Pelvic stability, 342–343, 344f
- Pelvis
- bony palpation of, 65–66, 65f, 66f
- Percussion
- for joint assessment, 72, 73f
- Perimysium, 27, 27f
- Peripheralization
- of symptoms, 388
- Pétrissage massage, 394b, 395, 396b, 396f
- Philosophy of chiropractic, 3
- Philosophy-based education, 5–6
- Physical examinations
- during JSDS evaluations, 50
- Physical therapies
- as adjunct chiropractic service, 4
- Physicians; *See* doctor of chiropractic (DC)
- Physiologic barriers, 67–72, 68f
- Physiologic joints
- description and illustration of, 20, 20f, 21f
- Pia mater
- in lumbar motion segment, 43–44, 44f
- Pinching
- or kneading pétrissage massage, 394b, 395, 396b, 396f
- Plantar fasciitis, 369
- Plumb line observation
- of lumbar spine, 238–245, 238f
 - of posture, 56, 57f, 57t, 147–151, 147f, 238–245, 238f, 239f
- Point pressure techniques
- classification of, 84, 85f
- Ponos, 4
- Positional release therapy (PRT), 394b, 400, 400f
- Positioning
- assisted and resisted, 124–126, 126f
 - doctor, 133, 133f
 - neutral, 126
 - patient, 124–127, 125f, 126f
 - principles of, 126–127
 - tables and equipment, 129–132, 129f, 130f, 131f, 132f
- Posterior longitudinal ligament (PLL), 17, 17f, 145, 146f
- Posterior root ganglion, 43–44, 44f
- Postisometric relaxation technique (PIR), 402–405, 404f, 405b, 405f
- Postural syndrome
- of pain, 389, 389b, 390f
- Posture
- evaluation of
 - during joint subluxation/dysfunction syndrome (JSDS) exam, 50, 55–56, 57f, 57t

Posture (*Continued*)

- methods, 56, 57f, 57t
 - plumb line observation, 56, 57f, 57t, 147–151, 147f, 238–245, 238f
 - scans to identify joint dysfunction, 147, 147b, 147f
 - and hip movements, 338–339, 340–341
 - scans, 147, 147b, 147f
 - spinal models and, 33–34
- Posture scans, 147, 147b, 147f
- Pressure pain threshold (PPT), 79–80, 80f
- Pressure tolerance (PTo), 79–80, 80f
- Primary care chiropractic physician
- defined by Council on Chiropractic Education (CCE), 35
- Primary respiratory mechanism, 392
- Progressive stretch lumbar rotation, 383–384, 385f
- Progressive stretch mobilization, 382
- Progressive stretch thoracic extension, 383, 384f
- Pronation
- of ankle and foot, 367–368, 368f
 - and joint motions, 14–15, 14f
- Prone positions; *See* adjustments
- Proprioceptive neuromuscular facilitation (PNF), 405–408, 406b, 407f, 408f
- Provocative tests
- description of, 71–72
 - for joint assessment, 74
 - to localize pain, 53, 54f
- Psychoneuroimmunology (PNI), 119
- Pubofemoral ligaments, 340, 340f
- Pulled elbow, 319

Q

Quality

- as goal for future, 9
- of joint range-of-motion, 72b

R

- Radial deviation, 329, 329f
- Radiographic analysis
- diagnostic rationale, 76b
 - functional x-ray exams, 77–78, 77f
 - fundamental clinical principles of, 79, 79b
 - lateral-flexion radiographs, 78, 78f
 - marking system limitations, 74, 76b
 - spinal x-ray exams, 76–77
 - spinography, 74–79, 75f
 - videofluoroscopy (VF), 78–79, 79b
- Radioulnar joint, 71t, 318, 318f
- RAND report, 8
- Range-of-motion (ROM)
- active
 - description and illustration of, 67–72, 68f
 - assessment
 - diagram illustrating, 68f
 - of elbow, 318t
 - goals and traction, 384–386, 385b
 - hypermobility *versus* instability affecting, 42t
 - and joint stability and connective tissues, 26–33, 26f
 - during JSDS evaluation, 50, 51, 51b
 - of knee joint, 353–354
 - of shoulder, 298, 299f

- Range-of-motion (ROM) (*Continued*)
 cervical spine, 59–60, 60f, 61f
 lumbar spine, 234f, 235–237, 235t, 236f, 237f
 McKenzie method, 59
 measurement procedures, 59–60, 60f, 61f, 62t
 mobilization goals of restoring, 382–383
 of neuromusculoskeletal (NMS) system, 59–60, 60f, 61f
 passive
 description and illustration of, 67–72, 68f
 motion scans, 147b, 150f
 quality goals, 72b
 of spine, 59–60, 60f, 61f
 thoracic spine, 147f, 189–190
 Receptor-tonus techniques, 408–409, 409b, 409f
 Recoil thrust, 137–138, 138f
 Red muscle, 27
 Referred pain, 53, 54f
 to ankle and foot area, 368, 370f
 elbow, 320f
 hip, 344–346, 344f
 knee, 356f
 Reflexes
 benefits/effects of adjustments on, 118, 118f
 Bennett (neurovascular), 413, 416f, 417f
 Chapman, 412–413, 414f, 415f
 muscle spasms, 394
 neurobiologic hypotheses on, 118
 neurolymphatic, 412–413, 414f, 415f
 neurovascular, 413, 416f, 417f
 and spinal nerves, 18–20, 19f
 Relaxation
 and deceleration, 27–28
 and massage, 394–398, 394b
 postisometric relaxation technique (PIR), 393–417, 394b, 404f, 405b, 405f
 “Release”, 400–401
 Reliability
 of palpation, 61–63
 summary of studies regarding, 62, 429t, 432t, 433t, 434t, 435t, 437t
 Repeated quick stretch
 and proprioceptive neuromuscular facilitation (PNF), 405–408, 406b, 407f, 408f
 Repetitive exercises, 92b
 Research
 early chiropractic, 7
 Resistance, 11, 12f
 and concentric contractions, 27–28
 Resisted adjustive methods
 versus assisted positioning, 124–126, 125f, 128f, 136t
 description of, 140–142
 illustration of, 128f
 Responsiveness
 of diagnostic tests, 53
 Restrictions
 joint, 47
 Retinacula, 339–340, 340f
 Retroversion, 338–339, 340–341
 Reversal of antagonists
 and proprioceptive neuromuscular facilitation (PNF), 405–408, 406b, 407f, 408f
 Reversal of direction, 405–408, 406b, 407f, 408f
 Rhythmic initiation
 and proprioceptive neuromuscular facilitation (PNF), 405–408, 406b, 407f, 408f
 Rib cage
 movements with respiration, 192–193, 193f
 movements with spine, 192, 192f
 Ribs; *See also* thoracic spine
 adjustment types for
 covered-thumb/costal push, 230, 231f
 hypothenar/costal push, 228, 228f, 229, 230f, 232, 232f
 ilial hypothenar/costal push, 230, 230f
 index/costal push, 228, 228f, 229, 229f, 231, 231f
 modified hypothenar/costal push, 229, 229f
 thenar/costal drop, 226, 227f
 web/costal push, 231, 231f
 respiration kinetics, 193
 Roll, 20, 21f
 Rotation
 body planes and axes associated with, 12–15, 13f, 13t
 cervical spine
 description and illustration of, 161, 161f
 effects of on lumbar segments, 25f, 248–251
 evaluation of lumbar spine, 237, 242, 243–245, 243f, 245f
 illustration of thrusts to induce, 138–139, 139f
 inducing, 181f, 182
 instantaneous axis of rotation (IAR), 21, 22f
 lumbar segment
 illustration of, 234f
 lumbar spine
 adjustment techniques/illustrations, 248–251, 248f, 250f, 251f
 movements, 13, 13f, 14f
 Rotational movements, 13, 13f, 14f
 Roulemont massage, 394b, 396, 396b, 396f
- S**
- Sacroiliac articulation
 dysfunction definition, 65
 palpation, 65
 Sacroiliac region
 summary of reliability studies regarding, 62, 432t, 433t, 434t, 435t, 437t
 Sagittal plane, 12–13, 13f, 13t, 14f, 125f
 Sagittal suture spread, 392, 392f
 Scanning-surface EMG, 81–82
 Schmorl’s node, 24, 32, 32f
 Schools
 chiropractic, 5–6
 Scope of practice
 chiropractic, 6
 “Screw-home” mechanism, 353–354, 354f
 Segmental motion palpation
 and end play
 cervical spine, 160t, 165, 165f, 167, 168–169
 lumbar spine, 242–245, 243f, 244f
 thoracic spine, 197–199, 198f, 199f
 Segmental range of motion (SROM)
 assessing and measuring, 50, 51b, 68f, 72b
 evaluation of cervical spine, 160t, 165–169, 165f, 166f, 168f
 of lower cervical spine, 159–161, 160t
 thoracic spine, 195, 195t
 of upper cervical spine, 155–157, 155t
 vertebral functional anatomy and
 C0–C1, 154–155, 155f, 155t, 156f, 165–169
 C1–C2, 155–157, 155t, 156f, 157f, 165–169
 C3–C7, 157–162, 157f, 158f, 160t, 165–169
 Segmental venous drainage, 46, 46f
 Semidirect thrusts
 definition of, 85b, 134–136
 Shear forces
 definition of, 13
 and joint biomechanics, 13, 24, 24f
 Shin splints, 368
 Short-lever thrusts
 and adjustment selection, 122b
 definition of, 85b
 illustration of prone, 87f
 segmental contact points, 134–136, 135f
 Shoulder, 294–315
 adjustive procedures
 acromioclavicular joint (sitting), 305b, 310, 311f
 acromioclavicular joint (supine), 305b, 310, 310f, 311f
 glenohumeral joint (prone), 304–307, 305b, 305f, 306f, 307f, 308f
 glenohumeral joint (sitting), 304–307, 305b, 309f
 glenohumeral joint (standing), 304–307, 305b, 309f
 glenohumeral joint (supine), 304–307, 305b, 305f, 306f, 307f, 309f
 scapulocostal articulation (side), 305b, 314–315, 314f, 315f
 sternoclavicular joint (sitting), 305b, 311–313, 314f
 sternoclavicular joint (supine), 305b, 311–313, 312f, 313f
 biomechanics of, 298, 299f
 capsular patterns, 71t
 close-packed positions for, 23t
 evaluation of, 298–302, 299f, 300f, 300t, 301f, 302f, 303f, 304f
 functional anatomy, 294–298, 295f, 296f, 297f, 297t
 Side effects
 of adjustive therapy, 94b
 Side-posture IVD debate, 103–105, 104f
 Skin
 soft tissue palpation of, 66–67, 66f, 67b, 67f
 Skin rolling, 148f, 396, 396f
 Skull
 center of gravity of, 158f
 Slide
 definition of, 13–15
 description and illustration of, 20, 21f
 Slouch-overcorrect exercise, 389, 390f
 Soft tissue approximation
 end feel, 70b
 Soft tissue injury, 39

Soft tissue manipulation
 classification of, 84, 85f
 effects of, 393–394
 functional techniques, 394b, 399–401, 400f, 401b
 manual resistance techniques, 394b, 401–413
 massage techniques, 394–398, 394b
 palpation, 50, 66–67, 66f
 types and procedures, 88–89, 89b
 Soft tissue palpation, 50, 66–67, 66f
 Somatic dysfunction
 definition of, 37b
 Somatic reflexes
 theory of altered, 45–46
 Somatosomatic reflexes
 theory of altered, 45–46
 Somatovisceral reflexes
 theory of altered, 45–46
 Specific spinal adjustments
vs. general, 87
 Spin, 20, 21f
 Spinal cord
 and spine function models, 33–34
 Spinal dysfunction and degeneration
 Gillet Model, 42
 Kirkaldy-Willis' Model, 42–43
 Spinal joint scans
 global range-of-motion (GROM), 147–148, 147b, 148f
 to identify JSDS, 151–152
 motion scans, 148–151, 149f, 150f, 151f
 pain scans, 147b, 148f, 149f
 posture scans, 147, 147b, 147f
 summary of, 147b
 Spinal joints
 evaluating function of, 146–151
 function and scanning, 146–151, 147b, 147f, 149f
 Spinal listings
 and variations, 47, 48f
 Spinal nerve root compression, 43–46, 43f
 Spinal nerves
 description and illustration of, 18–20, 19f, 20f
 in lumbar motion segment, 43–44, 44f
 Spinal palpation, 65–66, 65f, 66f
 Spinal physical capacity tests
 for joint subluxation/dysfunction syndrome (JSDS), 91–92, 92b
 Spinal stenosis
 pain caused by, 89–90
 Spinal x-ray examinations, 76–77
 Spine; *See also* cervical spine; lumbar spine; thoracic spine
 capsular patterns, 71t
 characteristics of, 33–34
 close-packed positions for, 23t
 mobilization
 examples, 383–384, 383f, 385f
 techniques, 381–384, 382b
 models
 of dysfunction and degeneration, 42–43
 of function, 33–34
 past and present theories concerning, 3–9
 range-of-motion assessment, 59–60, 60f, 61f
 summary of reliability studies regarding, 62, 432t, 433t, 434t, 435t, 437t

Spinous processes
 anatomy of, 146f, 233–234, 233f, 234f
 bony palpation of, 65–66, 65f, 66f, 148f, 149f
 Spiritualism, 3
 Spondylosis
 degenerative pathological sequence, 43, 43f
 Spondylotherapy, 417, 417f, 418t
 Sprains, 28, 93t, 368
 Standards of care
 guidelines in chiropractic, 8–9
 Static back endurance tests, 92b
 Static listing chart, 47, 48f
 Static palpation
 cervical spine, 162–164
 description of, 61–63
 lumbar region, 240, 241f
 thoracic spine, 195, 196f, 197f
 Stenosis
 degenerative pathological sequence, 43, 43f
 Sternoclavicular joint
 sitting adjustments, 305b, 311–313, 314f
 supine adjustments, 305b, 311–313, 312f, 313f
 Still, Andrew Taylor, 1, 3
 Straight chiropractic, 2
 Straight-leg raising test, 53
 Strain-counterstrain technique, 394b, 399, 400f
 Stretch
 defining connective tissue, 26–33
 Stroke
 precautions in adjustive therapy, 95, 95f, 96–100, 96f, 97f, 101–102, 101b, 102b
 Structural approach
 Carver's subluxation, 2
 Structural ligaments
 or membranes, 391–392
 Subluxation complex
 definition of, 37b
 Subluxation syndrome
 definition of, 37b
 diagnosing, 36
 Subluxation-based education, 5–6
 Subluxations
 definition discussion, 36–37
 definition of, 37b
 documentation of, 82f, 83f, 83t
 history of concept of, 2
 joint subluxation/dysfunction syndrome (JSDS)
 clinical evaluation of, 47–82
 diagnosis parameters, 47, 50–51
 diagnostic procedures, 52, 53
 examination procedures, 50–51, 51b, 54–59
 five diagnostic categories, 50
 gait evaluation, 55, 55b, 55f, 56b
 leg length evaluation, 56–59, 58f
 outcomes and measures, 53
 pain, 53, 54f
 patient observation, 54–59
 physical examination findings to support, 51b, 54–59
 postural evaluation, 55–56, 57f, 57t
 spinal listings and variations, 47, 48f

Subluxations (*Continued*)
 symptoms of, 53
 terminology and nomenclature
 confusion, 47
 original chiropractic theories regarding, 4
 vertebral subluxation complex (VSC)
 degenerative cycle, 42–43, 43f
 inflammatory and vascular
 components, 46–47
 joint instability and hypermobility, 41–43
 mechanical components of, 37b, 38–43
 models of spinal dysfunction and degeneration, 42–43
 neurobiologic components, 43–46
 terminology, 37b
versus vertebral subluxation/
 dysfunction syndrome, 37–47, 37b
 Suboccipital muscles, 154, 154f
 Supination
 of ankle and foot, 367–368, 368f
 and joint motions, 14–15, 14f
 Supine positions; *See* adjustments
 Surface electromyography, 81–82
 Swing phase, 368, 369f
 Synarthrotic joints
 structure and examples of, 15t
 Synovial fluid
 of facet joints, 29–30, 30f
 and joint biomechanics, 17–18, 18f
 Synovial joints; *See also* joints
 and joint biomechanics, 15
 ligamentous elements of, 17, 17f
 neurology of, 18–20, 19f, 20f
 terminology of disorders, 37b

T

Tapotement massage, 394b, 396–397, 397b, 397f
 Temperature
 assessment during JSDS evaluation, 50, 51, 51b
 effects of soft tissue manipulation on, 393–394
 skin assessment technique, 66f
 thermography, 80, 80f
 Temporomandibular joint (TMJ)
 adjustive procedures, 291–294, 291b, 292f, 293f, 294f
 biomechanics of, 286–288, 287f, 288b, 288t
 evaluation of, 288–289, 288b, 289b, 289f, 290f, 291f
 functional anatomy, 284–286, 284f, 285f, 286f, 286t
 Tendon sheaths
 soft tissue palpation of, 66–67, 66f, 67b, 67f
 Tendons
 effects of soft tissue manipulation on, 393–417
 soft tissue palpation of, 66–67, 66f, 67b, 67f
 Tennis elbow, 319
 Tensegrity systems, 34, 34f
 Tensile stresses
 on intervertebral discs (IVDs), 30–33, 32f

- Tension forces
 - and joint biomechanics, 24, 24f, 25f
- Tension trabeculation, 339, 339f
- Terminology; *See also* glossary
 - kinesiologic trunk and neck, 47, 48f
 - and nomenclature confusion, 47
- Texture
 - assessing during JSDS evaluation, 50, 51, 51b
 - skin assessment technique, 66f
- Theory of altered somatic and visceral reflexes, 45–46
- Theory of intervertebral encroachment, 43–44
- Therapeutic muscle stretching
 - classification of, 84, 85f
- Thermography, 80, 80f
- Thoracic spine
 - adjustment types at thoracocervical junction, 211–232, 212b
 - bilateral/thenar and hypothelar/transverse push, 213, 214f
 - hypothelar/transverse push, 212, 213f
 - thumb/spinous push, 211, 212f, 214, 214f, 215, 215f
 - adjustment types for costosternal region covered-thumb/costosternal push, 232, 232f
 - hypothelar/costosternal pull, 232, 233f
 - adjustment types for ribs
 - covered-thumb/costal push, 230, 231f
 - hypothelar/costal push, 228, 228f, 229, 230f, 232, 232f
 - ilial hypothelar/costal push, 230, 230f
 - index/costal push, 228, 228f, 229, 229f, 231, 231f
 - modified hypothelar/costal push, 229, 229f
 - thenar/costal drop, 226, 227f
 - web/costal push, 231, 231f
 - adjustment types in knee-chest position
 - hypothelar/spinous push, 220, 220f
 - hypothelar/transverse and bilateral hypothelar/transverse push, 220, 220f
 - adjustment types in prone positions
 - bilateral hypothelar/transverse push, 216, 217f
 - bilateral thenar/transverse push, 216, 216f
 - hypothelar spinous crossed thenar/transverse push, 219, 219f
 - unilateral hypothelar/spinous push, 217, 218f
 - unilateral hypothelar/transverse push, 140f, 141f, 218, 219f
 - adjustment types in sitting position
 - hypothelar/transverse pull, 223, 224f
 - adjustment types in standing position
 - thenar/transverse push, 225, 225f
 - thoracic long-axis distraction, 226, 226f
 - adjustment types in supine position
 - opposite-side thenar/transverse drop, 221, 221f
 - same-side thenar/transverse drop, crossed arm, 223, 223f
 - thenar/transverse drop, pump handle, 223, 224f
- Thoracic spine (*Continued*)
 - adjustments overview
 - flexion and extension, 209–210, 209f, 210f, 211f
 - lateral flexion dysfunction, 205f, 207f, 208–209, 208f, 209f
 - ribs, 211, 211f, 227f, 230f, 231f
 - rotational dysfunction, 204–208, 205f, 206f, 207f
 - bony palpation of, 65–66, 65f, 66f
 - contraindications and complications of adjustments, 102–103
 - evaluation of, 195–200
 - global range-of-motion, 195, 195t
 - joint play, 195–200, 197f
 - lateral flexion, 198–199, 199f
 - motion palpation, 195–200, 197f, 198f
 - observation, 195
 - posture, 195, 195f
 - rib motion palpation, 199–200, 201f
 - rotation, 198, 199f
 - segmental motion palpation and end play, 197–199, 198f, 199f
 - static palpation, 195, 196f, 197f
 - functional anatomy and biomechanics
 - kinetics, 191, 193
 - respiration kinetics, 193
 - of rib cage, 191–193, 192f, 193f
 - rib cage movements with respiration, 192–193, 193f
 - rib cage movements with spine, 192, 192f
 - and structures, 188–189, 188f, 189f
 - thoracocervical junction, 193, 194f, 194t
 - thoracolumbar junction, 193–194, 194f, 194t
 - of transitional areas, 193–194, 194f, 194t, 195t
 - identifying joint subluxation/dysfunction syndrome (JSDS) in, 151–152, 152b
 - overview of adjustments, 200, 211
 - PARTS isolation of dysfunction, 151–152, 152b
 - positioning for adjustments
 - knee-chest, 202, 202f, 220–221, 220f
 - prone, 200–210, 201f, 211, 212b, 212f, 213f, 215b, 216f, 218f, 219f, 228–229, 228f, 229f, 230f
 - side, 215, 215f
 - sitting, 191f, 202, 202f, 211, 212b, 214–215, 223–225, 224f, 231–232, 232f
 - standing, 204, 204f, 225–226, 225f, 226f
 - supine, 202–204, 203f, 204f, 211, 221–223, 221f, 223f, 224f, 226–232, 280–281
 - range-of-motion (ROM) and patterns, 147f, 189–190
 - flexion and extension, 189–190, 190f, 190t, 191, 191f
 - lateral flexion, 190, 190f, 191, 191f
 - rotation, 190, 191, 191f
 - spinal joint function and scanning, 146–151, 147b, 147f, 149f
 - structure of, 145–146, 146f
 - vertebrae structure, 145–146, 146f
- Thrusts
 - categorizing, 86
 - definition of, 85b, 137–142
 - force *versus* time, 107f
 - impulse, 138–139, 138f
 - motion-assisted techniques, 142–144
 - nonpause, 139
 - patient positioning for, 124, 125f
 - recoil, 137–138, 138f
 - selecting types of, 122b, 123f
 - techniques in adjustive therapy, 137–142, 138f, 139f, 140f, 141f
- Thumb carpometacarpal joints
 - capsular patterns, 71t
- Tibiofemoral joints
 - accessory movements of, 354–357, 356t, 358f
 - assessment of, 354–357, 356t, 357f
- Tibiofibular joints
 - accessory movements of, 354–357, 356t, 358f
 - adjustments, 362–364, 363f, 365f
- Tissue
 - texture of, 50, 51, 51b
- Tissue pull, 136–137
- Toes
 - close-packed positions for, 23t
- Tone
 - assessment during JSDS evaluation, 50, 51, 51b
 - and Palmer's nervous system efficiency model, 4
 - skin assessment technique, 66f
- Torque forces
 - definition of, 13
 - and joint biomechanics, 13, 24f, 25, 26f
- Trabecular patterns
 - and joint biomechanics, 15–16, 16f
- Traction
 - applications and process of, 88
 - definition of, 88, 384–386
 - and tissue pull, 136–137
- Traction/distraction
 - classification of, 84, 85f
- Transitional zones
 - of cartilage, 16, 16f
- Translational movements
 - description and illustration of, 13, 13f, 14f
 - hypermobility *versus* instability affecting, 42t
- Transverse friction massage, 394b, 397, 398f
- Transverse plane, 12–13, 13f, 13t, 14f, 125f
- Transverse processes
 - anatomy of, 146f, 233–234, 233f, 234f
 - bony palpation of, 65–66, 65f, 66f
- Trauma
 - to muscles and joints, 28
- Travell and Simons trigger point therapy, 408, 409–410, 411f
- Trigger point therapy, 409–410, 411f
- Trigger points
 - chart illustrating common, 411f
 - definition of, 394b, 399, 400f, 409–410
 - and receptor-tonus technique, 408–409, 409b, 409f
- Trophotropic responses
 - characteristics of, 119t

Trunk flexion, 237–238, 238f,
341–342, 341f
Tumors, 93t
Two-joint muscles, 341

U

Ulnar deviation, 329, 329f
Uncarthrosis, 93t
Unipennate muscles, 26–33, 27f
Upper cervical spine; *See* cervical spine
Utility
 of diagnostic tests, 53

V

Validity
 summary of palpation procedures, 63–65,
 440t
Vascular components
 of vertebral subluxation complex (VSC),
 46–47
Vector lines of drive, 135f, 137
Vertebrae
 anatomic structure of, 145–146, 146f,
 152–162, 153f, 156f, 157f, 158f,
 160f, 161f, 172f
 anatomical illustration of, 146f
 functional anatomy of
 lower cervical spine, 157–162, 157f,
 158f, 159f, 160f, 160t, 161f
 upper cervical spine, 152–157, 153f,
 155f, 155t
 intervertebral discs (IVDs)
 characteristics of, 30–33, 31f, 32f
 and spinal motion, 146f, 147b
 and spine function models, 33–34
 structures that compose, 145–146, 146f
Vertebral artery injury (VAI)
 with adjustive therapy, 95, 95f, 96–100,
 96f, 97f, 101–102, 101b, 102b

Vertebral column
 intervertebral discs (IVDs)
 characteristics of, 30–33, 31f, 32f
 and spine function models, 33–34
Vertebral subluxation complex (VSC)
 degenerative cycle, 42–43, 43f
 inflammatory and vascular components
 inflammatory reactions, 46–47, 46f
 vascular congestion, 46, 46f
 joint instability and hypermobility, 41–43
 mechanical components of, 37b, 38–43
 compressive buckling injuries, 41
 interarticular block, 40, 40f
 interarticular derangements, 40–41
 interdiscal blocks, 40–41, 41f
 joint fixation (hypomobility), 39–41
 joint manipulation, 38–39
 myofascial cycle, 39–40, 39f
 soft tissue injury, 39
 models of spinal dysfunction and
 degeneration, 42–43
 Gillet Model, 42
 Kirkaldy-Willis' Model, 42–43
 neurobiologic components, 43–46
 nerve root compression, 43–44
 theory of altered somatic and visceral
 reflexes, 45–46
 Theory of intervertebral encroachment,
 43–44
Vertebral subluxation/dysfunction syndrome
 definition of, 37–47
Vertebrobasilar ischemia (VBI)
 with adjustive therapy, 100–101, 101b
Vertebrobasilar insufficiency, 93t
Veterans Administration (VA)
 utilizing chiropractic, 7
Videofluoroscopy (VF), 78–79, 79b
Vis medicatrix naturae, 3–9, 3t
Viscera
 segmental innervation related to, 418t

Visceral manipulation
 classification of, 84, 85f
Visceral reflexes
 theory of altered, 45–46
Viscerosomatic reflexes, 45–46, 45b
Viscoelastic
 defining connective tissue, 26–33
Visual analog severity scales
 for pain, 53, 54f
Vitalism, 3

W

Wallenberg syndrome, 96–98
White muscle, 27
Wrists and hands
 adjustive procedures, 311, 334f, 335f,
 336f
 biomechanics of, 327–329, 328f, 328t,
 329f, 329t
 close-packed positions for, 23t
 cross-section of, 330f
 evaluation of, 329–333, 330f, 331f, 332f,
 332t, 333f
 functional anatomy, 326, 326f, 327f
 ligaments, 326, 327f, 328f
 muscles, 326, 328f, 328t
 osseous structures, 326, 326f, 331f
 three physiologic arches of, 329f

Y

Yellow ligaments, 17

Z

Zones
 of articular cartilage, 16, 16f
 deformation, 235f
 gliding, 16, 16f
 of parapsysiologic movement, 68,
 68f