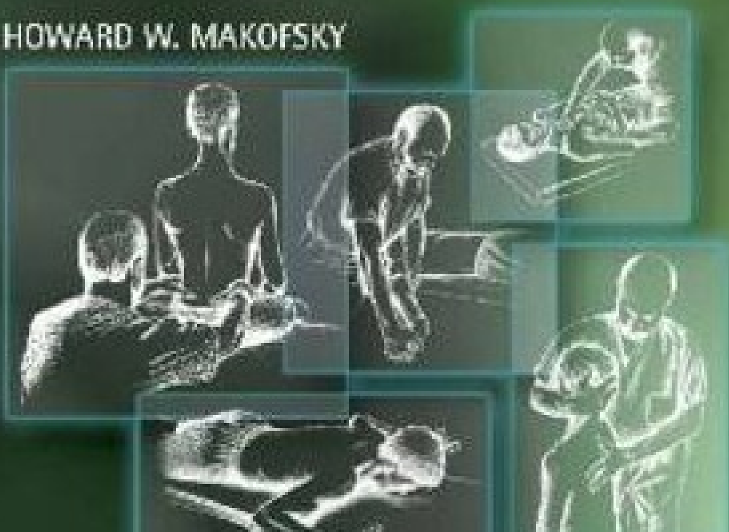
SECOND EDITION

Spinal Manual Therapy

An Introduction to Soft Tissue Mobilization, Spinal Manipulation, Therapeutic and Home Exercises

HOWARD W. MAKOFSKY



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Spinal Manual Therapy

An Introduction to Soft Tissue Mobilization, Spinal Manipulation, Therapeutic and Home Exercises

Spinal Manual Therapy: An Introduction to Soft Tissue Mobilization, Spinal Manipulation, Therapeutic and Home Exercises, Second Edition is an easy-to-follow manual of clinical techniques for the spine, pelvis, and temporomandibular joint. The text provides "tools" rather than "recipes" and immerses the reader in the process of "thinking as a manual therapist," rather than functioning as a technician. The clinical utility of this revised *second edition* combines the art and science of present day spinal manual therapy.

The focus of *Spinal Manual Therapy, Second Edition* is to provide clinically useful treatment techniques, while being mindful of the scientific literature related to the practice of spinal manual therapy. It is an ideal resource for all those interested in grasping the basics of spinal manual therapy and transferring that knowledge into practice within a clinical environment.

The hands-on approach taken by Dr. Howard W. Makofsky makes this new edition the go-to textbook for spinal manual therapy.

New to the *Second Edition:*
- New pictures of examination and treatment techniques with captions
- Additional case studies
- New evidence supporting spinal manual therapy
- Updated references throughout the text

This unique textbook has a plethora of clinical techniques, including the rationale for each of their use. With more than 300 figures, illustrations, and photographs for each examination/treatment technique for various regions of the body, students and clinicians learning manual therapy will benefit greatly from *Spinal Manual Therapy, Second Edition.*

Inside you'll find:
- Evaluation
- Soft tissue techniques
- Specific exercises
- Manipulative procedures
- Clinical problem solving

Spinal Manual Therapy, Second Edition mirrors a course on the introduction to spinal manual therapy and will be welcomed into physical therapy curricula as well as appreciated by clinicians when entering clinical practice.

Reviews from previous edition:

"...helps the reader to examine the patient using good manual skills to detect and treat somatic dysfunctions. I found it very interesting...the pictures are wonderfully helpful in demonstrating techniques and demonstrating exercises."

- Diane J. Cordeiro, PT, OCS, CFMT, *Advance for Physical Therapists and PT Assistants*

"Spinal Manual Therapy *is both comprehensive and user friendly. The plethora of figures will greatly enhance the learning experience. However, the quality of* Spinal Manual Therapy *surpasses solely that of the student level. In fact, new graduates, novices, and clinicians with limited exposure to orthopaedic manual therapy will greatly benefit from this book."*

- Jessica Palmer, SPT and Joshua Cleland, DPT, OCS, *The Journal of Manual and Manipulative Therapy*

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Erratum

In chapter 12, page 100, second column the text reads:

The authors conclude that, "Repetitive migraine attacks may lead to, or be the result of neoplastic changes in cortical and subcortical structures of the trigeminal somatosensory system."

It should read:

The authors conclude thar, "Repetitive migraine attacks may lead to, or be the result of neuroplastic changes in cortical and subcortical structures of the trigeminal somatosensory system."

Makofsky HW
Spinal Manual Therapy, 2nd ed.

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Dedication

To my wife Frances (Nak), whose faith and love of the scriptures inspire and strengthen me.

To four amazing kids, Katherine, Margaret, Russell, and Daniel,
two terrific sons-in-law, Daniel and Wesley,
and our precious granddaughter, Nora Grace.

To my mother, Mildred and late father, Abe Makofsky and to Margaret and Dr. Christopher Willis for wisdom and love.

To Bob, Nola, Sharon, Neil, Ally, and Cassidy for enduring support and encouragement.

To the children and fellow laborers at the Paradise Orphanage in Burkina Faso, West Africa.

To Christ my King!

In memory of Jeffrey J. Ellis, PT, manual therapist par excellence, who went home to be with His Lord on May 18, 2001.

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Section VI Pelvic Girdle

Section VII From the Classroom to the Clinic

Acknowledgments

First of all, I want to express my gratitude to my wife Frances and our dear friend Francine for putting up with me during the writing of this second edition of my book. Thanks for that cup of tea at just the right moment! Thanks also to my lovely daughters, Dr. Katherine Moore for help with the cases on pancoast tumor and multiple myeloma and Dr. Margaret Hanson for writing the Foreword.

Secondly, I'd like to thank John Bond, Brien Cummings, Debra Steckel, and all the folks at SLACK Incorporated for their patience and commitment to excellence. It's been a pleasure working with every member of the team.

For an outstanding job with photography, I'd like to acknowledge Noelle D'Arrigo. Your contribution Noelle was huge! Regarding the photo shoot, I'd also like to thank Neil and Sharon Solina for the use of their studio as well as Frances, Russell, and Daniel Makofsky for being such terrific models. I also wish to thank the medical library staff at NYCOM for assistance with difficult-to-find articles. It's always a pleasure working with you!

Because this textbook is based on 30 years of experience as a manual physical therapist, there are numerous friends, colleagues, and mentors that have contributed to the information contained in this text in some way, shape, manner, or form. Specifically, I'm referring to the physical therapists I've worked with at St. Charles Hospital, Braintree Hospital, and Southside Hospital and the wonderful faculties at Stony Brook, Touro College, and the New York Institute of Technology. Special acknowledgment goes to those colleagues who, in our manual therapy courses, have either shared in the teaching or assisted in lab. They are Lisa Morrone, Kevin Cerrone, Donald O'Brien, Peter Gambardella, Roger Lov, Mark Gugliotti, James Macaluso, Bernard Finnerty, Peter Douris, Cheryl Hall, Ilyse Flattau, Nicole Rinaldi, Joel Norman, Robert Shapiro, Renee Lemieux, Nechama Karman, Karen Friel, Kelley Sheehan, Ed Bezkor, Lisa Coors, Lisa Johnson, Robert Streb, and Susan Spagnoli (special thanks to Kevin Cerrone for all his work on the examination forms that appear in the Appendix).

Regarding those from whom I've learned much about the art and science of manual therapy, there are too many to name. Having said that, there are 7 primary individuals whose workshops, articles, textbooks, and DVDs are woven into the fabric of my practice and this text. They are the pillars upon which many a career and this book are built. They are Stanley Paris, James Cyriax, Freddy Kaltenborn, Philip Greenman, Mark Bookhout, Mariano Rocabado, and Robin McKenzie. Others who, for this author, have added significantly to this foundation through their teaching, research, publications, etc, include Ronald Melzack, Vladimir Janda, Carl Steele, Geoffrey Maitland, Paul Kimberly, John Mennell, Karel Lewit, Brian Mulligan, Fred Mitchell, Jr., Ola Grimsby, Gregg Johnson, Ed Stiles, Jeffrey Ellis, Catherine Patla, Alan Grodin, Rick Bowling, David Butler, Earl Pettman, Cliff Fowler, Jim Meadows, Olaf Evjenth, Robert Donatelli, Michael Wooden, Steve Kraus, Diane Plante, Nanci Machnik, Ad Warmerdam, Michael Helland, Alan Hayhoe, Leonard Goldstein, Gary Willner, Jan Prsala, Roz Sofer, Sondra Cooper, Marge Dorfman, Jeffrey Mannheimmer, Mark Dutton, Josh Cleland, Timothy Flynn, Julie Whitman, Gwendolen Jull, Carolyn Richardson, Julie Fritz, John Childs, Paul Hodges, Julie Hides, Anthony Delitto, Diane Lee, Chad Cook, Shirley Sahrmann, Rick Jemmett, Loren "Bear" Rex, Sandy Burkhart, Brian Miller, and Peter Fabian. Thanks for your brilliance and your passion!

Last, but certainly not least, I want to express my appreciation to all the physical therapy students at NYIT, Touro College, and Stony Brook I have had the privilege to instruct over the years and, God willing, will continue to instruct. It's because of you that teaching never feels like work and why, if I could, I would do it all over again. Cheers!

About the Author

Howard W. Makofsky, PT, DHSc is an Associate Professor in the Department of Physical Therapy at the New York Institute of Technology, Old Westbury, NY; an Adjunct Professor at the Touro College School of Health Sciences, Bay Shore and New York, NY; and a Clinical Assistant Professor of Physical Therapy at Stony Brook University, Stony Brook, NY. Dr. Makofsky holds a bachelor of science degree in human physiology from McGill University, a bachelor of science degree in physical therapy and a master of science degree in health sciences from Stony Brook University, and a doctor of health science degree from the University of St. Augustine for Health Sciences, St. Augustine, FL.

Dr. Makofsky has been practicing, researching, and teaching manual physical therapy for 30 years. He has more than 20 published articles in the scientific literature, and holds patents on the Occivator® and PostureJac®. Dr. Makofsky was on the editorial board of the *Journal of Manual & Manipulative Therapy* for several years and presently sits on the editorial board of the *Journal of Craniomandibular Practice*.

Dr. Makofsky and his wife, Frances, founded the not-for-profit 501(c)(3) public charity, Kaya's Kids, Inc, which provides financial support for the Paradise Orphanage in Burkina Faso, West Africa. Dr. and Mrs. Makofsky reside in Mastic Beach, NY during the school year, and in Advocate Harbour, Nova Scotia, Canada in the summer months. They have four grown children and one grandchild.

Preface

I feel fortunate to truly enjoy my work. It's been said that "timing is everything" and my involvement in the world of manual therapy over the past 30 years couldn't have occurred at a better time. From my graduation from physical therapy school in 1979 to the present, I've seen manipulative therapy go from being scorned to being a hot commodity. When, in 2004, the APTA Manipulation Task Force advocated the teaching of thrust in entry-level doctor of physical therapy programs, I was in disbelief. In 1979 we couldn't extend the lumbar spine let alone perform a grade V thrust! It was done back then, but mainly in private practice and rarely if ever in a hospital-based department (to this end, there are 3 high-velocity, low-amplitude thrust procedures taught in the second edition, 2 in the thoracic spine and one in the pelvis). For those who say that doctorally-trained physical therapists should not be thrusting joints, to them I say, "Who died and made you boss?"

Not only has the art of manual therapy become part of mainstream physical therapy, but the science is not far behind. To this end, I touch upon the development of clinical prediction rules, treatment-based classification, the regional-interdependence examination model, and provide 22 scientific studies in Chapter 26 to strengthen the case for the examination procedures and interventions covered in this text. Regarding evidence-based practice, I welcome and support it. However, it is still in its infancy and we must be careful, in the meantime, not to discard treatment techniques that may help a "fellow creature in pain" as we await the verdict of well-executed science.

For those of us who love the practice of manual therapy, the past 30 years have been an extraordinary time indeed. Unfortunately, some have drawn attention to themselves rather than to the art. No, we must always remember that we are but "links" in a wonderful "chain" and if, in our journey, we have insights or "connect some dots" along the way, we must remind ourselves that this healing art is not about us, but about the art and all who have collectively contributed to it, including our Creator who is the ultimate artist!

Writing a second edition of a textbook is like making a movie. If you don't like a particular scene, you get to go back and do it over. This is rarely the case in life and for this opportunity to make improvements to the original text, I am most grateful. In this second edition, the pictures all have captions and being electronic, they are sharper with easier viewing of fine detail. In addition, 7 studies have been added as well as 2 cases in Section VII, From the Classroom to the Clinic. Furthermore, an entire chapter has been devoted (Chapter 25) to the PostureJac, a device used around the globe for the relief of painful symptoms stemming from poor postural alignment. As with the original version in 2003, the second edition of *Spinal Manual Therapy* is intended to be a comprehensive lab manual for students learning spinal manual therapy, as well as clinicians in the field who are eager to embrace this exciting area of practice. That being said, it is not a "how-to" manual in the least. I have attempted to convey principles and concepts throughout the book such that the techniques become secondary. Techniques are based on style and preference. The advanced learner grasps these principles of evaluation and treatment and ultimately uses them to develop those manual techniques that work for him or her. The techniques in this book work for me and they are provided to get you started. They are not an end in themselves!

The absence of specific manipulative techniques to the upper cervical spine and sacrum is by design. The manual examination and treatment of these articulations require advanced theoretical knowledge, which is beyond the scope of this text. In addition, I have not included the vertebral artery test. This is explained further in Chapter 8, but basically we should not be using it as a rule-out test in light of its poor sensitivity. Furthermore, it introduces potential risk to the posterior cerebral circulation and the benefit in no way justifies the risk. Regarding special tests, I have opted to list them and leave that subject detail to the abundance of good books that are currently available on the topic. Please note that the thrust (pardon the pun) of this text is spinal manual therapy. I allude to the regional anatomy in each section, but this text is no substitute for a good anatomy book. In fact, my students can tell you how often I say that "anatomy is everything." If you know the structure of the human body, most of what we do as manual therapists can be figured out. As with the first edition of *Spinal Manual Therapy,* the terms *manipulation* and *mobilization* are used interchangeably, consistent with the 2001 edition of the *Guide to Physical Therapist Practice*. Unfortunately, many manual therapists around the globe continue to make a distinction, whereby mobilization refers to a nonthrust technique and manipulation to a high-velocity, low-amplitude procedure. In reality, all skilled passive therapeutic movements (eg, Maitland grades 1 to 5) are forms of manipulation, be they myofascial or arthrodial, and they are all performed for the similar purpose of improving mobility (ie, mobilization). Thanks to Dr. Stanley Paris, this useful simplification continues to work its way through the manual therapy community, but it will take time.

One final comment is needed. Manual therapy is not the intellectual property of any one profession. It belongs to all clinicians who have a license to touch. All that's required is that borrowed material be properly referenced and acknowledged. The many allusions to physical therapy in this text are based on my experience as a physical therapist and love for the physical therapy profession. Having said that, we welcome MDs, DOs, DCs, PTAs, and body workers to the learning of this material. I hope that you find it useful!

Howard W. Makofsky, PT, DHSc

Foreword

Manual therapy has been gaining increased importance among both students and practitioners in the field of physical therapy. Living in an era where computers and advancing technology have replaced so many jobs in our society, the power of human touch has yet to be duplicated by a machine. The expertise of a strong manual therapist cannot be replaced, and is invaluable to the patient with pain and/or dysfunction. However, the process of acquiring these skills can feel quite overwhelming for a student or new graduate. As a recent physical therapy student, the concept of "healing through my hands" was something that fascinated me. At the same time, I found the complexity of the spine and pelvis quite intimidating.

When I was introduced to *Spinal Manual Therapy* as a student, it didn't take long to realize that this text was different from most of my other books. As either current or former students, we can all relate to the countless textbooks we've been assigned to read through our years of schooling. How many of those books remained untouched, collecting dust on our shelves? Although many of those texts had valuable information, few had real practical value.

In this text, however, one can find relevant clinical pearls nestled in each page. It is written in a manner that is easy to understand with explanations that are simple and concise. The author speaks to the reader as if to a student sitting in on a lecture. Although the concepts and interventions are valuable for experienced clinicians, they are just as accessible to the student therapist. Rather than mere scientific theory, abstract ideas, and complex concepts, the pages of this text are full of material that can be put directly into clinical practice. Along with the author's 30 years of clinical experience, his emphasis on the scientific literature and current research provide the practitioner with the impetus to broaden and refine his or her own knowledge base.

I can recall countless occasions during my student clinical rotations when I would refer back to this textbook in between treating patients. I would place a patient on moist heat or electrical stimulation and then immediately turn to the text in search of guidance on how to approach my patient's neck, back, or facial pain. I would always come away with something useful, or at least a place to get started. I have had numerous classmates and fellow therapists tell me of similar experiences. I realized quickly that this text wasn't a recipe-type book, but rather a manual for critical thought, providing a framework for wise clinical decision making, and a way to view the patient's body as a whole system working together. The emphasis the book places on posture and analyzing the body through careful observation has proven to be incredibly useful as a new therapist.

The textbook addresses the cervical, thoracic, lumbar spine, and pelvis as well as the temporomandibular joint. In each of these sections, the reader is provided with well-illustrated examination and treatment techniques including appropriate therapeutic and home exercises for the patient. The text also provides numerous case studies to facilitate the clinical decision-making process and to help the student/therapist begin to see how the classroom material translates directly into clinical practice.

Spinal Manual Therapy: An Introduction to Soft Tissue Mobilization, Spinal Manipulation, Therapeutic and Home Exercises, Second Edition takes an eclectic approach to examining, diagnosing, and treating the body. The author integrates the ideas and concepts of some of the major contributors to the field of manual therapy including Paris, McKenzie, Maitland, Cyriax, Greenman, Kaltenborn, Rocabado, and many others. The text encourages the therapist to equip his or her "tool box" with a variety of tools. Rather than merely treating diagnoses, the therapist is encouraged to identify each patient's impairments in order to determine which treatment or combination of treatments is most effective for that patient. Dr. Makofsky teaches the student how to "think like a manual therapist" and how to be a "clinician rather than a technician." I recommend this book to all who seek to practice with the same philosophy.

Margaret Hanson, PT, DPT
Daughter and Former Student of the Author
St. Luke's Hospital
Kansas City, Missouri

Section I

Essentials of Spinal Manual Therapy

1

Vertebral Motion Dynamics

The Vertebral Motion Segment

The basic unit of spinal motion is the vertebral mo
tebrae—the superior and the inferior—and all related anatomic structures, including the intervertebral disc, 2 apophyseal joints, and various soft tissues. An example of a vertebral motion segment is the third cervical vertebra (C3) situated above the fourth cervical vertebra (C4). The nomenclature used to describe this union is the C3,4 motion segment. Other examples are T8,9 (thoracic spine) and L3,4 (lumbar spine). A junction or transitional segment is an area where one region of the spine is joined to a different region. Examples are the craniocervical, cervicothoracic, thoracolumbar, and lumbosacral junctions. The craniocervical junction is also known as the occipitoatlantal segment or O-A; the cervicothoracic junction is synonymous with C7,T1; the thoracolumbar junction with T12,L1; and the lumbosacral junction with L5,S1.

Physiologic Motion

Each of the 24 vertebrae (7 cervical, 12 thoracic, and 5 lumbar) have the ability to move in 3 planes of reference. The sagittal plane motions include forward bending or flexion and backward bending or extension, the frontal plane motions include side bending or lateral flexion to the right and left, and the horizontal plane motions include axial rotation to the right and left.

Motion Axes

Each of these 6 spinal motions can be considered rotations around or about an orthogonal axis (Figure 1-1). Forward and backward bending are rotations about the X or horizontal axis, side bending is a rotation about the Z or anteroposterior axis, and axial rotation occurs about the Y or vertical axis. The thumb, index, and middle fingers of one hand can be used to assist in recalling these 3 axes of spinal motion. The thumb pointing to the ceiling represents the Y or vertical axis, the middle finger flexed to 90 degrees at the metacarpophalangeal joint represents the X axis, and the index finger at a right angle to the middle finger, directed anteriorly, represents the Z axis (Figure 1-2).

Rule of Superior Motion

When manual therapists describe segmental motion, it is understood that the superior vertebra is mentioned first. For example, side bending right at the T5,6 motion segment suggests that the fifth thoracic vertebra (T5) is side bending right on T6. Most often this will be documented as T5,6 side bending right. However, some clinicians may describe this in short form as T5 side bending right. When only one vertebral level is noted, it denotes that segment's motion not under the level above but rather over the level below. Consequently, T5 side bending right refers to its motion relative to T6; L4 rotation left is motion relative to L5. This is the case whether spinal motion is initiated from above down or from below up. For example, trunk rotation that is initiated by rotating the lower extremities and pelvis

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Spinal Manual Therapy, 2nd ed. (pp 3-8)
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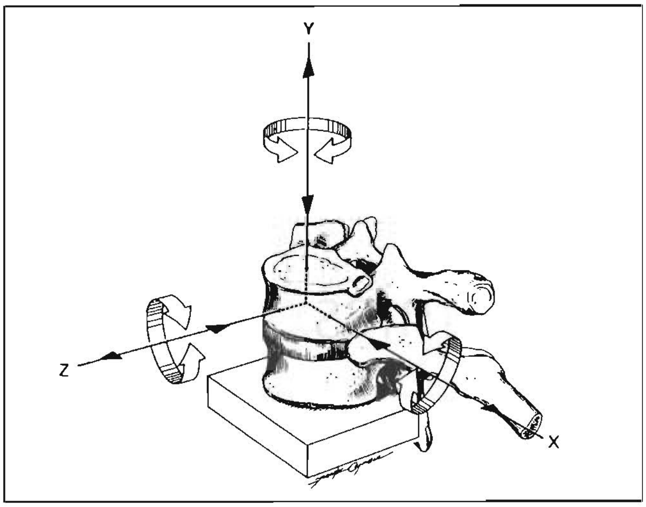

Figure 1-1. The three vertebral motion axes. (Reprinted with permission from Lee D. Biomechanics of the thorax: a clinical model of in vitro function. *J Man Manip Ther.* 1993;1(1):14.)

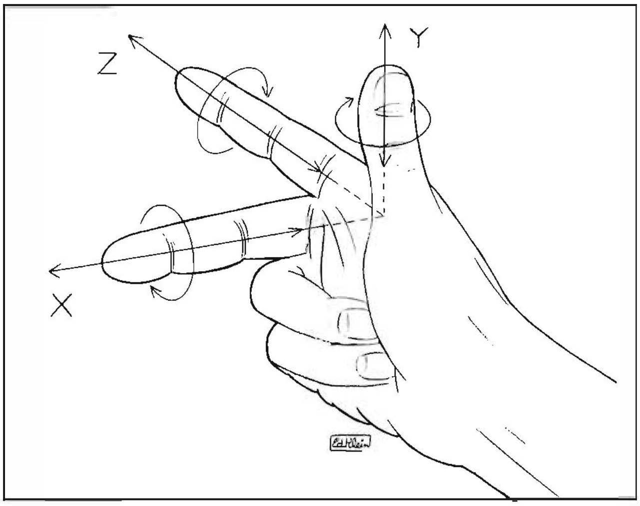

Figure 1-2. Manual illustration of the 3 cardinal axes. (Illustration by Ed Klein.)

to the right and proceeding up to and including T8 is still described as T7,8 rotation left by virtue of the fact that T7 is left rotated relative to T8.

Rule of Vertebral Body Motion

A vertebra's motion is always described by the direction of vertebral body motion and not spinous process (SP) movement. Consequently, a passive movement of the T11 SP to the left, which induces vertebral rotation to the right, is described as T11,12 rotation right because of the direction of vertebral body motion.

Fryette's Rules of Spinal Motion Coupling

Although the validity of Fryette's Rules is being questioned,[1] they continue to be taught within the osteopathic profession[2] and will be covered here.

Rule 1

When one or more motion segments are positioned in neutral (ie, loose packed) with the apophyseal (facet) joints idling in "easy normal," side bending and rotation are coupled to opposite sides (Figure 1-3). For example, in a neutral lordosis, side bending to the left from L1 through L5 is associated with Y-axis rotation to the right. Rule 1 is referred to as neutral or type 1 spinal mechanics (coupling). Neutral mechanics occur in all vertebral segments except from C2 through C7, where there is no true neutral position of the apophyseal joints. In the upper cervical spine (occiput-atlas-axis), type 1 spinal mechanics occur for different reasons (ie, based upon unique osseous and ligamentous characteristics). Although capable of type 1 spinal mechanics, the upper thoracic segments (T1-T4) tend to follow the lower cervical spine (type 2 spinal mechanics) in function; when rotation precedes side bending, type 2 or non-neutral coupling dominates throughout all levels of the thoracic spine.[2]

Rule 2

When a spinal motion segment is positioned in either flexion or extension such that the apophyseal joints are in apposition (ie, engaged), side bending to one side is coupled with Y-axis rotation to the same side (Figure 1-4). For example, side bending to the right at T7,8 from a position of trunk flexion is associated with T7 rotation right. Rule 2 is referred to as non-neutral or type 2 spinal mechanics (coupling). Non-neutral mechanics occur in all vertebral segments except in the upper cervical spine (occipitoatlantal and atlantoaxial joints) where type 1 mechanics prevail. However, Greenman[2] describes an exception to this rule in the lumbar spine, whereby type 1 mechanics prevail in the presence of L1-L5 extension.

Rule 3

When motion is introduced in one plane, the available motion in the remaining planes is reduced. For example, rotation of the head-neck is greater in an upright posture than it is in a slumped posture. Likewise, trunk side bending is greater in a neutral position of the spine than in a flexed or extended position of the spine.

The converse of this also applies (ie, if motion is increased in one plane, it will also be increased in the other planes as well). For example, if lumbar spine side bending is increased through manipulative therapy, then the other motions of flexion, extension, and rotation will increase as well.

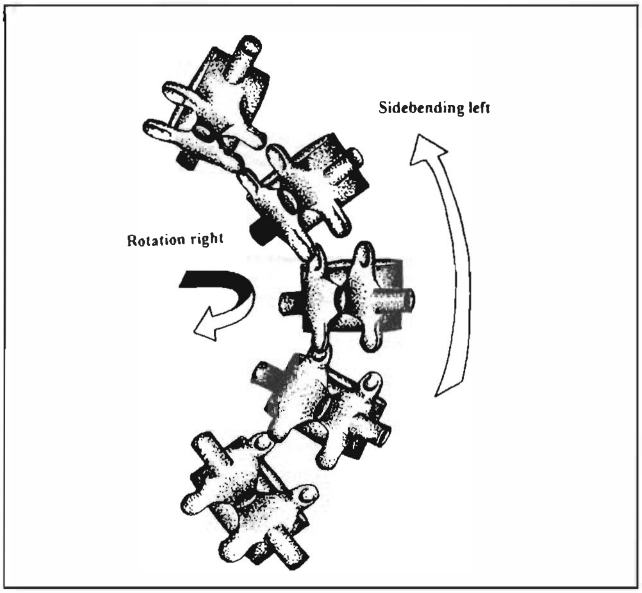

Figure 1-3. Type 1 spinal mechanics. (Reprinted from Gibbons P, Tehan P. *Manipulation of the Spine, Thorax, and Pelvis: An Osteopathic Perspective*. Edinburgh: Churchill Livingstone; 2000, by permission of the publisher Churchill Livingstone.)

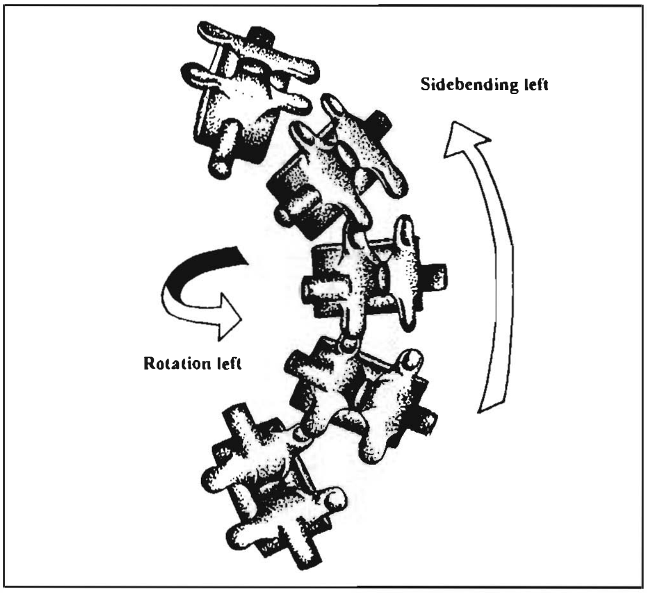

Figure 1-4. Type 2 spinal mechanics. (Reprinted from Gibbons P, Tehan P. *Manipulation of the Spine, Thorax, and Pelvis: An Osteopathic Perspective*. Edinburgh: Churchill Livingstone; 2000, by permission of the publisher Churchill Livingstone.)

Type 1 and 2 Impairment

Restricted spinal motion involving 3 or more segments in a neutral position of the trunk is referred to as type 1 or neutral impairment (ie, dysfunction).

For example, in a neutral trunk position a restriction in left side bending from T9 through T12 is associated with a restriction at the same levels in right rotation. This is also referred to as a type 1 rotoscoliosis, and its position can often be identified on an anteroposterior spinal radiograph.

Restricted spinal motion of one segment in a non-neutral position is referred to as type 2 or non-neutral impairment. For example, T3,4 is said to be FRS (flexed, rotated, and side bent) right when it is limited in the opposite directions (ie, extension, rotation, and side bending to the left). Conversely, L4,5 is said to be ERS (extended, rotated, and side bent) left when it is limited in flexion, rotation, and side bending to the right. These one-segment motion impairments may not be easily seen on a spinal radiograph but can be readily diagnosed through osteopathic segmental motion analysis.[2,3]

Apophyseal Joint Kinematics

Facet Opening

The term *facet opening* refers to the anterior and superior glide of the inferior articular process of the superior vertebra on the superior articular process of the vertebra below. For example, the facets are said to open bilaterally in spinal flexion; open on the left during flexion, side bending, and rotation to the right (Figure 1-5); or open on the right during flexion, side bending, and rotation to the left.

Facet Closing

The term *facet closing* refers to the posterior and inferior glide of the inferior articular process of the superior vertebra on the superior articular process of the vertebra below. For example, the facets are said to close bilaterally in spinal extension; close on the left during extension, side bending, and rotation to the left (Figure 1-6); or close on the right during extension, side bending, and rotation to the right.

Facet Gapping

The term *facet gapping* refers to the separation or distraction (traction) of the joint surfaces in a perpendicular direction. If a thoracic or lumbar facet gaps on the left, this implies that the inferior articular process of the superior vertebra separates away from the superior articular process of the inferior vertebra. Gapping of the facets generally occurs in the thoracic and lumbar spine in response to neutral rotation on the ipsilateral side (Figure 1-7). On the contralateral side of the rotation, the facets approximate each other as they are compressed together. No gapping occurs in either the upper (occiput-atlas-axis) or lower (C2-C7) cervical spine because of the absence of a neutral articular position.

Roll-Gliding

According to Kaltenborn,[4] the vertebral motion segment, not unlike the extremity joints, moves in a roll-glid-

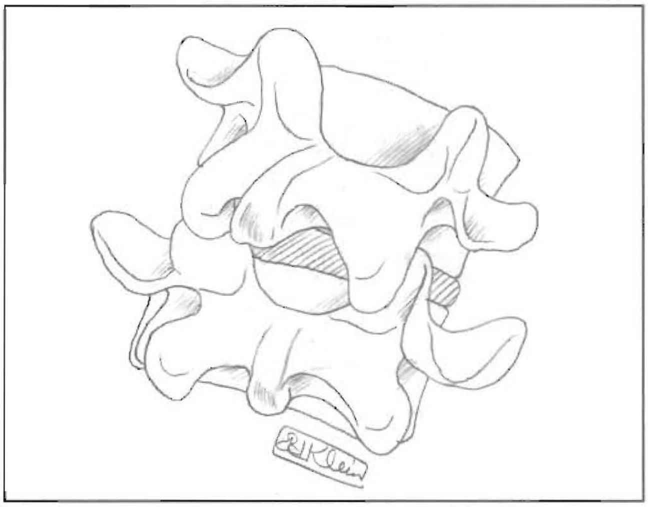

Figure 1-5. Facet opening of L4,5 on the left. (Illustration by Ed Klein.)

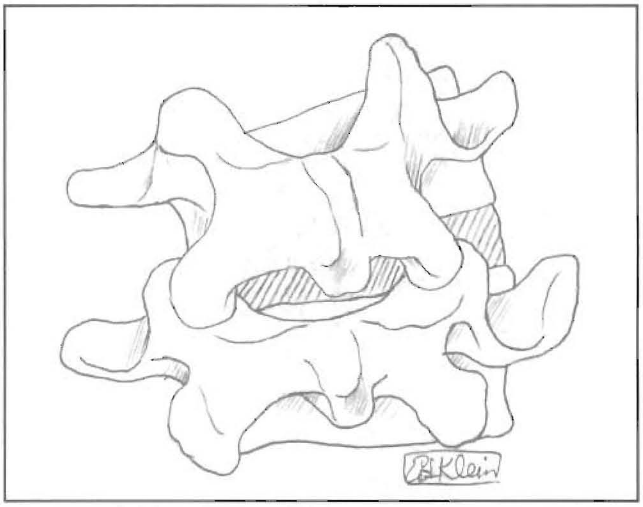

Figure 1-6. Facet closing of L4,5 on the left. (Illustration by Ed Klein.)

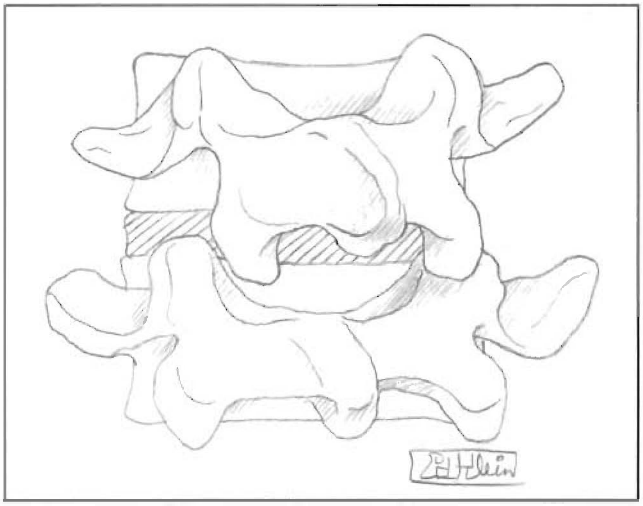

Figure 1-7. Facet gapping of L4,5 on the left. (Illustration by Ed Klein.)

ing fashion. Except for the occipital condyles, which are convex surfaces moving on the concave surfaces of the atlas, the remainder of the motion segments of the spine behave or function as a concave surface (superior vertebra) moving on a convex one (inferior vertebra). This suggests that the roll of the superior component (concave rule) will glide in the same direction on the inferior component below, whereas the inferior component (convex rule) will glide in the opposite direction of its roll. We have previously described the X, Y, and Z motion axes, but only with regard to rotation. However, to appreciate how a rigid body moves in space (ie, the helical axis of motion), we need to consider not only rotation about a given axis, but also the translation that occurs along a different axis (see Figure 1-1). For example, forward bending of the T7,8 motion segment involves anterior rotation (roll) of T7 about an X axis as well as anterior translation (glide) of T7 along the Z axis. Backward bending of T7,8 involves X-axis posterior rotation and Z-axis posterior translation of T7. For side bending of T7,8 about a Z axis, there is vertebral translation of T7 in the same direction along the X axis. The roll-gliding that occurs with Y-axis rotation is dependent upon the vertebral segment involved. At the atlantoaxial segment, axial rotation about the Y axis is associated with a craniocaudal translation along the same Y axis such that there is a slight loss of height as the extreme of rotation is reached. The vertical height is then restored when the head is rotated to neutral. Consequently, each vertebral motion segment has a total of 6 degrees of freedom—3 for rotation and 3 for translation.

In summary, it can be said that motion of the superior component of the motion segment demonstrates rotation and translation in the same direction, whereas the inferior component of the segment rotates and translates in opposite directions. If we accept the premise that the superior and inferior components of the motion segment have relative motions that are out of phase with each other, then it can also be said that the superior component of the segment will roll in one direction, while the inferior component will glide in the opposite direction. For example, backward bending of T5,6 involves a backward roll of T5 about the X axis with an anterior glide of T6 along the Z axis. This is not unlike an extremity joint in which a concave surface moves upon a convex one as at the trapezium-scaphoid joint in the midcarpal region of the wrist. Just as wrist extension involves a posterior roll of the trapezium with concurrent anterior gliding of the scaphoid, likewise T6 "dives" underneath the extending T5 as one would do at the beach in the presence of a formidable wave. Consequently, a mobilization/manipulation of T6 in a posteroanterior (PA) direction will improve backward-bending range at the T5,6 segment. Since translation is a mechanically simpler movement to perform manually, therapists routinely manipulate the inferior component of a segment

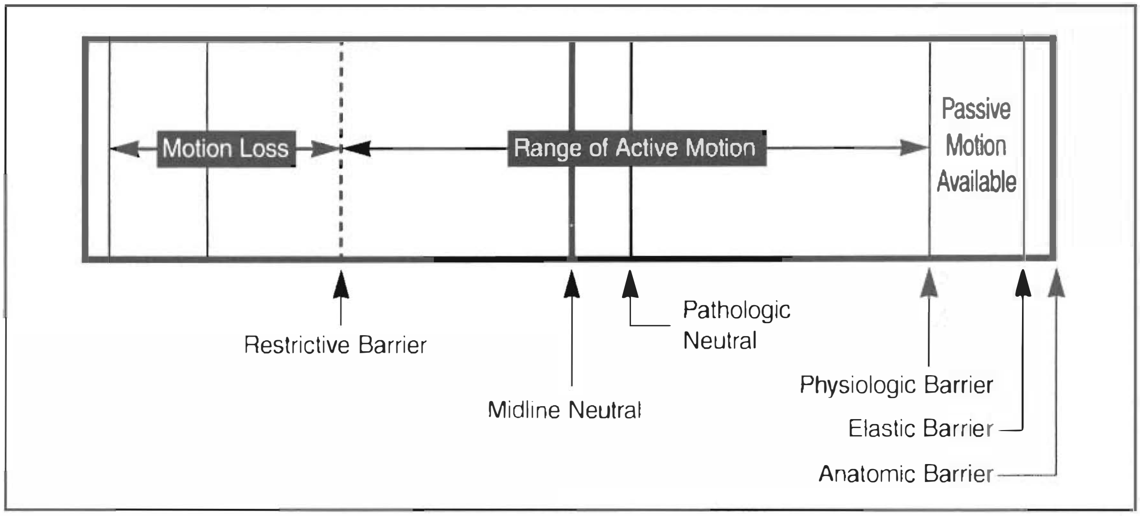

Figure 1-8. Normal and abnormal motion barriers. (Reprinted with permission from Flynn TW. *The Thoracic Spine and Rib Cage: Musculoskeletal Evaluation and Treatment.* Boston, MA: Butterworth-Heinemann; 1996.)

to achieve improvement in range. It is also common to perform a combination of roll-gliding in the spine with a simultaneous roll of the superior component while gliding the inferior component in the opposite direction. A second example involves the motion of side bending. Side bending left at L4,5 involves a Z-axis roll to the left of L4 and a glide of L5 along the X axis to the right. Consequently, a translational manipulation of L5 to the right under L4 or a manipulative combination of an L4 roll in left side bending with a concurrent glide of L5 to the right can be utilized to enhance the motion of left side bending at L4,5. Another commonly used term for rotation or roll is *overturning*; another term for translation or glide is *slide*.

Motion Barriers

There are 4 barriers[2] (3 normal and 1 abnormal) to joint motion (Figure 1-8).

Physiologic Barrier

The end of an active, voluntary effort in a normal joint is the physiologic barrier for that motion. Every movement in the body has an associated physiologic barrier.

Elastic Barrier

The elastic barrier is the point at which the soft tissue slack is taken up during a passive movement in a normal joint (ie, "the beginning of the end").

Anatomic Barrier

The anatomic barrier is the absolute end-point in the passive range of motion in a normal joint beyond which tissue injury occurs (ie, "the end").

Restrictive Barrier

The premature motion loss in an impaired joint is known as the restrictive barrier. It may represent a restriction at any point in the overall range of motion of a joint. It is associated with an abnormal end-feel (ie, hard or nonyielding versus resilient and supple). Restrictive barriers have multiple causes (ie, muscle splinting, capsular fibrosis, internal derangement, myofascial tightness) and are responsible for causing either a major motion loss when 50% or more of the range is restricted, or a minor motion loss involving less than 50% of the range of motion in a specific direction.

It is important to understand that the restrictive barrier presents as a range of restriction rather than as a definitive end-point. This restricted range spans from the initial sense of tension, which osteopathic physicians refer to as the "feather-edge," to the end-range of the restriction in which all the "slack" has been taken up. The feather-edge is the point used for localization purposes in osteopathic muscle energy technique, whereas the end-range of a restriction is challenged during certain joint manipulative procedures, including a small-amplitude, high-velocity thrust.

The restrictive barrier is an impairment that results from tissue pathology and can lead to functional limitation and disability if not given the appropriate intervention. The goal of manual therapy is to diagnose and correct these impairments so that the associated functional limitation and disability are minimized or ideally eliminated.

2

McKenzie's Three Syndromes

Postural Syndrome

According to McKenzie,[5-7] patients with postural syndrome are usually less than 30 years old and, by definition, are devoid of restrictive barriers. These patients develop symptoms that appear locally and usually adjacent to the spine. The pain is provoked by mechanical deformation of normal, healthy tissue when spinal segments are subjected to static loading over prolonged periods of time. The resulting pain disappears when the structure under load is released from tension.

The pain from postural syndrome is not induced by movement and is never referred to a distant site. Because there is no associated inflammation, it is never constant. Examination of these patients fails to reveal impairment because there is no underlying tissue pathology. The only consistent finding is pain provocation with static loading at end-range. Simply put, postural pain develops gradually when normal tissues are overstretched.

The most useful intervention is to correct the faulty alignment wherever it is found (ie, sitting, standing, lying, walking). This may also involve an ergonomic assessment of furniture, computer height, mattresses, pillows, etc, as well as an analysis of the patient's conditions at the worksite.

The long-term complication of postural syndrome is that it can eventually cause pathologic changes in the soft tissues with resultant impairment. However, this will not likely occur with proper instruction in correct posture, ergonomic intervention, and proper body mechanics.

Dysfunction Syndrome

An uncorrected postural problem will cause pathologic changes over time. For example, a 35-year-old computer operator who spends 8 hours per day in a forward head position will eventually develop adaptive shortening of the occipital extensor muscles. Likewise, the 40-year-old truck driver who spends 10 hours per day in a slumped sitting posture will eventually discover an inability to assume a normal lumbar lordosis in standing because of adaptive shortening of the trunk flexors.

As per the Nagi Functional Limitations Model,[8] these adaptive changes in connective tissue (ie, loss of hyaluronic acid/water, adhesions) represent pathophysiologic events that cause such macroscopic tissue impairment as restricted joint mobility, muscle weakness, and the faulty alignment that is often associated with imbalance in the musculoskeletal system. If the patient does not correct his or her impairment with the proper interventions, he or she can go on to develop functional limitations and disability, which can adversely affect performance at work, home, etc.

A distinguishing feature of the patient with dysfunction syndrome includes painful symptoms that tend to arise at the end of range rather than during movement. This patient has intermittent pain similar to the postural patient, but differs in that his or her soft tissues are abnormally tight. The symptoms are usually adjacent to the spine and are never referred distally except in the case of an adherent nerve root. Simply stated, the pain of dysfunction syndrome is produced immediately when shortened tissues are overstretched.

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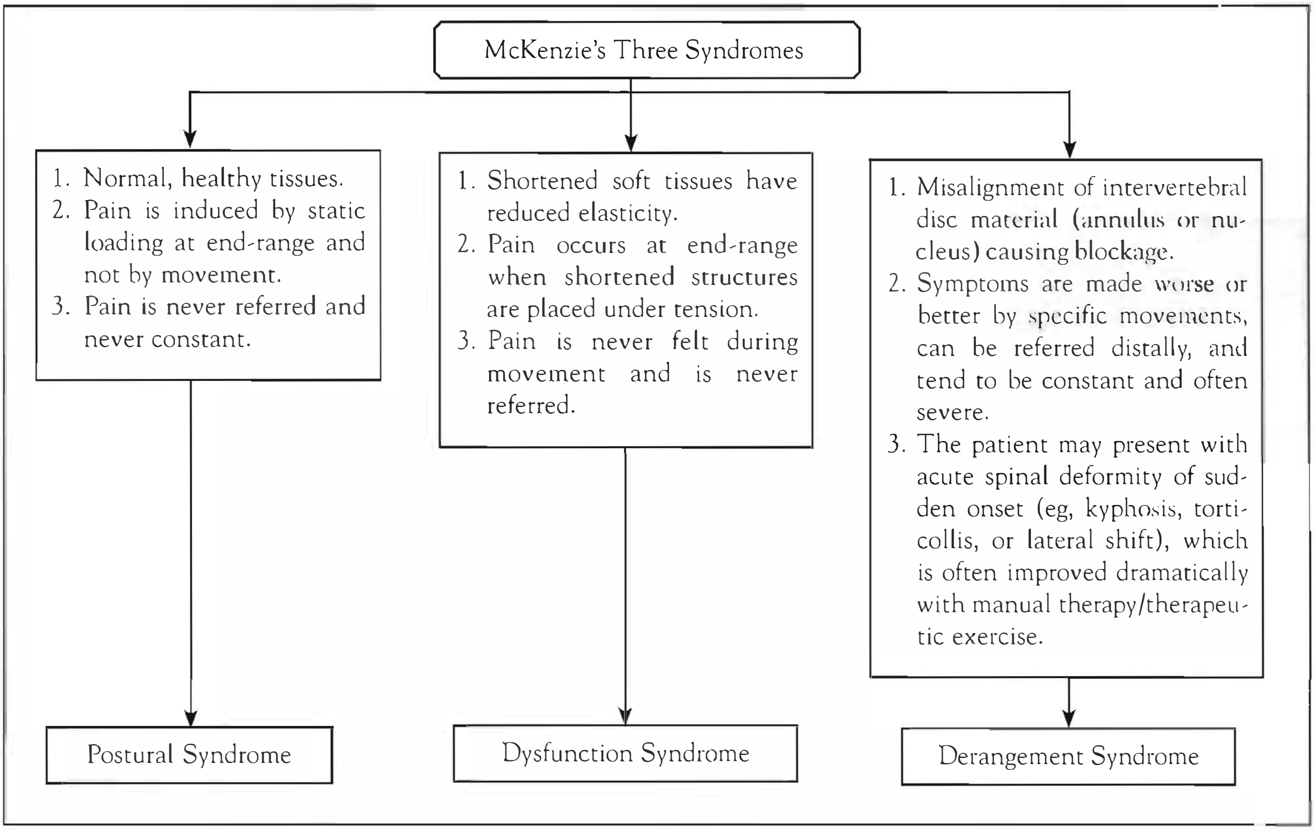

Figure 2-1. McKenzie's mechanical diagnosis of spinal pain and related symptoms.

As with postural syndrome, dysfunction syndrome also has a long-term complication. If untreated with the appropriate intervention (ie, manipulative therapy), it can cause more destructive pathology and result in the last of McKenzie's three syndromes, namely derangement syndrome. However, in some cases a traumatic event in the absence of preexisting dysfunction is enough to cause derangement of the intervertebral disc.

Derangement Syndrome

Characteristics of this syndrome can include neurologic signs and symptoms, pain during movement, acute deformity (eg, torticollis, lumbar kyphosis, lateral shift phenomenon), and pain that is severe and disabling. Patients with derangement syndrome often have a history of poor posture and progressive stiffness. It is believed that the lack of motion-based nutrition in conjunction with off-center loading on the intervertebral disc causes the displacement of disc material. The young are more likely to have a nuclear displacement, while those over the age of 50 tend to develop annular lesions. With the onset of degenerative disc disease, patients may develop clinical instability,[9,10] which requires stabilization training[11,12] of the hypermobile segment(s) in conjunction with manual therapy of the stiff, hypomobile segments above and/or below.

Patients with derangement syndrome (primarily occurring in the cervical and lumbar spine) often describe their neck and/or back as being "out." It is imperative that these patients be correctly diagnosed lest they be deprived of the correct intervention. The deranged disc requires an approach that is quite different from dysfunction syndrome and will not respond unless managed appropriately. The goals of intervention are as follows:

1. The derangement must be properly reduced.
2. The reduction must be stabilized in order for healing to occur.
3. Once the derangement is stable, lost function must be recovered.
4. The prevention of recurrence of the derangement must be emphasized.

The classification of spinal impairment into one of McKenzie's three syndromes (Figure 2-1) is just the beginning of establishing the correct intervention. There are further subclassifications of both the dysfunction and derangement syndromes. These are made during the evaluation process and are necessary in establishing the correct diagnosis. Though the theory behind McKenzie's approach[5-7] can and should be presented in every textbook on spinal manual therapy, it is not until the therapist attends a McKenzie workshop that a true understanding of this unique problem-based approach to spinal patients takes place.

3

Principles of Manual Examination, Diagnosis, and Intervention

Somatic Impairment

When asked about the manipulable lesion, osteopathic physicians for years described the osteopathic lesion.[13] This term has since has been replaced with the term *somatic dysfunction*. With the advent of the *Guide to Physical Therapist Practice*[8] and in keeping with the changes in terminology since then, the author of this text prefers the word *impairment* in this regard. The term *somatic impairment* will be used to describe an impairment of function in the neuromusculoskeletal system that is biomechanical in nature. It is a term to be contrasted with *disease*,[14,15] which is of a nonmechanical and pathological nature requiring the expertise of a physician for diagnosis and management. Whereas disease is within the realm of medicine and surgery, somatic impairment is within the realm of physical therapy. It develops as a result of tissue pathology, but the pathology is of a mechanical nature and can be traced to nonsystemic causes, including macrotrauma, cumulative microtrauma, immobilization, etc. Once disease[14,15] is ruled out (based upon a thorough history and physical exam), the next step for the manual therapist is to determine which of McKenzie's three syndromes[5-7] most accurately describes the patient (ie, postural, dysfunction, or derangement). This classification is important as it helps to establish direction for management of the spinal patient (see previous chapter for further details). In terms of somatic impairment, only the dysfunction and derangement syndromes apply, as postural syndrome implies the presence of normal tissues placed in abnormal positions for prolonged periods of time (ie, postural syndrome is not associated with somatic impairment).

Schafer and Faye[16] subclassify dysfunctions as either class I, II, or III and refer to them as fixations. Class I fixations are muscular in nature, class II fixations are related to the shortening of ligaments, and class III fixations represent true articular hypomobility. This classification system is based upon motion palpation and helps to determine the type of manual therapy that is utilized.

Stiles[17] emphasizes the area of greatest restriction (AGR) in his attempt to prioritize manipulative management. It is based upon the premise that areas of major hypomobility in the body are the "engines" that drive the entire system into an inefficient state in which impairment develops and symptoms result. Whereas a symptom-oriented approach to therapy addresses secondary and compensatory areas of impairment, a manually-oriented approach seeks to locate the AGR, even though it is usually asymptomatic and often found some distance away from the patient's complaint. It is the author's opinion that the AGR is most often found in the thoracic cage and hips. Most neck, shoulder, or low back pain that presents clinically would be better managed by identifying and treating the AGR rather than applying "fake, shake, and bake" therapy to the symptomatic areas of hypermobile compensation.

Joint mobility is evaluated with regard to the quality of motion, quantity of motion, end-feel,[4,18] and tissue reactivity.[4,19] Normal joints demonstrate smooth, friction-free, and interference-free movement and have a healthy degree of "play" at the end-range. In contrast, dysfunctional joints demonstrate hypo/hypermobility, friction, joint sounds, etc. They may feel blocked, restricted, or abnormally loose at

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the end-range. Panjabi[9,10] uses the term *clinical instability* to describe hypermobile joints that have an enlarged "neutral zone." This author prefers the term *destabilized,* because of the potential confusion with the use of instability in orthopedic surgery. Most approaches to manual therapy utilize the 0 to 6 scale for grading joint mobility where 0 = ankylosed, 1 = markedly hypomobile, 2 = slightly hypomobile, 3 = normal, 4 = slightly hypermobile, 5 = markedly hypermobile, and 6 = unstable.[4] Positionally, normally functioning joints are usually found in Panjabi's neutral zone, whereas dysfunctional joints often occupy end-range "border positions," which are associated with Panjabi's elastic zone.[9,10] Herein lies the important connection between joint malalignment (eg, forward head, hyperkyphosis, swayback) and impairment. Regarding the connection between somatic impairment and painful symptoms, the following paradigm has proven useful:

1. Normal tissues + Normal forces = Low likelihood of painful symptoms.
2. Normal tissues + Abnormal forces = Moderate likelihood of painful symptoms.
3. Abnormal tissues + Normal forces = Moderate likelihood of painful symptoms.
4. Abnormal tissues + Abnormal forces = High likelihood of painful symptoms.

With regard to the treatment approach espoused in this text, the skilled manual therapist seeks to stretch what is tight, mobilize/manipulate what is stiff, stabilize what is loose, and strengthen what is weak!

The CHARTS Method of Manual Examination

The **ART** method of physical examination has been the mainstay of the osteopathic diagnosis of somatic dysfunction for several years.[2] This diagnostic triad identifies 3 key components of a somatic dysfunction. They are as follows: **A** stands for asymmetry of related parts of the musculoskeletal system; **R** stands for range of motion of a joint, several joints, or region of the musculoskeletal system; and **T** stands for tissue texture abnormality of the soft tissues of the musculoskeletal system (eg, skin, fascia, muscle, tendon, ligament, joint capsule). It is believed that true somatic dysfunction demonstrates all 3 components of the triad. For example, hypertonicity of the right levator scapula muscle will be associated with the following findings: **A** – elevation of the right scapula, **R** – restricted cervical spine side bending left, and **T** – increased tone with shortening of the right levator scapula.

In the 1980s, the late Jeffrey Ellis[20] elaborated on the **ART** diagnostic triad by adding **C** for chief complaint, **H** for histories (eg, family, psychosocial, past medical, a description of the presenting problem, pharmacologic), and S for special tests (eg, neurologic, orthopedic, vascular, gait, functional capacity, radiologic, lab results). This resulted in the acronym **CHARTS**, which has gained widespread acceptance within the field of orthopedic physical therapy as an extremely useful tool in the examination/evaluation of patients presenting with somatic impairment.

An efficient way of collecting information about the patient's chief complaint is to use the o, p, q, r, s, t method. This consists of several questions, including the following:

- Onset – Did the problem have a sudden or an insidious onset?
- Pain – What makes it better or worse?
- Quality – What is the nature of the symptoms? (The adjectives used to describe the pain are quite helpful in diagnosing the problem.[21,22] Words such as *intense, radiating, severe, burning, shooting, shock-like, lancinating, piercing,* and *well localized* suggest pain of peripheral neurogenic—eg, radicular pain; words such as *deep, aching, diffuse, dull, boring, continuous, vague,* and *poorly localized* suggest pain of deep somatic or nociceptive origin; the words *throbbing* and *pulsing* suggest pain of vascular origin.)
- Radiating – How far down the extremity do the symptoms travel? The symptoms of McKenzie's dysfunction syndrome[5-7] do not generally travel past the elbow or knee, whereas the referred symptoms of a spinal derangement[5-7] can and often do. In this context, radicular pain, or pain arising from neurologic structures, must arise from the dorsal roots or the dorsal root ganglia.[21] The subject of neuropathic pain as related to complex regional pain syndrome types I (reflex sympathetic dystrophy) and II (causalgia), neuralgiform pain (eg, trigeminal neuralgia), are beyond the scope of this text.
- Severity – How intense (mild to severe) is the chief complaint? A visual analogue scale or a 0 to 10 pain intensity numerical rating scale (PI-NRS) is useful in determining pain intensity.
- Timing – Is the chief complaint constant, intermittent, or occasional?

Though it is not the role of the therapist to manage symptoms of visceral and pathologic origin, it is certainly the therapist's responsibility to recognize them so that the appropriate medical/surgical referral can be made. To this end, the student of manual therapy is encouraged to develop basic competency in the process of differential diagnosis in physical therapy.[14,15] Regarding acute versus chronic pain, the consensus of opinion is that pain persisting longer than 3 to 6 months is chronic.[23] However, the Quebec Task Force on Spinal Disorders[24] classifies patients into 1 of 3 stages based on duration of symptoms from onset: 1) Acute – less than 7 days, 2) Subacute – 7 days to 7 weeks, 3) Chronic – more than 7 weeks.

Direct Versus Indirect Technique

Osteopathic manipulative therapy (OMT) can be divided into 2 approaches.[2,13] Therapy that engages the motion barrier directly is referred to as direct technique. Examples include nonthrust joint manipulation, muscle energy technique, high-velocity/low-amplitude thrust, and direct fascial technique. Manipulative therapy, which moves away from the motion barrier in the direction of "ease" in the tissues, comprises those techniques that are known as indirect. Examples include strain/counterstrain, functional, facilitated positional release, integrated neuromuscular inhibition, and induration technique. This author developed a manual treatment method on the spine, which includes a combined indirect/direct approach known as position-assisted combination technique (PACT).[25]

The effective manipulator is skilled in both approaches and knows when "to go direct and when to go indirect." In general, direct techniques are applied to tissues that demonstrate contracture (ie, thick, fibrotic, and shortened tissues), whereas indirect techniques are more suited for states of contraction (ie, hypertonic, inflamed, and hyperalgesic). The author subscribes to the phrase "a time to hold and a time to scold" as related to child rearing. As a child at times needs comfort, but at others requires discipline, so too the soft tissues need an indirect approach that will settle them down when inflamed. However, when persistently tight in the absence of inflammation, they must be challenged with a direct technique that will release, elongate, and mechanically correct the underlying impairment. Because of the gentle nature of indirect techniques, they can be safely and effectively utilized in acute conditions in which direct techniques would be contraindicated. Therapists who have difficulty with "right brain" activities that require less analysis and more creative thought may have difficulty with the feeling-oriented indirect methods. However, the skills necessary to master these techniques can be learned by even the most "left brained" among us!

Sequencing Therapeutic Interventions

As stated previously, McKenzie's classification system[5-7] is extremely useful in directing therapy for the spinal patient. As covered in Chapter 2, the treatment sequence for derangement syndrome is as follows:

1. The derangement must be properly reduced.
2. The reduction must be stabilized in order for healing to occur.
3. Once the derangement is stable, lost function must be recovered.
4. The prevention of recurrence of the derangement must be emphasized.

To assist us with the sequencing of interventions in the management of the dysfunction patient, we will use a case study approach. Our patient is a 32-year-old female attorney. The patient is married to an accountant and has 2 children ages 3 years and 9 months. The patient was involved in a rear-end motor vehicle accident 6 weeks before presenting in the physical therapist's office for treatment. Ms. Jones reports chronic daily headaches as well as neck pain and stiffness. She is taking naproxen for pain and is wearing a cervical collar. The examination reveals moderate forward head posture; symmetrical limitation in neck rotation and side bending, moderate in nature; muscle hypertonus of the levator scapulae and suboccipital muscles, bilaterally; and moderate limitation of jaw opening with tenderness and tightness of the temporalis muscles, bilaterally. Neurologic examination for sensation, deep tendon reflexes, and muscle strength is normal.

The evaluation of Ms. Jones places her in practice pattern 4B in the *Guide to Physical Therapist Practice*,[8] which consists of soft tissue injuries of the cervical spine and temporomandibular joint (TMJ) involving pain, poor posture, and myalgia. The ICD-9 CM codes used for billing purposes are 723.1, 781.92, and 524.6. As per the *Guide*, the expected number of visits for this episode of care is 6 to 20.

The topic of sequencing intervention becomes relevant when considering how one proceeds with the management of Ms. Jones' symptoms and many somatic impairments. The recommended sequence of dealing with what appears to be a symptomatic dysfunction will now be covered in detail.

1. Reduce the patient's tissue reactivity. By reactivity, we are referring to the irritability[19,26] of the symptomatic area, which also correlates to the stage of tissue healing.[27,28] High levels of reactivity are present when pain precedes stiffness in the impaired range of motion, consistent with the inflammatory stage of healing. Low levels of reactivity are present when stiffness precedes pain, consistent with the late proliferative and remodeling stages; moderate levels are present when pain and stiffness simultaneously limit motion and is associated with the late inflammatory and early proliferative stages of healing. When high levels of tissue reactivity are present, indirect techniques are preferred to direct; used in conjunction with cryotherapy and electrotherapeutic modalities for the purpose of reducing pain, inflammation, and reflex-induced muscle splinting. However, if direct methods of manual treatment are selected, Maitland[19] grades 1 and 2 are recommended (see Chapter 6).
2. Restore impaired myofascial extensibility. Once the tissues can be moved without provoking pain and muscle splinting, it is time to commence connective tissue techniques, including myofascial release and direct fascial technique.[29] The soft tissues function as "guy ropes," and therefore the bony skeleton, being a series of struts, cannot assume optimal alignment and func-

tional mobility without normal extensibility within the myofascial system. A similar concept involves the muscle chain theory.[30] It is postulated that shortening of specific muscle chains results in postural deviations (eg, forward head/rounded shoulders posture). The 5 chains described include: 1) respiratory muscle chain consisting of the pectoralis minor, scalene, intercostal, diaphragm, and sternocleidomastoid (SCM) muscles; 2) posterior muscle chain consisting of the muscle groups from the soles of the feet through the leg, thigh, and spine; 3) antero-internal hip chain comprised of the iliopsoas, pectineus, gracilis, and the short and long adductors of the hip; 4) Antero-internal shoulder chain comprised of the pectoralis major, coracobrachialis, and subscapularis muscles; and 5) anterior arm chain formed by the upper trapezius and the flexors of the shoulders, upper arms/forearms, hands, and fingers. It is the author's belief that musculoskeletal motion loss within the spine and extremities is more often a problem of impaired myofascial extensibility than true articular dysfunction or derangement. If, however, joint motion is restricted, there is almost always an associated loss of myofascial extensibility. As a general rule, the connective tissue component of somatic impairment should always be treated first. The reason for this is that the unnecessary repeated manipulation of a joint destabilizes it and predisposes that joint to hypermobility. In addition, joint mobilization/manipulation in the presence of unresolved myofascial dysfunction is often met with failure. This has been the experience of those who thrust joints without first attending to the shortened myofascial elements. Rolf's concept[31] of postural "equipoise" depends on restoring normal extensibility to these soft tissue "guy ropes" just as the mast of a ship cannot be properly aligned unless its attaching stays and shrouds are functioning at their optimum length.

At this point, it is necessary to discuss the work of the late Vladimir Janda[32] from the Czech Republic. Based upon years of clinical experience, Janda believed that there are 2 groups of muscles in the body: 1) those in response to stress[33] (ie, overuse, misuse, disuse, and abuse) that become facilitated, hypertonic, and tight and 2) those in response to stress that become inhibited, hypotonic, and weak. Janda called the former, postural muscles and the latter, phasics. It is the postural muscles, such as the upper trapezius, levator scapulae, and SCM, that require soft tissue mobilization and stretching; this work must be done prior to articular manipulation as previously discussed. The phasic muscles such as the deep neck flexors (eg, rectus capitis anterior, rectus capitis lateralis, longus capitis, and longus colli) will require strengthening and will be dealt with later in the correct sequence. Having mentioned the Janda classification of muscles,[32,33] there is a growing trend toward reclassifying muscles[33,34] as either 1) stabilizers (local, global) and mobilizers, 2) global and local, 3) superficial and deep, 4) monoarticular and multiarticular, and 5) weightbearing and nonweightbearing. Needless to say, this is creating some level of confusion in the clinic. However, the important thing for the clinician is not how to classify muscles, but how to manage them in states of impairment (ie, stretch muscles that are tight, strengthen muscles that are weak, and improve muscle endurance when impaired).

3. Achieve normal joint mobility. Once the reactivity is reduced to a low level and the myofascial soft tissues have regained lost extensibility, it is now necessary to use manual therapy to normalize joint motion. The combined term *joint mobilization/manipulation* in this book is defined as, "A manual therapy technique comprising a continuum of skilled passive movements to a joint that is applied at varying speeds and amplitudes, including but not limited to a small amplitude/high velocity therapeutic movement" consistent with *Guide* terminology.[8] Consequently, the terms *manipulation* and *mobilization* will be used interchangeably in this text and may certainly apply to the myofascial tissues as well as capsuloligamentous structures. Though the term *manual therapy* is synonymous with manipulation, it embodies not only the art of manipulative therapy, but also the scientific foundation upon which the art is based.

   Now that we have defined our terms, it is important to inject some philosophy and ask what is the purpose and ultimate goal for the use of manipulation. Is it to reposition a bone that has become subluxed?[35] Is it to cure disease? No, it is much simpler than that. In the author's view, the purpose of manipulation—be it of a synovial joint of the spine or extremities or of the myofascial soft tissues—is simply to restore the normal joint play or accessory movements of a joint so that the physiologic/osteokinematic motion of the joint system can be returned to normal. When asked the same question, a panel of experts on the topic[36] stated the following: "The goal of manipulation is to restore maximal pain-free movement of the musculoskeletal system within postural balance." We have discussed the movement perspective in great detail; now we will proceed in our discussion of the importance of postural balance.

4. Attain orthostatic posture. McConnell[37] defines ideal posture as "optimal alignment with symmetrical loading of body parts." This is helpful, but we need something more specific. Johnson and Saliba[38] use the term *efficient state* and define it as, "A state where each body segment distributes weight, absorbs shock, has full available range of motion and independent control of movement to meet the functional needs of both stability and mobility." Buckminster Fuller,

an early 20th-century architect, discussed balance from a structural perspective.[39] He coined the term *tensegrity*, which is derived from the words *tension* and *integrity*. Whether in a building, suspension bridge, sailboat, tent, or the human body, tensegrity refers to the structural integrity arising from the synergy between balanced tension and compression. Perhaps the tensegrity model captures the true essence of "postural balance" and is really what manipulative therapy aims to achieve. In theory, the terms *postural balance*, *optimal alignment*, *efficient state*, and *tensegrity* make sense. However, on a clinical level, it is important to define their parameters. To assist us further in this regard, we refer to the work of F.M. Alexander.[40,41]

The Alexander technique is a means whereby each person can be taught the optimal use of his or her body. It involves a mind-body interaction in which we consciously inhibit inefficient movements to allow the body to generate movement and alignment that is taller, lighter, stronger, and more comfortable. Because optimal alignment and postural balance are among some of the benefits of Alexander's work, it behooves us to review his 4 concepts of good use:

a) Allow your neck/shoulders to release so that your head can balance forward and up.
b) Allow your torso to release into length and width.
c) Allow your legs to release away from your pelvis.
d) Allow your shoulders to release out to the sides.

A useful means for applying Alexander's principles of good use is the **AID** method, where **A** is for awareness (tuning into unnecessary muscle tension and poor postural habits), I is for inhibit (consciously cease doing the wrong thing), and D is for direction (thought processes that lead to balancing the head forward and up, lengthening and widening the torso, releasing the legs away from the pelvis, and releasing the shoulders to the sides). The aim of the AID method is to restore what Alexander called primary control[40,41] (ie, the intrinsic mechanism for balance, mobility, and support in the body, which is based on an optimal relationship between the head and spine in movement and in stillness). Feldenkrais[33] used the term *the potent state* and Rolf[31] *equipoise* to describe the ideal relationship between gravity and body posture. Based upon this author's understanding of ideal posture, the following concept has emerged: ideal human alignment consists of body posture that is balanced, efficient, and vertical, thus satisfying the biomechanical requirements of both static and dynamic function.

When the transition is made from imbalance to balance, from inefficient to efficient, and from a horizontally orientated alignment (long moment arms) to a vertical one (short moment arms), we will then see the improvement in symptoms that we seek for our patients. To operate in a clinical environment in which patients are treated more like "cattle" than the unique and wonderful creation that they truly are should be unacceptable. We can and should do better.

Achieving postural balance is not possible without the requisite work in reducing reactivity, restoring myofascial extensibility, and achieving normal joint mobility. The final step in the intervention sequence is to strengthen the weak phasic muscles.

5. Sensorimotor training and muscle strengthening procedures. At a well-attended manual therapy conference in 1985 in Boston, Dr. Sandy Burkhart made the statement, "In addition to being 'carpenters,' manual therapists need to be 'electricians' as well." That insightful comment represented a turning point for many in manual therapy practice, where the emphasis had always been on restoring the mechanics of the joint. With the Institute of Physical Art's integration of neurologic and orthopedic practice into a series of seminars on "functional orthopedics," as well as an explosive interest in "body work" (ie, Feldenkrais, Alexander, Rolf, Trager, Thai massage, Hanna, Pilates), manual therapists were suddenly interested in not only "fixing the hardware" but in "reprogramming the software." This new emphasis in manual therapy was good news for patients, as they were now able to maintain the improvement in joint mechanics by "retraining" the muscles of the body to move these more mobile structures in more efficient ways.

As discussed previously, the specific muscles that require sensorimotor re-education, strength/endurance work, etc are Janda's phasics that, under stress, become inhibited, hypotonic, and weak. In other muscle classification systems,[12,33,34] these are the stabilizers, local, deep, or weightbearing muscles. Examples of such muscles include the deep neck flexors, lower trapezius, multifidus, transversus abdominis, and the gluteus maximus and medius. One reason why muscle strengthening should be performed after manual therapy is because of the reflexogenic effects (ie, arthrokinetic reflex) of the articular mechanoreceptors on muscle tone.[42-46] For example, hip joint extension needs to be restored prior to strengthening the hip extensors in order to lessen hip-induced inhibition on the gluteus maximus.[45] Similarly T6-T12 extension needs to be restored prior to strengthening the lower trapezius muscles for similar reasons.[44] Consequently, it makes sense to mobilize/manipulate before strengthening in order to lessen and/or eliminate this "neural inhibition" on the muscle.

With the understanding that orthopedic patients could benefit from neurologic techniques came the realization that neurologic patients could likewise benefit from orthopedic/manual therapy techniques. This represented significant progress in the physical therapy profession, and it is the patient who benefits most from this integration.

Body Holism, Adaptive Potential, and Regional Interdependence

Sir William Osler,[47] the famous McGill physician, is quoted as saying, "It is more important to know the patient who has a disease than the disease that has the patient." This approach to patient care recognizes that the body functions as a whole, well-integrated unit. The basic components of the individual represent the "triad of health" and include physical structure, biochemical processes, and the mental/spiritual state.[48] When all 3 components are integrated and functioning normally, the individual is healthy and functional. However, when there is an imbalance in one or more areas, this represents "dis-ease," impairment, and/or disability.

When considering a "holistic" rather than a "localistic" approach to the patient, it is necessary to consider every factor that represents a source of potential "dis-ease" or imbalance to the patient. These include macrotrauma, cumulative microtrauma, psychological distress, nutritional deficiencies, infection, environmental and ergonomic influences, etc. It is always in the best interest of the patient to address as many of these "stressors" as possible. Commenting on the role of chronic overuse (ie, cumulative microtrauma), Sahrmann[49,50] states, "Musculoskeletal problems are seldom caused by isolated precipitating events, but are a consequence of habitual imbalances in the movement system."

The term that represents an individual's ability to cope with these negative influences is *adaptive potential*. In health, a person's adaptive potential is high; in states of impairment and/or disability, adaptive potential is low. The advantage of approaching the patient from a perspective of body holism is that intervention, whether it is physical, psychological, nutritional, etc, has the desired effect of restoring adaptive potential.
the patient's tolerance, and the result is improved overall health and wellness.

Stiles[47] discusses the clinical equation: Host + Disease = Illness.
tial. When it is compromised, the patient suffers; when it is improved, the patient benefits. As manual therapists, we may not have control over various disease states, but we can "fortify the host.
ic impairment and by referring him or her, when necessary, to other health care providers who can assist with the other health-limiting factors with which they deal.

The other aspect of a holistic approach to the treatment of musculoskeletal pain and dysfunction consists of addressing the entire region of impairment, which is now referred to as regional interdependence.[51] This model suggests that the patient's primary complaint may be related to impairments in regions distal or proximal to the region of the primary complaint.[52] For example, shoulder pain and dysfunction may be related to scapulothoracic impairment,[53] TMJ pain may be correlated to forward head posture (FHP),[54] and patellofemoral pain may be biomechanically traced to impairment of the hip.[55]
distal to the chief complaint may actually be "co-conspirators" rather than completely separate entities.[52] Though not an entirely new concept, the regional-interdependence examination model[51,5]
that needs to be appreciated by all practitioners of manual therapy. Otherwise, the biomechanical roots of the patient's pain may not be properly addressed.

Clinical Prediction Rules and Treatment-Based Classification

Clinical prediction rules (CPRs) are decision-making tools that contain predictor variables obtained from patient history, examination, and simple diagnostic tests. According to Beneciuk et al, CPRs derived from high-quality studies may have the best potential for use in clinical settings.[57] They assist clinicians with the diagnosis, prognosis, and appropriate management of a given disorder.[58,59] CPRs are developed using multivariate statistical methods and are designed to examine the predictive ability of selected groupings of clinical variables.
have been several published clinical prediction rules affecting the practice of spinal manual physical therapy. Childs et al[61] identified patients with low back pain most likely to benefit from high-velocity/low-amplitude thrust spinal manipulation; Hicks et al[62] collaborated on the preliminary development of a clinical prediction rule identifying low back patients most likely to respond to a stabilization exercise program; Cleland et al[63] identified a subgroup of patients with neck pain who would benefit from a combined approach of thoracic spine thrust manipulation, exercise, and patient education; and Cleland et al[64] developed predictors of short-term outcome in patients with cervical radiculopathy. Although an encouraging development towards evidence-based practice, CPRs are often based on a very distinct group that may or may not be reflective of a typical population of patients. In addition, many of the rehabilitation-based CPRs may have methodological weaknesses that can potentially undermine the utility of this instrument.[60] That being said, CPRs bring the practice of manual physical therapy a step closer to truly being an art based on science.

Before concluding this chapter with a brief discussion of contraindications, it is important to draw our attention to an important paradigm shift occurring within the field of orthopedic manual physical therapy.
which I speak is the transition away from the traditional

mechanism-based classification system to a treatment-based classification system. Whereas the mechanism-based classification system operates on the premise that the identified impairments are the cause of the associated musculoskeletal symptoms and therefore the correction of these somatic impairments should theoretically result in the improvement of such symptoms, the treatment-based system, utilizes a cluster of signs and symptoms to classify patients into subgroups with specific implications for management.[58] The advantages of the treatment-based classification system are that it is easily understood, clinically applicable, and straightforward. It is also eclectic in that it integrates various interventions including, but not limited to, mobilization/manipulation, stabilization exercises, and the McKenzie method of evaluation and treatment.[65-6] The controversy surrounding this paradigm shift involves whether or not to "sacrifice the sacred cow of the mechanism-based classification system" in light of the fact that its purported specificity (eg, vertebral segmental dysfunction) has been ingrained in the training of manual therapists for at least the past 3 millennia.[58]

Contraindications to Spinal Manual Therapy

The following conditions are contraindications to the use of direct joint manipulative techniques (ie, low- or high-velocity manipulation/mobilization, muscle energy, etc):

- Acute arthritis of any type
- Rheumatoid arthritis
- Acute ankylosing spondylitis
- Hypermobility/instability, including patients with generalized hypermobility as in Ehlers-Danlos syndrome
- Calvé-Perthes disease
- Fracture
- Ligamentous rupture
- Malignancy (primary or secondary)
- Osteomalacia
- Paget's disease
- Severe osteoporosis
- Osteomyelitis
- Tuberculosis
- Disc prolapse with serious neurologic impairment (including cauda equina syndrome)
- Evidence of involvement of more than 2 adjacent nerve roots in the lumbar spine
- Lower limb neurologic symptoms due to cervical or thoracic spine involvement
- Painful movement in all directions
- Infectious disease
- Depleted general health
- Patient intolerance
- Inability of the patient to relax
- Rubbery end-feel of the joint
- Undiagnosed pain
- Protective joint muscle spasm
- Segments adjacent to the level being manipulated that are too irritable or hypermobile to tolerate the stress of proper positioning prior to or during the manipulation.

In the event that any of these conditions are undiagnosed but present, the astute clinician is still protected providing he or she recognizes the level of reactivity in the pathologic tissues and acts accordingly. Regarding the use of indirect techniques, with the exception of frank disease, they may be effectively utilized in cases of high tissue reactivity because of their gentle nature. However, for the inexperienced novice, the above list should serve as contraindications to all manual techniques.

There are 2 axioms in the practice of medicine that are extremely useful in uncertain times of clinical practice. They are do no harm and when in doubt, don't!

To further assist in distinguishing pain of different origins, the reader is referred to Figure 3-1. It is helpful to remember that although technology has its place in physical therapy, as in medicine, there is no substitute for a good history and physical examination of the patient.

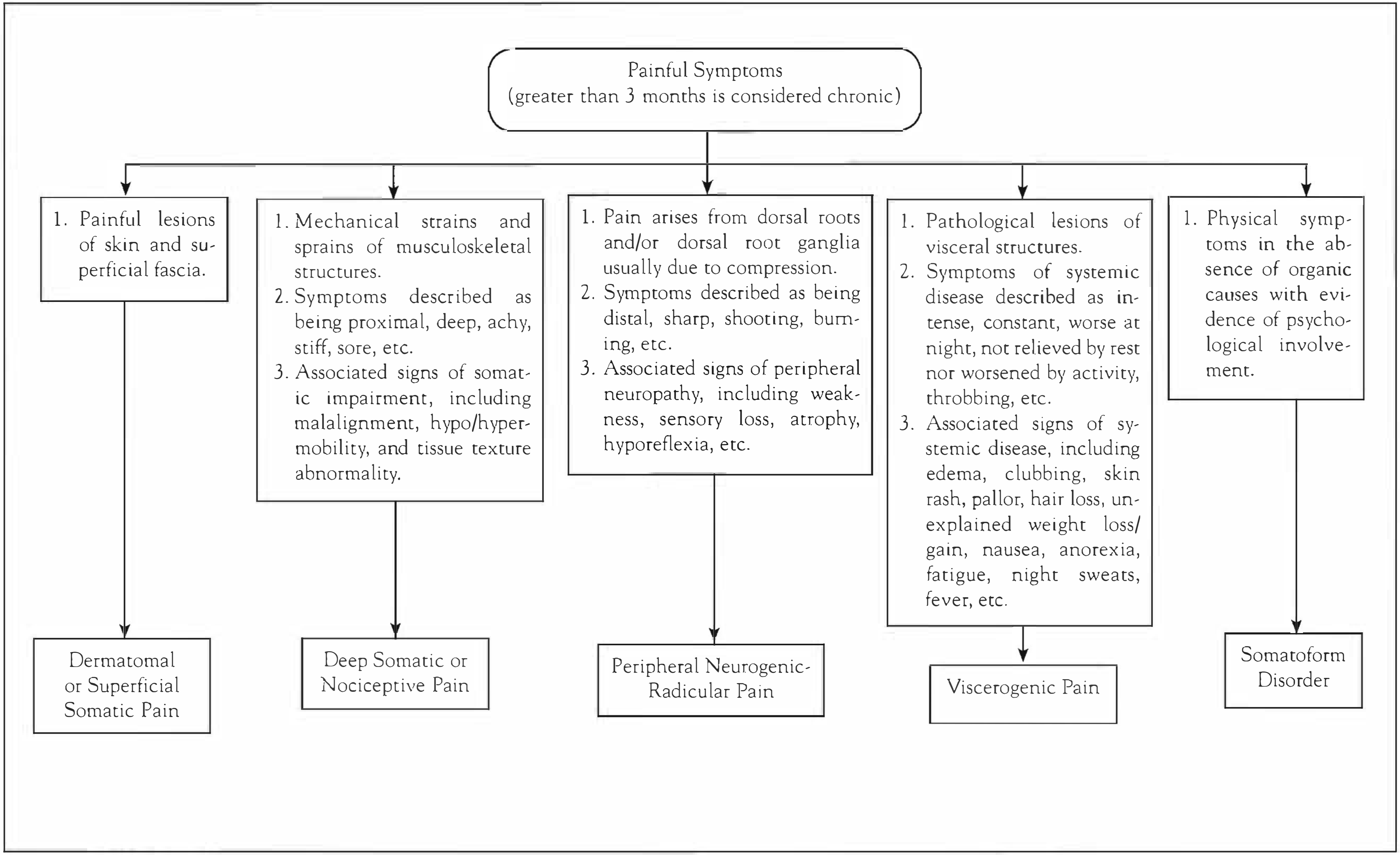

Figure 3-1. Differential diagnosis of painful symptoms.

References

1. Legaspi O, Edmond SL. Does the evidence support the existence of lumbar spine coupled motion? *J Orthop Sports Phys Ther.* 2007;37(4):169-178.
2. Greenman PE. *Principles of Manual Medicine.* 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2003.
3. Isaacs ER, Bookhout MR. *Bourdillon's Spinal Manipulation.* 6th ed. Woburn, MA: Butterworth-Heinemann; 2002.
4. Kaltenborn FM. *Manual Mobilization of the Joints, Vol. 1, The Extremities.* 6th ed. Minneapolis, MN: OPTP; 2006.
5. McKenzie R, May S. *The Lumbar Spine, Mechanical Diagnosis & Therapy. Vol. 1.* Waikanae, New Zealand: Spinal Publications New Zealand Ltd; 2003.
6. McKenzie R, May S. *The Lumbar Spine, Mechanical Diagnosis & Therapy. Vol. 2.* Waikanae, New Zealand: Spinal Publications New Zealand Ltd; 2003.
7. McKenzie RA. *The Cervical and Thoracic Spine, Mechanical Diagnosis and Therapy.* Waikanae, New Zealand: R.A. McKenzie; 1990.
8. Guide to physical therapist practice. 2nd ed. *Phys Ther.* 2001;81(1):1-768.
9. Panjabi M. The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. *J Spinal Disord.* 1989;5:383-389.
10. Panjabi M. The stabilizing system of the spine. Part II. Neutral zone and stability hypothesis. *J Spinal Disord.* 1992;5:393-397.
11. Richardson C, Hodges P, Hides J. *Therapeutic Exercise for Lumbopelvic Stabilization: A Motor Control Approach for the Treatment and Prevention of Low Back Pain.* 2nd ed. Edinburgh, UK: Churchill Livingstone; 2004.
12. Jemmett R. *Spinal Stabilization: The New Science of Back Pain.* 2nd ed. Halifax, Canada: Novont Health Publishing; 2003.
13. DiGiovanna EL, Schiowitz S, eds. *An Osteopathic Approach to Diagnosis and Treatment.* Philadelphia, PA: Lippincott Williams & Wilkins; 2004.
14. Goodman CC, Snyder TE. *Differential Diagnosis for Physical Therapists: Screening for Referral.* 4th ed. St. Louis, MO: Saunders Elsevier; 2007.
15. Boissonnault WG. *Primary Care for the Physical Therapist: Examination and Triage.* St. Louis, MO: Elsevier Saunders; 2005.
16. Schafer RC, Faye LJ. *Motion Palpation and Chiropractic Technique: Principles of Dynamic Chiropractic.* 2nd ed. Huntington Beach, CA: The Motion Palpation Institute; 1989.
17. Stiles EG. Clinical presentations and examples: an osteopathic perspective. In: Flynn TW, ed. *The Thoracic Spine and Rib Cage: Musculoskeletal Evaluation and Treatment.* Boston, MA: Butterworth-Heinemann; 1996.
18. DeConick S. *Orthopaedic Medicine: Cyriax Updated Value in Daily Practice. Part I: Clinical Examination and Diagnosis.* Minneapolis, MN: OPTP; 2003.
19. Maitland GD, Banks K, English K, Hengeveld E. *Maitland's Vertebral Manipulation.* 6th ed. Oxford: Butterworth-Heinemann; 2001.
20. Ellis JJ, Johnson GS. Myofascial considerations in somatic dysfunction of the thorax. In: Flynn TW, ed. *The Thoracic Spine and Rib Cage: Musculoskeletal Evaluation and Treatment.* Boston, MA: Butterworth-Heinemann; 1996.
21. Bland JH. Pain in the head and neck: where does it come from? *J Craniomandib Pract.* 1989;7(3):167-169.
22. Seaman DR, Cleveland C. Spinal pain syndromes: nociceptive, neuropathic, and psychologic mechanisms. *J Manipulative Physiol Ther.* 1999;22(7):458-472.
23. Hersh EV. Mechanisms of pain. In: Pertes RA, Gross SG, eds. *Clinical Management of Temporomandibular Disorders and Orofacial Pain.* Chicago, IL: Quintessence Publishing Co; 1995.
24. Spitzer WO. Scientific approach to the assessment and management of activity-related spinal disorders. A monograph for clinicians. Report of the Quebec Task Force on Spinal Disorders. *Spine.* 1987;12(7S):S16.
25. Makofsky HW. Position-assisted combination technique (PACT) in the management of type-II impairment in the thoracic and lumbar spine. *J Manual Manipulative Ther.* 2003;11(4):213-222.
26. Paris SV, Loubert PV. *FCO: Foundations of Clinical Orthopaedics, Seminar Manual.* St. Augustine, FL: Institute Press; 1999.
27. McGonigle T, Matley KW. Soft tissue treatment and muscle stretching. *J Manual Manipulative Ther.* 1994;2:55-62.
28. Dutton M. *Orthopaedic Examination, Evaluation, and Intervention.* 2nd ed. New York, NY: McGraw-Hill Companies Inc; 2008.

29. Andrade C-K, Clifford P. *Outcome-Based Massage, from Evidence to Practice.* 2nd ed. Baltimore, MD: Lippincott Williams & Wilkins; 2008.
30. Marques AP. *Cadeias Musculares: Um Programa para Ensinar Avaliacao Fisioterapeutica Global.* Sao Paulo, Brazil: Manole; 2005.
31. Rolf I. *The Integration of Human Structures.* New York, NY: Harper & Row Publishers; 1978.
32. Janda V. Muscles and motor control in cervicogenic disorders: assessment and management. In: Grant R, ed. *Physical Therapy of the Cervical and Thoracic Spine: Clinics in Physical Therapy.* 3rd ed. New York, NY: Churchill Livingstone; 2002.
33. Chaitow L, DeLany JW. *Clinical Application of Neuromuscular Techniques. Vol. 2, The Lower Body.* Edinburgh: Churchill Livingstone; 2002.
34. Richardson C, Hodges P, Hides J. *Therapeutic Exercise for Lumbopelvic Stabilization: A Motor Control Approach for the Treatment and Prevention of Low Back Pain.* 2nd ed. Edinburgh: Churchill Livingstone; 2004.
35. Huijbregts PA. Chiropractic legal challenges to the physical therapy scope of practice: Anybody else taking the ethical high ground? *J Manual Manipulative Ther.* 2007;15(2):69-80.
36. Dvorak J, Dvorak V, Schneider W, eds. *Manual Medicine.* Heidelberg, Germany: Springer-Verlag; 1985.
37. McConnell J. *McConnell Institute: The McConnell Approach to the Problem Shoulder.* Course Manual; 1994.
38. Johnson GS, Saliba VL. *Functional Orthopedics I: Soft Tissue Mobilization, PNF, and Joint Mobilization.* Steamboat Springs, CO: IPA Course Notes; 2001.
39. Barnes JF. Myofascial release: the missing link in traditional treatment. In: Davis CM, ed. *Complementary Therapies in Rehabilitation, Holistic Approaches for Prevention and Wellness.* Thorofare, NJ: SLACK Incorporated; 1997.
40. Conable B, Conable W. *How to Learn the Alexander Technique: A Manual for Students.* 2nd ed. Columbus, OH: Andover Road Press; 1992.
41. Caplan D. *Back Trouble: A New Approach to Prevention and Recovery.* Gainesville, FL: Triad Publishing; 1987.
42. Warmerdam A. *Manual Therapy Improve Muscle and Joint Functioning.* Wantagh, NY: Pine Publications; 1999.
43. Cleland J, Selleck B, Stowell T, et al. Short-term effects of thoracic manipulation on lower trapezius muscle strength. *J Manual Manipulative Ther.* 2004;12:82-90.
44. Liebler EJ, Tfano-Coors L, Douris P, et al. The effect of thoracic spine mobilization on lower trapezius strength testing. *J Manual Manipulative Ther.* 2001;9:207-212.
45. Yerys S, Makofsky, H, Byrd C, Pennachio J, Cinkay J. Effect of mobilization of the anterior hip capsule on gluteus maximus strength. *J Manual Manipulative Ther.* 2002;10:218-224.
46. Makofsky H, Panicker S, Abbruzzese J, et al. Immediate effect of grade IV inferior hip joint mobilization on hip abductor torque: a pilot study. *J Manual Manipulative Ther.* 2007;15(2):103-111.
47. Stiles EG. Clinical presentations and examples: An osteopathic perspective. In: Flynn TW, ed. *The Thoracic Spine and Rib Cage: Musculoskeletal Evaluation and Treatment.* Boston, MA: Butterworth-Heineman; 1996.
48. Valentine T, Valentine C. *Applied Kinesiology.* Rochester, VT: Healing Arts Press; 1987.
49. Sahrmann SA. *The Movement System Balance Theory: Relationship to Musculoskeletal Pain Syndromes.* Course Notes; 1990.
50. Sahrmann SA. *Diagnosis and Treatment of Movement Impairment Syndromes.* St. Louis, MO: Mosby-Year Book; 2002.
51. Wainner RS, Whitman JM, Cleland JA, Flynn TW. Regional interdependence: a musculoskeletal examination model whose time has come. *J Orthop Sports Phys Ther.* 2007;37(11):658-660.
52. Swartzlander B. Investigating a new model of assessing musculoskeletal disorder. *Advance Magazine for Physical Therapists and PT Assistants.* 2008;19(17):62.
53. Selkowitz DM, Chaney C, Stuckey SJ, Vlad G. The effects of scapular taping on the surface electromyographic signal amplitude of shoulder girdle muscles during upper extremity elevation in individuals with suspected shoulder impingement syndrome. *J Orthop Sports Phys Ther.* 2007;37(11):694-702.
54. Wright EF, Domenech MA, Fischer JR. Usefulness of posture training for patients with temporomandibular disorders. *JADA.* 2000;131:202-210.
55. Dierks TA, Manal KT, Hamill, J, Davis IS. Proximal and distal influences on hip and knee kinematics in runners with patellofemoral pain during a prolonged run. *J Orthop Sports Phys Ther.* 2008;38(8):448-456.
56. Vaughn DW. Isolated knee pain: a case report highlighting regional interdependence. *J Orthop Sports Phys Ther.* 2008;38(10):616-623.
57. Beneciuk JM, Bishop MD, George SZ. Clinical prediction rules for physical therapy interventions: a systematic review. *Phys Ther.* 2009;89(2):1-11.
58. Huijbregts PA. Clinical prediction rules: time to sacrifice the holy cow of specificity? *J Manual Manipulative Ther.* 2007;15(1):5-8.
59. Laupacis A, Sekar N, Stiell I. Clinical prediction rules: a review and suggested modification of methodological standards. *JAMA.* 1997;277:488-494.
60. Cook CE. Potential pitfalls of clinical prediction rules. *J Manual Manipulative Ther.* 2008;16(2):69-71.
61. Childs JD, Fritz JM, Flynn TW, et al. A clinical prediction rule to identify patients with low back pain most likely to benefit from spinal manipulation: a validation study. *Ann Intern Med.* 2004;141(12):920-928.
62. Hicks GE, Fritz JM, Delitto A, McGill SM. Preliminary development of a clinical prediction rule determining which patients with low back pain will respond to a stabilization exercise program. *Arch Phys Med Rehabil.* 2005;86:1753-1762.
63. Cleland JA, Childs JD, Fritz JM, Whitman JM, Eberhart SL. Development of a clinical prediction rule for guiding treatment of a subgroup of patients with neck pain: use of thoracic spine manipulation, exercise, and patient education. *Phys Ther.* 2007;87(1):9-23.
64. Cleland JA, Fritz JM, Whitman JM, Heath R. Predictors of short-term outcome in people with a clinical diagnosis of cervical radiculopathy. *Phys Ther.* 2007;87(12):1619-1632.
65. Pinto D, Cleland J, Palmer J, Eberhart SL. Management of low back pain: a case series illustrating the pragmatic combination of treatment- and mechanism-based classification systems. *J Manual Manipulative Ther.* 2007;15(2):111-122.
66. Childs JD, Fritz JM, Piva SR, Whitman JM. Proposal of a classification system for patients with neck pain. *J Orthop Sports Phys Ther.* 2004;34(11):686-700.
67. Fritz JM, Delitto A, Erhard RE. 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.* 2003;28(13):1363-1371.
68. Fritz JM, George S. The use of a classification approach to identify subgroups of patients with acute low back pain: interrater reliability and short-term treatment outcomes. *Spine.* 2000;25(1):106-114.
69. Delitto A, Erhard RE, Bowling RW. A treatment-based classification approach to low back syndrome: identifying and staging patients for conservative treatment. *Phys Ther.* 1995;75:470-485.

Section

II

Scapulothoracic Region

4

Examination and Evaluation of the Scapulothoracic Region

Posture

Consistent with the CHARTS methodology, the examination/evaluation of postural alignment is performed following the interview in which the chief complaint (C) and history (H) are recorded. This component of the examination consists of a detailed inspection for the presence of asymmetry (A). In the scapulothoracic region, this will be accomplished by analyzing posture in 3 ways. The patient will be observed from the side (lateral view), back (posterior view), and front (anterior view). The purpose of the postural assessment is to identify areas of potential impairment. Abnormal posture is characterized by alignment that is: imbalanced (sagittal, frontal, and horizontal planes) inefficient, and not in a vertical relationship with gravity. For example, a patient with accentuation of the thoracic kyphosis is likely to develop a restriction in extension and become destabilized in flexion. However, one should not assume that impairment of mobility exists based upon posture alone. Recall that it is the combined ART triad that signals somatic dysfunction (ie, impairment).

The standing lateral view of the scapulothoracic region enables the therapist to inspect the following structures for faulty alignment (Figure 4-1):

- Thoracic kyphosis (normal, increased, decreased). The upper (T1-T4), mid (T5-T8), and lower (T9-T12) thoracic regions should be assessed separately. Flattening of the curve represents an extended position, whereas an accentuated kyphosis represents a flexed position of the spine. Lee[1] approaches the thoracic spine 3-dimensionally as follows: a) Vertebromanubrial region (including T1, T2, ribs 1 and 2, and the manubrium), b) Vertebrosternal region (including T3, T4, T5, T6, T7, ribs 2 through 7, and the body of the sternum), c) Vertebrochondral region (including T8, T9, T10, and their respective costal cartilages which blend with the 7th costal cartilage above), and d) Thoracolumbar region (including T11, T12, and ribs 11 and 12). The advantage of Lee's approach[1] to the thoracic spine is to consider the entire thorax and not just the spine and scapulae (ie, vertebral column, shoulder girdle, ribs, and sternum).
- Scapular position in the horizontal plane (normal, abducted, or adducted).
- Scapular position in the sagittal plane (note an excessive anterior tilt or "tipping" with inferior angle prominence, which is confirmed in supine with anterior displacement of the shoulder versus the contralateral side). The normal scapula is flat against the thorax and rotated, about the X axis, 30 degrees anterior to the frontal plane.[2]
- Sternal angle or manubriosternal junction (should ideally have a slight upward inclination of approximately 30 degrees, but is often in a downward or depressed position).
- The sag of the rib cage (anterior lower than posterior) should not exceed approximately 30 degrees.
- Humeral head position. No more than one-third of the humeral head should be anterior to the

Makofsky HW
Spinal Manual Therapy, 2nd ed. (pp 23-31)

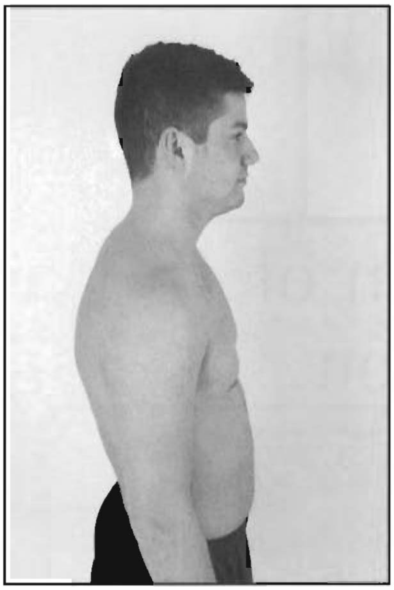

Figure 4-1. Lateral view.

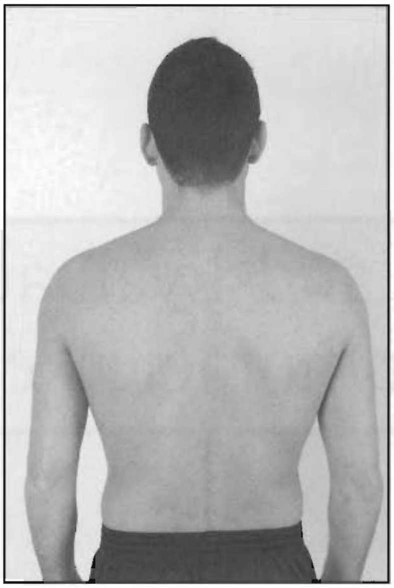

Figure 4-2. Posterior view.

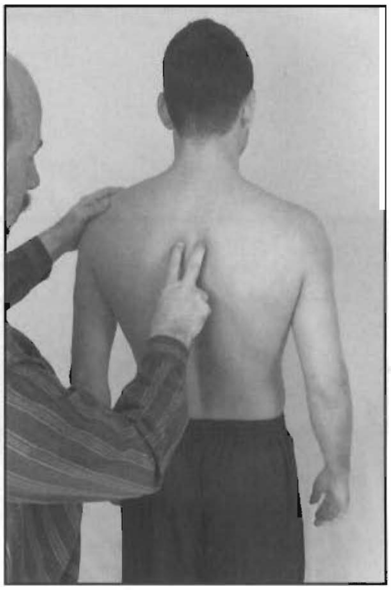

Figure 4-3. Paravertebral assessment for scoliosis.

acromion.[3] Anterior displacement of the humeral head suggests anterior glenohumeral joint hypermobility or posterior glenohumeral capsular tightness. Given a normal anatomic position of the humeral head in the glenoid fossa, the humeral head with respect to the shoulder girdle should be centered at the apex of 2 tangents extending laterally from the sternoclavicular joint anteriorly and the root of the scapular spine posteriorly.

A common postural problem seen in many patients, young and old alike, is a combination of shoulder girdle protraction/elevation, excessive scapular anterior tilt, sternal depression, and an increased mid/lower thoracic kyphosis. However, prior to assuming that an increased thoracic kyphosis has a postural or functional basis, structural causes of a pathological nature, such as Scheuermann's disease or adolescent kyphosis, ankylosing spondylitis, tuberculous spondylitis, osteoporosis, or fracture-dislocation, must be ruled out first.[4]

There are structural deformities of the chest wall[5] that may have significance in the evaluation of the pulmonary patient (eg, Harrison's sulcus, pigeon breast, and pectus excavatum), which are not of major consequence in the patient with somatic impairment. However, the presence of the barrel chest deformity,[5] although a sign of chronic obstructive pulmonary disease (COPD), represents a typical pattern of expiratory rib restriction that may derive some benefit from manual therapy. In addition to visual inspection of thoracic spine alignment, an architect's flexicurve[6] can be molded to the spine to measure the thoracic kyphosis. Another option for measuring the thoracic curve is with DeBrunner's kyphometer.[7]

The standing posterior view of the scapulothoracic region allows us to detect the following positional relationships (Figure 4-2):

- Scapular position in the frontal plane (the scapulae should be symmetrical and almost parallel to the spine; note elevation and upward or downward rotation). According to Sahrmann,[2] the shoulders should be slightly below the T1 level and the vertebral border of the scapula approximately 3 inches from the spine. Less than 3 inches is considered scapular adduction, while greater than 3 inches is considered abduction.
- Scapular position about the Y or vertical axis (external and internal rotation). Excessive internal scapular rotation about a vertical axis results in posterior displacement of the vertebral border (ie, "winging" of the scapula).
- Scoliosis or rotoscoliosis[8] of the upper, mid, and lower thoracic spine. Running the distal finger pads of the second and third digits of one hand down the thoracic spine paravertebrally (until blanching occurs) assists in the detection of a scoliotic curve (Figure 4-3).
- Asymmetry of posterior rib prominence.
- Contour of the neck-shoulder line. This line should be characterized as having a gentle slope.
- Waist angle acuity.
- Position of the upper extremities (eg, neutral, internally/externally rotated).

Common clinical findings related to malalignments/asymmetries in the posterior view include the following:

- Elevation with downward rotation of the scapula secondary to a combination of levator scapulae and pectoralis minor tightness (shoulders that are above the T1 level suggest scapular elevation).
- Anterior tilting or "tipping" of the scapula related to a combination of pectoralis minor tightness and/or weakness of the lower scapular stabilizers (ie, serratus

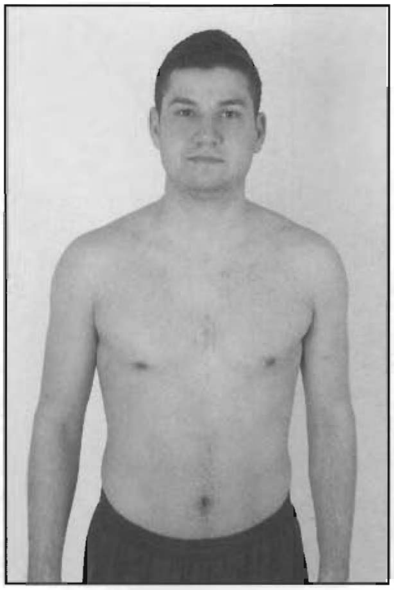

Figure 4-4. Anterior view.

anterior, rhomboids, middle and lower trapezius) associated with lower scapular prominence.

- "Winging" of the vertebral border of the scapula. According to Isaacs and Bookhout,[9] winging of the medial border of the scapula indicates weakness and lack of stabilization by the lower trapezius, serratus anterior, and rhomboid muscles. Weakness of the serratus anterior is often associated with flattening and restricted flexion in the midthoracic region, especially from T3 to T6.[9]
- Posterior rib prominence on the convex side of a mid/lower thoracic side bending curve related to type 1 or neutral spinal mechanics.
- "Gothic" shoulders or straightening of the neck-shoulder line[10] secondary to levator scapulae and upper trapezius tightness (when secondary to levator scapula tightness, the superior angle of the scapula will be higher than the acromion).
- Internally rotated upper extremities secondary to tightness of the latissimus dorsi, pectoralis major, etc.

In addition to the above functional malalignments/asymmetries, the therapist should be cognizant of the structural/pathological deviations in form that affect the scapulae. Examples include Klippel-Feil syndrome, which can cause bilateral scapular elevation, and Sprengel's deformity, another congenital deformity that is associated with an abnormally small/high scapula and poor development on the affected side.[4]

The final anterior view in stance (Figure 4-4) provides an analysis of these relationships:

- Clavicular alignment (the distal end of the clavicle should ideally be horizontal or only slightly elevated relative to the proximal end at the sternoclavicular joint; the clavicles should be symmetrical).
- The linea alba should be straight up and down.
- In males, symmetry of nipple height is assessed.
- The anterior aspect of the rib cage is observed for asymmetry (eg, asymmetry from rotoscoliosis).

Common anterior view asymmetries/misalignments include the following:

- Bilateral clavicular angulation in which the distal end of the clavicles are superior to the proximal attachment.
  der girdle, which is enhanced when the scapulae are also elevated. A unilateral angulation of the clavicle is seen when the shoulder girdle is elevated on the ipsilateral side.
- Asymmetric linea alba and nipple height consistent with the side-bending component of a rotoscoliosis.
- Anterior rib cage prominence on the concave side of a rotoscoliosis (the rotational component of the curve forces the ribs forward on the concave side and backward on the convex side of the curve as per type 1 spinal mechanics).

In the final analysis there are 4 abnormal postural patterns in the scapulothoracic region that are routinely encountered in clinical practice. They are as follows:

- Shoulder girdle protraction/elevation associated with an increased mid/lower thoracic kyphosis, sternal depression, and angulated clavicles.
  superior angle of the scapula, but not the acromion, suggests that the levator scapula is short; elevation of the entire scapula, including the acromion, infers that the upper trapezius is short.[2]
- Shoulder girdle protraction/depression associated with an increased mid/lower thoracic kyphosis, sternal depression, and angulated clavicles (pectoralis minor/major and latissimus dorsi muscles tend to be tight). The scapula is considered depressed when its superior angle is positioned lower than the second thoracic vertebra, implying that the upper trapezius muscle is long.[2]
- Scapular "winging" associated with flattening of the thoracic kyphosis, especially from T3 to T6.[9]
- Thoracic spine rotoscoliosis associated with an anterior rib prominence on the concave side of the curve and posterior rib prominence on the convex side of the curve. The shoulder girdle will tend to be higher on the convex side of the curve and the waist angle sharper on the concave side.

According to Kendall, et al[11] we must remember that hand dominance plays a role in spinal asymmetry such that an individual who is right-hand dominant would be expected to carry his or her right shoulder slightly lower and the right hip slightly higher as a normal variation.
the low shoulder is found on the nondominant side that our index of suspicion is raised.

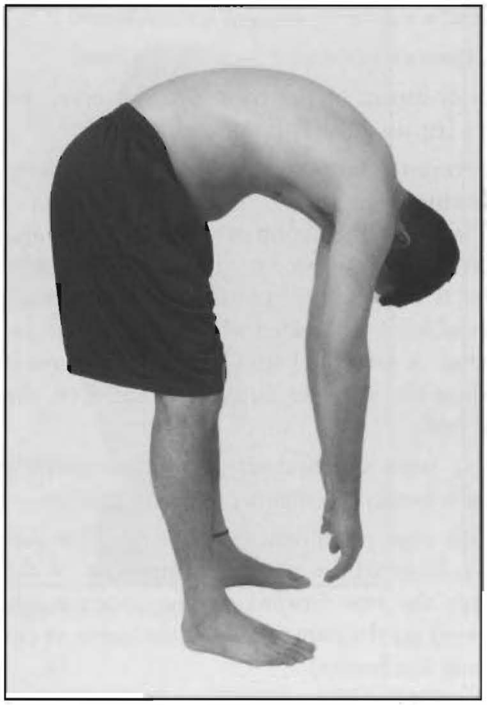

Figure 4-5a. Forward bending.

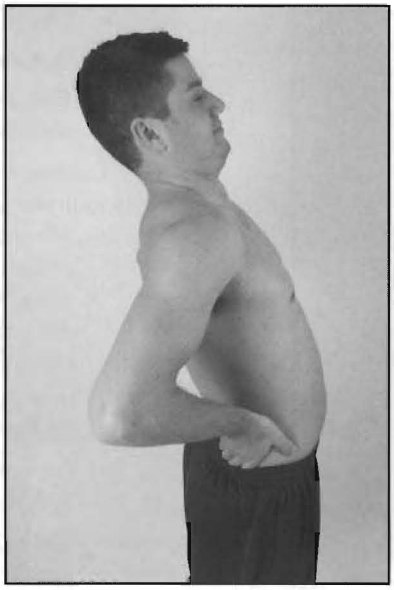

Figure 4-5b. Backward bending.

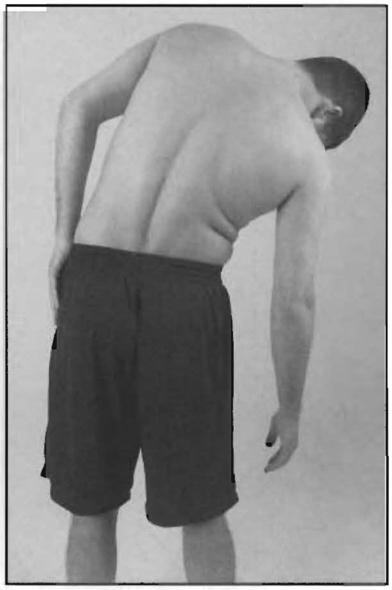

Figure 4-5c. Side bending right.

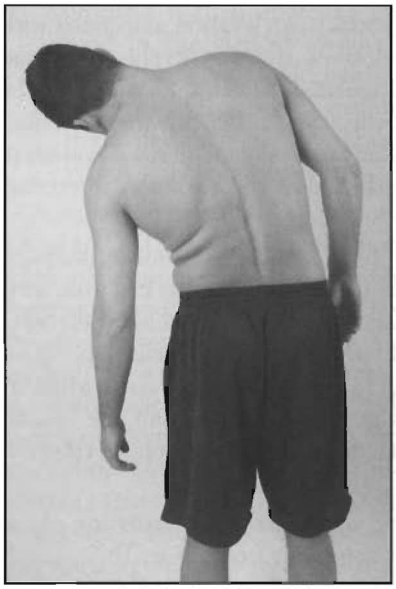

Figure 4-5d. Side bending left.

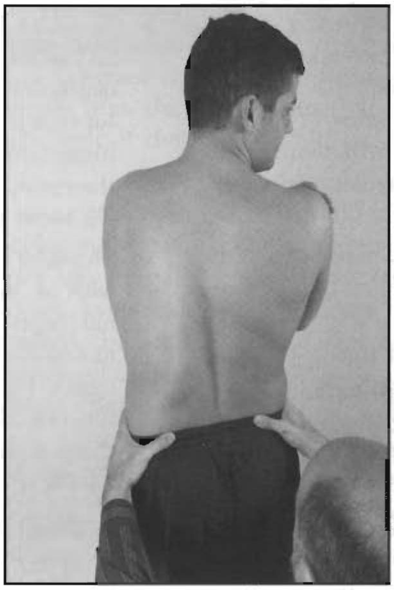

Figure 4-5e. Rotation right.

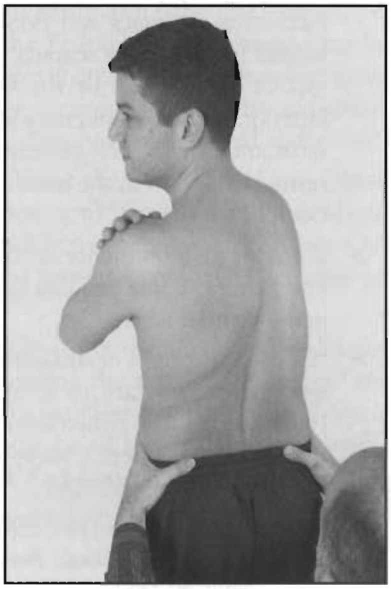

Figure 4-5f. Rotation left.

Active Movements

Now that C, H, and A have been completed, we can move onto R, which begins with an assessment of active range of motion. The examination of active thoracic movements consists of an analysis of 6 motions (Figures 4-5a to 4-5f). They are forward bending (ie, flexion), backward bending (ie, extension), side bending (ie, lateral flexion) to the right and left, and rotation to the right and left.

This part of the examination, as with the postural assessment, is performed while the patient stands. There is highly important information to be gleaned from the observation of active spinal motion. The following are a summary of points of which to take note:

1. The patient should stand in a comfortable and relaxed position in as close to the neutral position as possible.
2. Motion should start from the head and proceed to the neck and spine.
3. Though the quantity of movement is important and can be documented with inclinometers[12] (Figures

Figure 4-6a. Measuring thoracic flexion with inclinometers.

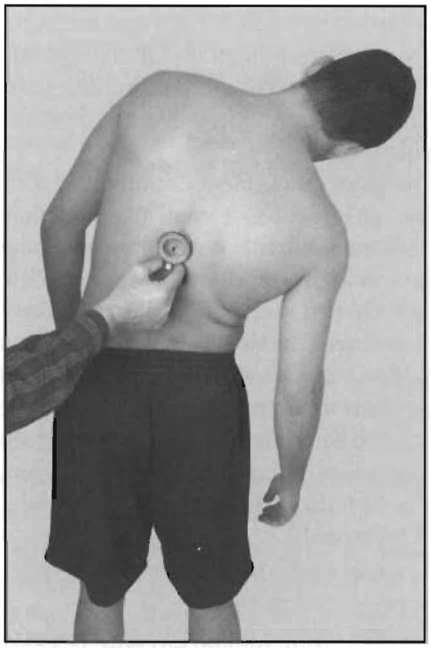

Figure 4-6b. Measuring thoracic side bending with an inclinometer.

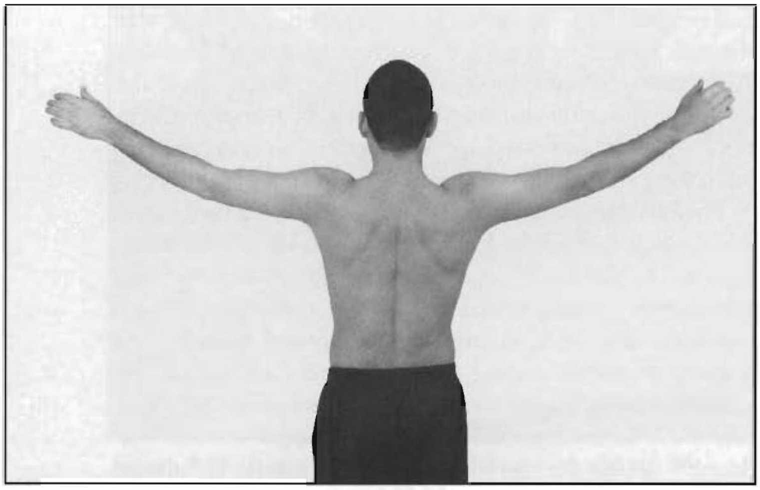

Figure 4-7. Bilateral scapulohumeral rhythm.

4-6a and 4-6b), it is the quality of motion that is most important to the manual therapist. For example, a patient may appear to have normal spinal flexion in that he or she can easily touch the floor. However, on closer inspection it is noted that it is the hamstrings that are flexible, whereas the spine demonstrates limitation of motion. The assessment of an active movement's quality requires skill in observation, which becomes better with practice. Optimal human motion is described as effortless, efficient, and smooth. It is without interference, restriction, or hypermobility. Whether the curve is anteroposterior as in forward and backward bending or mediolateral as in side bending, it should be a well-contoured and unbroken curve. Impaired movement is characterized by flat or straight lines that may cause effort and even pain. Motion loss in one area of the spine will cause another area to compensate and this is represented by pivot points or fulcrums. These areas of compensation tend toward hypermobility and may become symptomatic, while the areas of hypomobility remain stiff but are often asymptomatic. The mission of the manual therapist is to locate these stiff segments and to then decide which among them is the AGR. It is with this "culprit" lesion that we commence manipulative intervention.

4. By means of a comparison between pain and tissue stiffness, the therapist is able to determine the tissue's level of reactivity. This determination will serve as a guide in our choice of intervention later (ie, high reactivity will require indirect treatment methods and the use of pain-relieving modalities, whereas low reactivity responds better to direct techniques, as discussed in Chapter 3).
5. Whenever possible, a correlation between positional asymmetry and impaired mobility should be established. This correlation, in conjunction with tissue texture abnormality, provides the basis for diagnosing somatic impairment. For example, a correlation between an increased thoracic kyphosis from T5 to T10 and restricted backward bending in the same region has more clinical significance for the manual therapist than either one by itself.

The final aspect of active motion testing in the scapulothoracic region involves an assessment of scapulohumeral rhythm. This is accomplished by having the patient abduct both upper extremities in either the frontal plane or the plane of scapula, while the therapist observes scapular upward rotation from a posterior view (Figure 4-7). A normal response is to observe upward rotation of the scapulae through a range of 60 degrees beginning at approximately 30 degrees of shoulder abduction without "hiking up of the shawl area" or premature and/or excessive scapular abduction.[9] The levator scapulae and upper trapezius muscles often become tight by substituting for ipsilateral weakness of the lower trapezius, serratus anterior, and supraspinatus. This muscle imbalance has the potential to result in neck pain and headaches.[9,13] A common pattern, associated with subacromial impingement, is the combination of limited posterior scapular tilt, restricted scapular upward rotation, and scapular elevation during upper extremity elevation[9,14,15] resulting in inadequate clearance of the subacromial tissues under the coracoacromial arch. Janda's upper crossed syndrome[10] details the specific axioscapular muscle imbalance that accounts for this (eg, tight levator scapula and/or weak lower trapezius/serratus anterior). Sahrmann[2] points out that during shoulder abduction, in the presence of serratus anterior weakness, the inferior angle of the scapula fails to reach the midaxillary line of the trunk secondary to inadequate protraction on the affected side.

The connection between subacromial impingement and movement impairment of the scapulothoracic region has received much attention in the literature.[9,14-18] For those clinicians who attempt to manage shoulder impairment in general and subacromial impingement in particular without an appreciation of the influence of scapulothoracic posture, there no longer remains an excuse!

Passive Accessory Intervertebral Movements

There are 2 accessory or joint play motions in the thoracic spine that provide important information. They are PA and transverse pressures on the SPs. PA pressure on a thoracic SP induces extension, and transverse pressure on the side of the SP induces rotation. The purpose of performing passive accessory intervertebral movements (PAIVMs) is to identify motion restrictions in the 12 motion segments of the thoracic spine. When applying these manual forces, the therapist is reminded to use as little force as possible, but as much force as necessary. There are 4 components of the accessory motion assessment. They are the quality and quantity of the accessory motion, the end-feel[19,20] imparted to the therapist's manual contact at the end of the available range of movement, and tissue reactivity. The assessment of quality involves how the vertebral segment moves. Words such as *free*, *restricted*, *easy*, *hard*, *smooth*, and *rough* can be used here. The assessment of accessory joint quantity is based upon a 0 to 6 scale[20] previously discussed in Chapter 3. End-feel in this text is described as 1) normal—healthy tissue yield at end-range associated with normal joint mobility, 2) stiff—decreased tissue yield at end-range associated with hypomobility, or 3) loose—increased tissue yield at end-range associated with hypermobility. As mentioned in Chapter 3, tissue reactivity is described as high, moderate, or low based upon the relationship between pain and tissue stiffness. Tissue reactivity is a useful concept when deciding on the type of intervention to utilize (eg, pain-relieving modalities, indirect technique, grade 1 to 5 direct mobilization/manipulation).

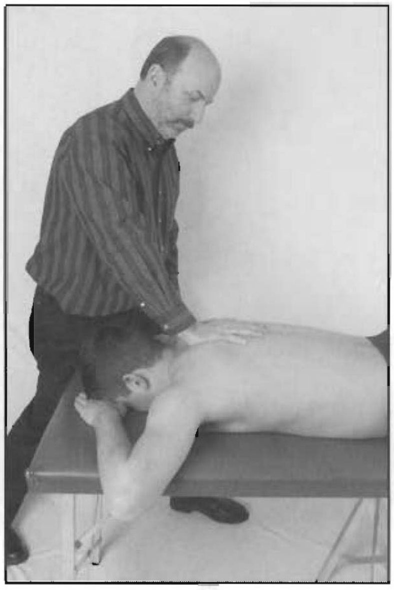

Figure 4-8. PA central spring T1-T4.

The PA accessory movement examination is classified as a "spring test," as it involves a small amplitude impulse over the SP. The therapist has the option of assessing superior vertebral motion with posterior rotation (ie, roll) or inferior vertebral motion with an anterior translation (ie, glide) because both forces induce spinal extension. However, considering that translations are easier to control and are more precise than rotations, the PA Central Spring Test is performed on the SP of the inferior vertebra, thus inducing segmental extension associated with facet closing (ie, PA translation of T4 induces T3,4 extension with bilateral closing of the T3,4 facets).

The PA central spring test for the assessment of thoracic extension is performed as follows:

1. The patient is prone lying with proper support provided. It is important that the thoracic spine be placed in a neutral position during testing.
2. The table height should be adjusted so that the therapist's middle finger reaches the top of the table when the therapist is standing.
3. T1 to T4 is best assessed with the therapist standing at the side of the table level with the patient's head-neck region and facing in a caudal direction. The therapist places his or her hand over the patient's spine, fingers pointing downward, such that the SP is cradled between the therapist's thenar and hypothenar eminence in the palmar groove (Figure 4-8).

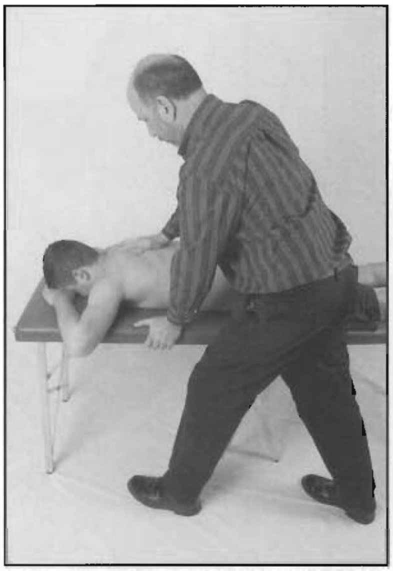

Figure 4-9. PA central spring T5-T12.

4. T5 to T12 is best assessed at the side of the table, but this time with the therapist facing toward the head end of the table. Once again the SP is cradled in the palmar groove between the thenar and hypothenar contacts, but this time with the therapist's fingers directed cranially (Figure 4-9). The therapist must demonstrate proper body mechanics at all times (ie, neutral pelvis; optimal head, neck, and spinal alignment, etc).
5. Due to the progressive inferior angulation of the SP in the thoracic spine from cranial to caudal, the therapist must incorporate a superiorly directed force below T3. A helpful landmark is to perform the PA spring anteriorly/superiorly toward the sternal angle. This will ensure that the translational motion occurs in the plane of the apophyseal joints.
6. The AGR is identified when somatic impairment is present.

A few surface landmarks are helpful in identifying the various spinal levels. The superior angle of the scapula is at the level of the T2 SP, the root of the scapular spine is at the level of the T3 SP, and the inferior angle of the scapula is at the level of the T7 SP. The most reliable method, however, is to locate the SP of C7 (vertebra prominens) and count down from there. The location of the cervical landmarks, including C7, will be covered in a subsequent chapter on the cervical spine.

The transverse pressure test for the assessment of segmental rotation is performed as follows:

1. The patient, lying prone, is again positioned in a comfortable, neutral posture with the table at the correct height for the therapist.
2. To assess rotation right from T1 to T12, the therapist stands on the patient's right side and slowly displaces the SP from right to left. This is accomplished by placing the passive thumb directly over the lateral aspect of the SP, which is reinforced by the active thumb. The movement for this procedure is not through the thumbs but through the upper extremities. If thumb contact is uncomfortable, the therapist can use a thenar eminence contact instead (Figure 4-10).

Figure 4-10. Transverse pressure T1-T12 from right to left.

3. As with the PA spring test, the therapist assesses the quality, quantity, end-feel, and tissue reactivity of the accessory range. If somatic impairment is present, the AGR needs to be identified. In the presence of segmental facilitation[21] (ie, local hyperalgesia, hypertonicity, and up-regulation of sympathetic activity), a technique known as "chasing the pain" has been used effectively. It consists of rapid transverse pressure oscillations for 30 to 60 seconds in the pain-free range. The segment is essentially "bombarded" with proprioceptive afferent input, which helps to "down-regulate" its neural facilitation and decrease painful symptoms. This process is repeated 2 to 3 times, increasing the amplitude of pain-free motion with each set of oscillations.

Passive Physiologic Intervertebral Movements

Passive physiologic intervertebral movements (PPIVMs) provide a means of evaluating physiologic motion of the spine on a segmental basis. As with PAIVMs, the quality, quantity, end-feel, and tissue reactivity are assessed at each motion segment of the thoracic spine (T1-T12). The examination includes the motions of forward and backward bending, side bending right and left, and rotation right and left.

Upper Thoracic Spine (T1 to T4)

With the patient in the sitting position, the therapist uses the head-neck region to induce the desired physiologic

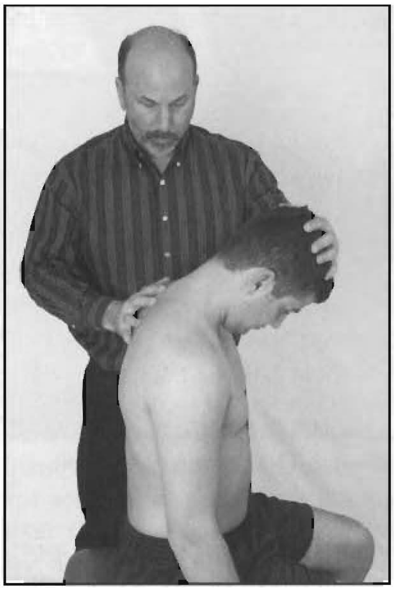

Figure 4-11a. PPIVM exam T1-T4 forward bending.

Figure 4-11b. PPIVM exam of T1,2 and T2,3 backward bending.

Figure 4-11c. PPIVM exam of T3,4 and T4,5 backward bending.

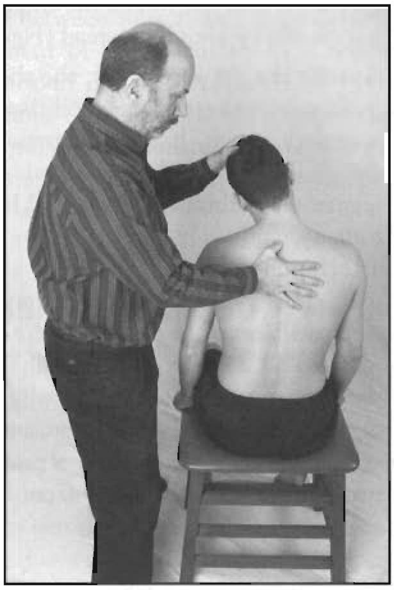

Figure 4-11d. PPIVM exam of T1-T4 side bending.

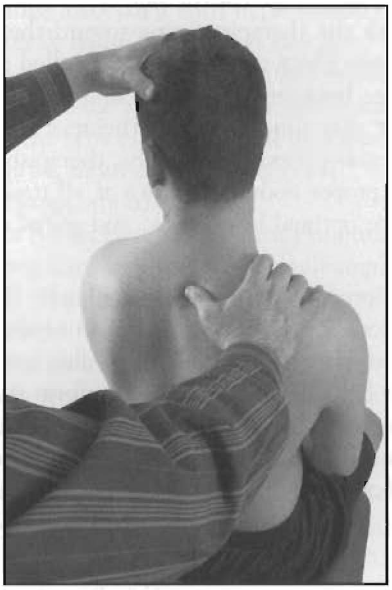

Figure 4-11e. PPIVM exam of T1-T4 rotation.

motions. While this is performed with one upper extremity (light cradling hold), the thumb or middle finger of the other hand is assessing intervertebral motion between the spinous processes (interspinous space or simply interspace) of T1,2; T2,3; T3,4; and T4,5. For the evaluation of side bending, the palpating contact is on the ipsilateral side of the movement; for rotation, the contact is on the contralateral side of the interspinous space (Figures 4-11a to 4-11e).

The most challenging of the 6 motions in the upper thoracic region is backward bending. In order to achieve upper thoracic extension, the therapist must induce upper cervical flexion (chin-tuck position), which will recruit lower cervical and then upper thoracic extension via head-neck retraction. If only the patient's head is extended, then the extension is confined to the craniovertebral region, which is unacceptable. In order to induce extension at the T3,4 and T4,5 segments, an overturning motion of the head-neck region into backward bending follows head-neck retraction. An alternative means of assessing side bending and backward bending is to translate the inferior vertebra of the segment opposite to the motion being assessed and thus "dive under the wave" (eg, PA pressure on the SP of T3 under T2 for T2,3 extension and a lateral pressure on the SP of T4 to the right under T3 to assess T3,4 side bending left). This is referred to as segmental roll-gliding.

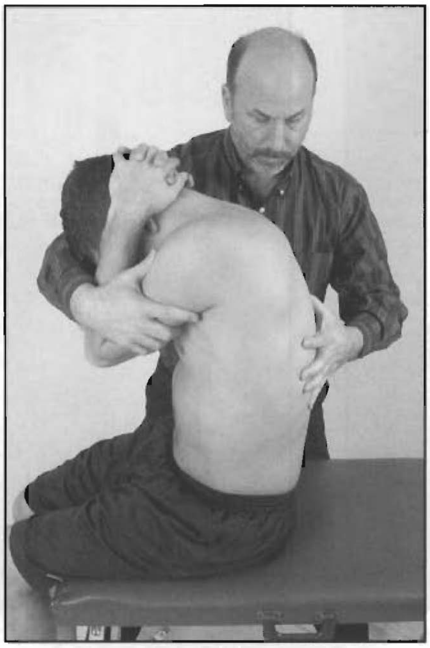

Figure 4-12a. PPIVM exam of T5-T12 forward bending.

Figure 4-12b. PPIVM exam of T5-T12 backward bending.

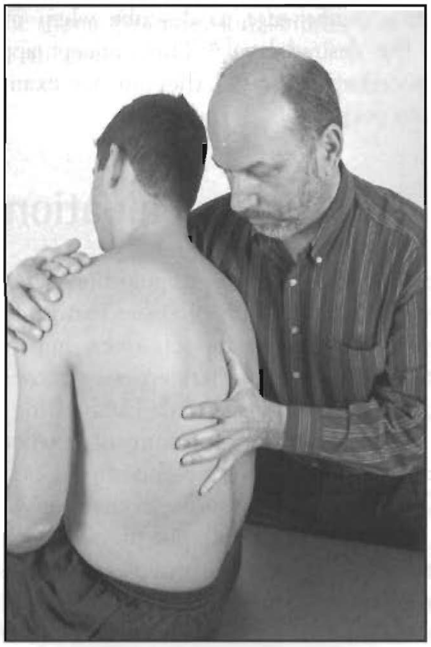

Figure 4-12c. PPIVM exam of T5-T12 side bending right.

Mid/Lower Thoracic Spine (T5-T12)

As with the upper thoracic spine, T5 to T12 is assessed at the interspinous space of each motion segment with a palpating finger (usually the thumb or middle finger), while physiologic motion is induced through the trunk with the other upper limb. During forward bending, separation of the SPs is assessed while backward bending assesses the approximation of the SPs. For side bending, the therapist's palpating contact is placed on the same side of the interspinous space as the side bending; for rotation, it is placed on the side opposite the direction of the movement. The patient and therapist's positions change from one movement to the next, as illustrated, but the principles remain the same as for the upper thoracic region. Some clinicians perform PPIVMs in both the sitting and recumbent positions, but a sitting examination alone is sufficient (Figures 4-12a to 4-12d). As with T1-T4, segmental roll-gliding can be performed when evaluating side bending and backward bending. This involves translating the inferior vertebra in the opposite direction to the physiologic motion being assessed (ie, "dive under the wave"). Thus the superior vertebra of the segment rolls while the inferior vertebra glides. The end-feel at the end of the translation range (ie, end-play) is very useful in detecting the presence of somatic impairment.

Passive Accessory Rib Mobility

Ribs 1 to 4

1. The first rib is found at the "summit" of the shawl area, halfway between the clavicle, anteriorly, and spine of the scapula, posteriorly. With the patient seated, the therapist passively left rotates the head-neck to the end of range with the left hand until motion arrives at the right first rib (Figure 4-13).
2. With the right hand positioned over the right upper trapezius, the therapist's right thumb applies a PA

Figure 4-12d. PPIVM exam of T5-12 rotation right.

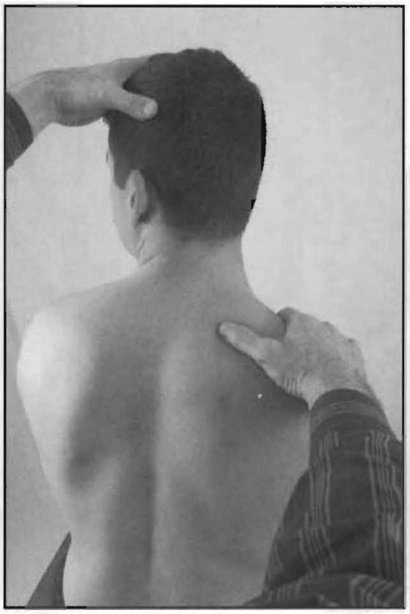

Figure 4-13. Assessment of ribs 1-4 on the right.

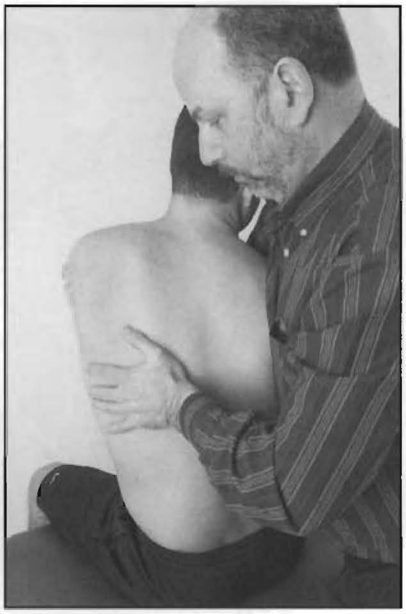

Figure 4-14. Assessment of ribs 5-12 on the left.

pressure to the right first rib just lateral to the costotransverse joint. The motion is assessed for quality, quantity, end-feel, and tissue reactivity and compared to the other side by simply reversing the maneuver for the left first rib.

3. Ribs 2, 3, and 4 on the right are similarly assessed by dropping the right thumb down to the desired rib level, just medial to the vertebral border of the scapula, and rotating the patient's head-neck region to the left until motion arrives at the thumb/posterior rib contact. Ribs 2, 3, and 4 on the left are assessed by reversing this maneuver.

Ribs 5 to 12

1. The seated patient's left hand is placed on his or her right shoulder.
2. The therapist reaches across the anterior chest wall and places his or her right hand on the patient's left shoulder while placing the left thumb, thenar eminence or pisiform contact against the medial aspect of the left-sided rib angle to be assessed. The therapist rotates the patient's trunk to the right until motion is perceived at the desired rib angle on the left (Figure 4-14).
3. At this point in the mobility exam, the therapist displaces each of the rib angles 5 to 12 sequentially in a transverse manner to the left. The exam is repeated on the right side by simply reversing the maneuver, and a comparison is made.

Localization to the desired level is crucial with the above passive accessory rib mobility tests. Osteopathic physicians use the term *feather-edge* to describe when motion first arrives at the desired level.[8] This concept applies to all manual procedures, whether they are for examination or intervention purposes.

Soft Tissue Palpation

The examination of the scapulothoracic region now progresses to the evaluation of tissue texture abnormality (T). There are 3 markers for soft tissue impairment that are worth noting. They are tenderness, tightness (ie, contracture), and tone (contraction). Establishing a baseline measure for the amount of pressure used when assessing tenderness is important during this aspect of the examination. Otherwise, false positive and negative errors are likely. In this regard, the therapist presses on the patient's anterior thigh with a light but firm pressure that should not be perceived by the patient as tender. If it is, then either the pressure is too strong or the other thigh should be used instead. It is this same nontender pressure that is used to assess the tissues of the scapulothoracic region. In addition to the presence of tenderness, the examiner is also evaluating the patient for muscle tightness and increased muscle tone (ie, the type associated with reflex-induced splinting or guarding and not that of spasticity from central nervous system disease).

The entire chest wall should be assessed. Structures to be examined include the following:

- Sternoclavicular joints
- Costosternal joints
- Costochondral junctions

- Intercostal spaces
- Xyphoid process
- Skin and superficial fascia
- Rectus abdominis
- Diaphragm
- Pectoral muscles and fascias, including the clavipectoral fascia
- Subclavius muscles
- Intercostal muscles
- Coracoclavicular ligament (conoid and trapezoid components)

The posterior and lateral aspects of the scapulothoracic region are examined next. Structures to be examined from superficial to deep include the following:

- Skin and superficial fascia
- Trapezius muscle (upper, mid, and lower fibers) and latissimus dorsi/thoracolumbar fascia
- Rhomboid major and minor, levator scapulae at the superior angle of the scapulae, supra/infraspinatus, teres major/minor
- Erector spinae (spinalis, longissimus, and iliocostalis at the rib angle)
- Transversospinales (semispinalis, multifidus, rotatores) in the medial groove of longissimus
- Costotransverse joints at the lateral edge of the longissimus thoracis muscle
- Intercostal spaces

In addition to the inspection for tenderness, tightness, and tone, the myofascial tissues of the scapulothoracic region and costal cage can also be examined for taut bands, trigger/tender points, swelling, fibrositic nodules, extensibility, and length.

Special Tests

The final category in the CHARTS process is special tests (S). It is here that all neurologic, vascular, integumentary, cardiopulmonary, and additional orthopedic procedures are performed. Examples include sensory/motor testing, thoracic outlet (inlet) tests, chest expansion, etc, all of which should demonstrate acceptable levels of diagnostic accuracy[22] (eg, sensitivity, specificity).

5

Connective Tissue Techniques for the Scapulothoracic Region (Myofascial Release, Direct Fascial Technique, and Friction)

Research over the past 10 years has provided the clinician with new insight into the pathophysiology and management of myofascial trigger points. The second edition of *Travell & Simon's Myofascial Pain and Dysfunction: The Trigger Point Manual*[23] proposes an "integrated hypothesis" purporting that the etiology of trigger points involves local myofascial tissues, the central nervous system (CNS), and biomechanical factors. McPartland[24] refers to trigger points as "contraction knots" and cites electromyography (EMG) research suggesting that trigger points represent regions of spontaneous electrical activity in a muscle secondary to motor end-plate dysfunction. As a consequence of sustained muscle contraction, local blood vessels are compressed, reducing local oxygen supply. The impaired local circulation coupled with the increased metabolic demands of the contracted muscle fibers results in the rapid depletion of local adenosine triphosphate (ATP). In addition to inhibiting ACH release from the nerve terminal, ATP also powers the Ca^{2+} pump, which returns calcium to the sarcoplasmic reticulum. Therefore, an "ATP energy crisis" not only increases ACH release, but also impairs the reuptake of Ca^{2+}, which increases the local contractile activity of the "contraction knot"—a vicious cycle.

In addition to the motor end-plate dysfunction theory, Shah et al,[25] using an in vivo microdialysis needle, found that the local biochemical milieu of the upper trapezius muscle in patients with active myofascial trigger points contained significantly higher concentrations of protons, bradykinin, calcitonin gene-related peptide (CGRP), substance P, tumor necrosis factor-alpha, interleukin-1 beta, serotonin, and norepinephrine as compared to normal subjects and asymptomatic patients with latent myofascial trigger points. The authors of this study conclude, "Exploration of the biochemical milieu of myofascial trigger points and normal muscle may help explain the pathogenesis, persistence, and amplification of myofascial pain." In addition, it is theorized that the nociceptive input from chronically active trigger points may have a sensitizing effect[26] on the CNS (ie, central sensitization) and thus play a role in such chronic pain states as chronic tension-type headaches,[27] migraine,[28] and fibromyalgia.[29]

Thoracic Inlet Release

The thoracic inlet (superior thoracic outlet) is the cephalic opening of the thoracic cage[8] through which pass the esophagus, trachea, major vessels of the neck and upper limb, vagus and phrenic nerves, the most inferior components of the brachial plexus, the sympathetic trunk and thoracic duct, with the dome of the pleura pushing up from below. Some have described several but not all the contents of the thoracic inlet as the "4 birds," namely the esophagoose, va-goose, azy-goose, and thoracic "duck."

Mechanically, the thoracic inlet is important because of its soft tissue influence on the sternum, ribs, clavicles,

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cervical and thoracic spine, scapulae, and upper extremities. Systemically, it is important because of its relationship to the major lymphatic ducts in the anterior chest wall (through which the lymphatic system for the whole body drains into venous circulation), as well as its role in pulmonary function and neural activity, particularly of the brachial plexus.[8] The borders of the thoracic inlet are the manubrium and the medial aspect of clavicles and first ribs, anteriorly; the vertebral body of T1, posteriorly (ie, vertebromanubrial region[1]). Because of its complex fascial network and the influence of postural factors (ie, forward head/rounded shoulders), the thoracic inlet is an area that is prone to developing somatic impairment. For this reason, it is the first of the soft tissue areas that is addressed in the management of patients with scapulothoracic impairment.

The thoracic inlet responds well to both direct and indirect treatment methods using a three-dimensional approach. In the presence of highly reactive tissues or when working with anxious patients, the indirect approach is preferred. However, when there is adaptive shortening with low tissue reactivity, direct technique is the treatment of choice. An extremely useful way of employing either method is with a palpation technique developed by Peter Fabian, PT known as the "4 Ms" procedure. In order of application, the first M stands for mold, the second for meld, the third for monitor, and the fourth for move.

With the patient positioned comfortably in supine and the therapist sitting at the head of the table, the therapist places one hand lightly over the sternal angle with the fingers and thumb spread over the upper ribs and sternoclavicular joints. The other hand is placed under the patient's upper thoracic region encompassing the cervicothoracic junction (Figure 5-1). Molding is the process of conforming one's hands to the patient's unique anatomic structure (ie, "anatomy to anatomy"). Melding is the process of "tuning in" to the tissues being palpated and involves the appreciation of contour, texture, tone, moisture, temperature, etc. It is a deeper form of palpation that involves sensitivity to function (ie, "physiology to physiology"). Monitoring separates a direct from an indirect technique. During the performance of an indirect approach, the therapist is sensing what osteopathic physicians refer to as "inherent tissue motion" or the "preferred tissue pattern." Inherent tissue motion or rhythm is a compilation of all the ongoing physiologic motions in the body that affect the neuromusculoskeletal system and produce fine movements. These include cardiovascular, respiratory, neuroreflexive, and craniosacral movements,[8] all occurring simultaneously. The therapist is simply monitoring these micromovements and noticing their combined direction, amplitude, and velocity.

The 2-hand palpation style enables the therapist to appreciate motion in 3 dimensions. This theoretically enlarges the receptive field of motion to all tissues between the therapist's hands due to the contiguity of myofascial structures. Monitoring tissue status in a direct technique is quite different. Here, the therapist performs a shearing motion with the top hand (the bottom hand anchors the tissues from below) in a multidirectional manner. However, the hand is not allowed to move over the tissues but rather "drags" the soft tissues with it until a barrier is reached. Ellis and Johnson[30] developed the "shear-clock" method of assessing superficial tissue motion. It involves tissue shear in 6 planes corresponding to the 12 hours of a clock (eg, 12 to 6, 1 to 7, 2 to 8, 3 to 9, 4 to 10, and 5 to 11). The therapist should not apply massage lotion or lubricant for this assessment lest motion over the skin occurs. The purpose of the "shear-clock" assessment is to identify restrictive motion barriers in the thoracic inlet and chest wall tissues that require direct myofascial mobilization.

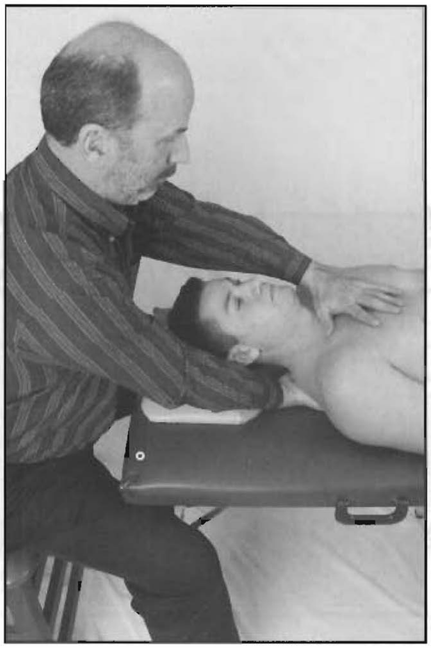

Figure 5-1. 4 Ms thoracic inlet release.

Moving is the final step in the 4 Ms procedure. To perform an indirect myofascial release technique, the therapist follows "ease" in the tissues. This induces neuromuscular relaxation and symptom reduction. The therapist is merely taking the tissues where they are most comfortable and thus enabling them to unwind. A useful analogy is to consider a stick floating on a stream as it meanders through the forest. Just as the stick follows the stream's current, the therapist follows the "current" of inherent tissue motion. Applying a slight degree of manual compression to the thoracic inlet will facilitate inherent tissue motion and enhance the efficacy of the indirect myofascial release.

Direct myofascial release techniques can be likened to a fullback on the football field. Unlike the halfback, who is quick and agile, looking for the openings on the field (ie, indirect approach), the fullback is strong and formidable, looking to run directly at anyone who dares to get in his way! So it is with direct technique. It is a means of releasing myofascial restrictions in the presence of contracture. It is used when stiffness is dominant and the tissue reactivity is

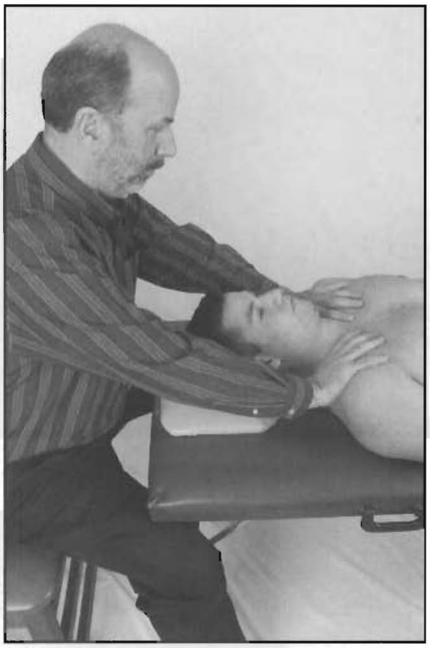

Figure 5-2. Thoracic inlet necklace release.

low. Another option is to perform the "necklace" technique (Figure 5-2), as described by Greenman,[8] in either a direct or indirect fashion as discussed previously. The author routinely performs these 3-dimensional thoracic inlet releases in the sitting position as well.

The author of this text recently developed the acronym SLOW to enable the therapist to conceptualize the main effects of soft tissue mobilization, whereby S is for soften and smooth, L is for loosen and lengthen, O is for open, and W is for warm. Regardless of the specific soft tissue technique utilized, the goal is always the same: to soften what is hard, to loosen what is tight, to open what is closed (eg, spaces, tunnels) and to warm what is cool (eg, areas affected by chronic sympathetic facilitation). In so doing, the therapist restores function and relieves painful symptoms emanating from dysfunctional neuromusuloskeletal tissues.

Anterior Chest Wall Fascial Techniques

The next group of soft tissue procedures are referred to as direct fascial techniques.[31] These soft tissue mobilizations have several physiologic effects, including enhanced circulation (eg, arterial, venous, and lymphatic), increased production of glycosaminoglycans and consequently increased hydration of the tissues, loosening of connective tissue adhesions (eg, cross-links), reduction of muscle hypertonicity, and viscoelastic elongation (ie, "creep"). Direct fascial techniques utilize a variety of manual contacts (eg, thumbs, palms, knuckles, forearms, finger pads, elbows) and apply them to intermuscular septa, musculotendinous junctions, tenoperiosteal junctions, postsurgical and post-traumatic

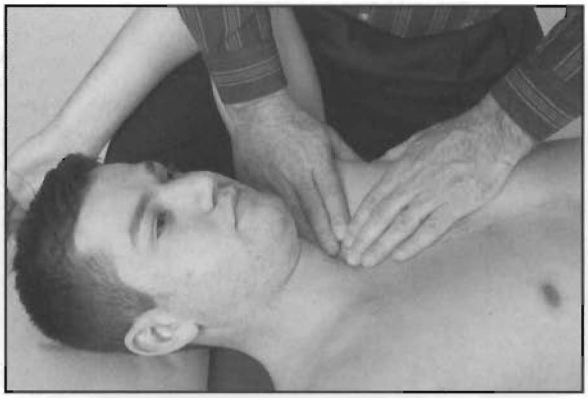

Figure 5-3. Muscle play anterior chest wall.

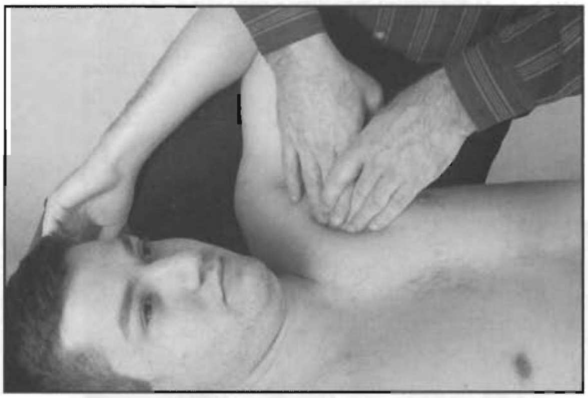

Figure 5-4. Strumming anterior chest wall.

scar tissue, fascial attachments, etc. Some of the names given to these techniques include "strumming," "sculpting," structural integration (Rolfing), connective tissue massage, myofascial manipulation, "ironing," deep tissue massage, soft tissue mobilization, etc.

There are several principles that guide the use of these effective soft tissue procedures, including the following:

- Commence each technique with the soft tissues in a loose or slackened state.
- Apply manual contacts in a direction perpendicular to muscle, tendon, and collagenous fiber orientation whenever possible.
- Combine all techniques with gentle oscillations, which are better received by the body than static pressure.
- Progress each technique into tissue length to accomplish full range of motion of the treated structures.
- The therapist must use a small amount of lubricant when employing these techniques. The author recommends Deep Prep II (Smith & Nephew, Germantown, WI).
- The contraindications listed in Chapter 3 apply to these direct treatment procedures as well.

The first 2 techniques illustrated are referred to as "muscle play" and "strumming." Muscle play (Figure 5-3) stretches the myofascial tissues of the anterior chest wall over the costal cage, whereas strumming (Figure 5-4) iden-

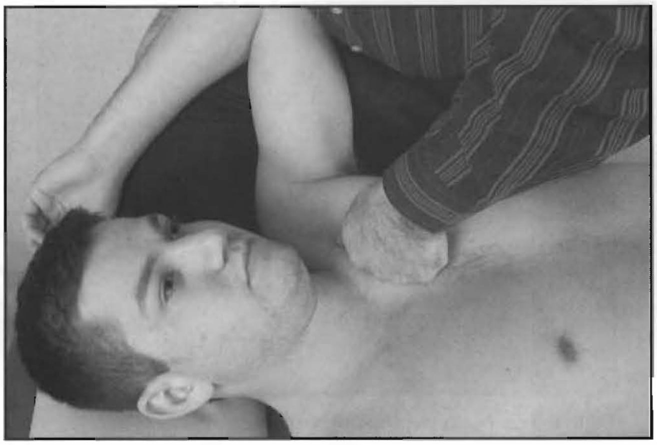

Figure 5-5. Steamroller anterior chest wall.

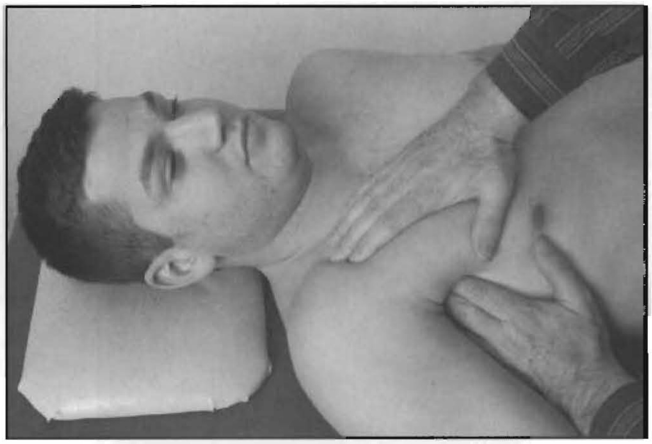

Figure 5-6. Pectoralis major and minor release.

Figure 5-7a. Chest wall strumming under stretch.

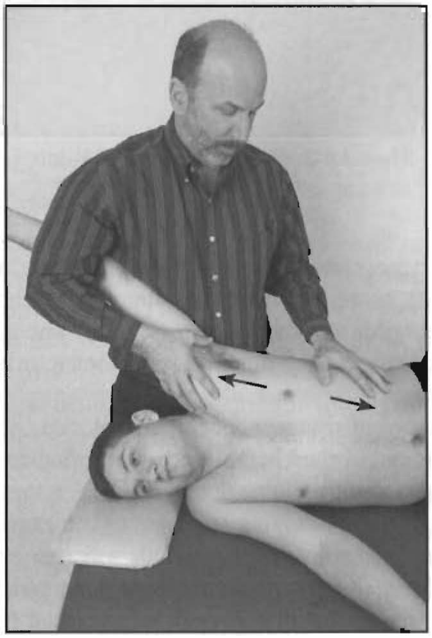

Figure 5-7b. Chest wall myofascial release under stretch.

tifies and treats localized regions of dysfunction, including taut bands. The muscle play contact consists of a "triangle" formed by the 2 thumbs and index fingers; strumming is accomplished by joining the third, fourth, and fifth fingers of both hands with the index fingers crossed on top and the thumbs out of the way.

The "steamroller" leads with the thumb and is followed by the proximal phalanges of the second through fifth fingers (Figure 5-5). In patients with high pain tolerance, the proximal interphalangeal (PIP) joints may follow the thumb for deeper tissue penetration. The steamroller is used to accomplish deep tissue massage under the clavicle and between the ribs.

The pectoralis major/minor fascial technique is a more aggressive maneuver that requires a willing patient who is able to tolerate a degree of mild discomfort. The therapist probes between the two pectoral muscles with one hand while drawing the major over the probing hand with the other. Once in the fascial plane between the muscles, the therapist scours the area for tight and thickened tissue and then attempts to free and soften through direct digital pressure with oscillations. Applying digital pressure in the expiratory phase of breathing allows for greater penetration (Figure 5-6).

The final combined direct fascial/myofascial release technique is one of the author's favorites. With the patient in the side lying position, the therapist carefully wedges his or her body between the patient's abducted/externally rotated upper limb and the patient's trunk (Figures 5-7a and 5-7b). Care must be taken to not cause impingement of the glenohumeral joint nor excessive anterior translation of the humeral head. The patient's only discomfort should be a stretching sensation across the anterior chest wall. In this position of pectoral elongation, the therapist's hands are

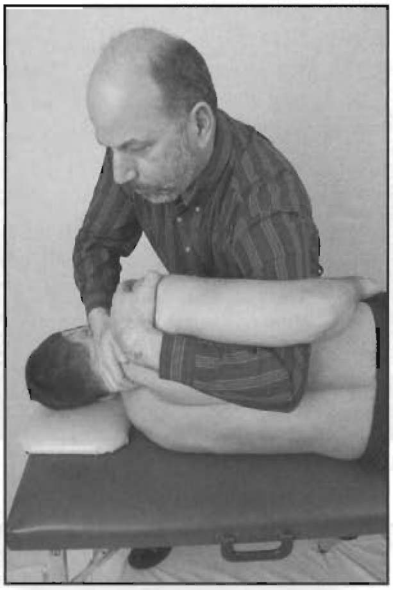

Figure 5-8a. Phase 1 scapular fascial technique.

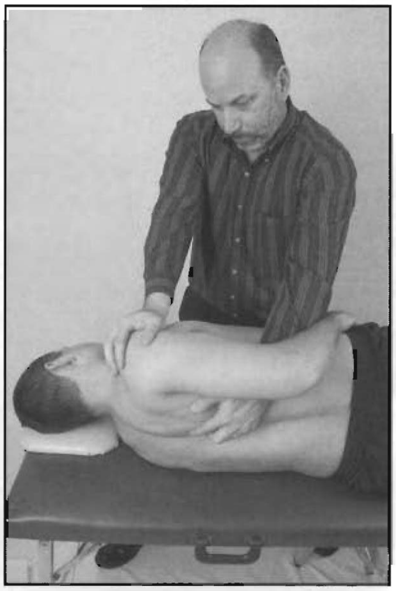

Figure 5-8b. Phase 2 scapular fascial technique.

free to perform muscle play, strumming, steamrolling, myofascial release (ie, manual stretching of myofascial tissues), postisometric stretching (ie, hold-relax), etc.

Scapular Fascial Techniques

Once the anterior and lateral structures of the chest wall are rendered more supple and mobile, the therapist can proceed to the myofascial attachments of the scapulae. The most common restrictions in scapular motion seen clinically are depression, adduction, upward rotation, and posterior tilting (ie, superior aspect of the scapula moves posterior and inferior as the inferior angle moves anterior and superior). These restrictions are due to the pull of tight postural muscles in conjunction with weakness of the lower scapular stabilizers. Consequently, the soft tissue component of these impairments must be managed in order to enable the scapulae to assume a neutral position on the costal cage.

To accomplish this, the direct fascial technique known as "framing the scapula," as taught by Cantu and Grodin,[32] will be employed. For phase 1, the patient is positioned in side lying as the therapist engages the restrictive motion barrier in scapular adduction/depression by grasping the patient's shoulder with the caudal-most hand. At the same time, the cranial-most hand performs a "raking" technique to the levator scapula and upper trapezius muscles (Figure 5-8a). As the tissues relax, the therapist takes up the slack toward increased depression, adduction, and posterior scapular tilt. (Note: The amount of each will vary from patient to patient and must therefore be managed on an individual basis.)

Phase 2 of "framing the scapula" involves switching hand position so that the cranial-most hand provides the motion against the barrier while the caudal-most hand performs the fascial technique (Figure 5-8b). The mobilizing hand

Figure 5-9. Subscapularis and serratus anterior release.

proceeds down the vertebral border of the scapula to the inferior angle, working the soft tissues, while simultaneously mobilizing the scapula into further depression, adduction, upward rotation, and posterior tilt. If possible, the therapist should consider an often forgotten movement of the scapula, which is rotation about the vertical Y axis through the acromioclavicular joint.[33] In patients with forward head/rounded shoulders posture (ie, Janda's upper crossed syndrome[10]), the scapulae tend to internally rotate as well as abduct. Consequently, scapular mobilization should incorporate scapular external rotation, along with adduction, into the intervention described above. Postisometric relaxation can be added to enhance this multiplanar mobilization of the scapula. This combined myofascial/scapular mobilization enables the scapula to function normally by extricating it from its previously abnormal positions of elevation, abduction, downward rotation, anterior tilt, and internal rotation.

There are 2 additional scapular techniques that are quite useful, especially related to shoulder impairment. The first is a direct fascial technique of the subscapularis muscle (Figure 5-9). The subscapularis is often a key component in "frozen

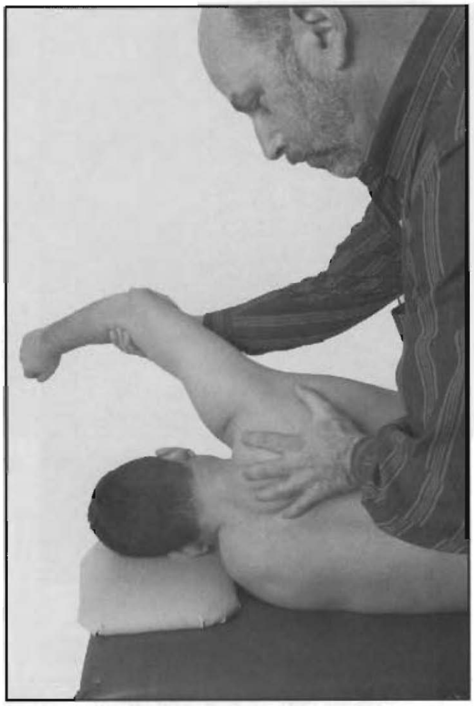

Figure 5-10. Scapulohumeral rhythm training.

shoulder" and its treatment often produces dramatic results. The therapist adducts and internally totates the patient's shoulder in order to gain access onto the ventral surface of the scapula along the posterior axillary wall. Digital pressure is applied to areas of increased density in order to release, soften, and inhibit muscle tone and tightness. As softening occurs, the shoulder is abducted and externally rotated; the direct fascial technique is repeated under stretch. As with all soft tissue procedures, the application of a hold-relax technique is a useful adjunctive tool in restoring myofascial length. While applying the SLOW acronym (discussed previously) to the subscapularis muscle, the interface of the subscapularis/serratus anterior on the ventral surface of the scapula should be inspected and, if necessary, treated with soft tissue mobilization (see Figure 5-9).

The second technique is of particular benefit to patients with shoulder impingement related to poor scapulohumeral rhythm. The patient is in the side-lying position with the upper limb at the side. The therapist grasps the scapula with one hand and the elbow with the other (Figure 5-10). Active assisted shoulder abduction is performed while the scapula is passively upwardly rotated and depressed. This provides clearance of the suprahumeral tissues under the coracoacromial arch and also gives the patient the sensation of normal scapulohumeral rhythm. The movement is progressed to an active effort, with the patient incorporating a conscious depression of the scapula as the arm is elevated past 90 degrees. This can then be practiced in standing with the assistance of a mirror to ensure that the scapula is not "hiked up" by the levator scapula and upper trapezius during shoulder elevation.[9]

Figure 5-11. Superficial fascial assessment.

Figure 5-12. Myofascial release posterior thoracic region.

Superficial Posterior Tissue Release

Myofascial release of the skin, subcutaneous tissue, and superficial fascia of the posterior scapulothoracic region is indicated when the examination reveals impaired mobility. The shear-clock method described previously is an excellent tool for both the diagnosis and management of superficial tissue restriction (Figure 5-11). Once found, the therapist uses the "hold one, move one" treatment by anchoring the tissues with one hand and applying a sustained tensional stretch with the other in the specific direction(s) of the restriction (Figure 5-12). As viscoelastic lengthening and plastic deformation occur, the therapist will feel a release of tension and "follow behind" the release until a new barrier is encountered. This process continues for several minutes until the restrictions have been satisfactorily managed. Whereas indirect technique looks for ease in the tissues, direct myofascial release seeks tissue "bind." Following the successful release of the principal barrier, new areas of bind are sought after and released accordingly.

Figure 5-13. CPR technique of erector spinae.

Figure 5-14. Forearm sweeping technique.

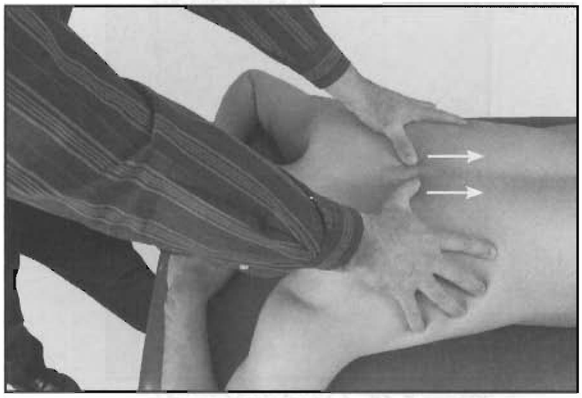

Figure 5-15a. Paraspinal sweep.

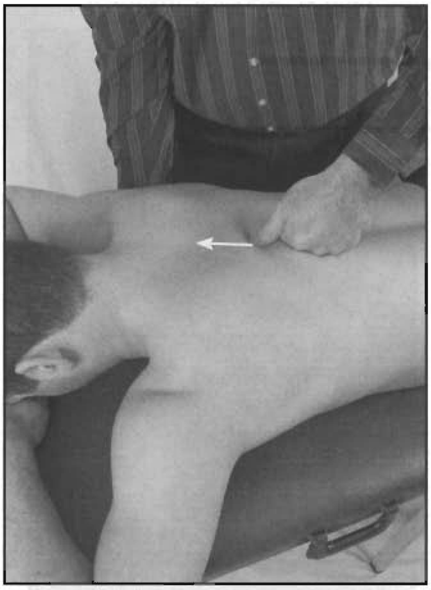

Figure 5-15b. Steamroller of transversospinalis.

Erector Spinae Fascial Technique

The myofascial treatment of the erector spinae muscles is accomplished with a variety of techniques, including strumming and muscle play. Another useful treatment method is termed the CPR technique because of how it resembles the manual method used during cardiopulmonary resuscitation (Figure 5-13).

The heel of the therapist's hand is placed in the medial groove of the longissimus thoracis and continues in a lateral direction, imparting a perpendicular stretch on all aspects of the erector spinae. A small amount of Deep Prep II is used to lessen skin friction. The therapist is encouraged to begin at the AGR and proceed from there. To reduce resting tension in the erector spinae, a neuromuscular technique,[34] referred to as forearm sweeping (Figure 5-14), is employed. Unlike direct fascial techniques, which are applied perpendicular to the orientation of the myofascial tissues, forearm sweeping, a form of stripping[31] massage, is applied parallel to the muscle fibers for the purpose of neuromuscular relaxation.

Transversospinalis Fascial Techniques

There are several techniques that address the deep spinal musculature found between the spinous and transverse processes (ie, the medial groove of longissimus). These techniques apply manipulative contacts in a direction that is caudal or cranial and thus at an oblique angle or at times perpendicular to the fiber orientation of the transversospinalis muscles (eg, semispinalis, multifidus, and rotatores). Such techniques include the paraspinal sweep, steamroller, and bilateral thumb oscillations (Figures 5-15a to 5-15c).

These fascial techniques are useful in detecting and treating hypertonicity and myofascial trigger points of the transversospinalis muscles. These areas of myofascial

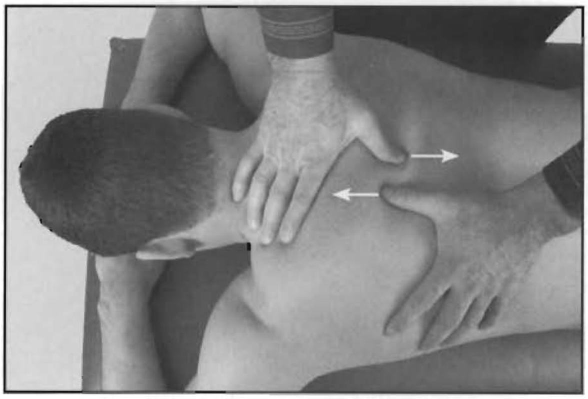

Figure 5-15c. Bilateral thumb oscillations.

Figure 5-16. Specific compression therapy.

Figure 5-17. Lateral myofascial release in the angry cat position.

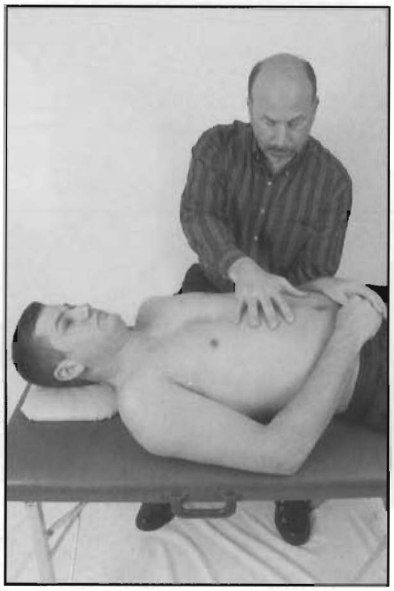

Figure 5-18. 4 Ms thoracic outlet release.

dysfunction feel like speed bumps or moguls. Greenman[8] refers to a speed bump in these deep fourth layer muscles as the "tootsie roll" sign. When these dysfunctional areas are identified, the thumb is used to apply PA pressure in the form of myotherapy or specific compression[31] (Figure 5-16), as well as circular friction.[31] Once the hypertonicity or myofascial trigger point is eradicated, the therapist continues to explore the medial groove of longissimus, in either a caudal or cranial direction, for other areas of myofascial dysfunction.

Once soft tissue treatment is rendered in prone, the previous techniques of the posterior scapulothoracic region can be applied in the "angry cat" position (quadruped on elbows with back arched upward) so that myofascial extensibility is restored throughout the full range of motion. In addition to enhancing thoracic flexion in the angry cat position, the therapist should also restore scapular abduction, in the flexed position, with the soft tissue techniques described previously, including myofascial release in a medial to lateral direction (Figure 5-17).

Respiratory Diaphragm Release (Thoracic Outlet)

The last of the connective tissue techniques is a 3-dimensional myofascial release of the respiratory diaphragm (Figure 5-18). The anatomic borders of the thoracic outlet (the caudal opening of the thoracic cage) consist of T12 posteriorly, the seventh through tenth costal cartilages anteriorly, and the 11th and 12th ribs laterally. The thoracic outlet is closed by the diaphragm, which separates the thoracic and abdominal cavities. As shown, this release involves both hands, with one hand placed under the thoracolumbar junction and the other hand placed over the respiratory diaphragm. As with the thoracic inlet release described earlier, the 4 Ms palpation technique is a useful way of performing either a direct or indirect technique to the diaphragm and its many attachments (ie, crural attachment to L1-L3, lower 6 ribs, psoas, and quadratus lumborum muscles). Mobility of this area is important for pulmonary, vascular, and musculoskeletal physiology; it is a common area for the development of myofascial impairment. As with thoracic inlet release, the thoracic outlet can also be treated while the patient sits, where the therapist has optimal control of his or her manual contacts.

To initiate an indirect release, slight compression of the patient's abdomen, between the therapist's hands, is applied.[35] The therapist's hands are then "directed" by inherent tissue motion into a succession of myofascial releases, which are complete when the tissues are supple and free of restriction.

Active Release Techniques

Also known as ART, active release techniques[31] consist of a manual, soft tissue diagnostic and treatment system developed by Michael Leahy, DC. They combine simultaneous movement with specific and deep neuromuscular techniques. Once soft tissue lesions are identified through observation of motion and palpation of the affected tissue, they are then treated through a combination of active or passive range of motion in conjunction with specific contact manipulation. Tissues are typically taken from a shortened to a fully lengthened position while the therapist's contact tension is maintained longitudinally along the tissue's fibers. Treatment response is often immediate and includes changes in tissue tension, texture, movement, and function. ART has made its greatest impact in the treatment of sports-related overuse injuries, cumulative trauma disorders, and peripheral nerve entrapments.

6

Manipulation of the Thoracic Spine and Ribs

There are several components to effective manipulative technique; however, the skilled manual therapist must pay particular attention to three. They are localization, control, and balance. If the T3,4 segment is restricted in flexion, the therapist must direct the greatest force at this motion segment and not elsewhere. To ensure that the technique is efficient and effective, the therapist must have maximal control of all points of leverage leading to the restricted area. Maximal, but tension-free, control of the patient's body is also necessary for optimal balance of both the therapist and patient. When these 3 factors are integrated and the applied force to the impaired joint is as gentle as possible, but as strong as necessary, the outcome is generally successful for the patient and satisfying for the therapist.

A skillful manipulation—whether it be a grade[36] 1, 2, 3, 4, or 5—is characterized as graceful, gentle, and purposeful. As for the patient, he or she must be relaxed, comfortable, and confident in the therapist's ability to relieve symptoms, as well as enhance healing and wellness. The main effect of manipulation is physiologic, but enhancement through the placebo effect is of great benefit to the patient. In general, manipulation, especially on the small joints of the spine and ribs, should be of short duration (ie, 30 to 60 seconds) lest the sensitive articular tissues react adversely. It is always wise to begin gently so that the patient's response to passive motion has a chance to be accurately assessed. Both PAIVMs and PPIVMs can be easily transformed into mobilization/manipulation techniques.

In February 2007, the American Academy of Orthopaedic Manual Physical Therapists formed a task force to standardize manual therapy terminology, beginning with manipulation.[37] As a result, the following recommendations were made for describing a manipulative technique:

- The rate of force application.
- Its location in the range of available motion.
- The direction of the force.
- The intended target of the force application.
- The resulting movement relative to the structure mobilized and the structure stabilized.
- The position of the patient.

Thoracic Flexion

The difference between PAIVMs/PPIVMs and manipulative technique is that the former involve the collection of data about motion (ie, quality, quantity, end-feel, and tissue reactivity), whereas spinal mobilization/manipulation is used to, "achieve maximal pain-free motion of the musculoskeletal system within postural balance."[38] When performing manipulation for the purpose of pain modulation (grades 1 and 2), the spinal contact is used for localization purposes and like PPIVMs is placed in the interspinous space. However, when manipulating to correct somatic impairment (grades 3 and 4), the spinal contact either provides a "block" of the inferior vertebrae ("hold one, move one" technique) or assists the manipulation by gliding the inferior vertebra in the direction opposite the roll of the superior component (roll-glide technique).

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Figure 6-1. Flexion mobilization of T1-T4.

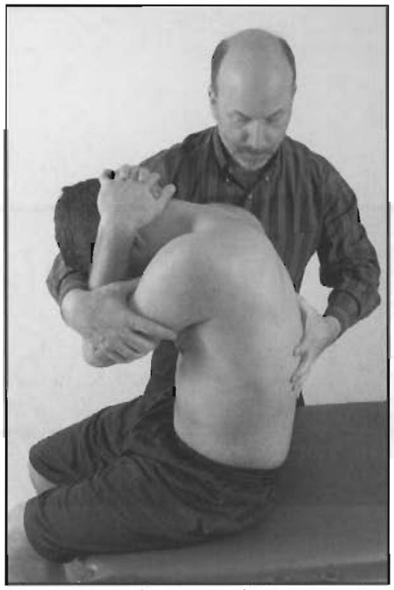

Figure 6-2. Flexion mobilization of T5-T12.

Improving spinal flexion from T1 to T4 is shown in sitting (Figures 6-1) but can easily be done in side lying as well. In order to mobilize T3,4 in flexion, the therapist induces flexion of T3 through the head-neck while the thumb or thenar eminence of the other hand prevents the SP of T4 from moving superiorly. This is achieved by blocking it at its superior aspect. Maitland's grades 1 to 4 are then applied in accordance with tissue reactivity.[36] Grade 1 manipulation is a small amplitude movement performed at the beginning of the accessory range. Grade 2 is a large amplitude motion performed within the range but not reaching its limit (at the beginning of the range it is expressed as 2– and deep into the range but not at the limit it is a 2+). Grade 3 is a large amplitude movement, similar to grade 2, but from mid to end-range (3– is a gentle "nudge" at end-range, whereas 3+ is a vigorous "knock"). Grade 4 is a small amplitude movement at the end of range, which can also be described as 4+ or 4–, depending on its vigor, as described for grade 3. Grade 1 manipulation is useful when tissue reactivity is high, grade 2 and 3 manipulations are used in the presence of moderate reactivity, and grade 4 techniques are applied to low reactive, stiffness-dominant tissues. Unlike grades 1 and 2, grades 3 and 4 involve passive movements into tissue resistance against the restrictive barrier. From a manipulative perspective, it is important to understand that grade 1 and 2 techniques require monitoring only for accurate localization (ie, at the interspinous space). This is because grade 1 and 2 techniques never engage the motion barrier and consequently stabilization of the inferior vertebra is not necessary. Conversely, when performing grade 3 and 4 hold one, move one mobilization/manipulation procedures, a block of the inferior vertebra is required. Without this necessary stabilization of the inferior vertebra, mobilization of the motion segment via the superior vertebra is rendered ineffective.

Flexion manipulation for the T5 to T12 region is similar to the upper thoracic spine with the obvious difference being the use of the patient's arms to induce flexion in this region of the spine (Figures 6-2). All flexion manipulations can be enhanced by adding a translational component via the trunk to the inferior vertebra in an anteroposterior (AP) direction, while the superior vertebra is flexed up and forward (ie, roll-glide).

Warmerdam[39] emphasizes the relationship between joint mobility and muscle strength (ie, the arthrokinetic reflex). Clinically, the serratus anterior muscle requires normal flexion from approximately T3 to T6 in order to function optimally.[9] Impaired mobility will weaken the serratus, while mobilization will restore it to its normal potential.

Thoracic Extension

The recovery of thoracic extension, especially from T6 to T12, is one of the most important applications of spinal manual therapy. There are many factors that contribute to this pattern of impairment, but perhaps the most common is poor sitting posture related to spending hours at the computer.

The manipulation of extension from T1 to T4 is performed on the seated patient. As noted with flexion above, grade 1 and 2 techniques require a monitoring contact in the interspinous space to ensure proper localization, whereas grade 3 and 4 procedures require a stabilizing or blocking force on the inferior vertebra. To perform an extension manipulation at the T2,3 segment, the therapist lightly

Figure 6-3. Extension mobilization of T1,2 and T2,3.

Figure 6-4. Extension mobilization of T3,4 and T4,5.

Figure 6-5. Segmental extensor strength training of T1-T4.

cradles the patient's head and induces extension down to T2 by gliding the head-neck dorsally (Figures 6-3). Meanwhile, the other hand has the option of either preventing the T3 SP from moving inferiorly on T4 for a grade 3 or 4 technique (hold one, move one) or enhancing the extension mobilization by performing a PA glide on the SP of T3 (roll-glide). Blocking the SP of T3 with either the thumb or thenar eminence at its inferior aspect is a mechanically simpler technique than performing a manipulative roll-glide, but the latter is the treatment of choice once the requisite skill is developed. It is this author's experience that T3,4 and T4,5 extension mobilization/manipulation requires an extension tilt or over-turning motion of the head-neck region in addition to a dorsal glide (Figure 6-4). Postmenopausal women who develop a matron's or dowager's deformity may derive benefit from gentle extension work in the T1-T4 region. The cervicothoracic region may also benefit because of its tendency to flex. Once extension is restored to the T1-T4 region, the segmental extensors (ie, the multifidus and semispinalis thoracis) can be strengthened with isometric training. This is achieved by localizing to the desired level and resisting the patient's attempt to push posteriorly for 3 to 5 seconds, 3 to 5 times (Figure 6-5).

Thrust Mobilization/Manipulation (Maitland Grade 5) for C7 to T4 Extension

In the absence of contraindications (see Chapter 3) and in the presence of low reactive, minor, end-range restrictions (between the elastic and anatomic barriers of motion), a grade 5 thrust manipulation to one or more of the C7 to T4 segments (eg, C7,T1; T1,2; T2,3; T3,4; and T4,5) may be utilized as follows:

1. The patient is seated with his or her fingers interlocked behind the cervical region with the elbows apart.
2. The therapist places a rolled towel between his or her sternum and the patient such that the top of the towel roll is located at the desired level of the segment to be thrusted. For example, if T1,2 is the desired segment to be thrusted, the top of the towel roll should be placed at the level of the T2 SP.
3. The therapist then weaves his or her hands through the patient's elbows such that the therapist's hands are placed over the hands of the patient (Figure 6-6).

Figure 6-6. T1-T4 thrust technique.

Figure 6-7a. Extension mobilization T5-T12 sitting.

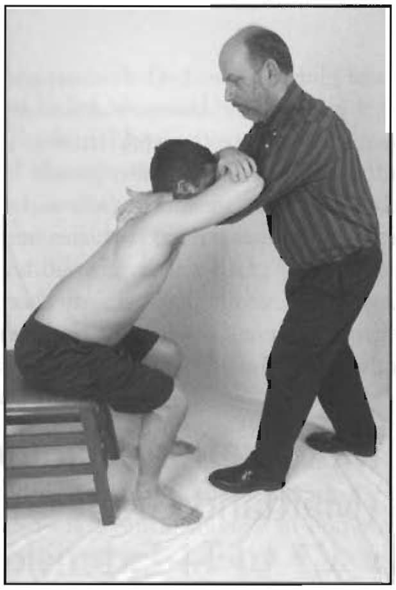

Figure 6-7b. The "weave" extension mobilization T5-T12.

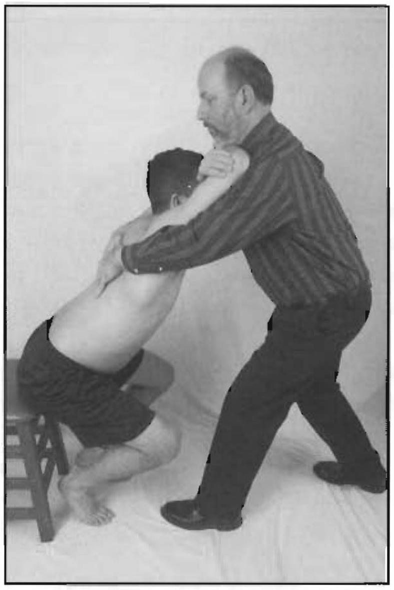

Figure 6-7c. The "hug" extension mobilization T5-T12.

4. Just prior to the thrust, the patient is brought through 2 to 3 cycles of alternating upper thoracic flexion and extension to fine-tune the timing of the thrust and to ensure patient relaxation.
5. When all the slack is taken up through the therapist's arms and hands in an upward direction and via the sternum in an anterior direction, the high-velocity/low-amplitude thrust is performed through the sternum. To achieve further neuromuscular relaxation for enhanced efficacy, the thrust can be performed following a deep breath on exhalation.

This traction/extension maneuver is often associated with an audible sound consistent with the sudden cavitation of one or more spinal joints. The patient must be relaxed; the thrust must be quick, of short amplitude, and not beyond the anatomic barrier of the joint. The grade 5 thrust is nothing more than range of motion at high speed against a minor end-range restriction.

There are several ways of improving extension in the mid/lower thoracic region. Increasing extension from T6 to T12 has been shown to enhance strength of the lower trapezius muscles in normal subjects.[40,41] Four methods, 3 in the sitting position and 1 in quadruped, are illustrated (Figures 6-7a to 6-7d). Although a hold one, move one technique is feasible here, in all 4 procedures illustrated, a roll-glide technique is being performed (ie, the inferior SP of the restricted

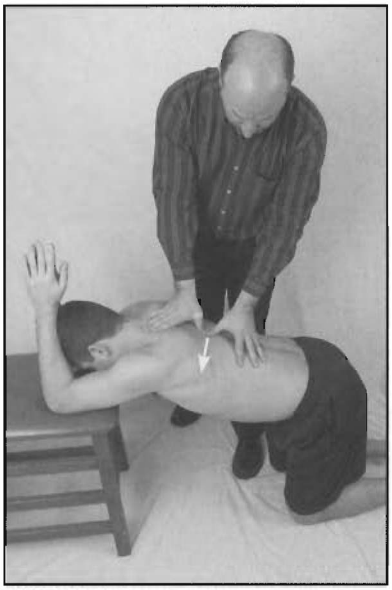

Figure 6-7d. Prayer position extension mobilization T5-T12.

Figure 6-8. T5-T12 thrust technique.

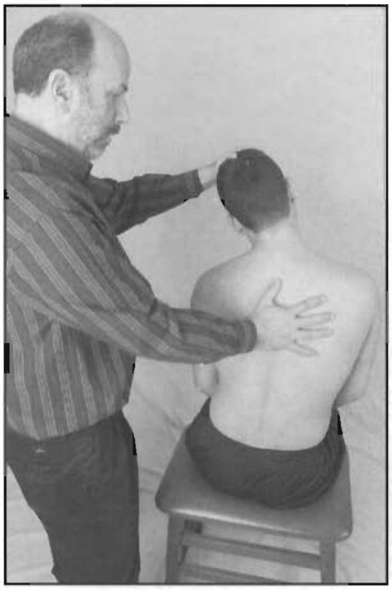

Figure 6-9. Side bending mobilization T1-T4.

motion segment is translated in an anterior direction along the Z axis as the superior vertebra is rotated into physiologic extension about the X axis); depending on the reactivity present, the appropriate Maitland grade is selected. To enhance the treatment's effectiveness, the patient may be asked to participate in the effort by actively extending the thoracic spine during grade 4 mobilizations. This active-assisted technique has been referred to as "mobilization with movement" by Mulligan[42] and "functional mobilization" and "physiologically enhanced mobilization" by Ellis and Johnson.[30] Its advantages include self-mobilization, lower trapezius and multifidus activation, and sensorimotor learning. Improving mid/lower thoracic extension not only benefits the thoracic spine directly, but is considered beneficial in managing posturally related cervical pain, interscapular pain, and headache,[43] as well as shoulder impingement[14-18] and temporomandibular disorders.[44]

The traction/extension thrust technique for T1 to T4 described above can be easily adapted for the T5 to T12 region. As illustrated (Figure 6-8), the patient and therapist's hand placement is different; the towel roll is placed lower down in the thoracic spine. Otherwise, it is similar and provides the therapist with an additional tool with which to achieve full pain-free range of motion in the lower thoracic spine.

Thoracic Side Bending

The mobilization of thoracic side bending is first illustrated for the upper thoracic region (Figures 6-9) and then for the mid/lower thoracic spine (Figure 6-10). For all side-bending manipulations, the therapist can start with the traditional hold one, move one approach and progress to the roll-glide technique. For example, a grade 3 or 4 T4,5 side-bending left maneuver with a hold one, move one approach involves mobilizing T4 to the left while blocking T5 on the left side of the SP. A roll-glide technique of T4,5 side bending left involves rotating T4 to the left on a Z axis while simultaneously translating the T5 SP to the right with the thumb or thenar contact. Although a transverse pressure at the apex of the SP from the neutral position will normally induce vertebral rotation, when it is applied at the base of the SP in conjunction with a lateral bending motion, it induces enhanced side bending.

From T5 to T12, the therapist has 2 options. One is referred to as the "push" technique (Figure 6-10a), while the other is the "pull" technique (Figure 6-10b). In both

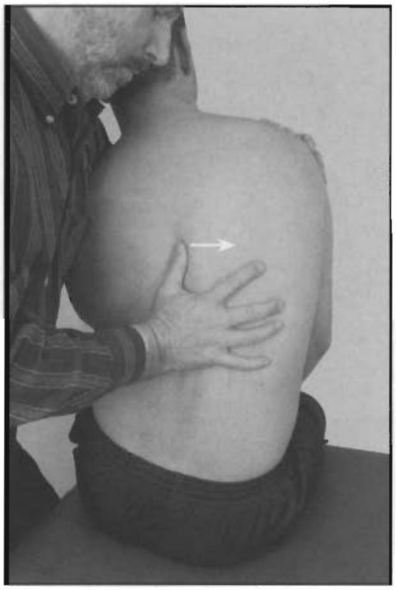

Figure 6-10a. Side bending push mobilization T5-T12.

Figure 6-10b. Side bending pull mobilization T5-T12.

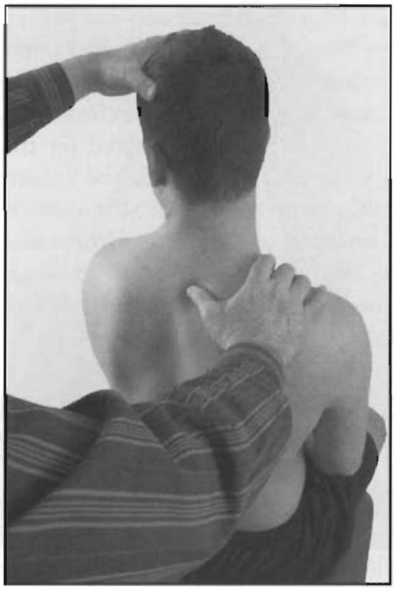

Figure 6-11. Rotation left mobilization T1-T4.

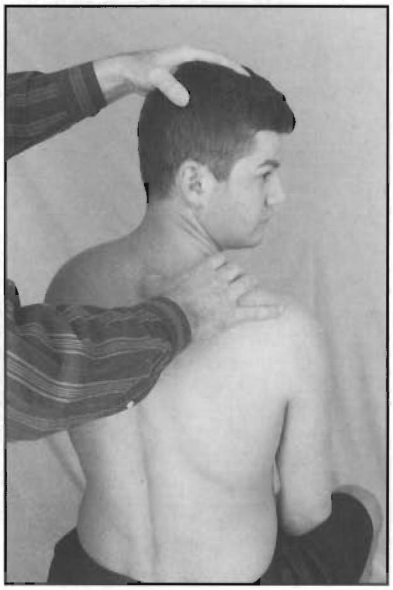

Figure 6-12. Pre-positioning for left rotation mobilization T1-T4.

procedures, the roll-glide manipulation is superior to the traditional hold one, move one approach.

Thoracic Rotation

As with the other thoracic manipulations/mobilizations covered previously, the difference between PPIVMs and manipulative intervention is found in the purpose behind the technique and the slight modification in hand position. Thoracic rotation manipulation is achieved via the hold one, move one approach in which rotation of the superior vertebra is induced with one hand while the other hand blocks the inferior vertebra (grades 3 and 4). For example, a left rotation manipulation at T2,3 involves inducing left rotation of T2 through the head-neck region as the therapist prevents T3 rotation left below (Figures 6-11). This blocking of T3 is achieved by placing the thumb or hypothenar contact against the SP of T3 on its left lateral aspect.

For rotational techniques, the concept of pre-positioning the segment is quite useful. For example, prior to a grade 3 or 4 T2,3 left rotation manipulation, the therapist rotates the patient's head-neck to the right (Figure 6-12). The therapist then places his or her right thumb against the SP of T3 on the right. The head-neck region is then rotated to the left with T3 fixed in a right rotated

Figure 6-13a. Pre-positioning for rotation left mobilization T5-T12.

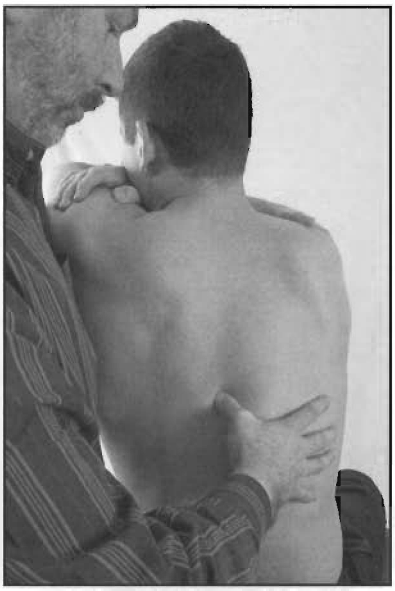

Figure 6-13b. Rotation left mobilization T5-T12.

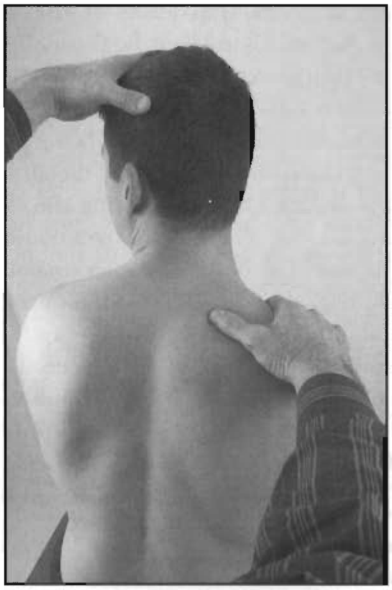

Figure 6-14. Mobilization of right ribs 1 to 4.

position. The advantage of pre-positioning is that the barrier is reached sooner and the surrounding joints are subjected to less stress. The same principles (ie, blocking, pre-positioning, etc) are applied to the T5 to T12 region; however, the trunk replaces the head-neck region as the lever arm (Figures 6-13a to 6-13b).

As emphasized earlier, all spinal manipulations, including grades[36] 1 through 5, start in the AGR and proceed from there. The major motion losses (50% or more) are treated first; the minor motion losses (less than 50%) are treated last.

Mobilization/Manipulation of the Rib Cage

The osteopathic diagnosis and treatment of respiratory and structural rib dysfunctions[8,9] is beyond the scope of this introductory textbook. The objective of this section is to provide the manual therapist with one useful technique for each of the 12 ribs. These techniques in conjunction with soft tissue/spinal manipulative procedures and therapeutic exercises will ensure that patients with scapulothoracic impairment receive a basic yet comprehensive approach to their condition.

To manipulate restriction of the right first rib, the therapist rotates the patient's head-neck region to the left while palpating the right first rib just lateral to the costotransverse joint with the right thumb. When motion arrives at the first rib, a PA-graded mobilization is performed with the thumb contact (fingers are draped over the shawl area for counterbalance). With practice, both hands move simultaneously to enhance the effectiveness of the procedure (Figure 6-14). As with the spine, the choice of which grade to use depends upon the tissue reactivity present.

The second, third, and fourth ribs are mobilized using the same procedure as for the first rib (ie, a graded PA pressure on the rib tubercle with simultaneous head-neck rotation).

The fifth through 12th ribs are treated as follows: to manipulate the right seventh rib by gapping its costotransverse joint, the patient grasps his or her left shoulder with the right hand and the therapist reaches across the front of the patient to grasp the posterior aspect of the right shoulder (Figures 6-15). The patient's trunk is then rotated to the left until motion arrives at the medial aspect of the angle of the right seventh rib (the rib angle is the most

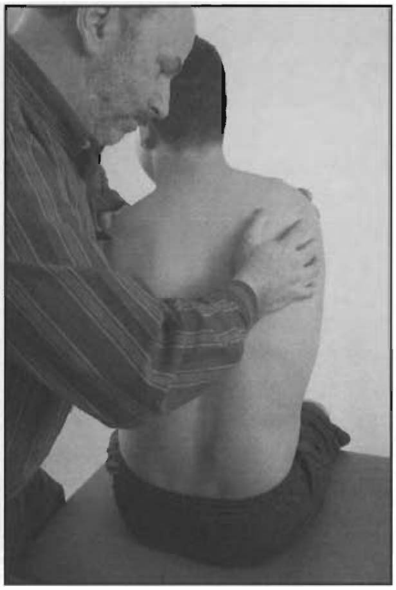

Figure 6-15. Mobilization of right ribs 5-12.

posterior aspect of the rib). The graded mobilization consists of a transverse pressure on the rib angle to the right with or without a simultaneous small amplitude rotation of the trunk to the left. The lower the rib, the greater the trunk rotation. To manipulate the fifth through 12th ribs on the left, all positions and movements are reversed.

There are additional rib mobilizations involving the costosternal joints that will not be covered in this text. Bookhout[45] uses AP mobilizations in this area of the costal cage to enhance shoulder girdle mobility in addition to managing adverse neural tension in the upper limb. Although manual therapy of the costosternal joints is beneficial, one should never directly mobilize the rib's costochondral junction lest pain and inflammation result.

Manipulation of the ribs affects the costotransverse, costovertebral, and costosternal joints as spinal manipulation affects the apophyseal joints. As with any manipulative procedure, there are both mechanical and neurological effects, but in the case of costal cage manipulation, there are beneficial effects on pulmonary function as well.

To enhance any of the manipulations described in this chapter, the therapist can add a postisometric relaxation component to the technique. This procedure, which osteopathic physicians refer to as muscle energy technique,[8,9] involves the use of a patient-activated, submaximal, isometric contraction at the very beginning of the restrictive motion barrier (ie, the feather-edge), which is in the opposite direction of the desired mobilization. Following a 6-second isometric contraction, the therapist mobilizes the affected segment until a new motion barrier is reached. This procedure is repeated 3 times prior to using the graded mobilizations that have been outlined above. For example, prior to applying a grade 4 extension manipulation at the T6,7 segment, the patient is asked to resist the therapist's attempt to extend the motion segment for a count of 6 seconds. This activates the flexors at the T6,7 segment isometrically. According to scientific theory,[46] and in keeping with the clinical experience of those trained in these procedures, this is followed by a period of reflex inhibition in which the muscles are amenable to being stretched. The value of this technique, prior to joint mobilization, is that muscle tone is reduced, thus enhancing the efficacy of the manipulation. In the presence of neuroreflexive muscle splinting, the use of either indirect treatment methods or postisometric relaxation is indicated. However, when reactivity is low and there is more contracture than contraction, then mobilization/manipulation alone is needed.

All sitting techniques of the thoracic spine and ribs should be performed with the patient's feet in firm contact with either the floor, a chair, or a stool. Otherwise, the patient will not be secure and therefore unable to fully relax.

7

Therapeutic and Home Exercises for the Scapulothoracic Region

The home exercise program (HEP) is as important if not more important than the manual therapy component of the intervention process. Having said that, the HEP should never consist of merely providing a handout to the patient. The HEP should be custom designed to address the specific needs of each patient. The patient requires individual instruction for each procedure in order to ensure its correct performance. The benefit to the patient is directly related to both the quantity and the quality of the home exercises; the time spent by the therapist facilitating the patient's independence in this process is a worthwhile investment.

The HEP should always be presented to the patient as an exercise prescription. This involves all aspects of the exercise, including number of repetitions, sets, and seconds held. It must also include instruction in warm-up, cool-down, injury prevention, first aid for managing flare-ups, etc. For stretching procedures, the patient is advised to stop at the first feeling of tissue resistance and to hold the stretch for 30 seconds.[47,48] It is wise to escalate patients up to 30 seconds by beginning at 5 to 10 seconds and working up from there. This is then repeated 3 times every 2 hours if possible. For strengthening exercises, the patient is advised to avoid any and all painful muscle contractions. The patient can begin with 10 repetitions, holding each contraction for 5 to 10 seconds. Strengthening exercises are usually performed no more than 3 times a day because working muscles need time to rest.

It is imperative that patients understand that the HEP is not optional. If they expect results, then they must "take their medicine!"

Doorway Stretch

The doorway stretch is an excellent way to stretch the myofascial structures of the anterior chest wall (eg, pectoralis major/minor and related fascia, clavipectoral fascia). The patient should be encouraged to explore various aspects of the chest wall in search of the area of greatest tightness. The doorway stretch can be performed bilaterally (Figure 7-1) or unilaterally. The patient must be careful not to stress the anterior capsule of the glenohumeral joint, which is already hypermobile in many patients.

Quadruped Flexion

In order to isolate spinal flexion in the upper, mid, and lower thoracic spine, the patient is instructed to perform self-mobilization in 3 distinct positions. To achieve flexion from T1 to T4, the patient is placed in the angry cat position with the ears in line with the elbows (Figure 7-2). To achieve flexion from T5 to T8, the patient is again placed in the angry cat position, but this time with the shoulders in line with the elbows (Figure 7-3). To achieve flexion in the lower thoracic spine, T9 to T12, the patient is instructed to extend his or her arms and sit back, buttocks to heels (Figure 7-4).

Once positioned correctly in proper alignment, the patient is instructed to "round the back" so that the thoracic kyphosis is increased. It is important that the patient understands the need to self-mobilize the region that lacks flexion. Simply flexing in an area that is already mobile is

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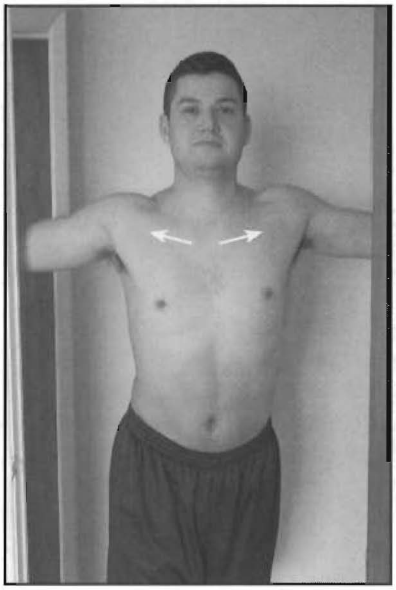

Figure 7-1. Doorway stretch.

Figure 7-2. Self-mobilization T1-T4 flexion.

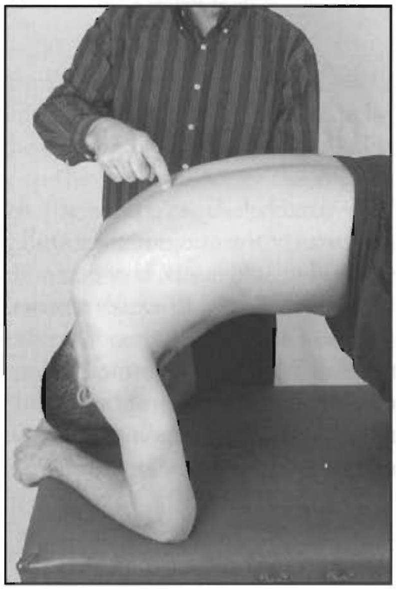

Figure 7-3. Self-mobilization T5-T8 flexion.

Figure 7-4. Self-mobilization T9-T12 flexion.

unproductive. The self-mobilization is held for 30 seconds and repeated 3 times every 2 hours.

Quadruped Extension

From the quadruped position, the patient's hands are placed in front of the patient to the point where thoracic extension begins to occur (Figure 7-5). The patient must have full range of shoulder flexion in order for this stretch to be effective. The patient rocks backward with the intention of drawing the chest to the floor and flattening the thoracic kyphosis. Three repetitions of 30 seconds each are performed, with the arms placed further in front of the patient with each stretch. For those who are able to tolerate a more vigorous stretch, the patient's hands can be placed on a chair or stool as illustrated (Figure 7-6). This also serves as an excellent stretch for the latissimus dorsi muscle.

Quadruped Side Bending

This stretch can address tightness of the latissimus dorsi, erector spinae, quadratus lumborum (QL), piriformis, and tensor fascia latae. It is also an excellent way to self-mobilize the thoracic and lumbar spine for increased side bending. Most patients require hands-on instruction before they

Figure 7-5. Self-mobilization T1-T12 extension.

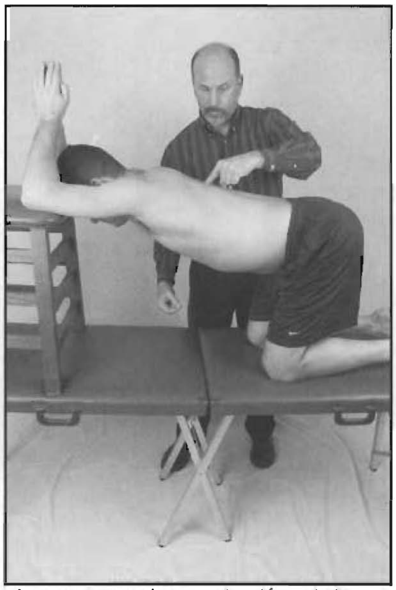

Figure 7-6. Advanced self-mobilization T1-T12 extension (prayer position).

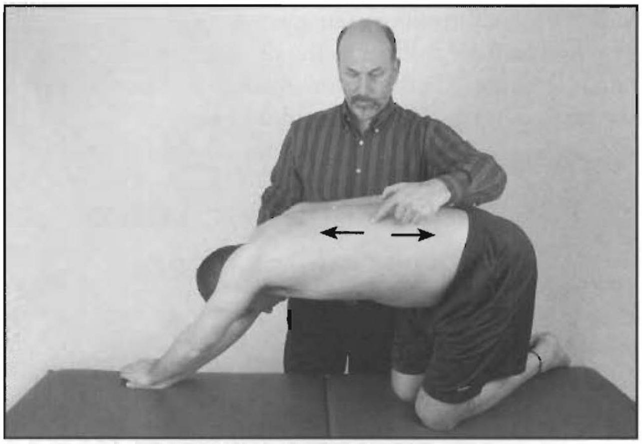

Figure 7-7. Thoracic side-bending stretch.

attain a proper stretch. The key is to "lean" into the convex side rather than "tilt" into it (ie, the shoulders should remain level). Patients often need to be reminded not to overstretch lest they "pay for it" later (Figure 7-7)!

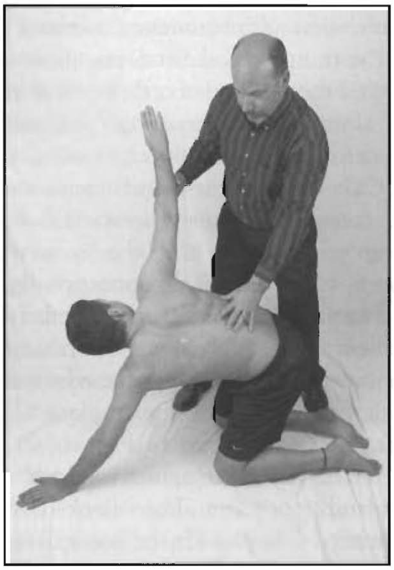

Figure 7-8. Thoracic rotation stretch.

Quadruped Rotation

The patient is advised to initiate motion with the eyes, followed by the head-neck, upper limb, and trunk. Sitting backward toward the heels reduces motion in the lower thoracic region as per Fryette's third rule. By adjusting hip position in this manner, the rotation can be biased to a given region of the thoracic spine (Figure 7-8).

Sensorimotor Training

Hanna[49] defined sensorimotor amnesia (SMA) as "the habituated state of memory loss of how certain muscle groups feel and how to control them." Many 20th century "body workers" (eg, Feldenkrais, Alexander, Hanna, Rolf, Pilates, Trager) contributed enormously to our understanding of mind/body connections, and physical therapists have been the conduits of much of this information into mainstream medicine.

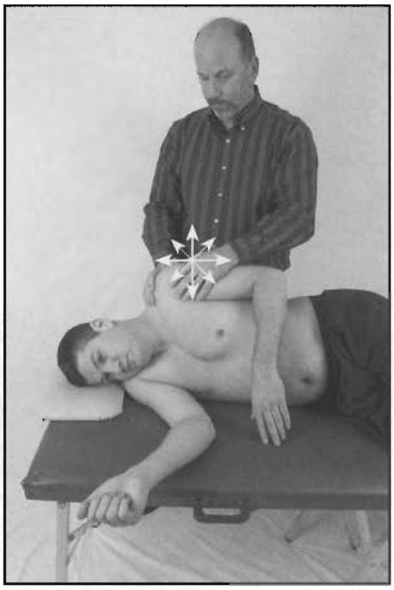

Figure 7-9. Feldenkrais shoulder clock.

Figure 7-10. Right scapular stabilization in quadruped.

The appreciation of neuromusculoskeletal impairment, as compared with musculoskeletal impairment alone, has greatly benefited the practice of orthopedic physical therapy. The purpose of sensorimotor training[50] is to reduce or eliminate pain in the neuromusculoskeletal system by helping the patient rediscover flexibility and ease of movement. Using an analogy from computers, manual therapy is to the "hardware" what sensorimotor training is to the "software." As mentioned earlier, it is the electrical connection that makes the difference between movement that is efficient and functional versus movement that is inefficient and dysfunctional.

While many options exist for the re-education of motion in the scapulothoracic region, a good place to start is with the Feldenkrais "clock" approach (Figure 7-9). In the side-lying position, the patient imagines a "clock" placed upon the shoulder and upper arm. The "clock" concept can be used in a variety of ways with the motion occurring in a clockwise or counterclockwise fashion as well as in imaginary "lines" that connect opposite ends of the "clock" (ie, 12 to 6, 1 to 7, 2 to 8, 3 to 9, 4 to 10, and 5 to 11). The sequence includes passive motion, followed by active-assisted, active, and resisted motion. It is crucial that all sensorimotor training commence with passive work so that the patient can develop a "template" for what the motion should feel like. Patient guidelines for successful sensorimotor training include the following:

- Perform the movements slowly and easily.
- Avoid excessive effort.
- Rest frequently.
- Pain and discomfort should never be experienced during an exercise.

The most common movement impairment seen in the scapulothoracic region is a combination of depression and retraction. It is this "down and back" motion that will require the most work. Another useful tool for muscle re-education is rhythmic stabilization. The shoulder girdle is placed in its neutral, physiologic position. The patient then attempts to maintain this position against a variety of forces in a variety of directions. These isometric contractions provide excellent feedback into the central nervous system for motor learning. Rhythmic stabilization is also applied with the patient in the side-lying position.

Strengthening the Lower Scapular Stabilizers

The final component of the intervention process is to revisit the weak phasic muscles to ensure that their motor strength is restored to normal. Otherwise, the imbalance persists and future impairment of function is likely. According to Janda,[10] the lower scapular stabilizers consist of the serratus anterior, rhomboids, and middle/lower trapezius. To activate the lower scapular stabilizers, with emphasis on the serratus anterior, Isaacs and Bookhout[9] place the patient in quadruped with the elbows slightly bent in order to level the shoulders and hips (Figure 7-10). The patient is then instructed to lift one hand slightly off the table to see if scapular winging occurs on the side of the support arm. Training weak lower scapular stabilizers consists of working the supported side, in neutral scapulothoracic alignment, until winging is reduced or absent. According to these same authors,[9] flexion restrictions of the thoracic spine, specifically T3 to T6, will need to be addressed before the serratus anterior will function optimally. The wall press is commonly used to strengthen the serratus anterior.[9] This exercise is performed standing with the patient's hands flat against the wall, at shoulder height, with the elbows straight. From the neutral position of the spine, the patient is asked to protract the shoulder girdle, flex the cervical spine, and tuck the pelvis under. The end-point of thoracic flexion is held for 5 to 10 seconds and repeated 10 times, 3 times per day. An alternative to the

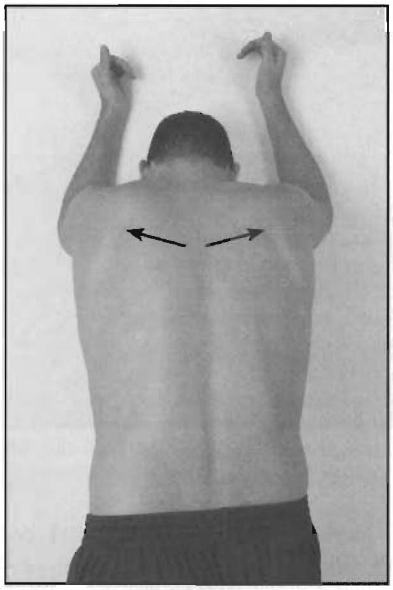

Figure 7-11. Wall slide for serratus anterior.

Figure 7-12. Manual training lower trapezius.

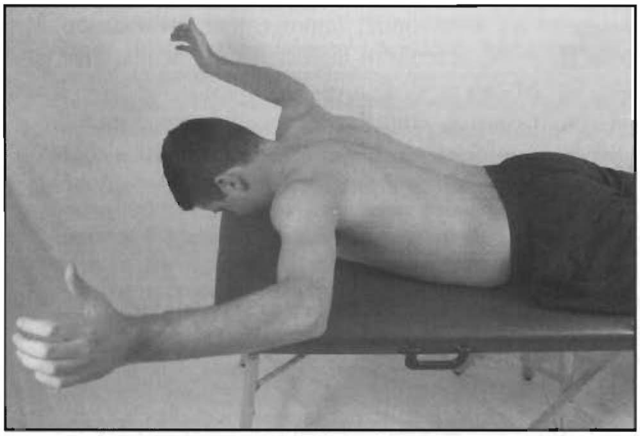

Figure 7-13. Bilateral lower trapezius training.

wall press for serratus anterior strengthening is the wall slide exercise.[51] As illustrated (Figure 7-11), the patient stands with his or her forearms, ulnar aspect, in contact with the wall and the shoulders and elbows at 90 degrees of flexion. The patient then slides the forearms up the wall while instructed to "bring your shoulders out and around as you slide up the wall." To enhance pressure against the wall while sliding the forearms upward, the patient is advised to assume the staggered-foot position (dominant foot against the base of the wall) and to transfer weight to the dominant foot while performing the wall slide.

To activate the lower trapezius muscle, Isaacs and Bookhout[9] recommend placing the patient in prone lying with the arm abducted to 125 degrees. The therapist then resists scapular adduction and depression at the inferior angle of the scapula (Figure 7-12). These same authors[9] note that extension restrictions in the thoracic spine, specifically T6 to T10, need to be corrected in order to achieve optimal activation of the lower trapezius. In order to bilaterally strengthen the rhomboids and middle/lower trapezius muscles simultaneously, the patient assumes the prone position with his or her upper limbs in approximately 145 degrees of abduction with the elbows flexed slightly. The patient then directs the elbows down and back toward the low back to recruit the scapular adductors and depressors while maintaining a neutral position of the craniovertebral region (slight chin-tuck), cervicothoracic junction, and lumbar spine (Figure 7-13). The combination of scapular upward rotation, adduction, and depression isolates the lower trapezius muscles. During this exercise the patient must be advised not to lower the upper limbs below 30 degrees of abduction lest the scapulae begin to rotate downward; any extension of the spine should occur in the thoracic rather than the lumbar region. In fact, a slight posterior pelvic tilt (PPT) is helpful in avoiding this tendency. In addition to this isotonic exercise, the lower trapezius muscles can be trained isometrically by having the patient maintain the adducted/depressed position of the scapulae while the upper limbs are slowly abducted back to approximately 120 degrees. The key to the successful execution of the return phase is that the patient must not elevate the shawl area (ie, upper trapezius and levator scapulae substitution) as the shoulders are being abducted. This exercise can also be performed while standing in front of a mirror to visually ensure that the shawl area is not "hiked up" during the return phase of abduction. Securing the scapulae in a position of adduction/depression with Leukotape P (Beiersdorf-Jobst Inc, Rutherford College, NC) is helpful in the retraining of scapular position, through increased kinesthetic awareness, as well as by directly inhibiting the upper and facilitating the lower trapezei[14] (Figure 7-14). When the patient is ready, dumbells can be added for advanced training (Figure 7-15). Furthermore, the author finds that the addition of bilateral, superior glide mobilization of the sternoclavicular joints during active scapular adduction/depression enhances the efficacy of lower trapezius training by increasing mobility of the shoulder girdle in the desired direction (Figure 7-16). Not illustrated, but also of clinical significance, is the component motion of posterior clavicular rotation,

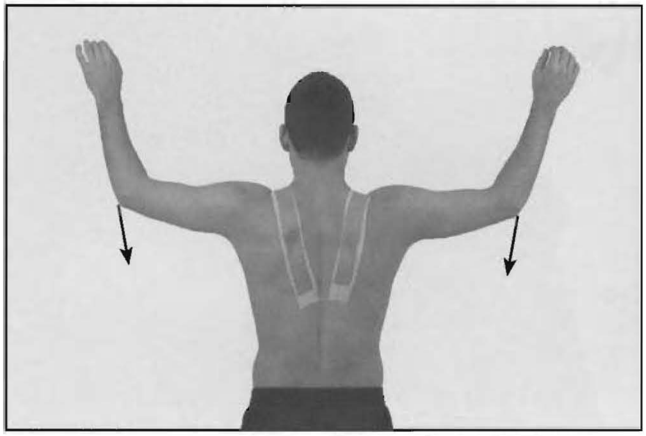

Figure 7-14. Lower trapezius training with scapular taping.

Figure 7-15. Lower trapezius training with dumbells.

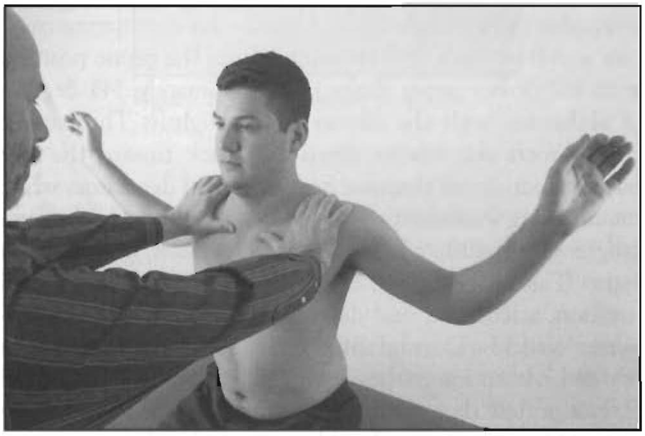

Figure 7-16. Sternoclavicular mobilization.

which is a requisite movement for scapular adduction and depression. The PostureJac (SomatoCentric Systems, Inc, Toronto, Ontario, Canada) can also be used to facilitate the rhomboids and middle/lower trapezius while inhibiting the upper trapezius[52,53] and will be dealt with in detail in Chapter 25.

For both the prone lying and standing positions, the patient takes 5 to 10 seconds to complete each of the 2 phases (isotonic and isometric) and performs 10 repetitions of the entire up and down cycle 3 times per day. Some patients find the Leukotape P so helpful in facilitating a corrected position of the scapulothoracic region that they keep it on for a few days at a time. A useful Alexander movement principle regarding upper extremity elevation is that motion should occur from distal to proximal (ie, fingers, wrist, elbow, and shoulder in that order). Furthermore, optimal active shoulder elevation should include the awareness of movement all the way into the sternoclavicular joint.

Lower trapezius training is a crucial component in the rehabilitation of shoulder impingement.[14,15,17,18,52-54] Without normal function of the lower trapezius, the scapulae will not adequately adduct, upwardly rotate, depress, and posteriorly tilt during upper limb elevation, resulting in the potential for subacromial impingement. In addition, it is critical for the scapula to maintain a stable platform upon which the humerus can move. Weakness of the lower scapular stabilizers may contribute to scapular hypermobility as well as compromise optimal positioning of the glenoid during elevation, thus interfering with normal scapulohumeral rhythm.[2,9,10,14-18,54]

Section II: Key Points

1. The thorax is a key region of somatic impairment (dysfunction) in patients with neck, back, and shoulder pain.
2. In the thoracic spine, McKenzie's dysfunction syndrome is much more common than derangement syndrome.
3. Follow the treatment sequence for dysfunction: tissue reactivity, myofascial extensibility, joint mobility, posture correction, and muscle function (motor control, strength, and endurance).
4. Thoracic extension (T6 to T12) and thoracic flexion (T3 to T6) are necessary for normal recruitment of the lower trapezius and serratus anterior muscles, respectively.
5. Stretch what is tight, mobilize what is stiff, and strengthen what is weak!

References

1. Lee D. *The Thorax: An Integrated Approach*. White Rock, BC, Canada: Diane G. Lee Physiotherapist Corporation; 2003.
2. Sahrmann S. *Diagnosis and Treatment of Movement Impairment Syndromes*. St. Louis, MO: Mosby-Year Book; 2002.
3. McConnell J. *The McConnell Approach to the Problem Shoulder*. Marina Del Rey, CA: McConnell Institute; 1994.
4. Solomon L, Warwick DJ, Nayagam S. *Apley's System of Orthopaedics and Fractures*. 8th ed. London: Arnold Publishing; 2001.
5. Hillegass EA, Sadowsky HS. *Essentials of Cardiopulmonary Physical Therapy*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2001.
6. Lundon K, Li A, Bibershtein S. Interrater and intrarater reliability in the measurement of kyphosis in postmenopausal women with osteoporosis. *Spine*. 1998;23(18):1978-1985.
7. Korovessis P, Petsinis G, Papazisis Z, Baikousis A. Prediction of thoracic kyphosis using the Debrunner kyphometer. *J Spinal Disorders*. 2001;14(1):67-72.
8. Greenman PE. *Principles of Manual Medicine*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2003.
9. Isaacs ER, Bookhout MR. *Bourdillon's Spinal Manipulation*. 6th ed. Boston, MA: Butterworth-Heinemann; 2002.
10. Janda V. Muscles and motor control in cervicogenic disorders. In: Grant R, ed. *Physical Therapy of the Cervical and Thoracic Spine*. 3rd ed. New York, NY: Churchill Livingstone; 2002.
11. Kendall FP, Kendall McCreary E, Provance PG, McIntyre Rodgers M, Romani WA. *Muscles: Testing and Function With Posture and Pain*. 5th ed. Baltimore, MD: Williams & Wilkins; 2005.
12. Cleland J. *Orthopaedic Clinical Examination. An Evidence-Based Approach for Physical Therapists*. Carlstadt, NJ: Icon Learning Systems; 2005.
13. Kuzmich D. The levator scapulae: making the con-neck-tion. *J Manual & Manipulative Ther*. 1994;2(2):43-54.
14. Selkowitz DM, Chaney C, Stuckey SJ, Vlad G. The effects of scapular taping on the surface electromyographic signal amplitude of shoulder girdle muscles during upper extremity elevation in individuals with suspected shoulder impingement syndrome. *J Orthop Sports Phys Ther*. 2007;37(11):694-702.
15. Hall CM, Thein Brody L. *Therapeutic Exercise Moving Towards Function*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2005.
16. Lukasiewicz AC, McClure P, Michener L, Pratt N, Sennett B. Comparison of 3-dimensional scapular position and orientation between subjects with and without shoulder impingement. *J Orthop Sports Phys Ther*. 1999;29(10):574-586.
17. Hertling D, Kessler RM. *Management of Common Musculoskeletal Disorders: Physical Therapy Principles and Methods*. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2006.
18. Dutton M. *Orthopaedic Examination, Evaluation, and Intervention*. 2nd ed. New York, NY: McGraw Hill; 2008.
19. DeConinck S. *Orthopaedic Medicine Cyriax, Updated Value in Daily Practice. Part I: Clinical Examination and Diagnosis*. Minneapolis, MN: OPTP; 2003.
20. Kaltenborn FM. *Manual Mobilization of the Joints. Vol. 1, The Extremities*. 6th ed. Minneapolis, MN: OPTP; 2006.
21. Korr IM, ed. *The Neurobiologic Mechanisms in Manipulative Therapy*. New York, NY: Plenum Press; 1978.
22. Cook CE, Hegedus EJ. *Orthopedic Physical Examination Tests: An Evidence-Based Approach*. Upper Saddle River, NJ: Pearson Prentice Hall; 2008.
23. Simons DG, Travell JG, Simons LS. *Travell & Simons' Myofascial Pain and Dysfunction. The Trigger Point Manual. Vol 1. Upper Half of Body*. Baltimore, MD: Williams & Wilkins; 1999.
24. McPartland JM. Travell trigger points-molecular and osteopathic perspectives. *JAOA*. 2004;104(6):244-249.
25. Shah JP, Phillips TM, Danoff JV, Gerber LH. An in vivo microanalytical technique for measuring the local biochemical milieu of human skeletal muscle. *J Appl Physiol*. 2005;99:1977-1984.
26. Fernandez-de-las-Penas C, Arendt-Nielsen L, Simons DG. Contributions of myofascial trigger points to chronic tension-type headache. *J Manual Manipulative Ther*. 2006;14(4):222-231.
27. Fernandez-de-las-Penas C, Alonso-Blanco C, Cuadrado ML, Gerwin RD, Pareja JA. Trigger points in the suboccipital muscles and forward head posture in tension-type headache. *Headache*. 2006;46:454-460.

28. Fernandez-de-las-Penas C, Cuadrado ML, Pareja JA. Myofascial trigger points, neck mobility and forward head posture in unilateral migraine. *Cephalgia*. 2006;26:1061-1070.
29. Wood P. Proposed models of fibromyalgia sub-types. *Practical Pain Management*. 2008;8(5):70-75.
30. Ellis JJ, Johnson GS. Myofascial considerations in somatic dysfunction of the thorax. In: Flynn TW, ed. *The Thoracic Spine and Rib Cage: Musculoskeletal Evaluation and Treatment*. Newton, MA: Butterworth-Heinemann; 1996.
31. Andrade CK, Clifford P. *Outcome-Based Massage*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008.
32. Cantu RI, Grodin AJ. *Myofascial Manipulation, Theory and Clinical Application*. 2nd ed. Gaithersburg, MD: Aspen Publishers; 2001.
33. Culham E, Peat M. Functional anatomy of the shoulder complex. *J Orthop Sports Phys Ther*. 1993;18(1):342-350
34. Chaitow L, DeLany JW. *Clinical Application of Neuromuscular Techniques. Volume 2: The Lower Body*. Edinburgh, UK: Churchill Livingstone; 2002.
35. Manheim C. *The Myofascial Release Manual*. 4th ed. Thorofare, NJ: SLACK Incorporated; 2008.
36. Maitland GD, Banks K, English K, Hengeveld E. *Maitland's Vertebral Manipulation*. 6th ed. Oxford: Butterworth-Heinmann; 2001.
37. Mintken PE, DeRosa C, Little T, Smith B. A model for standardizing manipulation terminology in physical therapy practice. *J Orthop Sports Ther*. 2008;38(3):A1-A6.
38. Dvorak J, Dvorak V, Schneider W, eds. *Manual Medicine*. Heidelberg, Germany: Springer-Verlag; 1985.
39. Warmerdam A. *Manual Therapy: Improve Muscle and Joint Functioning*. Wantagh, NY: Pine Publications; 1999.
40. Liebler EJ, Tufano-Coors L, Douris P, et al. The effect of thoracic spine mobilization on lower trapezius strength testing. *J Manual & Manipulative Ther*. 2001;9:207-212.
41. Cleland J, Selleck B, Stowell T, et al. Short-term effects of thoracic manipulation on lower trapezius muscle strength. *J Manual Manipulative Ther*. 2004;12:82-90.
42. Mulligan BR. Snags: mobilizations of the spine with active movement. In: Boyling JD, Palastanga N, eds. *Grieve's Modern Manual Therapy: The Vertebral Column*. 2nd ed. Edinburgh, UK: Churchill Livingstone; 1994.
43. Griegel-Morris P, Larson K, Mueller-Klaus K, Oatis CA. Incidence of common postural abnormalities in cervical, shoulder, and thoracic regions, and their association with pain in two age groups of healthy subjects. *Phys Ther*. 1992;72(6):426-431.
44. Wright EF, Domenech MA, Fischer JR. Usefulness of posture training for patients with temporomandibular disorders. *JADA*. 2000;131:202-210.
45. Bookhout MR. *Management of Thoracic Spine and Costal Cage Dysfunction With Manipulative Therapy, Myofascial Techniques, and Specific Exercises*. Bay Shore, NY: Course Manual; 1995.
46. Hammer WI. *Functional Soft Tissue Examination and Treatment Methods, New Perspectives*. 2nd ed. Gaithersburg, MD: Aspen Publishers, Inc; 1999.
47. Bandy WD, Irion JM. The effect of time on static stretch on the flexibility of the hamstring muscles. *Phys Ther*. 1994;74:845-852.
48. Bandy WD, Irion JM, Briggler M. The effect of time and frequency of static stretching on flexibility of the hamstring muscles. *Phys Ther*. 1997;77(10):1090-1096.
49. Hanna T. *Somatics*. New York, NY: Addison-Wesley Publishing; 1988.
50. Zemach-Bersin D, Reese M. *Audiotape Program User's Guide*. Berkeley, CA: Sensory Motor Learning Systems; 1983.
51. Hardwick DH, Beebe JA, McDonnell MK, Lang CE. A comparison of serratus anterior muscle activation during a wall slide exercise and other traditional exercises. *J Orthop Sports Ther*. 2006;36(12):903-910.
52. Goldstein LB, Makofsky HW. TMD/Facial pain and forward head posture. *Practical Pain Management*. 2005;5(5):36-39.
53. Goldstein LB, Makofsky HW. Deep cervical muscle dysfunction and head/neck/face pain. *Practical Pain Management*. 2009;9(1):30-33.
54. Prentice WE, Voight MI. *Techniques in Musculoskeletal Rehabilitation*. New York, NY: McGraw-Hill; 2001.

Section

III

CERVICAL SPINE

8

Examination and Evaluation of the Cervical Spine

As with the scapulothoracic region, the approach to examination and evaluation of the cervical spine in this text utilizes the CHARTS methodology. Although individual components of the CHARTS paradigm have been tested for accuracy and reliability (eg, special tests[1,2]), as a classification system it has not. An emerging system of classification for the cervical spine, which is based on published evidence, is referred to as treatment-based classification,[3,4] which is based on the goals of treatment and interventions used to achieve these goals, as compared to classifying patients by pathology or symptom distribution. This author finds it useful in organizing the examination findings into treatment-based categories, which are then used to guide intervention. For example, Childs et al[4] propose organizing key examination findings into the following classifications: 1) mobility, 2) centralization, 3) conditioning and exercise tolerance, 4) pain control, and 5) reduce headache. In the same article, Childs et al[4] also organize key signs and symptoms into categories associated with serious pathological neck conditions (eg, cervical myelopathy) and clinical "yellow flags" suggestive of heightened fear-avoidance beliefs. In addition to being evidence-based, treatment-based classification is eclectic and pragmatic. This is one to watch!

Posture

For the purpose of learning the specifics of a patient's structure, a compartmental approach is taken in which we separate the various anatomic regions from each other (eg, scapulothoracic, cervical, lumbar, and pelvic). However, in clinical practice the entire continuum of posture from head to toe must be integrated because of the interdependence of all body parts, which must be appreciated within a holistic paradigm.

Having said that, we come to the analysis of head and neck alignment. As in the scapulothoracic region, we will examine the standing patient from the side, back, and front. Employing the CHARTS methodology, the evaluation of posture provides much needed information on asymmetry (A). The importance of C and H was covered earlier in Chapter 3.

The standing lateral view of the cervical spine enables the therapist to inspect the following structures for faulty alignment (Figure 8-1):

- Head and neck position in the sagittal plane. The ear lobe to shoulder joint relationship can be assessed relative to forward head posture (FHP), which can be described as minimal, moderate, or severe. A posture grid or plumb-line can be used for greater accuracy. In addition to neutral posture being defined as a vertical axis from the ear lobe to midway through the shoulder joint,[5] the tragus and acromion should also be in a vertical relationship with gravity.[6]
- The position of the occiput. Note posterior cranial rotation (occipital extension) when present. Alexander teachers refer to this as "downward pull." The term for the preservation or recovery of the optimum dynamic relationship between the head and spine in movement and at rest is primary control. A recognized plane of reference for the assessment of head position is the "Frankfort plane." It suggests that

Makofsky HW
Spinal Manual Therapy, 2nd ed. (pp 63-76)

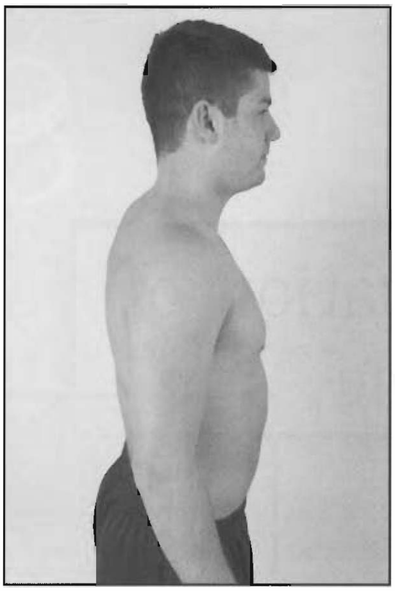

Figure 8-1. Lateral view of head and neck.

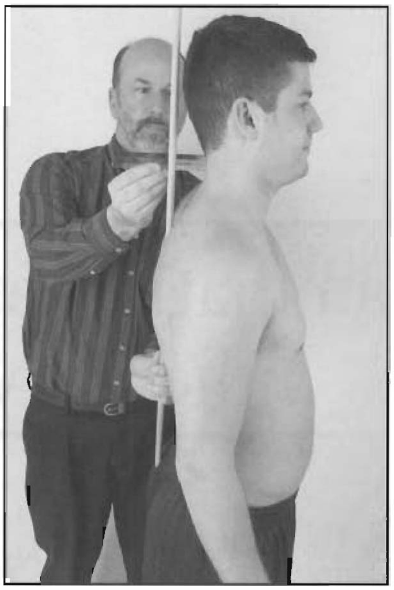

Figure 8-2. Rocabado head-neck measurement.

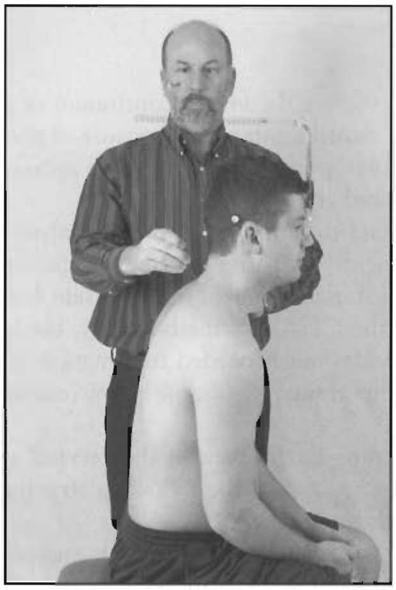

Figure 8-3. CROM measurement of forward head posture.

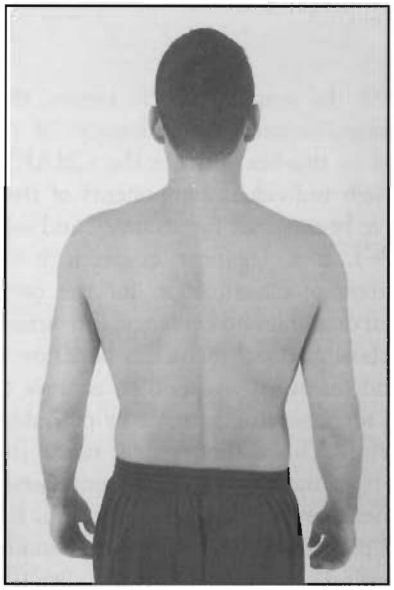

Figure 8-4. Posterior view of head and neck.

a line extending from the upper margin of the external auditory meatus to the inferior aspect of the orbit should be horizontal or parallel to the ground.

- The inferior orbit to manubrium relationship. This should ideally be a straight vertical line.
- Rocabado[7] recommends the use of a head-neck measure that involves extending a vertical tangent from the thoracic spine from which the perpendicular distance in centimeters is recorded to the midcervical lordosis (Figure 8-2). A distance of 6 cm represents the optimum head-neck to back relationship. In addition, the vertical thoracic tangent should ideally be in alignment with the occiput as well.
- Another option in measuring FHP (Figure 8-3) is to use the cervical range of motion (CROM) device (Performance Attainment Associates, Roseville, NJ).

Both the plumb-line method and the CROM device have demonstrated moderate to high intratester and intertester reliability in the evaluation of FHP.[8]

The standing posterior view (Figure 8-4) includes an assessment of the following:

- Occipital position in the frontal plane. The ears can be used to assess for a lateral tilt of the occiput; rotation of the head is noted by observing the face on one side. A type 1 head tilt involves contralateral head rotation (eg, congenital torticollis), while a type 2 tilt

Figure 8-5. Anterior view of head and neck.

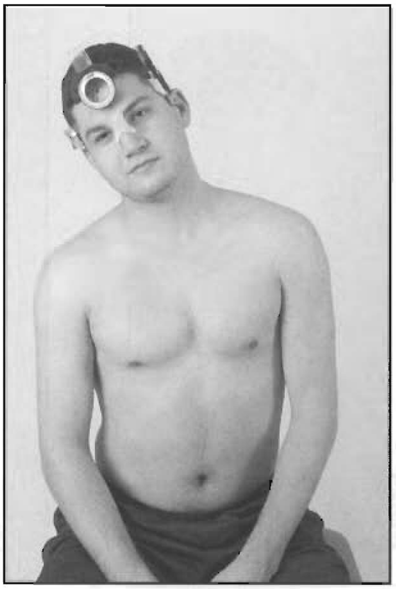

Figure 8-6a. CROM measurement of cervical side bending.

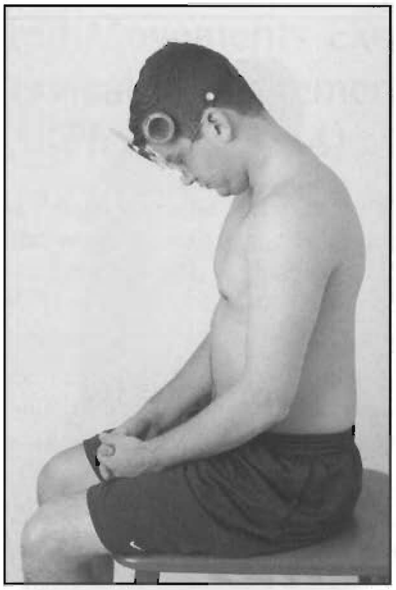

Figure 8-6b. CROM measurement of cervical flexion.

involves ipsilateral rotation (eg, acquired torticollis or wryneck).

➤ Lower neck position (C2 to C7). The most common asymmetry is a lateral shift to one side.

The standing anterior view of the head-neck region (Figure 8-5) is helpful in confirming a torticollis, but the assessment for craniofacial asymmetries is more relevant to the temporomandibular examination. In children with congenital torticollis, the face is often shorter on the side of the cervical concavity. However, this usually improves, as head posture is corrected in the developing child.

In patients with moderate to severe FHP, there is often an associated retrognathia of the mandible (ie, horizontal deficiency of the lower jaw). The lateral view demonstrates a convexity of the lower third of the craniofacial region. This is seen in children who are mouth breathers and in patients with juvenile rheumatoid arthritis. The connection between this finding, dental malocclusion, and adults with temporomandibular disorders will be covered in a subsequent chapter.

A plumb-line or posture grid can also be used for greater accuracy in both the anterior and posterior assessment of standing posture.

Active Movements

The examination of active cervical movements can be performed with a variety of methods, including the CROM device (Figures 8-6a to 8-6e), the universal goniometer, inclinometers, computerized motion diagnostics, visual estimation, etc. Although the CROM device is the preferred clinical goniometer for the cervical spine regarding its reliability,[9] therapists should learn the visual estimation method for 2 reasons. The first reason is that the CROM device is not available in all clinical situations. The second reason is that manual therapists need to develop the clinical skill of observing not only the quantity but also the quality of motion as discussed in Chapter 4. The skilled observer can detect things about human motion that a sophisticated goniometer or computer can never appreciate. It is true that outcomes are based upon numbers, but perhaps there are other numbers, in addition to degrees or centimeters, that are just as representative of improvement (eg, the Neck Disability Index,[10] which looks at 10 overall categories with all but 2 being directly

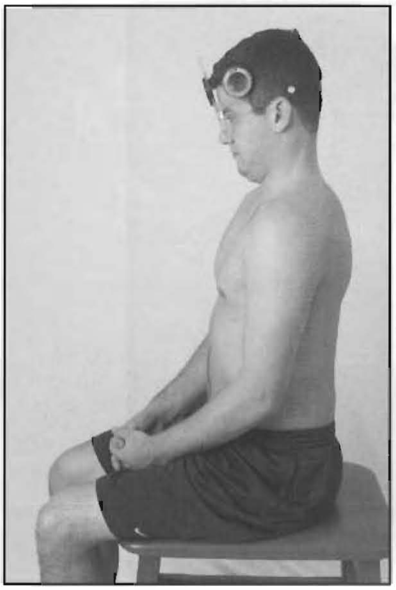

Figure 8-6c. CROM measurement of craniovertebral flexion.

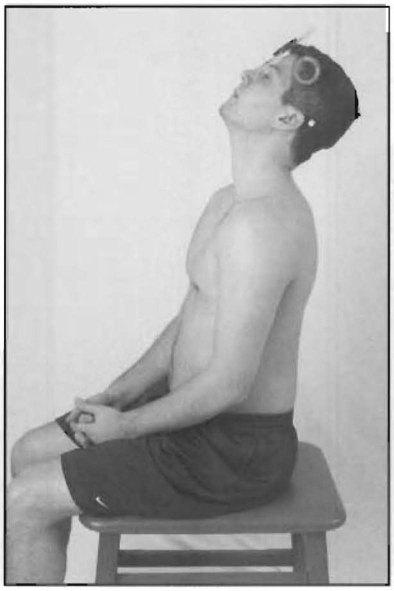

Figure 8-6d. CROM measurement of cervical extension.

Figure 8-6e. CROM measurement of cervical rotation.

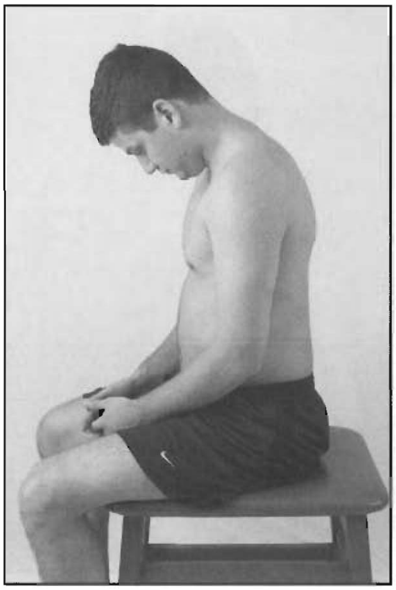

Figure 8-7a. Active cervical flexion.

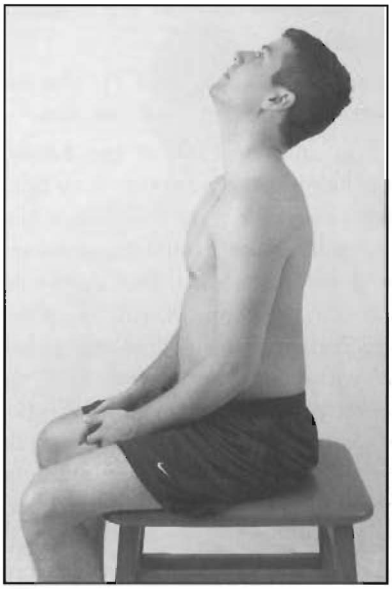

Figure 8-7b. Active cervical extension.

related to functionality; the Northwick Park[10]; the McGill Pain Questionnaire[10]).

Returning to the visual examination of active cervical spine mobility, there are 6 movements that the patient is asked to perform. They are flexion, extension, side bending left and right, and rotation left and right (Figures 8-7a to 8-7d). As with the remainder of the vertebral column, the therapist can refer to other texts for the normative values related to the quantity of each movement. Because the upper and lower cervical spine possesses different arthrokinematics[11,12] (ie, the upper cervical spine functions according to type 1 or neutral mechanics, whereas C2 to C7 follows type 2 or non-neutral mechanics), it is possible to identify the site of a dysfunction by observing coupled motions during active cervical rotation and side bending. For example, when observing active side bending to the right, the coupled motion of side bending right/rotation right emerges. This suggests that the O-A-A region is unable to move into left rotation while side bending right and consequently type 2 mechanics of the lower cervical spine prevails. Likewise, when observing active rotation left, the coupled motion of rotation left/side bending right emerges. This suggests restriction somewhere in the lower cervical spine (C2 to C7) and type 1 mechanics of the upper cervical spine dominates. When the neck is free of restriction, the Z-axis motions of side bending right and left remain within the coronal plane; the Y-axis motions of

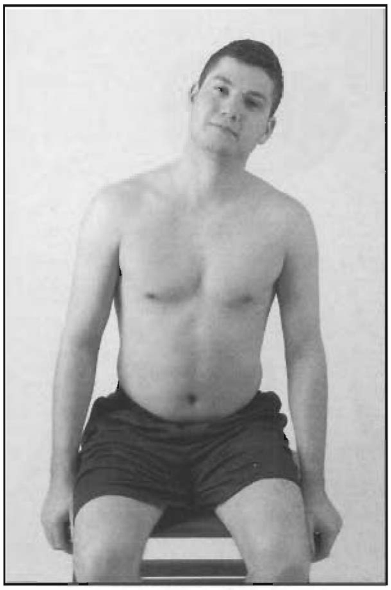

Figure 8-7c. Active cervical side bending.

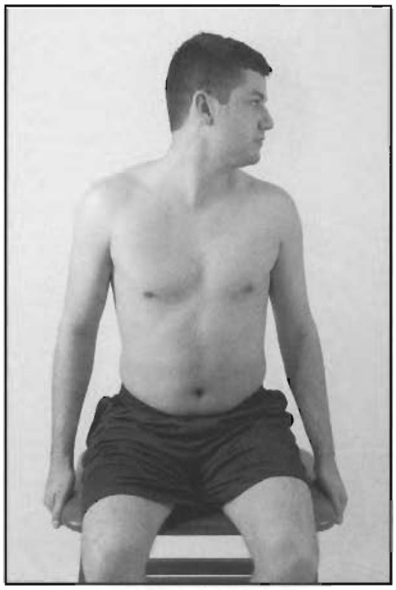

Figure 8-7d. Active cervical rotation.

rotation right and left remain within the horizontal plane. In addition to observing active cervical motion for the presence of restriction, the therapist can introduce coupled motion passively to test the upper and lower cervical region for dysfunction. For example, the quality, quantity, end-feel, and tissue reactivity of side bending right/rotation left can be tested for function of the upper cervical region; likewise side bending right/rotation right can be tested for function of C2 to C7. If the patient reports becoming light-headed or anxious at any point in the examination (possible signs of CNS ischemia), the head-neck region should be slowly returned to neutral and the examination discontinued. For ease of application, all techniques can be performed with the patient sitting.

The salient points of the active cervical motion examination include the presence of impairment (minimal, moderate, severe), the reactivity of the tissues, areas of suspected hypo/hypermobility, neuromuscular coordination, and the willingness of the patient to perform the motion. This last item speaks to the patient's motivation and may, in some cases of severe apprehension, indicate the presence of tissue pathology or systemic disease. Under the special tests section of the cervical spine examination (S), the use of radiologic, neurologic, orthopedic, and vascular procedures will be discussed relative to the diagnosis of nonmechanical and organic pathological conditions.

Repeated Movements Exam for Cervical Derangement (Phases 1 to 4)

During the interview process of the examination, indications of an intervertebral disc derangement become apparent. As discussed in Chapter 2, the hallmarks of a McKenzie derangement[13,14] include the following:

- Symptoms during movement as compared to a dysfunction that is at end-range.
- Symptoms that may be constant and severe as compared to intermittent and mild to moderate.
- Symptoms that start proximal, but with time become more distal (ie, below the elbow).
- Symptoms that have neurologic features (ie, burning, tingling, shooting, sharp, piercing, etc).
- The presence of an acute deformity (ie, torticollis or wryneck).

When the McKenzie cervical derangement syndrome is suspected, the therapist can then proceed to placing the patient in 1 of 7 categories. An overview of these 7 derangements is as follows:

- Derangement 1: Central or symmetrical pain across C5 to C7; rarely scapular or shoulder pain, no deformity.
- Derangement 2: Central or symmetrical pain across C5 to C7 with or without scapular and/or shoulder pain. Deformity of flattened or flexed cervical spine.

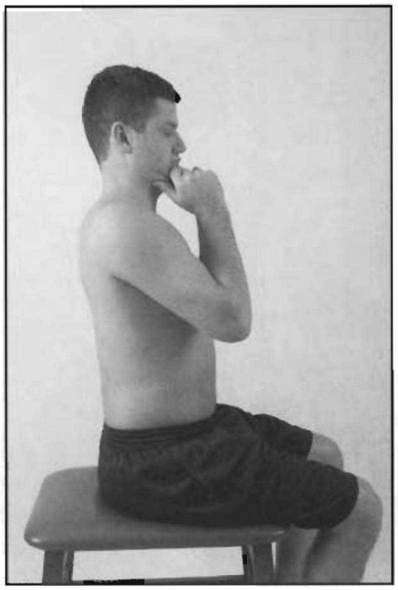

Figure 8-8a. Phase 1 head-neck retraction.

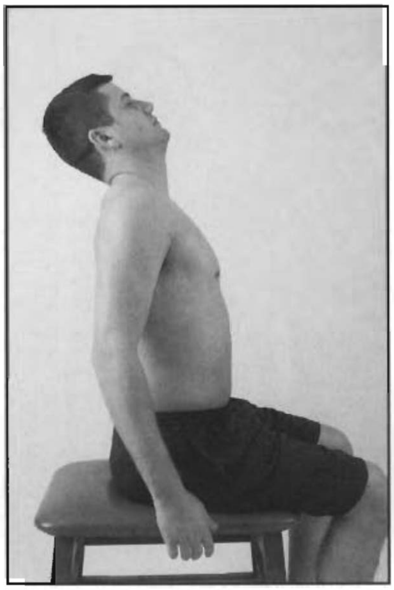

Figure 8-8b. Phase 1 head-neck retraction and extension.

- Derangement 3: Unilateral or asymmetrical pain across C3 to C7 with or without scapular and/or shoulder pain. No deformity is present.
- Derangement 4: Unilateral or asymmetrical pain across C5 to C7 with or without scapular and/or shoulder pain. With deformity of acute wryneck or torticollis.
- Derangement 5: Unilateral or asymmetrical pain across C5 to C7 with or without scapular and/or shoulder pain. Arm pain extends below the elbow. No deformity.
- Derangement 6: Unilateral or asymmetrical pain across C5 to C7 with or without scapular and/or shoulder pain. Arm pain extends below the elbow. Deformity of flattened or flexed cervical spine, acute wry neck, or torticollis.
- Derangement 7: Symmetrical or asymmetrical pain about C4 to C6 with pain occasionally referred to the anterior/anterolateral neck and throat. Obstruction of cervical flexion present.

The majority of cervical disc lesions occur at C5,6 followed by C6,7; the incidence of nerve root involvement is greatest at C6, followed by C7, C8, and C5 in decreasing order.[15]

The purpose of the repeated movements examination is to determine the responsiveness of the derangement to mechanical therapy. Theoretically, a contained disc displacement, be it annular or nuclear, should respond to the correct mechanical intervention with the centralization phenomenon (ie, symptoms become more proximal and therefore less distal) with the eventual resolution of all signs and symptoms. On the contrary, a noncontained disc herniation, as occurs in disc rupture, would not be expected to respond favorably to mechanical therapy. As discussed under contraindications (see Chapter 3), patients with neurologic signs should not be treated, but referred to the physician/surgeon for further consultation.

In the lower cervical spine, patients with derangements 1 through 6 are subjected to a series of mechanical phases, developed by the author, that begin with the simplest of procedures and progress to the more complex as needed. To achieve head-neck retraction, the index fingers and thumbs guide the motion; to prevent mandibular retrusion, the teeth are "lightly" clenched. Because derangement 7 is rare, it will not be addressed in this introductory textbook.

Phase 1

- Self-exam head-neck retraction. Upper cervical flexion/lower cervical extension (Figure 8-8a).
- Self-exam head-neck retraction followed by head-neck extension (Figure 8-8b).

Phase 2

- Self-exam head-neck rotation (Figure 8-9a).
- Self-exam head-neck side bending (Figure 8-9b).
- Self-exam combined head-neck retraction, rotation, and side bending (Figure 8-9c).
- Self-exam combined head-neck retraction, extension, rotation, and side bending (Figure 8-9d).

Phase 3

- Self-exam head-neck retraction in supine (Figure 8-10a).
- Self-exam head-neck rotation in supine (Figure 8-10b).

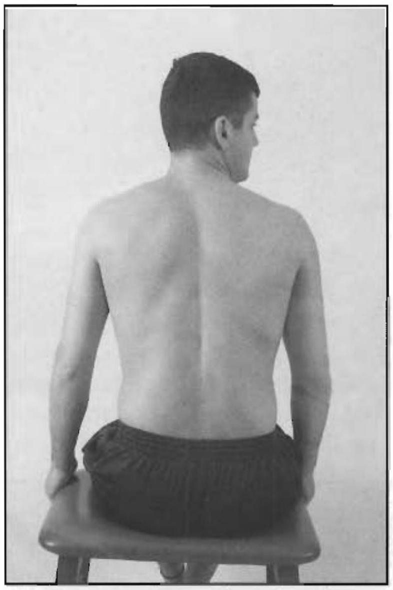

Figure 8-9a. Phase 2 head-neck rotation.

Figure 8-9b. Phase 2 head-neck side bending.

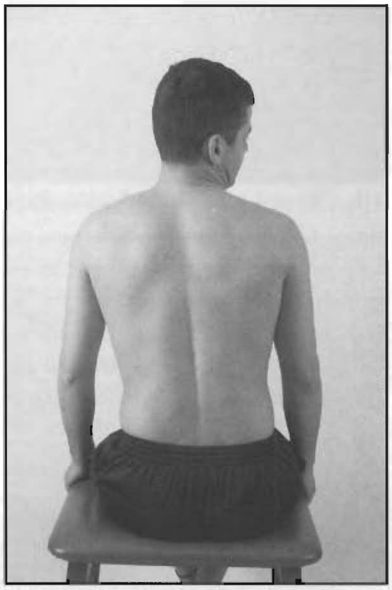

Figure 8-9c. Phase 2 head-neck retraction, rotation, and side bending.

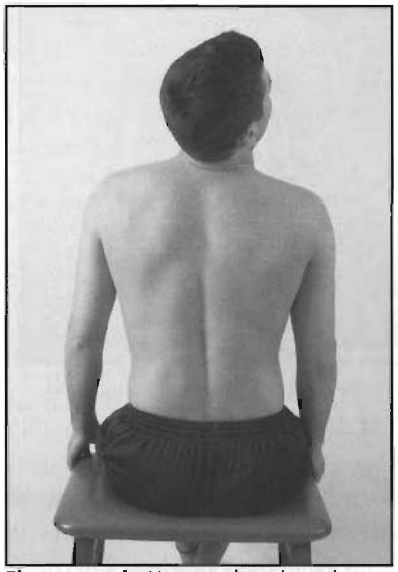

Figure 8-9d. Phase 2 head-neck retraction, extension, rotation, and side bending.

Figure 8-10a. Phase 3 head-neck retraction in supine.

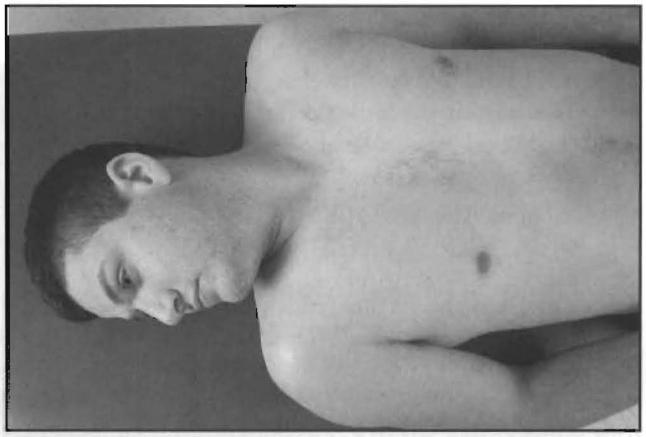

Figure 8-10b. Phase 3 head-neck rotation in supine.

Figure 8-10c. Phase 3 head-neck side bending in supine.

Figure 8-10d. Phase 3 head-neck retraction, rotation, and side bending in supine.

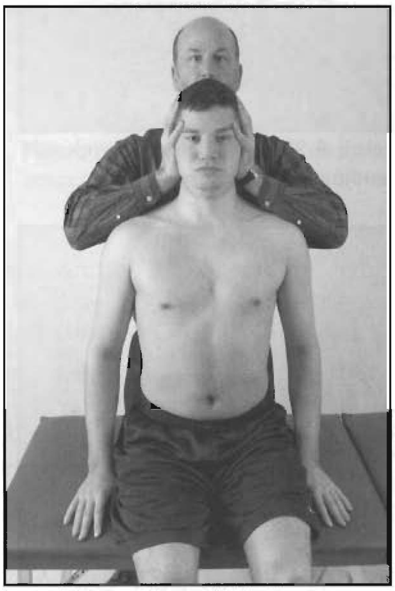

Figure 8-11a. Phase 4 traction and retraction in sitting.

- Self-exam head-neck side bending in supine (Figure 8-10c).
- Self-exam combined head-neck retraction, rotation, and side bending (Figure 8-10d).

Phase 4

- Therapist-assisted traction and retraction in sitting (Figure 8-11a).
- Therapist-assisted traction and retraction in supine (Figure 8-11b).
- Therapist-assisted traction, retraction, and extension in supine (Figure 8-11c).
- Therapist-assisted traction, retraction, extension, and rotation in supine (Figure 8-11d).

Guidelines to follow when performing the repeated movements exam include the following:

1. Sagittal plane movements (retraction and extension) are attempted prior to lateral compartment movements (rotation and side bending).

Figure 8-11b. Phase 4 traction and retraction in supine.

Figure 8-11c. Phase 4 traction, retraction, and extension in supine.

Figure 8-11d. Phase 4 traction, retraction, extension, and rotation in supine.

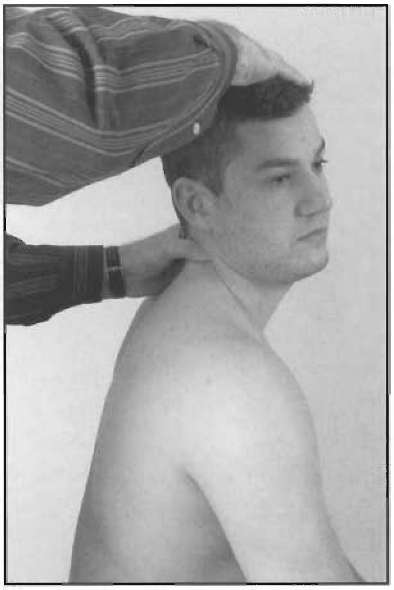

Figure 8-12a. Assessment of C2-C7 facet closing on the right.

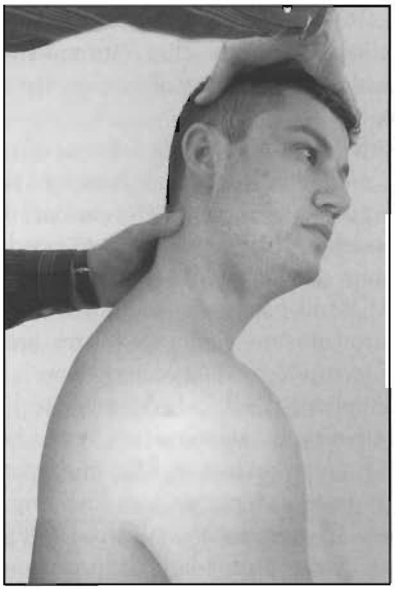

Figure 8-12b. Assessment of C2-C7 facet opening on the right.

2. Self-treatment is always attempted prior to therapist-assisted technique.
3. Sitting intervention is more functional than recumbent and should be attempted first.
4. After each set of 10 repetitions, the patient's symptoms are reassessed relative to the location and the intensity of the distal-most symptom. A 0 to 10 scale for rating intensity of the distal-most symptom is suggested. Any proximal migration of the distal-most symptom toward the cervical area (centralization) is considered a successful outcome and that motion(s) should be continued.
5. Progression to the next phase is suggested when the patient reaches a plateau.
6. If at any time the distal-most symptom is referred more distally (peripheralization), treatment should stop and the patient's intervention taken back to the previous phase if possible.

The repeated movements exam is used to accomplish the first goal of managing a derangement, which is to reduce it. The remaining 3 goals will be addressed in Chapter 11.

Apophyseal Joint Opening/ Closing (C2 to C7)

The following arthrokinematic examination (PPIVMs and PAIVMs) of the apophyseal joints of the lower cervical spine was developed by Mariano Rocabado. As mentioned previously, the arthrokinematic examination of the upper cervical spine is more suitable for advanced coursework and will not be covered at this time.

Apophyseal joint kinematics, including facet opening and closing, were reviewed in Chapter 1. The unilateral evaluation of cervical apophyseal joint motion is unlike any of the other spinal mobility tests in that the lower cervical region is the only area where the apophyseal or facet joints of the spine can be directly palpated. There are some guidelines that will hopefully elucidate the key aspecrs of this useful examination tool. The technique will be described for the patient's right side. The therapist stands on the right side of the sitting patient; the therapist's left hand lightly palpates the C2,3 facets with the thumb and the distal phalanx of the middle finger over the right and left facets, respectively (the C2,3 facets are at the level of the SP of C2, between the SCM muscle, anteriorly, and the upper trapezius, posteriorly). The therapist controls head-neck motion with the right hand over the cranial vertex. The key to the effectiveness of this procedure is proper localization to the appropriate joint level. For the assessment of C2,3 motion on the right, the head-neck region is rotated to the right until motion "arrives" at the left thumb. At this point, the therapist "rocks" the head-neck into combined extension/right side bending for the evaluation of facet "closing" (Figure 8-12a), then proceeds to "rock" the head-neck into combined flexion/left side bending for the evaluation of opening (Figure 8-12b). The patient's ears provide a useful landmark for establishing the direction of the forward and backward rocking motion (ie, the closing motion is in line with the right ear, while the opening motion is in line with the left). In addition to placing the left thumb over the C2,3 facet joint to assess for opening and closing restrictions, the left hand can also induce translation of C3 under the rolling C2 vertebra to assess for roll-gliding restriction of the C2,3 motion segment. Facet closing of C2,3 on the right is associated with translation of C3 to the left; opening with translation of C3 to the right. The information attained from this technique, as with other joint mobility tests, consists of the quality and quantity of motion, the end-feel, and tissue reactivity.

To assess the remainder of the lower cervical spine, the head-neck is rotated down to each level and the process is repeated. To assess the left side, the patient's head-neck is rotated to the left and all contacts are reversed.

In the lower cervical spine, flexion, rotation and side bending (FRS) and extension, rotation and side bending (ERS) impairments are common. There are no type 1 impairments from C2 to C7 because there is only type 2 mechanics. Applying the Rocabado sitting technique to our understanding of type 2 impairments, it can be said that a closing restriction represents an FRS impairment, whereas an opening restriction represents an ERS impairment. For example, the C4,5 segment is considered an FRS left when the right apophyseal joint is limited in closing. Limitation in arthrokinematic closing on the right is associated with limitation in osteokinematic extension, rotation, and side bending to the right. If C4 cannot freely extend, rotate, and side bend to the right, then it must be FRS left (the cause of cervical spine closing restriction is controversial with such possibilities as apophyseal joint irregularity, disc derangement, and meniscoid entrapment). Conversely, limited opening of the right C2,3 facet results in impairment of combined flexion, rotation, and side bending left of C2 on C3. Consequently, its position is opposite its restriction and is considered ERS right.

Jeffrey Ellis taught the **FOES** acronym for remembering the side of involvement in type 2 lesions. FOES stands for flexion opposite extension same. Consequently, the involved joint is on the opposite side with an FRS impairment, and the involved joint is on the same side with an ERS impairment ("o" can also refer to stuck open and "s" to stuck shut).

The previous sections on active cervical movements, repeated movements, and apophyseal joint kinematics are under the range of motion (R) category of the CHARTS examination. We will now proceed to tissue texture abnormality (T).

Soft Tissue Palpation

A review of cervical spine landmarks will prove helpful prior to the examination of relevant soft tissue structures in the head and neck region.

- External occipital protuberance. Bony prominence on the occiput at the level of the superior nuchal line.
- Inferior nuchal line. The inferior aspect of the occipital ridge.
- Mastoid process. Bony temporal bone prominence behind the ear.
- External jugular tubercle. Bony prominence on either side of the occiput just below the inferior nuchal line.
- SP of C2. The first palpable SP in the cervical spine (the posterior arch of atlas is not easily palpable).
- SP of C6. The next easily palpable SP in the cervical spine (the SPs of C3, C4, and C5 are small); upon extension of the head-neck, the SP of C6 translates forward.
- SP of C7 (vertebra prominens). The largest of the cervical SPs, which does not translate forward upon head-neck extension.
- Transverse process of C1 (atlas). Midway between the angle of the mandible and the mastoid process.
- Hyoid bone. Superior to the thyroid cartilage ("Adam's apple") in the anterior neck region.

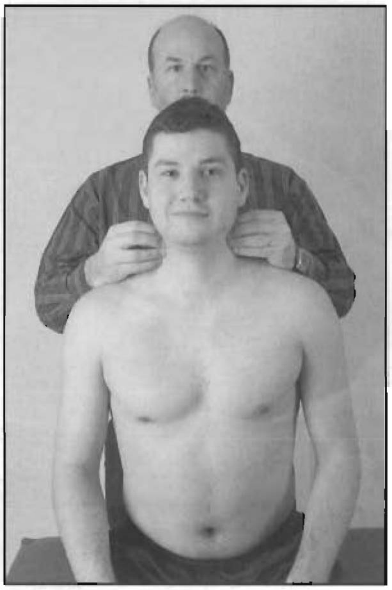

Figure 8-13. Palpating the SCM muscles.

The soft tissue examination of the cervical region inspects myofascial, articular, and neural structures for tissue texture abnormality. The presence of tenderness, tightness, and tone is recorded.

Anterior cervical palpation includes the following structures:

- Hyoid bone. Assess motion side to side.
- Supra/infrahyoid muscles.
- SCM muscles from the mastoid process to both the sternal and clavicular attachments (Figure 8-13).
- Scaleni muscles (anterior, middle, posterior). Palpated at the lateral edge of the midbelly of the SCM muscle. Contralateral side bending of the head-neck tightens the ipsilateral scaleni, making them easier to palpate.

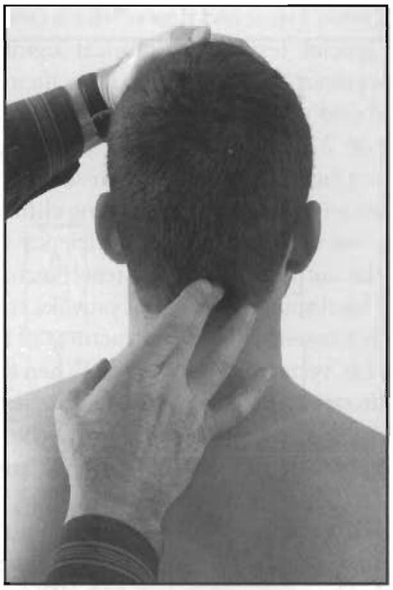

Figure 8-14. Palpating the right greater occipital nerve.

- Inferior clavicular region. Assessing attachment of the pectoralis major and fascia, clavipectoral fascia, superficial layer of the cervical fascia, subclavius muscle, and the coracoclavicular ligament (conoid and trapezoid ligaments).
- Pectoralis minor tendon. The coracoid process is palpated in the deltopectoral groove and the pectoralis minor tendon is accessed inferior to the coracoid. A deep inhalation will tauten the tendon, making it easier to palpate.

Posterior cervical palpation includes the following structures:

- Upper trapezius muscle. The therapist inspects for taut bands and myofascial trigger points.
- Levator scapula muscle. Palpated from the vertebral border of the scapula between the superior angle and root of the scapular spine, to the upper 4 vertebrae of the cervical spine.
- Posterior cervical muscles (splenius capitis/cervicis, semispinalis capitis/cervicis, longissimus capitis/cervicis, multifidi, and rotators). No attempt is made to distinguish one individual muscle from another. Palpation proceeds from caudal to cranial.
- Suboccipital muscles (rectus capitis posterior major/minor, rectus capitis lateralis, inferior/superior oblique). Slight passive extension of the occiput relaxes the superficial muscles, allowing access into the deeper suboccipital region. No attempt is made at this point to identify the individual muscles.
- Greater occipital nerve.[7] There are 4 potential sites of impingement:
  - In the upper trapezius
  - In the semispinalis capitis
  - Under the inferior oblique
  - Between the occiput and posterior arch of C1 when the occipitoatlantal space, as seen on a lateral radiograph, is less than 4 mm[16]

The optimal site for testing irritability of the greater occipital nerve is where it becomes subcutaneous, approximately 2 to 3 cm inferior and lateral to the external occipital protuberance. The forehead is stabilized with one hand; with the thumb or middle finger of the other hand, the nerve is compressed for approximately 10 seconds (Figure 8-14). Both sides are tested for irritability. A positive test consists of nerve-type discomfort (eg, burning, paresthesia, sharp pain) in the distribution of the nerve or over the ipsilateral eye where it has an anastomosis with the supraorbital branch of the ophthalmic division of the trigeminal nerve. A positive response is suggestive of a greater occipital neuralgia, which can be mistakenly diagnosed as migraine.

Special Tests

For the sake of clarity, this section will be organized as follows:

1. Neurologic
   a. Myotomes (C1 to T1)
   b. Dermatomes (C2 to T2)
   c. Deep tendon reflexes (biceps, triceps, brachioradialis)
   d. Cranial nerve testing[17] (I – identify various odors; II – visual field testing; III – upward, downward and medial gaze; IV – downward and lateral gaze; V – corneal reflex, face sensation, clench teeth; VI – lateral gaze; VII – close eyes tight, smile, whistle, puff cheeks; VIII – hear watch ticking, hearing tests, balance tests; IX - gag reflex, ability to swallow; X – gag reflex, ability to swallow, say "Ahhh;" XI – resist shoulder shrug; XII – tongue protrusion (observe for deviation)
   e. Upper limb tension tests 1, 2, 3, and 4
   f. Slump sit test (Figure 8-15)
   g. Upper motor neuron lesion (Babinski's sign)
   h. Valsalva's test (reveals space-occupying lesions in the cervical canal)
   i. Myelopathy hand[18]
   j. Hallpike-Dix maneuver for posterior or anterior canal benign paroxysmal positional vertigo (BPPV), roll test for horizontal canal BPPV, and Semont ("Liberatory") maneuver for BPPV secondary to posterior canal cupulolithiasis[19]
2. Orthopedic
   a. Spurling's compression test (maximal cervical compression)
   b. Cervical distraction test (relieves nerve root compression)
   c. Craniocervical flexion test,[20] neck flexor mus-

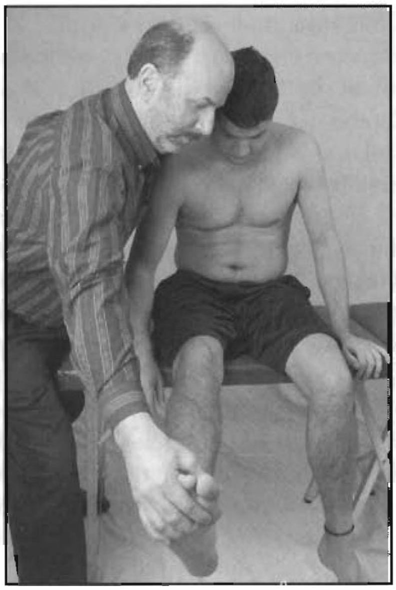

Figure 8-15. Slump sit test.

cle endurance test,[21] or the flexor endurance test (test of deep cervical flexor muscle endurance)

d. Cervical rotation lateral flexion (CRLF) test for a superiorly subluxed first rib[12]
e. Functional assessment (ie, the Neck Disability Index[10])
f. Neck torsion test[17,19] or Fitz-Ritson test[22] for cervicogenic dizziness (see Figures 12-3a and 12-3b)
g. Nine-point Beighton scale[23] for generalized hypermobility

3. Vascular
   a. Vertebrobasilar insufficiency (5 Ds, 3 Ns, 1 A)[24-27]
   b. Roos test for thoracic inlet (outlet) syndrome[1,10,28]
4. Physician based
   a. Radiologic – cervical x-ray series with mobility films, magnetic resonance imaging (MRI), magnetic resonance angiography (MRA), computerized axial tomography (CAT) scan with or without contrast, myelogram, etc.
   b. Electrodiagnosis (electromyography, conduction velocity, etc)
   c. Lab work (complete blood cell count, erythrocyte sedimentation rate, rheumatoid factors, HLA-B27 antigen, Lyme test, Epstein-Barr virus, antinuclear antibodies, etc)
   d. Tissue biopsy
   e. Sleep studies (sleep apnea, fibromyalgia/chronic fatigue, etc)
   f. Psychiatric/psychological evaluation

Regarding the abridged special tests' sections throughout this text, the reader is referred to other textbooks such as Cook and Hegedus,[1] Cleland,[2] Dutton,[10] Magee,[28] Konin et al,[29] and Gross, Fetto, and Rosen[30] for a complete review of relevant special tests, their clinical significance, and diagnostic accuracy (eg, sensitivity, specificity, predictive values, likelihood ratios). The author would, however, like to comment on 2 of the above items. The subject of vertebral artery testing is controversial. In addition to having poor test sensitivity[31] and therefore being clinically unhelpful in ruling out vertebrobasilar insufficiency (VBI), it has never been the author's practice to teach techniques in an introductory level spinal course that provoke transient ischemia to the brainstem and other structures of the posterior cranial fossa (ie, vertebral artery tests). When analyzing the risk-to-benefit ratio, there is not enough benefit to justify the risk. Consequently, at the basic level the 5 Ds, 3 Ns, 1 A[24-27,32] approach will suffice—any patient who presents with undiagnosed **D**izziness, **D**iplopia, **D**ysarthria, **D**ysphagia, **D**rop attacks, **N**ausea, perioral **N**umbness, **N**ystagmus, and **A**taxia should be seen by a neurologist to rule out VBI. The relationship between VBI and the cervical spine is such that blood flow in the vertebral artery is reduced physiologically at the level of C1,2 when the head-neck region is rotated or rotated and extended to the contralateral side.[33,34] This response is time dependent; therefore, patients should not be placed in these positions for extended periods of time. In those patients diagnosed with VBI or in the elderly, the extremes of these positions should be avoided entirely. In patients with diagnosed BPPV, therapy may include working through these positions that provoke dizziness,[19] but this should not be attempted without medical clearance (eg, Epley maneuver).

Regarding the potential risks of cervical manipulation (eg, cervical arterial dysfunction,[26,31,32,35-37] tissue disruption), the techniques presented in this book do not involve high velocity thrust to the neck nor the excessive use of force; if the contraindications to manual therapy (discussed in Chapter 3) are respected, the patient is at no time placed at risk for serious injury. Having said that, a working knowledge of cervical arterial dysfunction[24,26] (ie, disease of the vertebrobasilar system, the internal carotid arteries, and the circle of Willis, including local dissection, atherosclerotic events, vessel injury, as well as nonischemic and ischemic events) is highly recommended for practitioners involved in any form of manual therapy to the cervical spine. Such knowledge equips the therapist to recognize the presence of this serious condition so that patients can be referred to the appropriate medical specialist for management.

The second item that warrants discussion, relative to special tests, is the need to identify clinical instability when it exists. As a general rule, vertebral horizontal translation of greater than 3.5 mm on a flexion or extension x-ray and/or angular vertebral rotation of more than 11 degrees indicates the presence of segmental instability.[38-41] The upper cervical spine[10,42-44] in patients with rheumatoid arthritis, ankylosing spondylitis, Down syndrome, Grisel's syndrome, os odontoideum, Klippel-Feil syndrome, and a history of

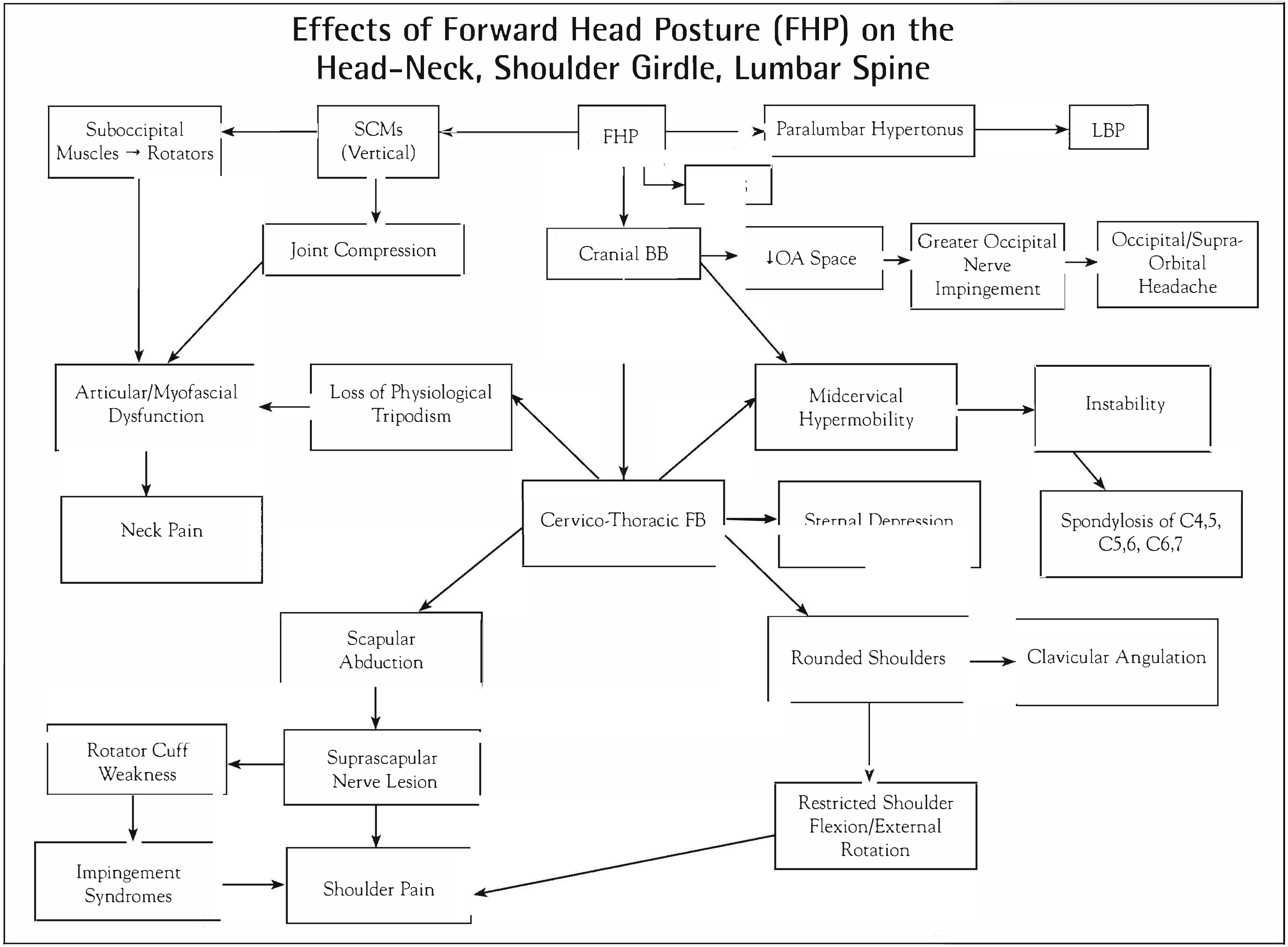

Figure 8-16. Global effects of FHP. SCM= sternocleidomastoid; LBP= lower back pain; TOS= thoracic outlet syndrome; OA= occipitoatlantal.

macrotrauma (eg, whiplash-associated disorders[20]) must have the atlantodental interval (ADI) assessed with lateral radiographs, including a flexion view, for signs of hypermobility/instability. In a typical adult, the ADI should be no greater than 3 mm. Patients are typically placed in hard collars with an ADI of more than 3 mm and considered for spinal fusion when greater than 5 mm.

Adverse Effects of Forward Head Posture

In ideal human anatomy, the head is anteriorly positioned with respect to the vertebral column. The term *forward head* implies excessive displacement of the head relative to the spine whereby the presence of abnormal muscle tensions may develop. Rocabado[41] uses the term *tripodism* to describe the normal balance that exists in the lower cervical spine (ie, C2 to C7) when vertebral function takes place at the posterior third of the intervertebral disc and the 2 apophyseal joints are parallel. In this balanced state, each member of the "tripod" (ie, the intervertebral disc anteriorly and the 2 facet joints posteriorly) bears equal weight. With forward head posture (FHP), tripodism is lost as the upper cervical spine at the occipitoatlantal junction extends (ie, "backward head") and the lower cervical spine and cervicothoracic junction flex (ie, "forward neck"), thus reducing the craniovertebral angle.
shifts forward onto its anterior aspect and away from the apophyseal joints.
suboccipital compression; lower cervical hypermobility, especially from C3 to C6 due to slackening of the nuchal ligament; off-center loading on the nucleus pulposus; elevation of the first 2 ribs from increased scaleni tension; and posterior/superior displacement of the mandible, which will be addressed in a subsequent chapter on the TMJ.
clinical perspective, these changes in head-neck alignment, in essence destabilizing one's posture, introduce the possibility of developing cervicogenic[20,45,47,48] and tension-type headaches,[45,49,50] midcervical clinical instability (leading to osteoarthrosis and spinal stenosis),[41,42,48,51] cervical disc derangement,[14] thoracic inlet (outlet) syndrome,[52] osteoporotic spinal deformity,[53] shoulder impingement,[54,55] and low back pain.[56] Other posturally related conditions include swallowing impairment,[16,57] reduced costal cage expansion during inhalation,[58] temporomandibular disorders,[59,] and fibromyalgia.[6]
such postural malalignment may have systemic effects as well.[62,63]

In addition to the loss of physiologic tripodism, forward displacement of the head increases the torque on the cervical spine. For example, given that the average head weighs 10 pounds, the torque on the cervical spine will increase by a factor of 10 for every inch of forward displacement (ie, torque = force x distance). Consequently, a forward head of 3 inches results in the equivalent of 30 in. lbs. of torque, whereas a 5-inch anterior displacement of the head results in 50 in. lbs. of torque on the neck in the direction of flexion. This nonphysiologic posture, in turn, places excessive demands on the cervical erector spinae muscles, which must produce an equivalent counter-torque for postural support.[64] Related to the global effects of FHP on the body (Figure 8-16), Alexander believed that the tensing of muscles in the neck (suboccipital/cervical extensors) results in the tensing of muscles of the whole body.[65]
simple experiment shows the veracity of this concept. In the forward head position, the extensors of the head-neck and spine can be felt to contract as far down as the lumbosacral junction. This appears to be a stabilizing response to gravity as the head-neck region is displaced forward. In addition, the shoulder girdle protracts, the rib cage sags forward, and the glenohumeral joints lose mobility. More indirect effects of FHP include hip adduction/internal rotation and rearfoot pronation of the feet.

Obviously, there is a large segment of the population that never experiences the untoward consequences of FHP as outlined above. However, as adaptive potential is compromised in response to different forms of stress[66]
misuse, abuse, and disuse), the likelihood of developing these ailments becomes greater. Much of what is done to improve health and ameliorate suffering in this book is based upon the balance of head, neck, and spinal alignment and the reduction, if not the elimination, of FHP. The subject of posture will be dealt with in greater detail in Chapter 25.

9

Connective Tissue Techniques and Stretching Procedures for the Cervical Spine

Lateral Neck Fascial Technique

This direct fascial technique (Figure 9-1) is useful for treating the levator scapula, upper trapezius, and posterior cervical muscles. The patient's occiput is placed on a head block or towel roll to create space under the cervical concavity. The examiner stabilizes the patient's head-neck region by placing one hand on the patient's forehead, while the other hand "rakes" through the soft tissues in a cross-fiber direction. A small amount of Deep Prep II or similar soft tissue massage cream is useful.

The therapist begins in the upper thoracic region and progresses cephalward into the upper neck area. An oscillatory motion can be added for additional soft tissue relaxation.

Deep Neck Fascial Technique

This direct fascial technique (Figure 9-2) is directed toward the deeper spinal muscles in the medial groove of longissimus. With the patient's occiput resting on the therapist's anterior forearms, the flexed PIP joints of both hands once again "rake" through the soft tissues from the upper thoracic region to the craniovertebral region. Upon encountering increased tone or tightness, the therapist maintains a superiorly directed force with the addition of oscillatory motion until the tension and/or tightness has abated. At the end of the caudal to cranial "sweep," the therapist imparts a gentle traction force on the occiput to stretch the posterior occipitoatlantal space. Several cycles can be applied as tolerated. This technique is excellent preparation for inhibitive occipital distraction, which is to follow.

Inhibitive Occipital Distraction

This procedure is a combination of direct fascial technique and manual traction. The first phase involves the use of digital compression for the purpose of inhibiting tone in the occipital extensors. The therapist supports the patient's occiput in his or her palms, with the second through fifth digits making contact with the skull over the inferior nuchal line (Figure 9-3a). The patient is asked to relax, breathe in through the nose and out the mouth, and imagine a quiet and tranquil scene that will enhance overall relaxation. As the subcranial soft tissues soften, the therapist is ready to progress the patient to the second phase. Now that the tissues have "let go" of their contraction, the occiput is distracted away from C1 by pulling it along the table in a cephalward direction toward the therapist. This separation of the occiput from the atlas creates more space at the occipitoatlantal junction, posteriorly, and essentially decompresses the region, including the greater occipital nerve. This sequence of neuromuscular inhibition followed by occipital distraction is repeated several times until the tissue slack has been removed.

Osteopathic practitioners describe a similar technique known as condylar decompression.[11] In addition to the inhibitive distraction described above, they incorporate a lateral release of the area. This lateral release (second phase of the technique) is achieved as the manual thera-

Makofsky HW
Spinal Manual Therapy, 2nd ed. (pp 77-84)

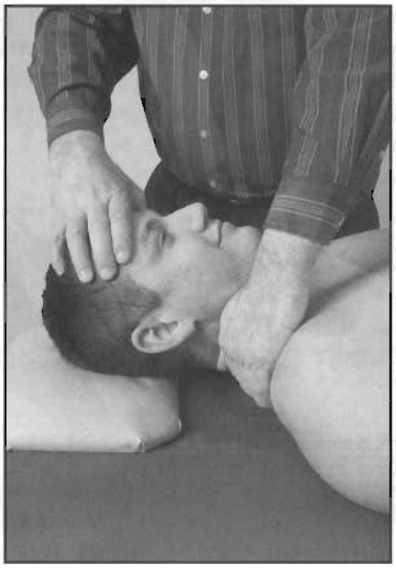

Figure 9-1. Lateral neck fascial technique.

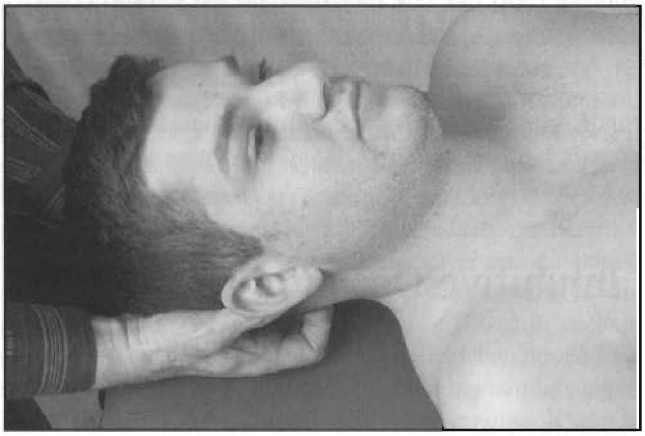

Figure 9-3a. Inhibitive occipital distraction phase 1.

pist increases bilateral forearm, wrist, and hand supination and brings both elbows together. The digital pressure is maintained until a release of tension is felt, especially the sensation of softening on each side of the occipital bone (Figure 9-3b).

At this point, the patient is progressed to the third phase in which the occiput is lifted off the therapist's palms and supported solely on the distal finger pads of all the extended digits save the thumbs (Figure 9-3c). This increase in pressure achieves further inhibition and allows additional separation of the occiput away from the atlas for maximal patient benefit. Progression to the third phase may not be possible in those individuals who find the increase in pressure uncomfortable (ie, pain, headache, and dizziness may result). Compression of the vertebral artery is avoided by maintaining pressure over the inferior nuchal line rather than between the occiput and atlas.

The author has modified the Paris technique of "inhibitive distraction" to arrive at its present form.[67]

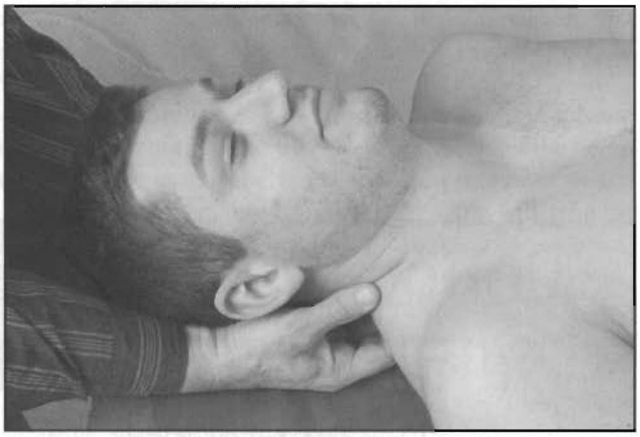

Figure 9-2. Deep neck fascial technique.

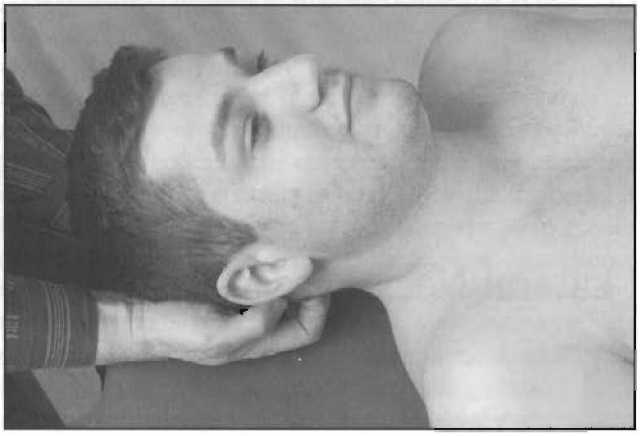

Figure 9-3b. Condylar decompression phase 2.

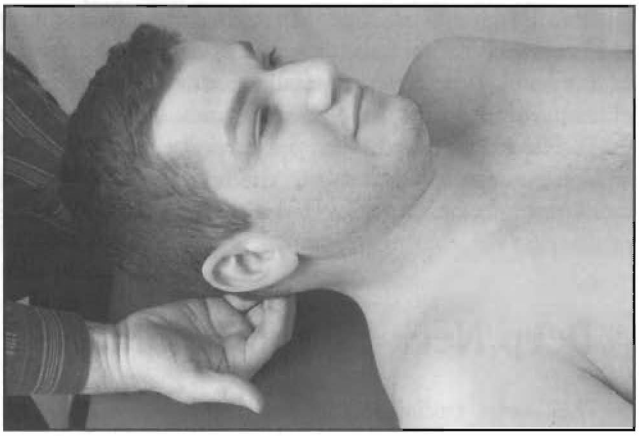

Figure 9-3c. Inhibitive occipital distraction phase 3.

Manual Traction/Functional Technique

The advantages of manual over mechanical traction include localization, feedback, specificity, and patient comfort. Some of the physiologic effects of traction include decompression of articular, neurologic, and vascular structures; soft tissue stretching; and mechanoreceptor

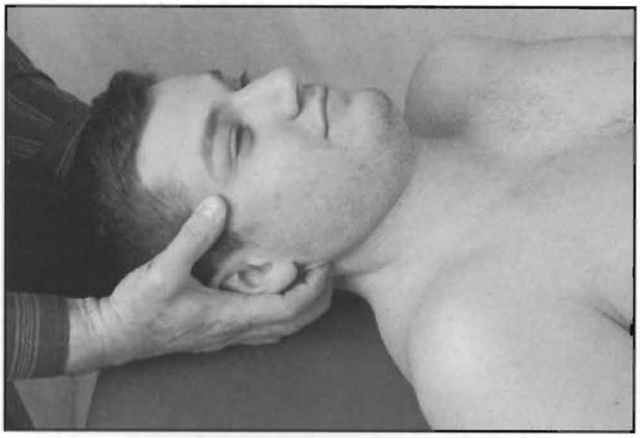

Figure 9-4. Manual cervical traction functional technique.

Figure 9-5. Right upper trapezius stretch.

stimulation for the relief of pain and reduction of muscle tone. It has been the author's experience that 5 minutes of effective manual traction is far superior to 20 minutes of mechanical traction.

To enhance the effectiveness of manual traction (Figure 9-4), the author has combined it with an osteopathic functional technique known as balance and hold.[11] The purpose of this "indirect" maneuver[68] is to reduce neuromuscular tone to a minimum prior to the application of traction. This "preparation" phase will enhance the efficacy of traction in that the muscular resistance to traction is minimized. This is a major problem with mechanical traction, as a "tug of war" struggle between muscle tone and traction leaves the patient caught in the middle.

The therapist's fingers monitor head-neck muscle tone with the fifth fingers on the suboccipitals, the fourth fingers on the posterior cervicals, the third fingers on the scaleni, the second fingers on the SCMs, and the thumbs on the temporalis muscles. At this point, the therapist seeks "ease" in head-neck side bending, rotation, and flexion-extension. When ease is found in one plane (eg, side bending right or left), the other motions are "stacked" one on the other in a final position of maximal tissue ease. This preferred tissue pattern represents the neuromuscular rest position (functional neutral) of the head-neck region and is the optimal starting position for manual traction. For one patient, this position may consist of 5 degrees side bending right, 7 degrees rotation left, and 5 degrees extension. For another, this position may be 3 degrees side bending right, 6 degrees rotation right, and 5 degrees extension. There are as many neuromuscular rest positions as there are patients to assess and treat. It is this tuning in process that makes functional technique so interesting and effective.

The actual traction is performed along the adjusted vertical axis of the head-neck region in a cephalward direction. For manual traction, the therapist has a choice of Kaltenborn[69] grade 1 (ie, support of the head and neck to achieve loosening), grade 2 (ie, to the end of the tissue slack), and grade 3 (ie, beyond the slack to patient tolerance). Between 5 and 10 repetitions of the appropriate grade should be applied in a "ramping" manner in both directions. The actual traction should be held for approximately 10 seconds.

At this point in the process, the balance and hold technique is repeated. As nociception and muscle splinting are reduced, the adjusted vertical axis and the true vertical axis of the head-neck approach each other. The therapist must, however, apply manual traction in functional neutral and not force the head-neck region into anatomic neutral. The therapist must also be careful not to squeeze the head too tight, especially during the more rigorous grade 3 traction.

The next 5 sections address specific stretches of the main muscle groups of the head-neck region. Each stretch will incorporate the postisometric relaxation concept (osteopathic muscle energy[11]) for enhanced treatment efficacy. When performing these procedures, therapists need to be careful not to inflame a healed cervical disc derangement or create one through the use of excessive force. If at any time patients report a peripheralization of their symptoms, the stretch should be immediately stopped. All stretching techniques will be performed in the supine position.

Upper Trapezius Stretch

The author recommends the use of contralateral side bending to stretch the fibers of the upper trapezius muscle. A stretch (Figure 9-5) will be described for the right side. The patient's right shoulder is depressed until the slack is removed, at which time the head-neck region is passively left side bent to the restrictive barrier with the therapist's left hand. The patient is then given the command, "Don't let me move your right shoulder down." Following a submaximal, isometric contraction of the right upper trapezius of 6 seconds duration, the right shoulder is depressed further (ie, the slack is taken up). It is important to wait a few seconds before moving into the new range as this allows for maximal postfacilitation inhibition of the muscle. The contraction-relaxation cycle is repeated 3 times. To stretch the left upper trapezius muscle, all contacts and instructions

Figure 9-6a. Right anterior and middle scalene stretch.

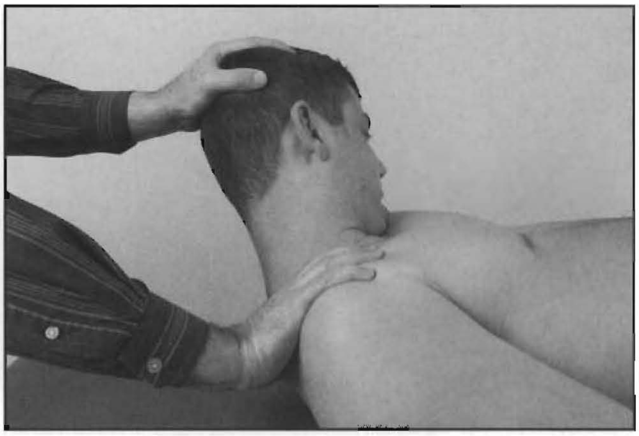

Figure 9-6b. Right posterior scalene stretch.

are reversed accordingly. To avoid technique-related injury, the patient is never stretched to the point of pain; peripheral symptoms should not be permitted, especially on the stretched side; and the head-neck region is always returned slowly to the midline.

Scaleni Stretch

Kinesiologically, the anterior, middle, and posterior scalene muscles have been treated, for the most part, as one combined system.[5] However, Evjenth and Hamberg[70] functionally separate the anterior and middle from the posterior scalene muscle. They ascribe the movements of flexion, ipsilateral side bending, and contralateral rotation to the anterior and middle scalenes and extension, ipsilateral side bending, and ipsilateral rotation to the posterior scalene. Consequently, 2 different stretches will be performed—one for the anterior and middle and one for the posterior scalene.

The anterior and middle scalenes, together, affect all levels of the lower cervical spine (ie, C2-C7). The optimal stretch of these 2 muscles is achieved with the head-neck region off the end of the table. To stretch the right side, the motions of head-neck retraction, left side bending, and right rotation are combined. The right first rib is held in depression by the therapist stabilizing the first rib with his or her right hand (Figure 9-6a). The patient is asked to resist passive side bending to the left. Following the postisometric relaxation, the head-neck is repositioned in further left side bending. The greater the head-neck retraction off the end of the table, the more effective is the stretch. For additional effectiveness, the stretch should be coordinated with the exhalation phase of breathing.

The posterior scalene affects the lower 3 levels of the cervical spine (C5, C6, and C7). To stretch the right side, the head-neck is flexed, left side bent, and left rotated as the first 2 ribs are stabilized with the therapist's right hand (Figure 9-6b). The therapist's command to the patient is, "Don't let me move you" as the therapist attempts to move, through a "shot put-like" motion, further into this combined position. As the patient's extensors, right side benders, and right rotators relax, the head-neck is taken further into the range of motion. This stretch can also be coordinated with exhalation for enhanced effectiveness.

Regarding the integration of different treatment approaches, hypertonicity and/or tightness of the middle scalene is associated with FRS[11,12] impairment from C2 to C7. Conversely, posterior scalene hypertonicity or tightness can be found with ERS[11,12] impairments in the neck from C5 to C7. With ERS impairments, the side of the restricted facet joint correlates well with ipsilateral posterior scalene dysfunction. However, the relationship between FRS impairment and middle scalene dysfunction is not as straight forward. For example, it appears that middle scalene tightness on the right has the potential to result in an FRS left impairment from C2 through C6. The explanation relates to the middle scalene's limiting affect on anterolateral translation of the inferior vertebrae to the opposite side. Regarding the influence of the scalenes on the first and second ribs, hypertonicity of the anterior scalene is thought to cause first rib superior subluxation, whereas posterior scalene hypertonicity is thought to contribute to a superiorly laterally flexed second rib.[11,12]

Care should be taken not to aggressively stretch the scaleni as the brachial plexus emerges between the anterior and middle scalene muscles and nerve entrapment here is a possibility. Entrapment of the lower trunk of the brachial plexus affects nearly all fibers of the ulnar nerve and some fibers of the median nerve. Patients with lower trunk compression complain mainly of paresthesia in the fourth and fifth digits, ulnar side of the hand, and occasionally of the forearm. Another type of nerve entrapment is possible when the C5 and C6 nerve roots pierce the middle scalene instead of passing between it and the anterior scalene.[71] The long thoracic nerve arises from the ventral rami of C5, C6, and C7. Because C5 and C6 frequently come together and pierce the middle scalene, tightness or aggressive stretching of the middle scalene has the potential to cause a long thoracic nerve palsy with weakness of the serratus

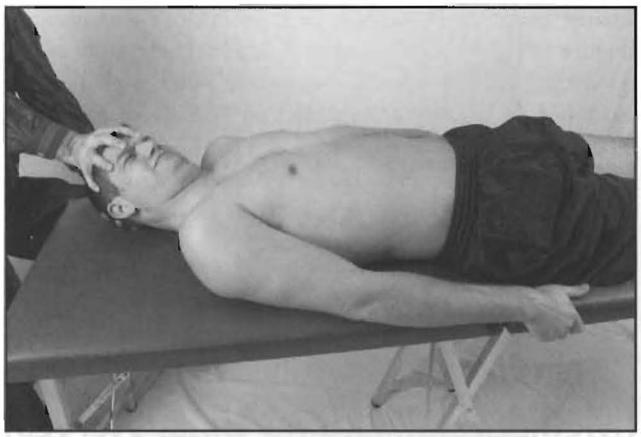

Figure 9-7. Right SCM stretch.

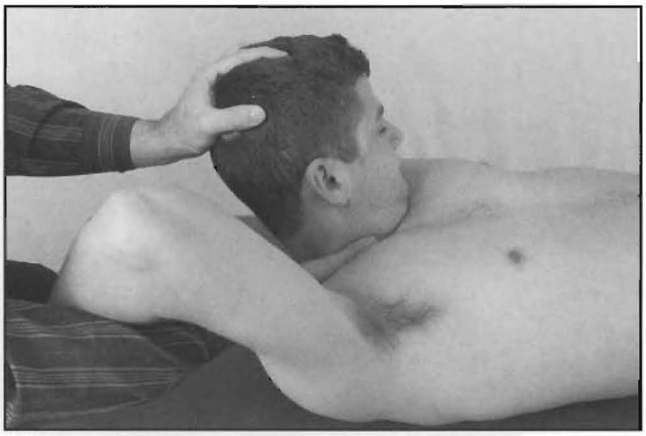

Figure 9-8. Right levator scapula stretch.

anterior and resultant winging of the scapula.[72] In addition, the dorsal scapular nerve also pierces the middle scalene and, if compressed by hypertrophy, spasm, or aggressive stretching, weakness of the rhomboids and levator scapula can develop. This has the potential to result in abnormal shoulder motion and mild scapular winging, leading to shoulder and neck pain.[73]

One final clinical note is that bilateral tightness of the scaleni muscles flexes the lower cervical spine and contributes to FHP.

Sternocleidomastoid Stretch

As with the scalene muscles, bilateral tightness of the SCMs also contributes to FHP. The bilateral stretch will be covered with the other occipital (capital) extensors below.

The unilateral SCM stretch is similar to the anterior/middle scalene stretch. The difference is the occipital flexion component, which was unnecessary for the scaleni because they have no occipital attachment. To stretch the right SCM, the therapist places his or her right hand on the patient's forehead while the left hand grasps the occiput. To stabilize the distal attachments, the patient's right hand holds the side of the table. The first phase of the stretch involves combined head-neck retraction, left side bending, and right rotation. The second phase incorporates occipital flexion, which is accomplished by a simultaneous push of the right hand and pull of the left hand (Figure 9-7). Care must be taken not to flex the lower cervical spine, as this will undermine the stretch. A useful image for the therapist is to envision the head rotating around an imaginary axis running through the patient's ears. This will ensure that the upper and not lower cervical spine is flexed during the stretch.

The isometric component is directed toward occipital extension. This is accomplished by having the patient look up and back with his or her eyes (oculocervical reflex). Following the 6-second contraction, the occiput is passively flexed. This cycle is repeated 3 times. If the technique is effective, the patient should feel a stretch at the right mastoid process.
may encroach upon the pharyngeal airway, patients need to indicate any respiratory distress immediately.

Levator Scapula Stretch

Tightness of the levator scapula muscle adversely affects the scapula, cervical spine, and shoulder complex.[74] It, like the posterior scalene, can cause ERS dysfunction in the cervical spine, albeit at higher levels (ie, C2, C3, and C4), and fixation of the atlas, resulting in headache and dizziness. In the shoulder complex, tightness of the levator scapula will contribute to downward rotation of the scapula. If upward rotation of the scapula becomes impaired, subacromial impingement may occur due to poor clearance of these tissues under the coracoacromial arch. Consequently, normal length of the levator is key to normal upper quarter physiology.[7]

There are different options for stretching this muscle, but the author's preferred method is to incorporate scapular upward rotation and depression from below and combined head-neck flexion and contralateral rotation/side bending from above. Because pushing (shot put-like motion) is preferable to pulling, this technique (Figure 9-8) involves guiding the head from below such that the head-neck region is pushed toward the contralateral side (nose to opposite hip). This follows the initial set-up in which the upper limb on the stretched side is elevated with the hand grasping the top of the table. To ensure scapular upward rotation and depression, the therapist holds the superior angle down with the radial aspect of the first metacarpophalangeal joint (MCP) contact (lateral knife-edge). If possible, the therapist should attempt to add posterior scapular tilt to depression/upward rotation with the first MCP contact. The head-neck region is then flexed to the contralateral side to the motion barrier. The patient is then asked to resist further motion for a count of 6 seconds fol-

Figure 9-9. Occipital extensor stretch.

lowed by movement of the head-neck region to the new barrier while the scapula is prevented from elevating and downwardly rotating. Of all the neck stretches, the levator stretch, along with the posterior scalene stretch, has the most potential to cause disc injury secondary to lower cervical flexion with contralateral rotation. Consequently, the patient must be monitored continuously for radicular symptoms. As usual, the cervical spine must be returned to neutral slowly to avoid facet joint compression.

Occipital Extensor Stretch (Suboccipital, Posterior Cervical, and Sternocleidomastoid Muscles)

The restoration of Alexander's primary control (ie, optimal and tension-free alignment of the head, neck, and upper back) is dependent on restoring normal length to the occipital extensors. Prior to restoring length to these muscles, the requisite myofascial extensibility, covered previously, should be attained.

The therapist uses a force-couple contact (as explained with the SCM stretch) with one hand under the occiput and the other hand placed on the forehead with the fingers directed caudally. The motion is that of occipital flexion, with the lower cervical spine in a neutral or slightly retracted position on the table. To ensure optimal stabilization of the lower cervical spine, the patient is instructed to grasp the sides of the table with both hands. Through the spinal "corkscrew" mechanism, scapulothoracic/lower cervical axial extension leads to upper cervical flexion and consequently stretching of the occipital extensors is enhanced. As with the SCM stretch, the therapist is advised to envision an imaginary axis running through the patient's ears about which the stretch occurs. This will ensure that the upper and not lower cervical spine comes into a flexed position. The isometric contraction of the occipital extensors is achieved with the patient's eyes looking up and back against therapist resistance for 6 seconds. The technique (Figure 9-9) is complete after 3 hold-relax-stretch cycles. As with the SCM stretch, the patient's pharyngeal airway must not be unduly compressed. A useful option to the bilateral stretch is to bias the force to the more restricted side. Some therapists use this unilateral technique as a form of occipitoatlantal flexion mobilization. When used as either a bilateral or unilateral flexion mobilization, both hands participate simultaneously.

Neurodynamic Mobilization (Median, Radial, and Ulnar Nerves)

The upper limb tension tests (ULTT 1, 2, 3, and 4) were mentioned under special tests (see Chapter 8),[28] but were not described or illustrated. Consequently, this section will provide information related to both the examination and intervention of adverse neural tension in the brachial plexus. Consistent with the philosophy of this textbook, this presentation will be streamlined to provide only the essentials on the topic.

Neurodynamic testing/intervention of the upper limb is recommended for patients presenting with nonirritable conditions of the head, neck, thoracic spine, and upper extremities.[76,77]

on neural structures, but in so doing other extraneural soft tissue structures are stressed as well. Contraindications include irritable conditions, inflammation, spinal cord signs, malignancy, nerve root compression, peripheral neuropathy, and complex regional pain syndrome (ie, reflex sympathetic dystrophy).

Median Nerve (ULTT1, ULTT2)

Two positions for the right median nerve will be described and illustrated, one in which shoulder elevation is blocked and the other in which shoulder girdle depression is utilized. In the ULTT1 (Figure 9-10), the supine patient's right upper limb is positioned sequentially as follows: shoulder abduction with restraint on shoulder girdle elevation, elbow extension, shoulder external rotation/forearm supination, wrist extension, and finger/thumb extension. Contralateral cervical side bending can be added if additional tension is needed.

The production of symptoms alone is not noteworthy (eg, deep ache, tingling, stretch), but rather the reproduction of the patient's symptoms. The earlier in the sequence the symptoms occur, the greater the likelihood of neural impairment.

The ULTT2 again stresses the median nerve, but this time shoulder girdle depression is included (Figure 9-11). The supine patient is positioned diagonally such that the right shoulder is at the edge of the table with the feet pointed to the left. The movement sequence of the right upper limb is as follows: shoulder girdle depression in slight abduc-

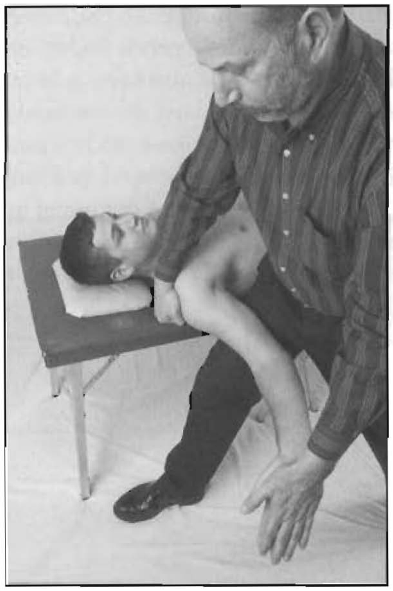

Figure 9-10. Upper limb tension test 1.

Figure 9-11. Upper limb tension test 2.

Figure 9-12. Upper limb tension test 3.

tion, elbow extension, forearm supination, wrist extension, and finger/thumb extension. Contralateral cervical side bending can be added as above, if necessary.

Radial Nerve (ULTT3)

To perform a test/intervention of the right radial nerve, a supine patient is positioned as above for the ULTT2. The therapist incorporates the following movements in sequential order: shoulder girdle depression, elbow extension, shoulder internal rotation/forearm pronation, wrist flexion, ulnar deviation, and thumb flexion (Figure 9-12). As above, it is the reproduction of the patient's symptoms that is significant.

Ulnar Nerve (ULTT4)

The patient is positioned in supine without being placed diagonally. For the right upper limb, the movement sequence is as follows: wrist extension, forearm pronation, elbow flexion, shoulder external rotation/abduction, and shoulder girdle depression (Figure 9-13).

As mentioned previously, contralateral head-neck side bending can be used to enhance all the above gliding movements for both examination and intervention purposes.

With regard to the treatment of peripheral nerve dysfunction, the therapist has the option of using either the "slider" or "tensioner" maneuvers.[78] The slider technique has been described as nerve "flossing" and involves sliding of the nerve along its bed without nerve elongation. The tensioner technique, however, involves elongating the entire length of the tract and is therefore a form of stretching. Because the risk of nerve injury is greatly reduced with the slider, it is the author's treatment of choice. Once the nerve dysfunction is identified, "flossing" is performed for approximately 1 minute. If symptoms persist after several treatment sessions, a gently applied tensioner technique can be attempted

Figure 9-13. Upper limb tension test 4.

at the point in the range just before the patient's symptoms appear. It should be done in a gentle on/off manner for 5 to 10 cycles. The therapist must also keep in mind that peripheral nerves traverse tunnels, spaces, myofascial tissues, etc. Consequently, connective tissue techniques are usually necessary in conjunction with neural mobilization in order to achieve the desired restoration of normal mobility in the nervous system. Because of the sensitive nature of nerve tissue, patients are not generally instructed in self-mobilization lest injury result.

10

Cervical Spine Manual Therapy

As previously mentioned, manipulation of the cervical spine will not include the occipitoatlantal and atlantoaxial joints. The examination and treatment of the upper cervical spine is usually taught to students and therapists who have completed coursework in basic spinal manual therapy. The reasons for this are many and include the complexity of craniovertebral kinematics, the influence of craniovertebral motion on vertebrobasilar blood flow (posterior circulation[24]), the risk of treating undiagnosed upper cervical instability, and the relationship between the high cervical segments and the upper cervical spinal cord. However, the author is confident that the interventions covered in this textbook will enable the treating therapist to successfully manage the majority of cases seen without incurring the risk of the advanced upper cervical procedures.

Apophyseal Joint Closing Restriction

The restriction of apophyseal joint closing from C2 through C7 is managed similarly to the examination technique described in Chapter 8 (Figure 10-1). However, the intervention, unlike the examination, involves the use of muscle energy technique[11,12] (postisometric relaxation or hold-relax) as well as graded mobilization against the restrictive motion barrier. It is hypothesized that a short duration, submaximal isometric contraction is followed by the down-regulation of muscle tone.[79] Consequently, reduced tone allows for an increase in range of motion in the opposite direction (eg, an isometric contraction of the biceps is followed by reduced biceps tone, resulting in improved range in elbow extension). The manual traction/functional technique, described in the previous chapter, is an effective means of preparing the facet joints for FRS correction (it must be kept in mind that the "closing" of a spinal facet joint must be performed carefully because of the associated compression and with minimal force lest symptoms become exacerbated).

For the successful management of a C2,3 FRS left, the therapist localizes the combined motions of extension, rotation, and side bending right to the feather-edge of the restrictive barrier by pulling the head towards the right ear with the right hand while gently palpating the right C2,3 facet joint with the left thumb. The command given to the patient is, "Don't let me move you," as the therapist attempts to move the head-neck back and to the right (ie, the patient resists the therapist's force by attempting to move the left ear towards the left axilla). This 6-second isometric contraction is followed by a relocalization against the closing barrier of C2,3 and repeated for a total of 3 cycles. Following the muscle energy technique (MET), the dysfunctional segment is reassessed as in Chapter 8. If motion has been restored to the segment, the therapist is finished. However, if restriction persists, graded mobilization/manipulation for 30 to 60 seconds is performed. As with the thoracic spine, grade 1 and 2 techniques require a monitoring contact over the left C2,3 facet joint for localization. However, grade 3 and 4 techniques require a stabilizing force on C3 (ie, a manual block) against which C2 is "closed." Again, as with thoracic mobilization/manipulation covered in Chapter 6, a hold one, move one or roll-glide

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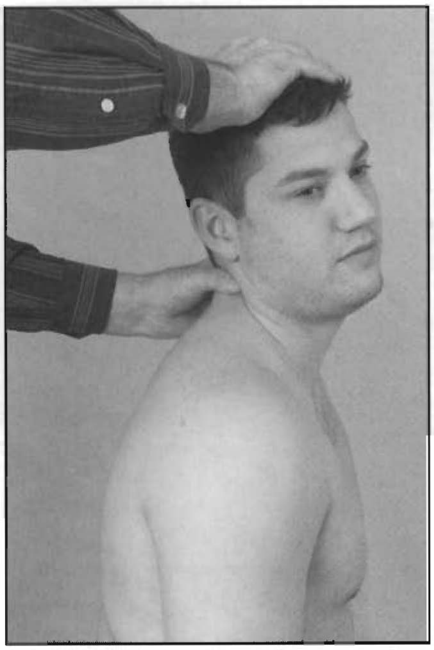

Figure 10-1. FRS left correction C2-C7.

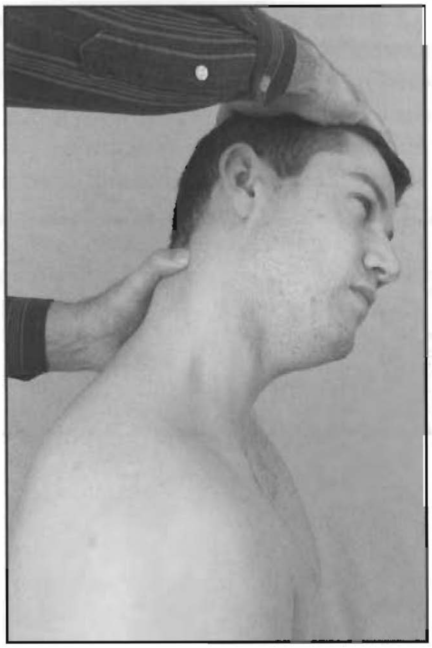

Figure 10-2. ERS right correction C2-C7.

technique is utilized based on preference and skill level. The author's preferred manipulation involves a roll-glide in which C2 is rolled over C3 in a diagonal plane back and to the right, as C3 is translated under C2 diagonally forward and to the left.

Apophyseal Joint Opening Restriction (Extended, Rotated, and Side Bent—ERS)

The treatment of an ERS right impairment at C2,3 is similar to the examination procedure for an apophyseal joint opening restriction (see Chapter 8). Once the head-neck region is localized to the feather-edge of the restrictive barrier in C2,3 opening on the right, the therapist proceeds with the MET (Figure 10-2). As described above for an FRS left, there are 3 steps to the MET. They are 1) localization to the feather-edge of the restrictive barrier, 2) a therapist-controlled short duration/submaximal isometric contraction by the patient in the opposite direction(s), and 3) relocalization to the new restrictive motion barrier. These 3 steps are then repeated 3 times and the dysfunctional segment is reassessed. If at this point the ERS impairment persists, the therapist has the option of performing graded mobilization/manipulation for 30 to 60 seconds.

Students often question the head-neck position in this technique. Though it is true that the combined physiologic motions of C2 flexion, rotation, and side bending to the left will open the C2,3 facet on the right, it is also true that rotation of the head to the right in no way interferes with the mechanics of this technique. This is because upper cervical kinematics are out of phase with those of the lower cervical spine, and even though the head is rotated right, the mechanics from C2 to C7 will necessitate that left side bending be coupled with left rotation (type 2 mechanics). Although it is possible to perform this technique with the head rotated to the left, the principles of localization, balance, and control are optimal when performed as illustrated with the head rotated to the right (see Figure 10-2).

Graded mobilization/manipulation for an ERS right requires the therapist to passively direct the patient's head toward his or her left ear with either 1) a right C2,3 facet joint monitoring contact with the left thumb for grades 1 and 2, 2) a blocking contact on the left side of C3 with the left middle digit (hold one, move one) for grades 3 and 4, or 3) a gliding motion of C3 under C2, towards the therapist, with the left hand (roll-glide), for grades 1 through 4.

The Rocabado sitting technique described in this chapter is used as an intervention for all FRS and ERS impairments from C2 through C7. Although the supine muscle energy techniques[11,12] can be equally effective, with the advantage of reduced muscle activity, the sitting approach has the advantage of ease of application (ie, no table required) and optimal three-dimensional control of the cervical structures. In addition, people tend to spend more time sitting than recumbent; therefore, sitting interventions are more functionally oriented.

Regarding cervical derangements, there appears to be a correlation between FRS impairment and disc herniations especially at C5,6 and C6,7. For example, an FRS right at C5,6 may lead to either a contained or noncontained disc herniation posterolaterally on the left and vice versa for an FRS left. Although the McKenzie approach has established merit in management of derangement syndrome,[13,14] the osteopathic muscle energy[11,12] approach is also useful in this regard. If MET is to be used in the management of cervical derangements, the patient's symptoms should be continuously monitored as a means of assessing improvement (centralization) or worsening (peripheralization) of the patient's condition. At no time should MET be used for this purpose if frank neurologic signs are present (eg, sensory loss, muscle weakness, atrophy, areflexia). In addition, signs of upper motor neuron disease (eg, Babinski's sign) and/or cervical arterial dysfunction[26] are an absolute contraindication to any form of cervical spine manual therapy in addition to those listed in Chapter 3.

One final comment is in order. In the author's opinion, there is no need for thrust procedures in the cervical spine. Being the most mobile region of the vertebral column, the cervical spine should be handled with gentle forces lest clinical instability[41,42] develops. In addition, the vertebrobasilar (posterior circulation) and internal carotid/circle of Willis (anterior circulation) arterial systems are at some risk of injury with thrust procedures compared to MET and nonthrust techniques, which are just as effective without the associated risk.[24-]

11

Therapeutic and Home Exercises for the Cervical Spine

Chin-Tuck

There are 2 variations of the chin-tuck that are taught to patients: the basic chin-tuck and the "corkscrew" chin-tuck. The basic chin-tuck is a simple way of achieving occipital flexion and elongation of the occipital (capital) extensors. The patient should be positioned in standing with his or her back to the wall. The patient is asked to imagine a "rope" attached to the back of the head, approximately half-way between the top of the head and the most inferior aspect of the occiput (anatomically, this corresponds to the junction of the sagittal and lambdoid sutures known as lambda). This rope, when tightened, pulls the occiput out of "downward pull" in a "forward and up" direction and simultaneously causes the chin to move back and slightly down toward the hyoid bone, which is just above the Adam's apple in the throat. According to Alexander,[80-82] this restoration of "primary control" has the effect of lengthening and widening the torso. Placing the index finger just below the nose (Figure 11-1) helps to guide the motion of craniovertebral flexion.

The patient's head-neck region is in contact with the wall at all times. The stretch is held for 30 seconds at the point where the tissues at the back of the skull begin to feel the stretch (as with the other self-stretches, the patient begins with a 5- to 10-second stretch and progresses up to 30 seconds as tolerated). The basic chin-tuck is performed 3 times and repeated every 2 hours. As with all self-stretches, the patient must not elicit pain at any time. The most common error seen among patients is that they flex not at the occipitoatlantal junction but in the midcervical region. This movement is not only ineffective in restoring normal head, neck, and spinal alignment, but contributes to the problem of midcervical hypermobility. To avoid this, the patient is given a visual aide to assist with occipitoatlantal flexion. The patient is asked to place an imaginary "axis" through the ears as he or she rotates around it. This, in conjunction with keeping the eyes level and not looking down toward the floor, ensures that the motion occurs in the upper and not lower cervical area. Eye-head coordination[20] can be enhanced by having the patient look up as the chin is tucked in. This will train the patient to look up with the eyes without having to extend the occiput, which will assist with maintaining neutral posture of the head-neck and spine.

The advanced variation, known as the corkscrew chin-tuck, is so named because of how it resembles the workings of a wing style corkscrew (Figures 11-2). As illustrated, the head, neck, and spine of the corkscrew are driven cephalward into length by depression of its "shoulder complex" as per Newton's Third Law (ie, "To every action there is always an equal and opposing reaction"). In the human structure, this "ratcheting up" of the head, neck, spine, and sternum is believed to occur at the rib articulations and driven by a reverse action contraction of the lower trapezius muscles (Figure 11-3). As the shoulder girdle is depressed or retracted, the rib angles are depressed. However, the costovertebral, costotransverse, and costosternal joints move in a cephalward direction, providing an upward "lift" to the spine and sternum in the opposite direction. The converse is also true; relaxation of the lower trapezius muscles allows for shoulder girdle elevation/protraction, which causes the torso to functionally shorten. The clavicles at the

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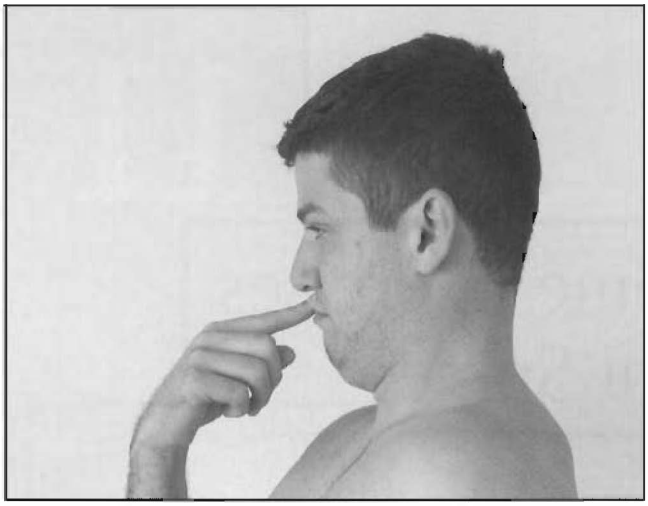

Figure 11-1. Basic chin-tuck.

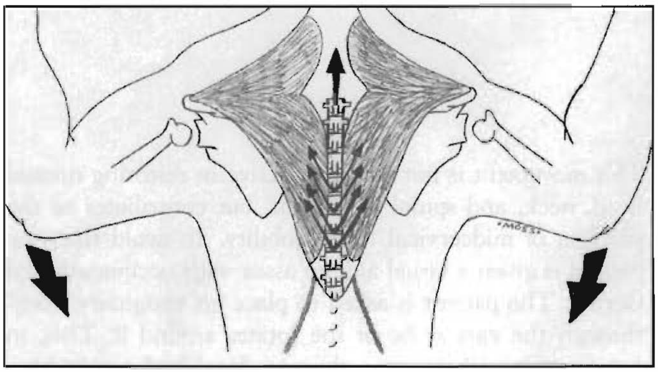

Figure 11-3. Lower trapezius vertebral lifting mechanism. (Illustration by Neil Moss.)

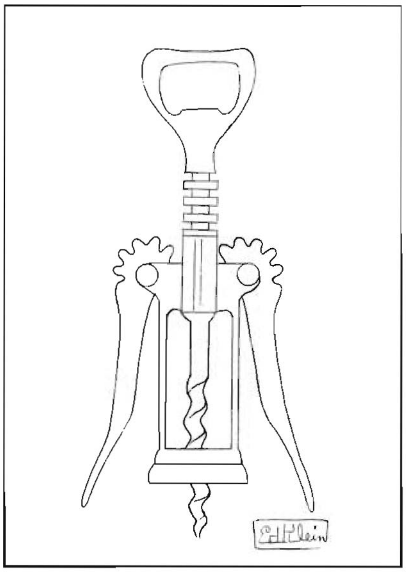

Figure 11-2. Spinal "corkscrew" principle. The spine is lengthened. (Illustration by Ed Klein.)

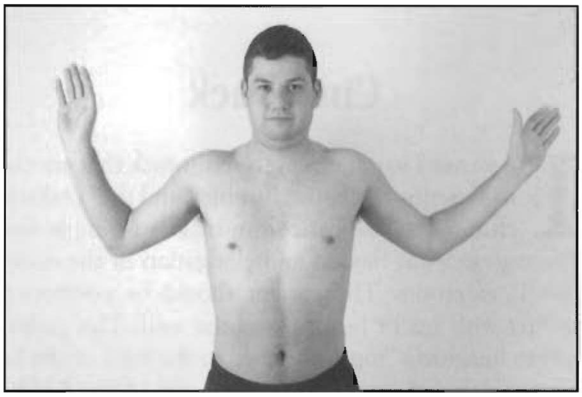

Figure 11-4. Corkscrew chin-tuck.

sternoclavicular joints most likely contribute to this corkscrew mechanism as well (ie, with shoulder girdle depression/retraction, the sternal ends of the clavicles provide a "lift" to the trunk, whereas with elevation/protraction of the shoulder girdle, they allow a "collapsing" down of the torso). As the spine functionally lengthens in response to shoulder girdle depression/retraction, the occiput naturally flexes on the cervical spine. This is because the lower cervical spine extends with spinal lengthening, causing the upper cervical spine to flex (the upper and lower cervical spine are out of phase such that upper cervical flexion causes lower cervical extension and vice versa). Consequently, there is a correlation between depression/retraction of the shoulder girdle and flexion of the craniovertebral region.

The corkscrew chin-tuck exercise is similar to the basic chin-tuck except for the shoulder girdle component (Figure 11-4). As the patient performs a "framing the doorway" motion of the upper extremities, he or she again imagines a rope pulling the back of the head forward and up as the chin approaches the hyoid bone just superior to the thyroid cartilage. It is believed that the descending shoulders and scapula enhance the chin-tuck as hypothesized in the spinal corkscrew principle described above. This exercise can be performed as a stretch or as a strengthening maneuver. When performed for strengthening purposes, it is done 10 times, with a hold of 5 to 10 seconds, 3 times per day. Another variation on the corkscrew chin-tuck (not illustrated) is to have the sitting or standing patient place his or her hands, with fingers interlocked, on top of the head with the elbows in the plane of the scapula. As the scapulae are adducted or depressed, the head is forced cephalward against the resistance of the patient's hands. In this manner, the spinal corkscrew is strengthened and posture is "jacked-up!"

Based upon this spinal corkscrew principle, the Occivator (SomatoCentric Systems, Inc, Toronto, Ontario, Canada), a postural/exercise retraining device, has been developed by this author.[83,84] As illustrated (Figure 11-5), its purpose is to lengthen the spine and improve postural alignment by directing the occiput up and forward on the neck as the shoulder girdle is simultaneously directed down and back.

Figure 11-5. Posture correction utilizing the Occivator.

In addition to its obvious mechanical effects, this newly developed system helps to break poor postural habits while establishing new ones.

Management of Cervical Derangement (Phases 1 to 4)

The self-treatment model for a lower cervical derangement is based upon the patient's response to the repeated movements exam previously covered in Chapter 8. Because the photographs of the various phases (1 to 4) are the same for both examination and treatment, those taken previously will not be repeated here. The reader is, therefore, encouraged to cross-reference the appropriate treatment phase with the corresponding photograph in Chapter 8.

The management of a disc derangement is more of an art than a science. The author trusts that the following suggestions will serve as guidelines for the treating therapist:

1. The patient must be responsive to this intervention (ie, the patient must demonstrate the McKenzie centralization phenomenon during the repeated movements exam).
2. The phase selected for the home program is ideally phase 1. The intervention phase is escalated to the higher phases only when required.
3. The patient must demonstrate proficiency with self-treatment in the clinic before he or she can be trusted to perform it at home.
4. The patient must stop the exercises if the symptoms peripheralize; however, a mild increase in intensity is permitted as long as it is in a centralized direction.
5. Head-neck extension in supine is permissible only under therapist supervision and not at home. This is because of the adverse effect of cervical backward bending on vertebral artery blood flow as well as possible spinal canal narrowing in those with spinal stenosis. In the clinic, signs of distress can be monitored, but at home, over the end of the bed, serious complications without assistance may ensue. Sitting extension at home is permitted (phase 2) because the patient can easily alter head and neck position without difficulty, if necessary.
6. Therapist-assisted technique (phase 4) is used as a last resort. Self-treatment is always the preferred approach with McKenzie.
7. The patient must be committed to performing the home program every 2 hours. The number of repetitions depends on the response to treatment. At least 3 sets of 10 repetitions are recommended; however, additional repetitions are allowed, providing the symptoms are improving.
8. In addition to the repeated movements component of derangement reduction, the patient needs to concentrate on maintaining the reduction in order to allow healing to occur. In this regard, instruction in proper posture (eg, avoid forward head positions) and the use of a cervical support pillow are mandatory. It is imperative that the cervical lordosis be preserved day and night lest the deranged tissue be reinjured. There are many such pillows on the market. There are 3, in particular, that the author currently recommends. The Tempur-Pedic Swedish neck pillow (Tempur-Pedic Inc, Lexington, KY), the Mediflow water-based pillow (Mediflow Inc, Markham, Ontario, Canada), and the McKenzie roll (OPTP, Minneapolis, MN).

Once the derangement is reduced and stabilized, the final goals are to recover lost function and prevent recurrence. The recovery of function will be addressed in subsequent home exercises. The prevention of recurrence is multifactorial, including postural correction, normalizing strength of the weak phasic muscles, addressing ergonomic factors at home and in the workplace, stress management, etc.

Regarding the management of derangements that do not respond to mechanical therapy, the next step would be pain management in a multidisciplinary pain clinic for those patients who are not surgical candidates. For those patients who are surgical candidates, referral to a spine surgeon is the next step. The indications for surgery include intractable pain and suffering; frank neurologic signs (ie, sensory loss, reflex changes, muscle weakness, atrophy, Babinski's sign, etc); diagnostic confirmation of pathology with MRI, MRA, CAT scan, discography, myelography, etc; and failed conservative therapy. This final item does not mean the use of pain-relieving modalities alone, but in conjunction with manual therapy and therapeutic exercise. The decision regarding spinal surgery is often a difficult one and requires the combined input of a team of professionals working together for the good of the patient. Having said that, spine surgery today is safer, more effective, and less costly than in

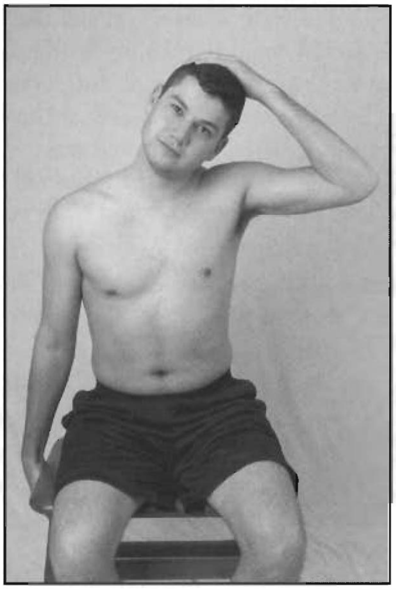

Figure 11-6. Right upper trapezius stretch.

the past. Surgical procedures developed in the past 10 years have revolutionized spine surgery practice and it may not be the last resort that it once was.[85,86]

Active Cervical Range of Motion

The use of cardinal plane active range of motion exercises is especially useful for those patients who cannot tolerate the other home exercises described in this chapter. Patients who are elderly and those with pathology of the spine, such as rheumatoid arthritis, ankylosing spondylitis, severe osteoporosis, etc, can experience the benefit of movement therapy without the risk of tissue disruption or injury (providing that their condition is not so severe as to preclude the use of active motion). The patient's instructions include the following:

1. Assume an upright head-neck position in either sitting or standing.
2. Turn slowly to the right until a slight stretch is experienced; return to the midline position and repeat 3 times.
3. Turn slowly to the left until a slight stretch is experienced; return to the midline position and repeat 3 times.
4. Tilt the head-neck region to the right (ie, ear to shoulder) and repeat 3 times.
5. Tilt the head-neck region to the left and repeat 3 times.
6. Bend the head-neck forward beginning with a chin-tuck and repeat 3 times.
7. Perform a chin-tuck followed by backward bending of the head-neck region; repeat 3 times. If at any time the patient becomes dizzy or apprehensive when tilting his or her head backward, he or she is to stop immediately. These symptoms may be the early warning signs of cerebral anoxia.[11]

These exercises are to be performed at least 3 times per day, but this can be modified by the therapist as indicated.

Upper Trapezius Self-Stretch

To stretch the right side (Figure 11-6), the sitting patient is instructed to grasp the bottom of the chair with the right hand. With the left hand, the patient pulls the head-neck region toward the left shoulder (the motion involves straight side bending to the side opposite the stretch). The patient is instructed to stop at the first indication of a stretch, hold for 30 seconds, return slowly to the start position, and repeat 3 times every 2 hours. At no time should peripheral symptoms in the right upper limb be experienced. Experiencing these symptoms would suggest either an active derangement and/or adverse neural tension that should be avoided in this simple muscle stretch.

For the more coordinated and intelligent patient, a self-muscle energy component can be added to the stretch for enhanced efficacy (eg, a 6-second isometric contraction of the right upper trapezius followed by the stretch).

Scaleni Self-Stretch

As with the distinction in function made between the anterior and middle with the posterior scalene in Chapter 9, so too the self-stretch for the scalene muscles must separate the anterior and middle components from the posterior one. The self-stretch for the right anterior and middle scalenes is similar to the upper trapezius stretch described above; however, the patient's head-neck region is positioned in retraction (upper cervical flexion and lower cervical extension), side bending left, and slight rotation right (Figure 11-7a).

The right posterior scalene self-stretch involves head-neck flexion, side bending, and rotation to the left (Figure 11-7b). The purpose of grasping the chair with the right hand is to maintain first and second rib depression during the stretch. Both stretches are held for 30 seconds and repeated 3 times every 2 hours. Again, the patient must be careful not to overstretch and should stop immediately if symptoms peripheralize.

Sternocleidomastoid Self-Stretch

To stretch the right SCM, the patient's right hand grasps the bottom of the chair. The initial phase is to tilt the head-neck region to the left shoulder in a position of retraction and slight right rotation. The next phase is what separates

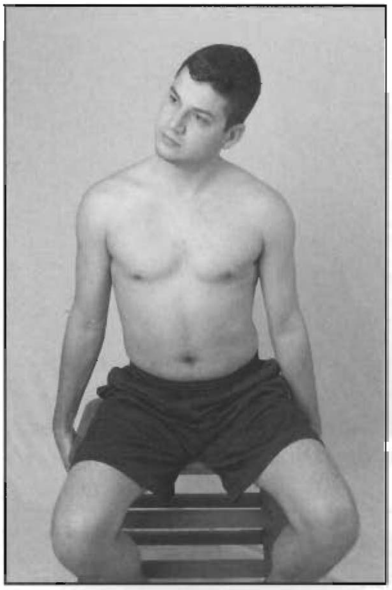

Figure 11-7a. Right anterior and middle scalene stretch.

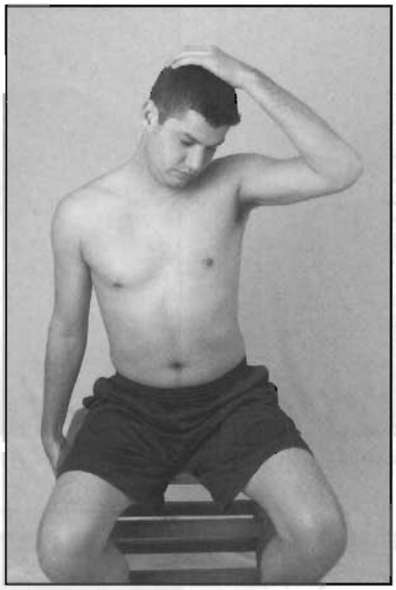

Figure 11-7b. Right posterior scalene stretch.

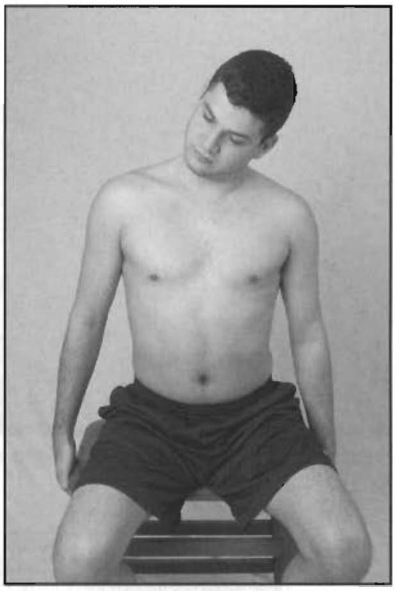

Figure 11-8. Right SCM stretch.

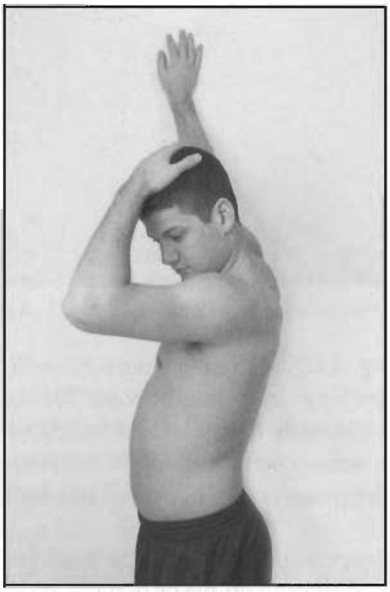

Figure 11-9a. Right levator scapula stretch.

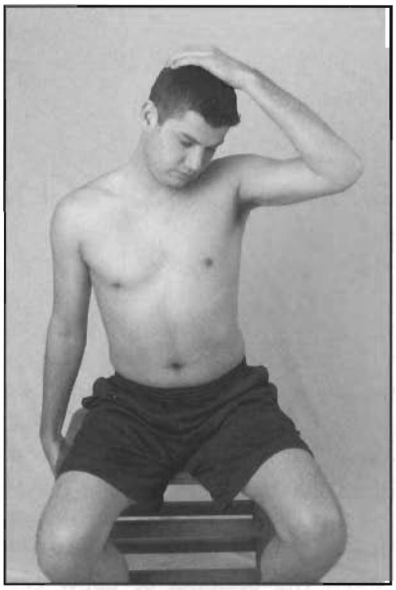

Figure 11-9b. Right levator scapula stretch modified.

the SCM from the anterior/middle scalene stretch. The patient, who is already in a slight chin-tuck, accentuates it further while simultaneously moving the head into further left side bending/right rotation until a stretch can be felt at the right mastoid process (Figure 11-8). As with previous stretches, the patient must proceed to the point of the initial stretch and go no further. The stretch is repeated 3 times, holding each stretch for 30 seconds, every 2 hours (this number of sessions through the day may not be feasible for some patients, but it emphasizes the need to do them often).

Levator Scapula Self-Stretch

The last of the self-stretches concludes with the levator stretch. To stretch the right side, the standing patient places the right arm against the wall in full abduction. This places the right scapula in upward rotation. With the left hand, the patient pulls the head-neck into combined flexion, rotation, and side bending to the left (Figure 11-9a). If there is a coexisting shoulder condition that precludes full abduction, the levator self-stretch can be modified to include scapular depression instead. This is achieved by having the sitting patient grasp the bottom of the chair as with the other stretches (Figure 11-9b).

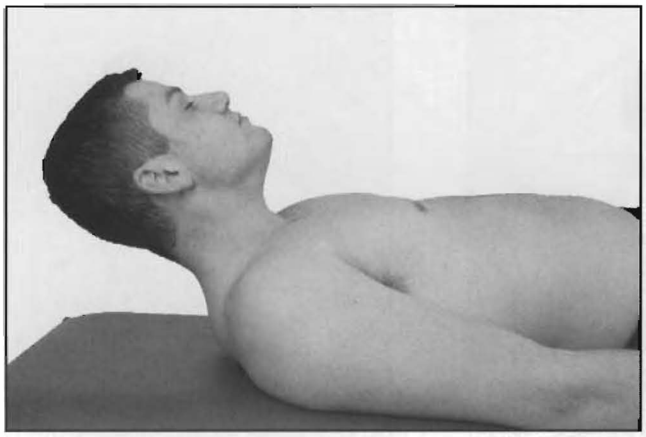

Figure 11-10. Example of weak deep neck flexors.

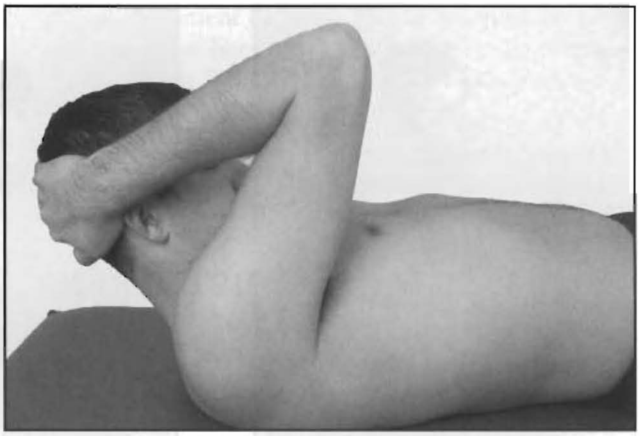

Figure 11-11a. Training of deep neck flexors phase 1.

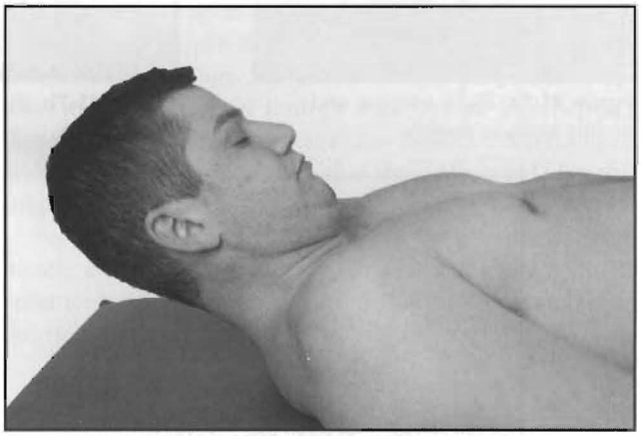

Figure 11-11b. Training of deep neck flexors phase 2.

Because the posterior scalene and levator scapulae stretches incorporate lower cervical flexion, the therapist and patient need to exercise caution. It is possible to exacerbate a latent derangement on the stretched side. Therefore, the patient must not overstretch, stop at the first indication of peripheral symptoms, return slowly to the start position, and perform a few prophylactic neck retractions to protect against disc disturbance.

Cervical Strengthening Exercises

The phasic muscles that require strengthening and endurance training in the head-neck region are the upper cervical or deep neck flexors and the lower cervical segmental extensors. It is only when these muscles are strong and possess good endurance that the tendency towards FHP (ie, "backward head/forward neck") can be overcome. When these muscles are weak, the patient demonstrates occipital extension upon attempting to flex the head-neck region, rather than flexion, suggesting that substitution with the sternocleidomastoid muscles is taking place (Figure 11-10).

To strengthen the occipital or upper cervical flexors (ie, rectus capitis anterior, longus capitis, and rectus capitis lateralis muscles), the supine patient is instructed to perform passive upper cervical spine flexion followed by lower cervical flexion (phase 1) with his or her fingers interlocked behind the occiput (Figure 11-11a). The patient then progresses from passive to active assisted to active flexion (phase 2) without the assist from his or her hands (Figure 11-11b). The deep neck flexor of the lower cervical spine, the longus colli, is recruited when the lower cervical spine is flexed with the head in the chin-tuck position.

A deficiency in endurance of the deep neck (cervical) flexor muscles (longus capitis, rectus capitis anterior, rectus capitis lateralis, and longus colli) is associated with neck pain, forward head posture, as well as cervicogenic and tension-type headache.[20,47,51,87-90] Endurance of these muscles can be tested with the craniocervical flexion test[20] (CCFT), using the Stabilizer pressure biofeedback device (Chattanooga Group Inc, Chattanooga, TN) or the flexor endurance test, which demonstrated excellent intratester reliability (intraclass coefficient of 0.92 for women and 0.93 for men).[21] The flexor endurance test[21] involves the following steps:

1. The subject assumes the supine hook-lying position with hands resting on his or her abdomen.
2. The subject is then asked to raise his/her head just enough to allow the tester to slide the widths of the index and middle finger of one hand, one on top of the other, under the subject's head at the most posterior aspect of the occiput.
3. The subject is allowed to rest his/her head-neck on the examiner's fingers.
4. The subject is then asked to "tuck the chin completely" (craniocervical flexion) and to raise the head just off the examiner's fingers (cervical flexion). The examiner gently moves his or her fingers side to side under the subject's head, providing a tactile reminder for maintaining proper head-neck position during the test (Figure 11-11c).

Figure 11-11c. The flexor endurance test.

Figure 11-11d. Training of deep neck flexor muscle endurance.

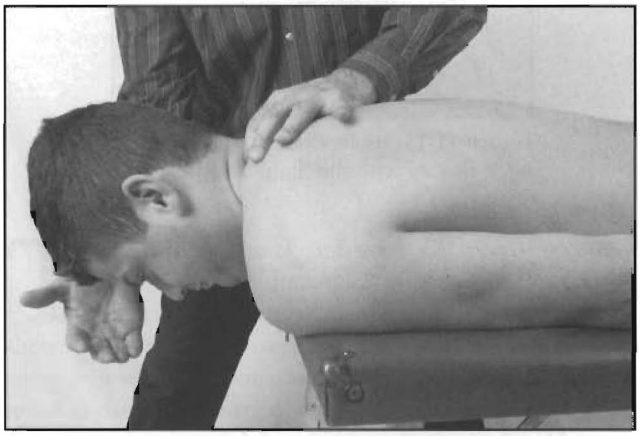

Figure 11-12a. Training lower cervical extensors while prone.

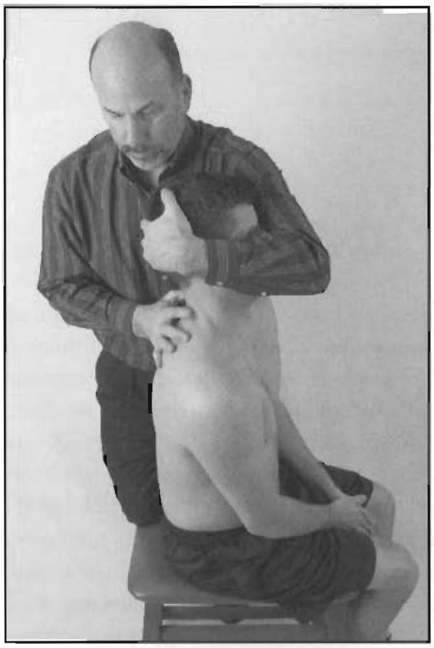

Figure 11-12b. Training lower cervical extensors while sitting.

5. Time is started when the subject's head is raised off the tester's fingers and ended when any of the following conditions are met:
   a. The subject experiences pain and is unwilling to continue.
   b. The subject reaches the end of endurance and is unwilling to continue.
   c. The examiner determines that the chin-tuck has been lost.
   d. The examiner determines that the subject raises the head (flexes the neck while still in a chin-tuck) such that the tester's fingers no longer maintain contact.

Endurance of the deep neck flexors is trained by having the patient maintain a chin-tuck position over the end of the table for progressively longer periods of time (Figure 11-11d). In the beginning, therapist support will be needed. However, with improvement, the patient should be able to maintain this position for at least 10 seconds without shaking or anxiousness.

To strengthen the lower cervical segmental extensors (ie, semispinalis cervicis and multifidus), the prone patient's head-neck region is placed over the end of the table as the therapist localizes axial extension to the C4 through C7 levels, one segment at a time (Figure 11-12a). Once properly localized to the barrier of bilateral apophyseal joint extension, the therapist withdraws his or her forehead support and the patient performs an isometric contraction of the segmental extensor muscles. Through bilateral facet palpation, the therapist ensures that the patient activates the desired segmental extensor muscles. Similar segmental extensor training can be performed in sitting as well (Figure 11-12b).

For either the upper cervical flexors or the lower cervical segmental extensors, the patient, following competency in the clinic, can perform self-strengthening at home. He or she can do 10 repetitions, holding each repetition for 5 to 10 seconds, repeating 3 times per day.

In addition to the postural realignment function of the Occivator mentioned previously in this chapter, it is also used to enhance the strength of the occipital flexor muscles, lower cervical segmental extensors, and the lower scapular stabilizers, simultaneously.

A second device developed by this author, the PostureJac, has the advantage of being a portable posture-retraining device and will be covered in detail in Chapter 25.

Lastly, the Stabilizer alluded to previously, has an air-filled pressure sensor that monitors the slight flattening of the cervical lordosis (Figure 11-13). In addition to its role in the CCFT,[20] the Stabilizer is also useful as a biofeedback tool in the retraining of motor control, strength, and endurance of the deep neck flexors.[20]

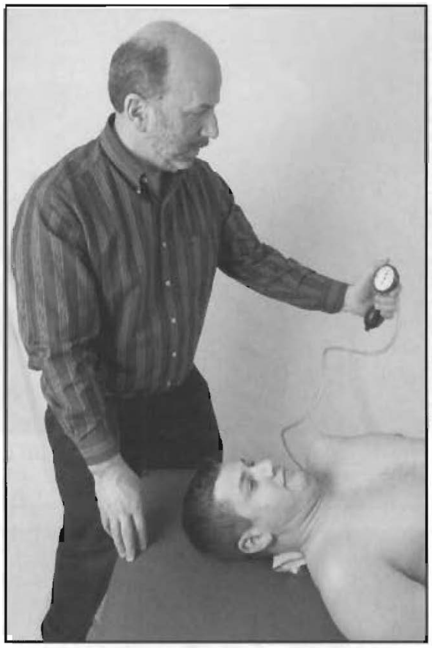

Figure 11-13. Biofeedback of the deep neck flexors with the Stabilizer.

12

The Role of the Cervical Spine in Headache and Dizziness

The head and neck are areas of intense postural reflex activity.[10] Examples include the tonic neck, cervicocollic, cervicorespiratory, cervicosympathetic, cervico-ocular, and trigeminocervical reflexes to name a few.[91] Consequently, in the presence of cervical spine impairment, particularly in the uppermost segments, there is the potential for many systems to be adversely impacted.[10,91] oss

dizziness are common features of cervical impairment, injury, or disease. Their cervical causes are of great interest to manual therapists. In this chapter, the role of the cervical spine in both headache and dizziness will be explored. Porterfield and DeRosa[95] state, "The neurosciences of the cervical spine have a degree of complexity found in no other region of the axial skeleton." We will certainly be exposed to some of this complexity in this chapter.

Headache

Headache of cervical origin (ie, cervicogenic headache [CGH]) accounts for 15% to 20% of all chronic and recurring headaches, and up to 70% of individuals with frequent intermittent headache (eg, 50 million in the United States) report associated neck pain.[88,96-98]

To better understand the role of the cervical spine in CGH[88,] and its contribution to other forms of chronic headache, it is necessary to review our current understanding of the neuroanatomy of the upper cervical spinal cord.

The spinal nucleus of the trigeminal nerve (ie, fifth cranial nerve) consists of 3 parts: pars oralis, pars interpolaris, and pars caudalis (Figure 12-1). The pars caudalis is the most caudal of the 3 and merges imperceptibly with the dorsal horns of the upper 3 cervical spinal cord segments, consisting of the marginal zone, substantia gelatinosa, and the nucleus proprius. The spinal tract of the trigeminal nerve descends caudally through the medulla oblongata as far as the C4 level. Fibers from the spinal tract terminate in the gray matter of the pars caudalis and upper 3 cervical cord segments. Bogduk[92,102]

of interconnecting gray matter of the pars caudalis and the upper cervical dorsal horns as the "trigeminocervical nucleus." This nucleus is defined not by intrinsic features, but by the afferent input it receives from the spinal tract of the fifth cranial nerve. Because it incorporates the neuroanatomic structures responsible for pain transmission and receives afferent input from trigeminal and upper cervical nerves, the trigeminocervical nucleus can be seen as the nociceptive nucleus for the entire head and upper neck. In addition, Mannheimer and Rosenthal[94] report that the entire trigeminocervical complex includes not only the fifth cranial nerve, but also receives input from the 7th, 9th, 10th, 11th, and 12th cranial nerves as well. The clinical significance of these scientific discoveries is summarized by Jull,[103] who states, "Through the convergence of cervical and trigeminal afferents on common neurons in the trigeminal nucleus, any structure innervated by any of the upper three cervical nerves may refer pain into the head and face." 99-
Furthermore, Jull[88] describes "bi-directional interactions" between trigeminal and upper cervical afferents within the trigeminocervical nucleus. Consequently, this may explain not only head and face pain of upper cervical origin, but also neck symptoms of trigeminal origin (eg, migraine).

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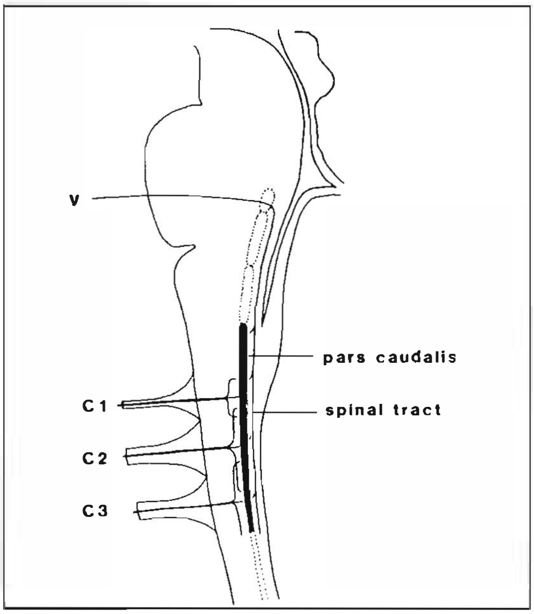

Figure 12-1. The trigeminocervical nucleus. (Reprinted with permission from Bogduk N. Cervical causes of headache and dizziness. In: *Grieve's Modern Manual Therapy*. 2nd ed. New York, NY: Churchill Livingstone; 1994.)

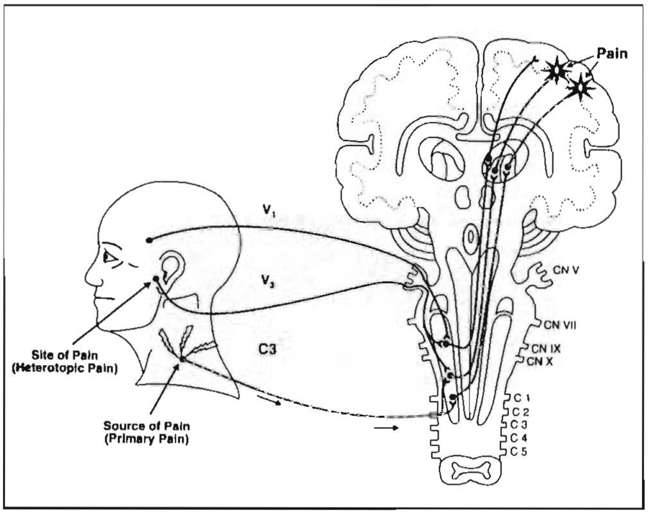

Figure 12-2. Head and temporomandibular joint/facial pain of cervical origin. (Reprinted with permission from Okeson J. *Orofacial Pain: Guidelines for Assessment, Diagnosis, and Management*. Chicago, Il: Quintessence Publishing; 1996.)

Although the pathophysiology of CGH is not completely understood, Bogduk[92] believes that there is sufficient "circumstantial evidence" pointing to the convergence between nerves that innervate the head and nerves that innervate the cervical spine as the "foundational mechanism." He goes on to say that this is not simply convergence between trigeminal and cervical afferents, for in addition to innervation by the trigeminal nerve, the head is also innervated by cervical nerves. For example, the occiput and regions as far forward as the coronal suture are innervated by the greater occipital nerve, the lesser occipital nerve, and the greater auricular nerve, whereas the forehead and orbital regions are innervated by the trigeminal nerve. Consequently, CGH perceived anterior to the coronal suture implies convergence between cervical and trigeminal afferents; CGH posterior to the coronal suture suggests convergence between certain cervical and other cervical afferents.[92]

To support the concept of upper cervical pain referral into the head and face, Bogduk[102] cites several studies in this regard and then states the following, "These experiments clearly demonstrate the capacity of experimental painful stimuli in the upper neck to produce pain in the head. It is possible, therefore, that pathological painful lesions of any of the structures innervated by the upper cervical nerves are equally capable of producing such referred pain" (Figure 12-2).

When considering the role of the cervical spine in headache, there are 2 possible connections. The first involves direct pain referral from upper cervical spine disease or somatic impairment (ie, CGH). The second involves the indirect role of the upper cervical region in other forms of chronic headache, including tension-type, migraine with and without aura, posttraumatic headache (PTH), and analgesic rebound headache. The role of the cervical spine in temporomandibular disorders (TMD) will be addressed in a subsequent chapter.

CGH is a form of secondary headache arising from painful dysfunction or disease of the cervical spine, particularly the upper 3 segments. In 2004, the International Headache Society (IHS) accepted CGH as a discrete headache type, as published in the 2nd edition of the *International Classification of Headache Disorders*.[104] Prior to that, the Cervicogenic Headache International Study Group established diagnostic criteria for CGH in 1990 and again in 1998.[105] The following are the current IHS diagnostic criteria for CGH[89,98-101]:

1. Pain localized in the neck and occiput, which can spread to other areas in the head, such as the forehead, orbital region, temples, vertex, or ears, usually unilateral.
2. Pain precipitated or aggravated by specific neck movements or sustained postures.
3. At least one of the following:
    a. Resistance to or limitation of passive neck movements,
    b. Changes in neck muscle contour, texture, tone, or response to active and passive stretching and contraction, and/or
    c. Abnormal tenderness of neck musculature.
4. Radiological examination reveals at least one of the following:

a. Movement abnormalities
b. Abnormal posture
c. Fractures, congenital abnormalities, bone tumors, rheumatoid arthritis, or other distinct pathology (not spondylosis or osteochondrosis).

In addition, the presence of painful upper cervical joint dysfunction, accompanied by impairments in the deep cervical flexors, scapular postural muscles, and cervical kinaesthesia, suggests that the headache is of cervical origin.[97,106] Headache characteristics include moderate to severe, nonthrobbing, and nonlancinating pain, usually starting in the neck and eventually spreading to the oculofrontotemporal area on the symptomatic side. CGH is, in principle, a unilateral headache, but it may become bilateral over time. The frontotemporal pain may at times exceed the neck/occipital pain. In the initial phase, the headache is usually episodic; later it becomes chronic with a fluctuating quality. Occasionally, patients with CGH also report nausea, phonophobia/photophobia, dizziness, blurred vision, difficulty swallowing, and ipsilateral edema in the periocular area. However, these "attack-related phenomena" are not the major features of this headache. Diagnostic anesthetic blockade of the greater/lesser occipital nerves, C2 and C3 roots, third occipital nerve, facet joints, and lower cervical roots and branches on the symptomatic side should temporarily abolish the pain of CGH. However, Jull[88] suggests that there are problems of specificity with diagnostic blocks and are therefore "not fail-safe for the diagnosis of CGH." Of the 3 spinal segments involved with CGH (ie, OA, AA, and C2,3), the C2,3 facet joints are thought to play the most significant role.[107] Having said that, other researchers provide support for the role of C1,2 segmental dysfunction in CGH. Specifically, Hall and Robinson found that subjects with CGH have an average of 17 degrees less rotation toward the headache side in the flexion-rotation test (FRT) in contrast to subjects with no headache. The FRT, which identifies restriction of rotation at the C1,2 segment, has a sensitivity of 86% and a specificity of 100% for detecting CGH.[108] Studies have also shown a connection between CGH, FHP, weak and poor endurance of the deep cervical flexors, facet joint arthropathy, cervical spine trauma, and joint hypo/hypermobility including clinical cervical spine instability.[20,42,47,89,109] Consequently, the role of spinal manual therapy and specific exercise as an intervention for CGH is gaining momentum. In fact, the *Evidence Report: Behavioral and Physical Treatments for Tension-Type and Cervicogenic Headache* from the Duke University Evidence-Based Practice Center published in 2001 concluded the following: "Cervical spine manipulation was associated with significant improvements in headache outcomes in trials involving patients with neck pain and/or neck dysfunction and headache."[110] Schoensee et al,[107] investigating the effect of upper cervical mobilization on the frequency, duration, and intensity of cervical headaches, concluded that manual therapy was effective as an intervention for headaches of cervical origin. In addition to the role of manual therapy in the management of headaches of cervical spine origin, Jull[20,111] emphasizes the importance of specific retraining of the upper cervical flexor muscles, the lower trapezius, and serratus anterior, combined with postural retraining as well as ergonomic and lifestyle advice.

There is some controversy regarding the role of the cervical spine in such primary headache conditions as migraine and tension-type headache. However, several studies have established a correlation between chronic tension-type headache and 1) FHP[49,50]; 2) neck mobility[50,90,112]; 3) reduced deep cervical muscle strength and endurance[45,113]; and 4) active myofascial trigger points in the suboccipital muscles, upper trapezius, SCM, and temporalis muscles.[49,114] Regarding episodic tension-type headache, the following somatic features have been identified versus a healthy nonheadache control group[115]: 1) smaller craniovertebral angle (ie, FHP); 2) decreased neck mobility; and 3) more active myofascial trigger points in the upper trapezius, SCM, and temporalis muscles.

In addition, there is a growing body of knowledge suggesting that the musculoskeletal system does in fact play a role in the pathogenesis and management of migraine,[116] including a recent study showing that subjects with unilateral migraine had a significantly greater number of active trigger points on the same side as the migraine as well as a greater forward head posture in both the sitting and standing positions versus healthy controls.[117] As far back as 1995, Hack et al[118] identified a fibrous connection between the rectus capitis posterior minor muscle and the posterior atlanto-occipital membrane, which attaches to the cranial dura mater. This proposed "myodural bridge" shed light on the connection between subcranial muscle tension and migraine. In a study by Marcus et al,[119] postural abnormalities were more prevalent in patients with migraine and tension-type headache than in the controls. Karpouzis et al[120] showed that a history of head, neck, and back injury was the most commonly reported circumstance related to the onset of chronic headache in 1013 patients; Silberstein et al[121] demonstrated a clinically favorable response to pericranial injection of botulinum toxin type A with reduced migraine frequency, severity, acute medication usage, and associated vomiting. Whereas most neurology-based textbooks and articles view muscle contraction as a consequence of migraine, Silberstein et al[121] raise the possibility that muscle contraction may play a role in migraine pathogenesis through some "as of yet unknown effect on the sensory system." Thus, we see an increasingly important role of the cervical spine in headache diagnosis and management emerging in the scientific literature. Whether this role is as an etiologic factor in primary headache pathogenesis or secondary to the neurochemical pain pathophysiology expressed in migraine and tension-type headache remains to be determined.

Moskowitz[122] proposed a mechanism whereby an upper cervical impairment can give rise to a throbbing vascular headache. This mechanism involves the activation of trigeminal sensory fibers in the brainstem, which in turn trigger an efferent pathway through the facial nerve to the greater superficial petrosal nerve. The greater superficial petrosal nerve provides the autonomic connection by innervating autonomic pathways in the cranial vasculature. Some have used this and other similar physiologic mechanisms[123] to suggest a major role of the cervical spine in migraine. However, the literature does not support this concept. A more plausible argument, and the one to which the author subscribes, is that upper cervical spine impairment (eg, OA, AA, and/or C2,3 joint dysfunction, forward head posture, myofascial trigger points, greater occipital nerve entrapment) is one of many factors in migraine pathogenesis leading to what is known as central sensitization[109,114,117,124-126] (ie, somatosensory hypersensitivity). Similar to the role of emotional stress, dietary triggers, sleep deprivation, hypoglycemia, hormonal factors in women, etc, the presence of chronic upper cervical spine impairment, leading to nociceptive-neuronal hyperexcitability of the trigeminocervical nucleus, has the potential to trigger a migraine attack. Nocturnal bruxism[127] and fibromyalgia[124] are thought to trigger migraine in a similar manner.

Whereas migraine was once thought to be a function of intracranial/extracranial vasodilatation (ie, Wolff's vascular theory[125,128]), it is now believed that migraine is a complex disorder of CNS regulation of pain-producing intracranial structures (ie, neurovascular malregulation leading to neurogenic inflammation[125,129] of the trigeminovascular complex). Based upon this neurovascular theory [109,125,128] and given that migraine in known to run in families and affect a large segment of the population (30 million Americans), this author considers the following the best definition of migraine to date, "A common, disabling malfunction of the pain-regulating mechanism of the brain." It is beyond the scope of this chapter to provide a detailed analysis of migraine pathophysiology. However, it is important for manual therapists to realize that migraine is enormously complex and that the presence of cervical impairment is not the "whole ball of wax." In addition to abnormal afferent input from the upper cervical area (mainly through the opthalmic division of the trigeminal nerve[92,94,102,103]), the trigeminocervical nucleus receives afferent input from the extensive trigeminovascular system, which is thought to be abnormal in patients suffering from migraines. There is strong evidence to suggest that a neurochemical imbalance in serotonin (5-HT) plays a key role in this abnormality.[109,125,128] Plasma serotonin has been shown to fall at the onset of a migraine attack, and the fact that reserpine (a serotonin-depleting agent) precipitates migraine is further evidence that falling serotonin and migraine are related.[128] In addition, the relief that migraine sufferers obtain from the 5-HT agonists is another indication of the serotonin-migraine connection.[125,128]

The take-home message from this crash-course in brain neurochemistry is that migraine is multifactorial. The input from an impaired upper cervical region is one of many factors. Migraine, whether with or without aura, is primarily a disturbance within the trigeminal system, with the greatest pathophysiology emanating from the trigeminovascular junctions at the base of the brain and in the dura mater.[109,121,122,125,128] Supporting the role of the trigeminal system in migraine, DaSilva et al[129] recently demonstrated structural changes with MRI (eg, thickness) in the somatosensory cortex of migraine sufferers compared to age and gender-matched controls. The most significant thickness changes were noticed in the caudal somatosensory cortex, where the trigeminal area is somatotopically represented. The authors conclude that, "Repetitive migraine attacks may lead to, or be the result of neoplastic changes in cortical and subcortical structures of the trigeminal somatosensory system." There appears to be little to no benefit of manual therapy during an attack of migraine, but between episodes there is significant benefit. By correcting somatic impairment throughout the head, neck, TMJ, and upper back,[130] there will be less nociceptive input into the trigeminocervical nucleus. This "de-facilitation" will have the net effect of raising the central pain threshold for the head and upper neck region and hopefully have a beneficial effect on the frequency, duration, and severity of migraine.

There is an effective nonmedicinal strategy that can be employed to abort an extracranial vascular headache. According to Willis,[131] a tourniquet is applied around the head just above the ears. The best time to use this method is just prior to the headache, but it can be used during the migraine, providing that the scalp is not overly sensitive to pressure (ie, allodynia[125]). The tightness of the tourniquet is to be moderate in nature and it can be left in place for several hours. The principle behind this method is based upon Laplace's law, where $T = Pr$. T represents the circumferential tension within the vessel wall, P represents the pressure gradient across the vessel, and r stands for the radius of the vessel. Because vasodilatation increases T during migraine, the arterial wall is stretched and becomes inflamed and painful. When T is decreased with the tourniquet, by decreasing P and r, the stretch on the vessel wall is lessened and the headache diminishes. This is a useful method in patients who cannot tolerate migraine medication.

Regarding PTH,[109,125,128] the role of the cervical spine cannot be ignored. Although there is a strong correlation between mild head injury and PTH, there is also a large percentage of PTH patients who have a history of cervical spinal injury as well. The term *posttraumatic migraine* has been used to describe the onset of migraine following mild head injury. However, according to Packard,[132] "trauma probably never causes migraine." Instead he attributes the onset of migraine following head injury to a temporary worsening of preexisting migraine related to a nonspecific stress reaction or to a "complicating neck sprain," which may aggravate pre-existing migraine as well. Because the symptoms of PTH include physical, psychological, and cognitive aspects, its management must involve a multidisciplinary approach. Jensen et al[133] demonstrated a superior effect of manual therapy over cold packs in the treatment of PTH. Using a combination of spinal mobilization, high velocity thrust, and muscle energy techniques, the manual therapy group demonstrated a more rapid decline in the pain index and overall use of analgesics compared with the cold pack group.

There are 2 remaining chronic headache types to discuss relative to the role of the cervical spine. The first is cluster headache and the second is analgesic abuse headache.[109,128,130] Although the exact mechanism of cluster headache remains uncertain, Hildebrandt and Jansen[134] reported on 2 middle-age males in whom chronic intermittent hemicrania associated with ciliary injections, lacrimation, and rhinorrhea (typical symptoms of cluster headache) were successfully treated with surgical decompression of the C2 and C3 nerve roots. In one case, a pannus-like layered network of veins with arterial supply was the culprit; in the other case, it was a network of veins. This study illustrates the point that there may be an upper cervical component in some cases of cluster headache. Whether somatic impairment can cause the symptom complex noted in the above 2 cases of vascular compression is unknown, but certainly the possibility exists.

The abuse of both over-the-counter and prescription analgesics for chronic headache management is a serious health problem. Although not always to blame, drug-induced factors are often the cause of what has been referred to as transformational migraine[135] (ie, the transformation of periodic migraine, that over time, takes on a more frequent and then continuous pattern). Srikiatkhachorn et al[136] demonstrated that chronic paracetamol administration in laboratory animals resulted in 5-HT depletion that, in turn, produced readaptation of the 5-HT 2a receptor. This change in the 5-HT 2a serotonin receptor may be an important mechanism related to the loss of analgesic efficacy, ultimately resulting in the daily complaints associated with analgesic abuse. Analgesic abuse headache is finally receiving the attention it deserves and may be prevented or reversed by avoiding the chronic use of analgesic medication. This means that therapists must do a better job of providing nonmedicinal headache relief to their patients. The normalization of head, neck, TMJ, and spinal function[130] will go a long way toward achieving this goal and consequently spare at least some, if not many, the nightmare of the chronic head, neck, and face pain, in addition to the many other adverse effects associated with analgesic abuse (eg, gastrointestinal, kidney, and liver damage).

The author, as with much of this textbook, has intentionally not included an extensive review of the basic science material on this topic. The reader is encouraged to scan the references in order to broaden his or her knowledge of the subject.

Dizziness

Dizziness associated with cervical spine movement impairments may be secondary to VBI, the vestibular system, the visual system, or from cervical spine structures (ie, cervicogenic dizziness [CD][137,138]). The term *dizziness* will be used, generically, in this chapter to include the following symptoms:

- Vertigo: A sensation that the environment is spinning (external), or that the individual is spinning (internal).
- Presyncopal lightheadedness: A feeling that one is about to pass out.
- Disequilibrium: A sensation of imbalance or unsteadiness (more prominent in standing).

Dizziness can have central, peripheral, or systemic causes.[139-141] Peripheral causes include peripheral vestibulopathy, peripheral vestibular disorder (eg, benign paroxysmal positional vertigo), Meniere's disease, labyrinthitis, labyrinthine concussion, vestibulotoxic drugs, perilymph fistula, etc. Central causes include demyelinating disease, tumors, seizures, VBI, migraine-related vertigo, transient ischemic attack, minor brain injury, and CD. Systemic causes of dizziness include endocrine disease (hypothyroidism, diabetes), pharmacologic side effects (anticonvulsants, antihypertensives, tranquilizers, analgesics, muscle relaxants, etc), and the many causes of presyncope (eg, hypoglycemia, panic, vasovagal episode, hypotension, cardiac arrhythmias, Valsalva's maneuver, etc).

Generally, true vertigo indicates a disorder of the inner ear, vestibular nerve, brainstem, or cerebellum, whereas VBI presents with presyncope and CD with disequilibrium.

The diagnosis of VBI is straight forward when the 5 Ds, 3 Ns, and 1 A[139,140,142] are present (see special tests section of Chapter 8). However, when only dizziness is present (which is sometimes the case) diagnosis is difficult. The diagnosis of BPPV is also straight forward. It is common in middle age, but in about 15% of cases there is a relationship to head trauma.[19,139,142,143] The patient typically develops severe vertigo when turning over or first lying in bed. The episodes last less than a minute and the patient can find another position in which he or she is asymptomatic. As soon as he or she moves, however, another attack is provoked. There are 2 theories as to how BPPV occurs. One is canalithiasis and the other cupulolithiasis.[19,140,142] Canalithiasis, caused by free-floating otoconia in one of the semicircular canals, is thought to be the more common of the two. Clinically, the onset of vertigo associated with cupulolithiasis has less latency due to the fact that the otoconia are deposited directly on the cupula (ie, vertigo occurs without significant delay when provoked as compared to canalithiasis). The Hallpike-Dix maneuver (88% sensitivity, 100% specificity) is used to test patients suspected of having BPPV affecting the posterior or anterior canals, whereas the roll test detects horizontal canal BPPV.[19,144,145] The treatment of BPPV is

best managed with physical procedures geared toward either removing debris from the affected canal or decreasing symptoms through habituation.[19]
demonstrated efficacy with BPPV affecting the anterior and posterior canals, whereas the log roll maneuver is effective in managing horizontal canal BPPV.[19,145]
lithiasis (especially of the posterior semicircular canal) is suspected, the Semont ("Liberatory") maneuver is the technique of choice. The Brandt-Daroff habituation exercises are a useful tool for those who have difficulty tolerating the canalith repositioning maneuvers mentioned above.[19,141,145] A detailed description of the above-mentioned diagnostic and treatment procedures for BPPV is beyond the scope of this text. The reader is directed to the references provided.

CD is a sensation of altered orientation in space and disequilibrium originating from abnormal afferent activity from the neck.[19,13]
vestibular dysfunction and, therefore, rarely results in true vertigo. Signs and symptoms of CD include the following:

- Intermittent positioning-type dizziness precipitated by head and neck movement.
- No latency period (ie, onset of symptoms is immediate upon assuming the provoking position).
- The duration is anywhere from minutes to hours.
- Dizziness is fatigable with repeated motion.
- Associated signs and symptoms include nystagmus, neck pain, suboccipital headaches, and occasionally paresthesia in the trigeminal distribution.
- Possible head-neck malalignments, such as forward head and torticollis.
- Segmental impairment of the upper cervical spine.
- Positive neck torsion test.

CD is often associated with whiplash-associated disorders,[20] which can make diagnosis difficult as BPPV and VBI can also be trauma related.[146,147] CD has also been reported in advanced cases of cervical arthritis, herniated cervical discs, and head trauma. In the latter, complaints of ataxia, unsteadiness of gait, and/or postural disequilibrium are the most common.

The pathophysiology of CD appears to involve abnormal afferent input to the vestibular nuclei from damaged joint receptors in the upper cervical region, resulting in a false sense of motion. Aspinall[139] attributes CD to a disturbance of the tonic neck reflexes from a distortion of the normal afferent input to the vestibular nuclei from the neck. Herdman[19] suggests that inflammation or irritation of the cervical roots or facet joints would lead to a mismatch among vestibular, visual, and cervical inputs. This "multisensory mismatch" would then give rise to the symptoms of CD, especially during movements of the head-neck region. Isaacs and Bookhout[12] relate CD to abnormal muscle tone in cervical musculature or following mobilization of the cervical spine, when proprioceptive feedback does not match

Figure 12-3a. The Fitz-Ritson or neck torsion test with the head-neck turned to the left.

ocular and vestibular sensations. Wapner et al[148] discovered that the sensation of tilting or falling could be evoked by electrical stimulation of the cervical muscles. Gray[149] found that CD could be relieved by injecting local anesthetic into the posterior cervical muscles.
claim that abnormal afferent input from the cervical region results in patient-perceived dizziness.
that she doubts whether cervical lesions have a "profound effect" on the oculomotor and vestibular systems, but goes on to say, "There is evidence that treatment of cervical dysfunctions can lead to decreased symptoms of dizziness and improvements in postural stability."

This discussion will conclude with a description of a clinical assessment tool for CD known as the neck torsion test[19,144,145] (90% sensitivity, 91% specificity), which is similar to the test developed by Fitz-Ritson.[150]
seated on a stool that rotates (Figures 12-3a and 12-3b). The therapist stands behind the patient and holds the patient's head steady.
tion to prestretch the cervical musculature. With the patient's eyes closed, the body is rotated to either side with the feet. This motion essentially rotates the neck to either side while the semicircular canals are motionless.
ness must therefore be of cervical spine origin. Fitz-Ritson found that the patients who responded best to manipulative treatment were those who suffered upper cervical joint problems, along with muscle trauma in that region.
the theory that CD arises from abnormal afferent input from the receptors of the upper cervical spine.

According to Jull and colleagues,[20] patients presenting with neck pain, with or without complaints of dizziness, lightheadedness, or feelings of unsteadiness, should be examined for impairments in the postural control system.

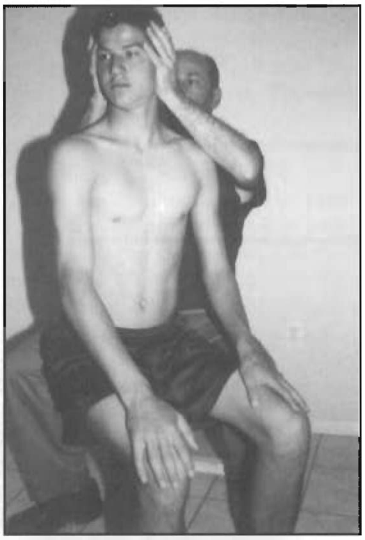

Figure 12-3b. The Fitz-Ritson or neck torsion test with the head-neck turned to the right.

This examination includes the following:

- Tests of cervical joint position sense
- Balance
- Oculomotor control

The oculomotor assessment incorporates the assessment of all aspects of eye movement including the ability to maintain gaze while moving the head (gaze stability), eye follow while keeping the head still (smooth-pursuit), and maintaining gaze when the eyes and head are moving (eye-head coordination). The reader is referred to the text by Jull, Sterling, Falla, Treleaven, and O'Leary for more information on the assessment and treatment of disturbances in the postural control system[20] (ie, sensorimotor control).

Regarding the complexity of the cervical spine from a neuroanatomical perspective, the clinician must be cognizant of the multiple inputs and influences that affect the somatic structures of the neck. They include, at a minimum, vestibular,[19] visual,[20,151] limbic,[152] craniomandibular,[153,154] respiratory,[57,155] and visceral.[11] In addition, migraine headache is believed to cause pain and muscle hypertonicity in the head-neck region.[128] Consequently, all potential sources of cervical spine pain, including pathological causes (eg, undiagnosed fractures), must be identified and managed if the patient's condition is to improve. This may necessitate referral to a neurologist, neurosurgeon, orthopedic surgeon, internist, ophthalmologist, ear, nose and throat specialist, dentist, dental surgeon, psychiatrist, etc.

Section III: Key Points

1. The cervical spine is the most mobile region of the vertebral column and is prone to developing clinical spinal instability (Panjabi).
2. McKenzie's derangement syndrome occurs often in the cervical spine, primarily at C5,6 and C6,7.
3. Upper cervical spine impairment may cause headache and dizziness, whereas lower cervical impairment may be the source of referred pain into the scapula, chest wall, and upper limb.
4. Avoid performing thrust manipulation in the upper cervical spine. The benefit does not justify the risk!
5. Forward head posture has been linked to many conditions and needs to be taken seriously.

References

1. Cook CE, Hegedus EJ. *Orthopedic Physical Examination Tests: An Evidence-Based Approach.* Upper Saddle River, NJ: Pearson Prentice Hall; 2008.
2. Cleland J. *Orthopaedic Clinical Examination: An Evidence-Based Approach for Physical Therapists.* Carlstadt, NJ: Icon Learning Systems; 2005.
3. Huijbregts PA. Clinical prediction rules: time to sacrifice the holy cow of specificity? *J Manual Manipulative Ther.* 2007;15(1):5-8.
4. Childs JD, Fritz JM, Piva SR, Whitman JM. Proposal of a classification system for patients with neck pain. *J Orthop Sports Phys Ther.* 2004;34(11):686-700.
5. Kendall FP, Kendall McGreary E, Provance PG, McIntyre Rodgers M, Romani WA. *Muscles: Testing and Function With Posture and Pain.* 5th ed. Baltimore, MD: Williams & Wilkins; 2005.
6. Andrade GH, Azevedo DC, Lorentz I, et al. Influence of scapular position on cervical rotation range of motion. *J Orthop Sports Phys Ther.* 2008;38(11):668-673.
7. Rocabado M. Diagnosis and treatment of abnormal craniocervical and craniomandibular mechanics. In: Solberg WK, Clark GT, eds. *Abnormal Jaw Mechanics: Diagnosis and Treatment.* Chicago, IL: Quintessence Publishing; 1984.
8. Hickey ER, Rondeau MJ, Corrente JR, Abysalh J, Seymour CJ. Reliability of the cervical range of motion (CROM) device and plumb-line techniques in measuring resting head posture (RHP). *J Manual Manipulative Ther.* 2000;8(1):10-17.
9. Fletcher JP, Bandy WD. Intrarater reliability of CROM measurement of cervical spine active range of motion in persons with and without neck pain. *J Orthop Sports Phys Ther.* 2008;38(10):640-645.
10. Dutton M. *Orthopaedic Examination, Evaluation, and Intervention.* 2nd ed. New York, NY: McGraw-Hill Companies, Inc; 2008.
11. Greenman PE. *Principles of Manual Medicine.* 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2003.
12. Isaacs ER, Bookhout MR. *Bourdillon's Spinal Manipulation.* 6th ed. Boston, MA: Butterworth-Heinemann; 2002.
13. McKenzie R, May S. *The Lumbar Spine, Mechanical Diagnosis & Therapy. Volume 1.* Waikanae, New Zealand: Spinal Publications New Zealand Ltd; 2003.
14. McKenzie RA. *The Cervical and Thoracic Spine, Mechanical Diagnosis and Therapy.* Waikanae, New Zealand: R.A. McKenzie; 1990.
15. Kramer J. *Intervertebral Disc Diseases: Causes, Diagnosis, Treatment, and Prophylaxis.* 2nd ed. New York, NY: Thieme Medical Publishers; 1990.
16. Rocabado M. Biomechanical relationship of the cranial, cervical, and hyoid regions. *J Craniomandib Pract.* 1983;1(3):61-66.
17. Vidal P, Huijbregts P. Dizziness in orthopaedic physical therapy practice: history and physical examination. *J Manual Manipulative Ther.* 2005;13(4):221-250.
18. Ono K, Ebara S, Fuji T, Yonenobu K, Fujiwara K, Yamashita K, Myelopathy hand: new clinical signs of cervical cord damage. *J Bone Joint Surg.* 1987;69B(2):215-219.
19. Herdman SJ. *Vestibular Rehabilitation.* 3rd ed. Philadelphia, PA: F.A. Davis Company; 2007.
20. Jull G, Sterling M, Falla D, Treleaven J, O'Leary S. *Whiplash, Headache, and Neck Pain: Research-Based Directions for Physical Therapies.* Edinburgh: Churchill Livingstone; 2008.
21. Olson LE, Millar AL, Dunker J, Hicks J, Glanz D. Reliability of a clinical test for deep cervical flexor endurance. *J Manipulative Physiol Ther.* 2006;29(2):134-138.
22. Fitz-Ritson D. Assessment of cervicogenic vertigo. *J Manipulative Physiol Ther.* 1991;14(3):193-198.
23. Jansson A, Saartok T, Werner S, Renstrom P. General joint laxity in 1845 Swedish school children of different ages: Age and gender-specific distributions. *Acta Paediatr.* 2004;93(9):1202-1206.
24. Savitz SI, Caplan LR. Vertebrobasilar disease. *N Engl J Med.* 2005;352:2618-2626.
25. Coman WB. Dizziness related to ENT conditions. In: Grieve GP, ed. *Modern Manual Therapy of the Vertebral Column.* Edinburgh, UK: Churchill Livingstone; 1986.
26. Kerry R, Taylor AJ, Mitchell J, McCarthy C, Brew J. Manual therapy and cervical arterial dysfunction, directions for the future: a clinical perspective. *J Manual Manipulative Ther.* 2008;16(1):39-48.
27. Huijbregts P, Vidal P. Dizziness in orthopaedic physical therapy practice: classification and pathophysiology. *J Manual Manipulative Ther.* 2004;12(4):199-214.

28. Magee DJ. *Orthopedic Physical Assessment.* 5th ed. St. Louis, MO: Saunders Elsevier; 2008.
29. Konin JG, Wiksten DL, Isear JA, Brader H. *Special Tests in Orthopedic Examination.* 3rd ed. Thorofare, NJ: SLACK, Incorporated; 2006.
30. Gross J, Fetto Rosen E. *Musculoskeletal Examination.* 2nd ed. Cambridge, MA: Blackwell Science; 2002.
31. Richter RR, Reinking MF. Clinical question: How does evidence on the diagnostic accuracy of the vertebral artery test influence teaching of the test in a professional physical therapist education program? *Phys Ther.* 2005;85(6):589-599.
32. Meadows J. *Orthopedic Differential Diagnosis in Physical Therapy: A Case Study Approach.* New York, NY: McGraw-Hill; 1999.
33. Zaina C, Grant R, Johnson C, Dansie B. Taylor J, Spyropolous P. The effect of cervical rotation on blood flow in the contralateral vertebral artery. *Man Ther.* 2003;8(2):103-109.
34. Kuether TA, Clark WM, Barnwell SL. Rotational vertebral artery occlusion: a mechanism of vertebrobasilar insufficiency. *Neurosurgery.* 1997;41(2):427-433.
35. Di Fabio RP. Manipulation of the cervical spine: Risks and benefits. *Phys Ther.* 1999;79(1):50-65.
36. Haldeman S, Kohlbeck FJ, McGregor M. Unpredictability of cerebrovasular ischemia associated with cervical spine manipulation therapy. *Spine.* 2002;27(1):49-55.
37. Ernst E. Adverse effects of spinal manipulation: a systematic review. *J R Soc Med.* 2007;100:330-338.
38. Kasai Y, Morishita K, Kawakita E, Kondo T, Uchida A. A new evaluation method for lumbar spinal instability: passive lumbar extension test. *Phys Ther.* 2006;86(12):1661-1667.
39. Weinstein JN, Rydevik BL, Sonntag VKH, eds. *Essentials of the Spine.* New York, NY: Raven Press; 1995.
40. Panjabi MM. Clinical spinal instability and low back pain. *J Electromyogr and Kinesiol.* 2003;13:371-379.
41. Rocabado, M. The importance of soft tissue mechanics in stability and instability of the cervical spine: a functional diagnosis for treatment planning. *J Craniomandib Pract.* 1987;5(2):130-138.
42. Olson KA, Joder D. Diagnosis and treatment of cervical spine clinical instability. *J Orthop Sports Phys Ther.* 2001;31(4):194-206.
43. Pettman E. Stress tests of the craniovertebral joints. In: Boyling JD, Palastanga N, eds. *Grieve's Modern Manual Therapy: The Vertebral Column.* 2nd ed. Edinburgh: Churchill Livingstone; 1994.
44. Mintken PE, Metrick L, Flynn T. Upper cervical ligament testing in a patient with os odontoideum presenting with headaches. *J Orthop Sports Phys Ther.* 2008;38(8):465-475.
45. Fernandez-de-las Penas C, Perez-de-la-Heredia M. Performance of the craniocervical flexion test, forward head posture, and headache clinical parameters in patients with chronic tension-type headache: a pilot study. *J Orthop Sports Phys Ther.* 2007;37(2):33-39.
46. San Agustin CT, Wilmarth MA, Raymond J, Hilliard TS. The amount and variation of craniovertebral angle changes in college-aged students using one-strapped and two-strapped backpacks and bags. *Orthop Pract.* 2003;15(3):30-33.
47. Watson DH, Trott PH. Cervical headache: an investigation of natural head posture and upper cervical flexor muscle performance. *Cephalalgia.* 1993;13:272-284.
48. Paris SV. Cervical symptoms of forward head posture. *Top Geriatr Rehabil.* 1990;5(4):11-19.
49. Fernandez-de-la-Penas C, Alonso-Blanco C, Cuadrado ML, Gerwin RD, Pareja JA. Trigger points in the suboccipital muscles and forward head posture in tension-type headache. *Headache.* 2006;46:454-460.
50. Fernandez-de-las-Penas C, Alonso-Blanco C, Cuadrado ML, Pareja JA. Forward head posture and neck mobility in chronic tension-type headache: a blinded, controlled study. *Cephalalgia.* 2005;26:314-319.
51. Falla D, Jull G, Hodges PW. Feedforward activity of the cervical flexor muscles during voluntary arm movements is delayed in chronic neck pain. *Exp Brain Res.* 2004;157:43-48.
52. Kreig L. Thoracic outlet syndrome: Pathology and treatment. *J Manual Manipulative Ther.* 1993;1(2):52-59.
53. Keller TS, Harrison DE, Colloca CJ, Harrison DD, Janik TJ. Prediction of osteoporotic spinal deformity. *Spine.* 2003;28(5):455-462.
54. Ludewig PM, Cook TM. Alterations in shoulder kinematics and associated muscle activity in people with symptoms of shoulder impingement. *Phys Ther.* 2000;80(3):276-289.
55. Solem-Bertoft E, Thuomas K-A, Westerberg C-E. The influence of scapular retraction and protraction on the width of the subacromial space. *Clin Orthop Relat Res.* 1993;296:99-103.
56. Cacciatore TW, Horak FB, Henry SM. Improvement in automatic postural coordination following Alexander technique lessons in a person with low back pain. *Phys Ther.* 2005;85(6):565-578.
57. Jankelson RR. *Posture, Airway & the Tongue in Clinical Dentistry.* Kent, WA: Myotronics, Inc; 2006.
58. McMaster MJ, Glasby MA, Singh H, Cunningham S. Lung function in congenital kyphosis and kyphoscoliosis. *J Spinal Disord Tech.* 2007;20(3):203-208.
59. Wright EF, Domenech MA, Fischer JR. Usefulness of posture training for patients with temporomandibular disorders. *JADA.* 2000;131:202-210.
60. Evcik D, Aksoy O. Relationship between head posture and temporomandibular dysfunction syndrome. *J Musculoskel Pain.* 2004;12(2):19-24
61. Hiemeyer K, Lutz R, Menninger H. Dependence of tender points upon posture – a key to the understanding of fibromyalgia syndrome. *J Manual Medicine.* 1990;5:169-174.
62. Lennon J, Shealy CN, Cady RK, Matta W, Cox R, Simpson WF. Postural and respiratory modulation of autonomic function, pain, and health. *Amer J Pain Management.* 1994;4(1):36-39.
63. Freeman JT. Posture in the aging and aged body. *JAMA.* 1957;165(7):843-846.
64. Calliet R. *Soft Tissue Pain and Disability.* 3rd ed. Philadelphia, PA: FA Davis; 1996.
65. Conable B, Conable W. *How to Learn the Alexander Technique: A Manual for Students.* 2nd ed. Columbus, OH: Andover Road Press; 1992.
66. Chaitow L, DeLany JW. *Clinical Application of Neuromuscular Techniques. Vol. 2, The Lower Body.* Edinburgh, UK: Churchill Livingstone; 2002.
67. Paris SV. *Course Notes, The Spine: Etiology and Treatment of Dysfunction Including Joint Manipulation.* Atlanta, GA: 1979.
68. Chaitow L. *Positional Release Techniques.* Edinburgh, UK: Churchill Livingstone; 2002.
69. Kaltenborn FM. *Manual Mobilization of the Joints, Vol. 1, The Extremities,* 6th ed. Minneapolis, MN: OPTP; 2002.
70. Evjenth O, Hamberg J. *Muscle Stretching in Manual Therapy: A Clinical Manual. Vol. II The Spinal Column and Temporomandibular Joint.* Alfta, Sweden: Scand; 1984.
71. Simons DG, Travell JG, Simons LS. *Travell & Simons' Myofascial Pain and Dysfunction: The Trigger Point Manual. Vol 1. Upper Half of Body.* Baltimore, MD: Williams & Wilkins; 1999.
72. Schmitt L, Snyder-Mackler L. Role of scapular stabilizers in etiology and treatment of impingement syndrome. *J Orthop Sports Phys Ther.* 1999;29(1):31-38.
73. Tubbs RS, Tyler-Kabara EC, Aikens AC, et al. Surgical anatomy of the dorsal scapular nerve. *J Neurosurg.* 2005;102:910-911.

74. Kuzmich D. The levator scapulae: making the con-neck-tion. *J Manual Manipulative Ther.* 1994;2(2):43-54.
75. Diener I. The effect of levator scapula tightness on the cervical spine: proposal of another length test. *J Manual Manipulative Ther.* 1998;6(2):78-86.
76. Butler DS. *Mobilization of the Nervous System.* Melbourne, Australia: Churchill Livingstone; 1991.
77. Butler DS. *The Sensitive Nervous System.* Adelaide, Australia: Noigroup Publications; 2000.
78. Herrington L. Effect of different neurodynamic mobilization techniques on knee extension range of motion in the slump position. *J Manual Manipulative Ther.* 2006;14(2):101-107.
79. Hammer WI. *Functional Soft Tissue Examination and Treatment by Manual Methods: New Perspectives.* 2nd ed. Gaithersburg, MD: Aspen Publishers; 1999.
80. Conable B, Conable W. *How to Learn the Alexander Technique: A Manual for Students.* 2nd ed. Columbus, OH: Andover Road Press; 1992.
81. Brennan R. *The Alexander Technique Workbook, Your Personal System for Health, Poise, and Fitness.* London: Element Books Ltd; 2002.
82. Caplan D. *Back Trouble: A New Approach to Prevention and Recovery.* Gainesville, FL: Triad Publishing; 1987.
83. Goldstein LB, Makofsky HW. TMD/facial pain and forward head posture. *Practical Pain Management.* 2005;5(5):36-39.
84. Augustine C, Makofsky HW, Britt C, et al. Use of the Occivator for the correction of forward head posture and the implications for temporomandibular disorders: a pilot study. *J Craniomandib Pract.* 2008;26(2):1-8.
85. Coric D, Adamson T. Minimally invasive cervical microendoscopic laminoforaminotomy. *Neurosurg Focus.* 2008;25(2):E2.
86. Li J, Yan DL, Zhang ZH. Percutaneous cervical nucleoplasty in the treatment of cervical disc herniation. *Eur Spine J.* 2008;17(12):1664-1669.
87. Fernandez-de-las-Penas C, Perez-de-Heredia M, Molero-Sanchez A, Miangolarra-Page JC. Performance of the craniocervical flexion test, forward head posture, and headache clinical parameters in patients with chronic tension-type headache: A pilot study. *J Orthop Sports Phys Ther.* 2007;37(2):33-39.
88. Jull G. Diagnosis of cervicogenic headache. *J Manual Manipulative Ther.* 2006;14(3):136-138.
89. Petersen SM. Articular and muscular impairments in cervicogenic headache. *J Orthop Sports Phys Ther.* 2003;33(1):21-30.
90. Harris KD, Heer DM, Roy TC, Santos DM, Whitman JM, Wainner RS. Reliability of a measurement of neck flexor muscle endurance. *Phys Ther.* 2005;85(12):1349-1355.
91. Murphy DR. Normal function of the cervical spine II, neurophysiology and stability. In: Murphy DR, ed. *Conservative Management of Cervical Spine Syndromes.* New York, NY: McGraw-Hill Companies, Inc; 2000.
92. Bogduk N. Cervicogenic headache: anatomic basis and pathophysiologic mechanisms. *Current Pain and Headache Reports.* 2001;5:382-386.
93. Wrisley DM, Sparto PJ, Whitney SL, Furman JM. Cervicogenic dizziness: a review of diagnosis and treatment. *J Orthop Sports Phys Ther.* 2000;30(12):755-766.
94. Mannheimer JS, Rosenthal RM. Acute and chronic postural abnormalities as related to craniofacial pain and temporomandibular disorders. *Dent Clin North Amer.* 1991;35(1):185-208.
95. Porterfield JA, DeRosa C. *Mechanical Neck Pain. Perspectives in Functional Anatomy.* Philadelphia, PA: WB Saunders; 1995.
96. Hall T, Chan HT, Christensen L, Odenthal B, Wells C, Robinson K. Efficacy of a C1-C2 self-sustained natural apophyseal glide (SNAG) in the management of cervicogenic headache. *J Orthop Sports Phys Ther.* 2007;37(3):100-107.
97. Zito G, Jull G, Story I. Clinical tests of musculoskeletal dysfunction in the diagnosis of cervicogenic headache. *Manual Therapy.* 2006;11:118-129.
98. van Duijn J, van Duijn A, Nitsch W. Orthopaedic manual physical therapy including thrust manipulation and exercise in the management of a patient with cervicogenic headache: a case report. *J Manual Manipulative Ther.* 2007;15(1):10-24.
99. Hanten WP, Olson SL, Lindsay WA, Lounsberry KA, Stewart JK. The effect of manual therapy and a home exercise program on cervicogenic headaches: a case report. *J Manual Manipulative Ther.* 2005;13(1):35-43.
100. Horn C, Smith KL. Cervicogenic headache part II: clinical examination, findings, and approaches to management. *J Manual Manipulative Ther.* 1997;5(4):171-175.
101. Hall T, Briffa K, Hopper D. Clinical evaluation of cervicogenic headache: a clinical perspective. *J Manual Manipulative Ther.* 2008;16(2):73-80
102. Bogduk N. Cervical causes of headache and dizziness. In: Grieve GP, ed. *Modern Manual Therapy of the Vertebral Column.* 2nd ed. Edinburgh: Churchill Livingstone; 1994.
103. Jull GA. Headaches of cervical origin. In: Grant R, ed. *Physical Therapy of the Cervical and Thoracic Spine: Clinics in Physical Therapy.* London: Churchill Livingstone; 1988.
104. IHS: Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders. 2nd ed. *Cephalalgia.* 2004;24(Suppl 1):9-160.
105. Sjaastad O, Fredriksen A, Pfaffenrath V. Cervicogenic headache: diagnostic criteria. *Headache.* 1998;38:442-446.
106. McDonnell MK, Sahrmann SA, Van Dillen L. A specific exercise program and modification of postural alignment for treatment of cervicogenic headache: a case report. *J Orthop Sports Phys Ther.* 2005;35(1):3-15.
107. Schoensee SK, Jensen G, Nicholson G, Gossman M, Katholi C. The effect of mobilization on cervical headaches. *J Orthop Sports Phys Ther.* 1995;21(4):184-190.
108. Hall T, Robinson K. The flexion rotation test and active cervical mobility: a comparative measurement study in cervicogenic headache. *Manual Therapy.* 2004;9:197-202.
109. Swerdlow B. *Whiplash and Related Headaches.* Boca Raton, FL: CRC Press; 1999.
110. McCrory DC, Penzien DB, Hasselblad V, Gray RN. *Evidence Report: Behavioral and Physical Treatments for Tension-Type and Cervicogenic Headache.* Durham, NC: Duke University Evidence-Based Practice Center; 2001.
111. Jull G. Management of cervical headache. *Manual Therapy.* 1997;2(4):182-190.
112. Kidd RF, Nelson R. Musculoskeletal dysfunction of the neck in migraine and tension headache. *Headache.* 1993;33(10):566-569.
113. Moraska A, Chandler C. Changes in clinical parameters in patients with tension-type headache following massage therapy: A pilot study. *J Manual Manipulative Ther.* 2008;16(2):106-112
114. Fernandez-de-las-penas C, Arendt-Nielsen L, Simons D. Contributions of myofascial trigger points to chronic tension-type headache. *J Manual Manipulative Ther.* 2006;14(4):222-231.
115. Fernandez-de-las-penas C, Cuadrado ML, Pareja JA. Myofascial trigger points, neck mobility, and forward head posture in episodic tension-type headache. *Headache.* 2007;47(5):662-672.
116. Saper JR. Chronic daily headache: a clinician's perspective. *Headache.* 2002;42(6):538-542.
117. Fernandez-de-las-Penas C, Cuadrado ML, Pareja JA. Myofascial trigger points, neck mobility, and forward head posture in unilateral migraine. *Cephalalgia.* 2006;26:1061-1070.
118. Hack GD, Koritzer RT, Robinson WL, Hallgren RC, Greenman PE. Anatomic relation between the rectus capitis posterior minor muscle and the dura mater. *Spine.* 1995;20(23):2484-2486
119. Marcus DA, Scharff L, Mercer S, Turk DC. Musculoskeletal abnormalities in chronic headache: a controlled comparison of headache diagnostic groups. *Headache.* 1999;39:21-27.
120. Karpouzis KM, Spierings E. Circumstances of onset of chronic headache in patients attending a specialty practice. *Headache.* 1999;39:317-320.

121. Silberstein S, Mathew N, Saper J, Jenkins S. Botulinum toxin type A as a migraine preventive treatment. *Headache*. 2000;40:445-450.
122. Moskowitz MA. The neurobiology of vascular head pain. *Ann Neurol*. 1984;16:156-168.
123. Olesen J. Clinical and pathophysiological observations in migraine and tension-type headache explained by integration of vascular, supraspinal, and myofascial inputs. *Pain*. 1991;46:125-132.
124. Wood P. Proposed models of fibromyalgia sub-types. *Practical Pain Management*. 2008;8(5):70-75.
125. Young WB, Silberstein SD. *Migraine and Other Headaches*. New York, NY: Demos Medical Publishing LLC; 2004.
126. Bendtsen L. Central sensitization in tension-type headache: Possible patho-physiological mechanisms. *Cephalalgia*. 2000;29:486-508.
127. Shankland WE. Migraine and tension-type headache reduction through pericranial muscular suppression : a preliminary report. *J Craniomandib Pract*. 2001;19(4):269-278.
128. Saper JR, Silberstein S, Gordon CD, Hamel RL, Swidan S. *Handbook of Headache Management: A Practical Guide to Diagnosis and Treatment of Head, Neck, and Facial Pain*. 2nd ed. Baltimore, MD: Lipincott Williams & Wilkins; 1999.
129. DaSilva AF, Granziera C, Snyder J, Hadjikhani N. Thickening in the somatosensory cortex of patients with migraine. *Neurology*. 2007;69:1990-1995.
130. Morrone L. *Overcoming Headaches and Migraines: Clinically Proven Cure for Chronic Pain*. Eugene, OR: Harvest House Publishers; 2008.
131. Willis GC. *Headache. Lecture Notes*. Maizuru General Hospital, Kyoto Prefecture, Japan, 1990.
132. Packard RC. Post-traumatic headache: more than just a headache. *Headache Quarterly*. 2001;12:99-100.
133. Jensen O, Nielsen F, Vosmar L. An open study comparing manual therapy with the use of cold packs in the treatment of posttraumatic headache. *Cephalalgia*. 1990;10:241-250.
134. Hildebrandt J, Jansen J. Vascular compression of the C2 and C3 roots—yet another cause of chronic intermittent hemicrania? *Cephalalgia*. 1984;4:167-170.
135. Mathew NT, Stubits E, Nigam M. Transformation of episodic migraine into daily chronic headache: an analysis of factors. *Headache*. 1982;22:66-68.
136. Srikiatkhachorn A, Tarasub N, Govitrapong P. Effect of chronic analgesic exposure on the central serotonin system: a possible mechanism of analgesic abuse headache. *Headache*. 2000;40:343-350.
137. Wrisley DM, Sparto PJ, Whitney SL, Furman JM. Cervicogenic dizziness: a review of diagnosis and treatment. *J Orthop Sports Phys Ther*. 2000;30(12):755-766.
138. Kondratek M, Creighton D, Krauss J. Use of translatoric mobilization in a patient with cervicogenic dizziness and motion restriction: a case report. *J Manual Manipulative Ther*. 2006;14(3):140-151.
139. Aspinall W. Clinical testing for cervical mechanical disorders which produce ischemic vertigo. *J Orthop Sports Phys Ther*. 1989;11(5):176-182.
140. Meadows J. *Orthopedic Differential Diagnosis in Physical Therapy*. New York, NY: McGraw-Hill, 1999.
141. Yardley L, Donovan-Hall M, Smith HE, Walsh BM, Mullee M, Bronstein AM. Effectiveness of primary care-based vestibular rehabilitation for chronic dizziness. *Ann Intern Med*. 2004;141(8):598-605.
142. Huijbregts P, Vidal P. Dizziness in orthopaedic physical therapy practice: classification and pathophysiology. *J Manual Manipulative Ther*. 2004;12(4):199-214.
143. Willis GC. *Acoustic Nerve: Deafness, Tinnitis, and Vertigo. Neuroanatomy of the VIII Cranial Nerve and Connections. Lecture Notes*. Maizuru General Hospital, Kyoto Prefecture, Japan, 1990.
144. Vidal P, Huijbregts P. Dizziness in orthopaedic physical therapy practice: history and physical examination. *J Manual Manipulative Ther*. 2005;13(4):221-250.
145. Vidal P. *Examination and Intervention of Dizziness: The Vestibular System and Orthopaedics*. Brentwood, TN: Paul Vidal & Cross Country Education, Inc; 2005.
146. Oostendorp R, VanEupen A, VanErp J, Elvers H. Dizziness following whiplash injury: a neuro-otological study in manual therapy practice and therapeutic implication. *J Manual Manipulative Ther*. 1999;7(3):123-130.
147. Endo K, Ichimaru K, Komagata M, Yamamoto K. Cervical vertigo and dizziness after whiplash injury. *Eur Spine J*. 2006;15:886-890.
148. Wapner S, Werner H, Chandler KA. Experiments on the sensory-tonic field theory of perception: effect of extraneous stimulation of the visual perception of verticality. *J Exp Psych*. 1951;42:351-357.
149. Gray LP. Extralabyrinthine vertigo due to cervical muscle lesions. *J Laryngology*. 1956;70:352-361.
150. Fitz-Ritson D. Assessment of cervicogenic vertigo. *J Manipulative Physiol Ther*. 1991;14(3):193-198.
151. Vernon H. *Upper Cervical Syndrome: Chiropractic Diagnosis and Treatment*. Baltimore, MD: Williams & Wilkins; 1988.
152. Janda V. Muscles and motor control in cervicogenic disorders. In: Grant R, ed. *Physical Therapy of the Cervical and Thoracic Spine*. 3rd ed. New York, NY: Churchill Livingstone; 2002.
153. Urbanowicz M. Alteration of vertical dimension and it effect on head and neck posture. *J Craniomandib Pract*. 1991;9(2):174-179.
154. Chakfa AM, Mehta NR, Forgione AG, Al-Badawi EA, Lobo S, Zawawi KH. The effect of stepwise increases in vertical dimension of occlusion on isometric strength of cervical flexors and deltoid muscles in nonsymptomatic females. *J Craniomandib Pract*. 2002;20(4):264-273.
155. Makofsky HW. Snoring and obstructive sleep apnea: does head posture play a role? *J Craniomandib Pract*. 1997;15(1):68-73.

Section

IV

TEMPOROMANDIBULAR JOINT

13

Examination and Evaluation of the Temporomandibular Joint

Posture

The analysis of craniomandibular alignment or posture is a complex science that requires expertise in general dentistry, orthodontics, oral and maxillofacial surgery, as well as in physical medicine.[1-3] For those therapists with advanced training in the TMJ, including an understanding of cranial osteopathy, the analysis of craniofacial structure is an essential component of the examination. However, at the introductory level, more emphasis is placed on the analysis of mandibular range of motion, soft tissue palpation, and the influence of the cervical spine and posture on the craniomandibular region then on structural alignment, including the assessment of dental occlusion. That being said, the basic examination of the TMJ/facial region should note the following:

- Facial type (eg, a longer dolichocephalic face versus a rounder brachicephalic one)
- Deviations from the normal orthognathic position, including horizontal deficiency of the lower jaw (ie, retrognathia) as well as horizontal excess of the mandible (ie, prognathia), as observed from the side. Whereas the orthognathic profile has a straight appearance, the retrognathic mandible appears convex; the prognathic jaw concave.
- From the front, the height of the mandibular ramus (from gonial angle to the head of the condyle) should be compared from left to right for asymmetry. If for example, the left ramus is longer, the patient's face will appear convex on the left; concave on the right. This may predispose the patient's right TMJ toward hypomobility; the left towards hypermobility upon opening of the mouth.

As mentioned, the examination of dental occlusion is beyond the scope of this introductory textbook. However, certain dental concepts[1-3] are useful in terms of understanding the role of head-neck posture in both craniomandibular kinesiology and pathokinesiology. The term *maximum intercuspation* (MIP) refers to the position of the upper and lower teeth in the fully clenched state of the upper and lower jaws. It is a function of tooth anatomy and geometry and is unaffected by transient changes in head-neck position. The term *vertical dimension of occlusion* (VDO) refers to the distance from the nose to the chin with the teeth in MIP. It, too, is a structurally determined dental relationship that is unaffected by anything other than occlusion. The dental profession alone has exclusive rights by virtue of their training and expertise to manage pathology, impairment, functional limitation, and disability related to MIP and VDO. Having said that, there are other dental concepts that are influenced by functional factors, including head-neck posture, that clearly fall within the domain of the physical therapy profession. Five such concepts that are related and that clearly fall within the functional realm are mandibular rest position, interocclusal or freeway space, the habitual pathway of closure, initial tooth contact position, and the vertical dimension of rest (VDR). Though many would argue that these concepts are also dental in nature, there is no doubt that extradental factors (eg, head-neck posture) also play a role. For example, it has been established that head-neck extension exerts a posterior force on the mandible, which changes the pathway of mandibular

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Figure 13-1a. TMJ palpatation during mandibular depression.

Figure 13-1b. Active mandibular depression from the side.

Figure 13-1c. Measuring mandibular depression.

closure and shifts the initial tooth contacts posteriorly.[4] With regard to head-neck posture, it has been demonstrated that FHP exerts a superiorly directed force on the mandible which alters the rest position of the mandihle and decreases freeway space as well as VDR.[5] Consequently, the basic examination of mandibular posture must include an inspection of the influences from below, namely an examination of the cervical and scapulothoracic region as previously covered in this text.

Active Mandibular Movements

There are 4 active movements of the mandible that will be assessed. They include mandibular depression (opening), lateral excursion to the right and left, and protrusion.

When assessing depression of the mandible, the examiner must do the following:

1. Palpate the lateral poles of the mandibular condyles for joint sounds (Figure 13-1a).
2. Observe for mandibular deflections and deviations (see Figure 13-1a) from the front.
3. Observe for premature and/or excessive anterior mandibular translation from the side (Figure 13-1b).
4. Measure (Figure 13-1c) the maximal interincisal opening (MIO).

The normal TMJ (Figure 13-2) is freely moveable, friction-free, and noise-free.[6] However, in the impaired TMJ there are basically 3 types of joint sounds that can be palpated. They are clicking, crepitus, and a popping sound or "thud." Most clicks are single, short duration noises associated with a reducing disc displacement. They can be palpated during opening or closing and may occur at any point in the opening/closing cycle. When a TMJ demonstrates both opening and closing clicking, the term *reciprocal clicking* is used. This is a sign of an anterior disc displacement (ADD) with reduction (Figure 13-3a). The opening click is typically more pronounced than the closing click, which may require auscultation with a stethoscope in order to be heard. This is in contrast to an ADD without reduction (ie, a closed-lock of the TMJ) in which joint clicking is absent (Figure 13-3b). Reciprocal clicking must be distinguished from the clicking that occurs secondary to an articular surface defect. Whereas an articular surface defect click will occur at the same point in the opening and closing cycle, reciprocal clicking rarely occurs at the same point in both opening and closing. The opening click usually occurs beyond 20 mm and the closing click occurs just before the teeth meet in occlusion. Crepitus is a grating or gravelly noise associated with degenerative joint disease of which the TMJ is not excluded. A loud popping noise or thud palpated at the end of opening indicates TMJ hypermobility. This occurs as the disc and mandibular condyle, together, translate past the articular eminence of the temporal bone. This hypermobility can be confined to the TMJ or be a generalized state of increased motion throughout the body. When the disc/condyle complex translates anterior to the articular eminence and cannot return to its normal anatomic position, it is considered dislocated or an open-lock (Figure 13-4).

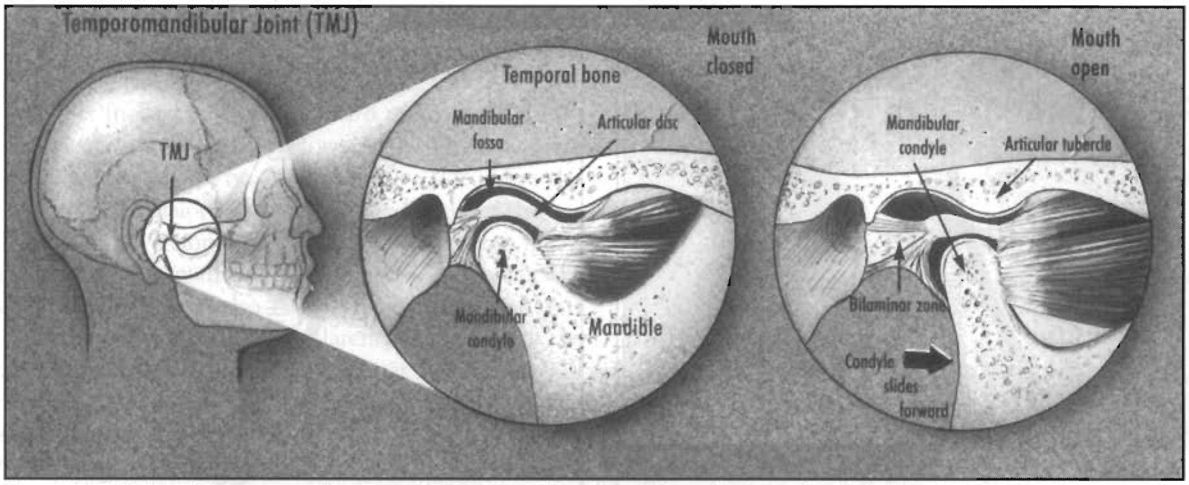

Figure 13-2. The TMJ at rest and in mouth opening. (Reprinted from Morrone L, Makofsky H. The TMJ home exercise program. *Clinical Management in Physical Therapy*. 1991;11(2):20-26, with permission of the American Physical Therapy Association.)

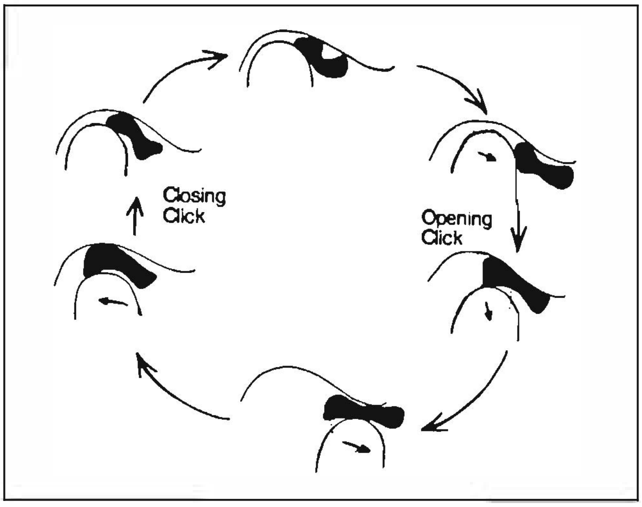

Figure 13-3a. Right TMJ anterior disc displacement with reduction.

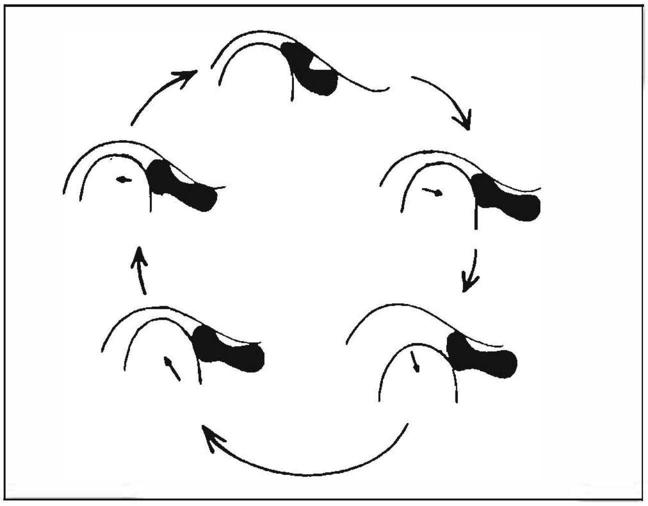

Figure 13-3b. Right TMJ anterior disc displacement without reduction (closed-lock).

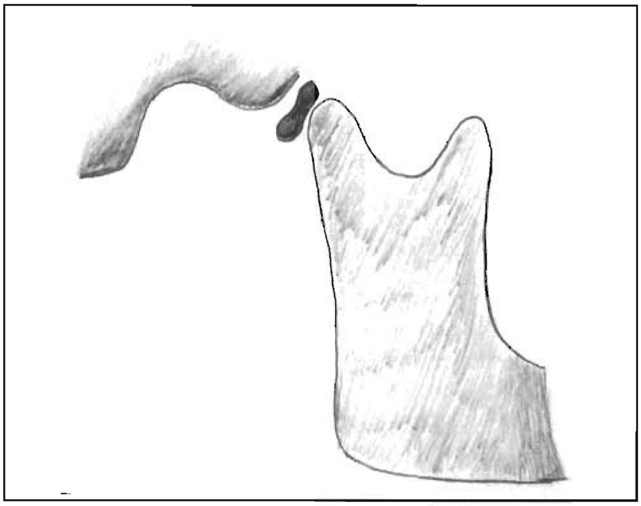

Figure 13-4. Right TMJ dislocation (open-lock).

The second aspect of examining depression involves observing deflections and deviations of the mandible. The mandible is said to deflect when it shifts from its midline position to either the right or the left side and fails to return to the midline (Figure 13-5). Deflections occur when the mandibular condyle has restricted anterior translation on the ipsilateral side or excessive translation on the contralateral side. For example, if translation is restricted on the right, a deflection will occur to the right; if excessive on the right, a deflection will occur to the left (usually toward the end of range). The pathology leading to impairment of translation is most often due to either unilateral capsular tightness or an ADD without reduction. Although the underlying pathology is different (ie, capsular versus intracapsular), the deflection of the depressing mandible to the side of impairment is the same.

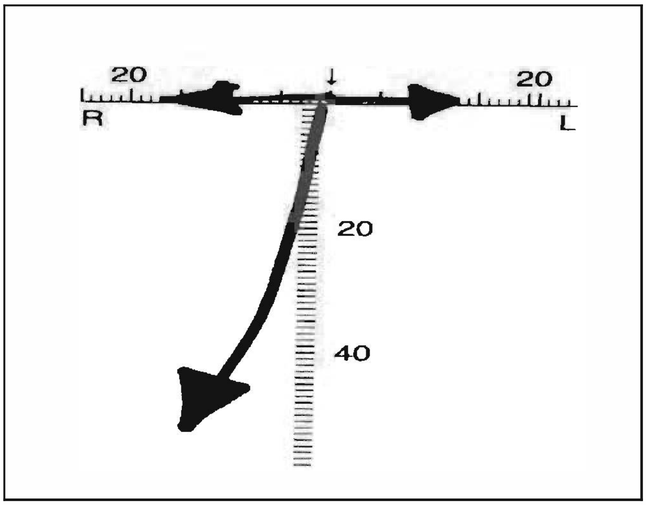

Figure 13-5. Mandibular deflection to the right.

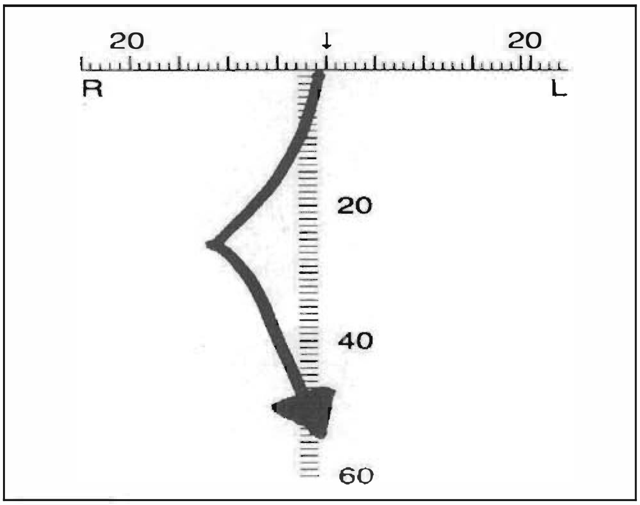

Figure 13-6. Mandibular deviation to the right.

Figure 13-7a. Lateral excursion left.

Figure 13-7b. Lateral excursion right.

The mandible is said to deviate when it shifts to one side of midline during opening but then returns to the midline as opening continues (Figure 13-6). Deviations during opening, when correlated to ipsilateral reciprocal clicking, are usually secondary to an ADD with reduction. Whereas the anteriorly displaced disc causes a shift of the lower jaw to the affected side (due to a momentary interruption of mandibular translation), the return of the mandible to midline occurs when the displaced disc is reduced. This disc reduction (ie, normalization of position) produces the characteristic opening click. The closing click occurs when the condyle slips off the posterior aspect of the disc, usually at the end of closing.

Mandibular depression should also be assessed from the side (see Figure 13-1b). For simplicity's sake, depression of the mandible can be divided into 3 phases. The initial phase of opening consists of an X-axis rotation. The middle phase consists of a combination of X-axis rotation and translation of the mandible along the Z or anteroposterior axis, and the final phase of opening consists primarily of further anterior translation along the Z axis. It is believed that rotational motion within the TMJ occurs in the inferior joint compartment between the head of the condyle and the articular disc, whereas translation or sliding motion occurs in the superior compartment of the TMJ between the disc and articular eminence of the temporal bone. A common pattern seen in patients with TMD is premature or excessive translation. It is this premature or excessive translation that causes mechanical stress and strain within the tissues of the TMJ, leading to the common development of hypermobility.

The normal range of mandibular depression MIO is between 40 and 50 mm, as measured between the upper and lower anterior incisors (see Figure 13-1c). In the absence of a metric ruler, the patient is asked to place his or her knuckles between the upper and lower teeth in a sideways manner. One knuckle opening is hypomobile, 2 is low normal, 3 is high normal, and 4 tends toward hypermobility.

Normal range for mandibular lateral excursion is 8 mm to either side. A metric ruler can be used, but an easier method involves observing the lower lip frenulum as the lower jaw moves from side to side (Figures 13-7a and 13-7b). The therapist's gloved hand pulls the lower lip down to expose the frenulum. With the teeth slightly apart, the patient moves his or her mandible to the right and then to the left. Since the anterior upper incisor is approximately 8 mm in width, normal lateral excursion involves the lower lip frenulum clearing the upper anterior incisor on each respective side. Thus the patient's lateral excursions can be evaluated without the use of a ruler if desired. If impairment of motion is present, it can be described as minimal, moderate, or severe. Conversely, excessive motion should also be noted. For those patients who have difficulty coordinating lateral excursion of the mandible, placing the tongue on the upper back molar will assist with lateral motion to that side. A distinction should be made between restricted mobility and incoordination.

The final active mandibular movement to assess is protrusion (Figure 13-8). The examination of mandibular protrusion includes palpating the lateral poles of the condyles for joint sounds, observing the motion for deflections and deviations, and measuring the quantity of motion present. Normal protrusion should obtain to 8 mm. A simple way of assessing this is to ask the patient to place his or her lower teeth anterior to the upper teeth. If this can be achieved, then the motion has normal range. Abnormal motion can be described as minimal, moderate, or severe limitation or hypermobility. The clinical interpretation of joint sounds and deflections/deviations in protrusion is similar to the same findings in the opening and closing cycle of the mandible as discussed previously.

Mandibular retrusion is not tested actively, but it can be assessed passively as a TMJ provocation test. This can be done in supine or sitting by means of a gentle up and back motion of the mandible. Pain in one or both TMJs suggests the presence of inflammation. Easy does it!

Intraoral Joint Play Motion

There are 2 indications for the use of intraoral joint play motion testing. One is suspected capsular hypomobility and the other is the likelihood of an ADD without reduction or a closed-lock (see Figure 13-3b). Because the TMJ is more often a disorder of hypermobility than hypomobility, manual therapists must be careful not to subject these tissues to unnecessary mechanical stress.

The indications of capsular hypomobility are as follows:

- A history of macrotrauma to the jaw with subsequent inflammation and/or a history of jaw immobilization following surgery, infection, or as an intervention for TMD.
- The presence of a capsular pattern when impairment is unilateral (ie, restricted depression associated with deflection to the affected side, restricted lateral excursion to the contralateral side, and restricted protrusion with mandibular deflection to the affected side).

In the presence of bilateral impairment, the mandible will not deflect nor deviate, but will demonstrate limitation in the normal range of depression, lateral excursion to either side, and protrusion.

Figure 13-8. Mandibular protrusion.

The indications of an intracapsular closed-lock are a prior history of reciprocal clicking and intermittent closed locking. Though a closed-lock can occur following a single macrotrauma, it usually occurs in response to cumulative microtrauma over a period of time and is associated with such parafunctional activities as bruxism, nail biting, gum chewing, and other nonessential activities that stress and strain the internal supportive structures of the TMJ.

Consequently, intraoral joint play testing of the TMJ is helpful in confirming the diagnosis of TMJ hypomobility and is useful in distinguishing a tight capsule from a nonreducing disc displacement (though a closed-lock results in hypomobility, its precursor, the ADD with reduction, is actually a form of hypermobility between the condyle and articular disc). Though an MRI examination is the gold standard for the diagnosis of a closed-lock, the MRI should not be ordered unless TMJ surgery is being considered. The difference between a tight capsule and a closed-lock relative to intraoral joint play testing is twofold. The trained manual therapist is usually able to detect a difference in the end-feel. Whereas the tight capsule has a slight degree of "creep" or "give" at the end-range, the nonreducing disc derangement is less yielding and is often associated with muscle splinting, which makes the end-feel even firmer. However, the more significant distinction between the two is found in the response to manipulation. Whereas the tight capsule gains millimeters, the closed-lock gains centimeters of increased motion. This distinction holds true whenever an internal derangement is reduced and a joint is "unlocked" (eg, the knee, spine, elbow).

There are 3 joint play motions of the TMJ that will be assessed intraorally. They are long axis distraction, lateral glide, and anterior glide. The therapist stands on the side opposite the joint to be mobilized and stabilizes the head while monitoring the affected joint with either the middle or index finger. The gloved thumb of the other hand is placed intraorally on the mandibular arch with the index finger alongside the body of the mandible extraorally. This

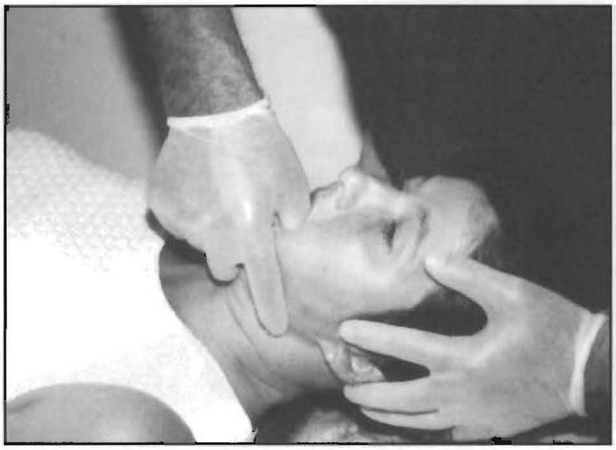

Figure 13-9. Right TMJ intraoral mobility testing.

Figure 13-10a. Temporalis muscle.

Figure 13-10b. Masseter muscle.

Figure 13-10c. Medial pterygoid muscle.

examination technique is demonstrated with the patient in the supine position (Figure 13-9), but it can also be performed with the patient sitting.

For each of the intraoral movements tested, the quality, quantity (0 to 6 scale covered previously), tissue reactivity, and end-feel are assessed. Long axis distraction involves separation of the mandibular condyle away from the temporal fossa in a caudal direction, lateral glide involves translatoric motion in a straight lateral direction, and anterior glide consists of a translatoric motion in a forward or protrusive direction. Because of the potential for the cusps of the mandibular teeth to cause discomfort to the therapist's thumb, it is suggested that a sterile gauze pad be used as a cushion.

Soft Tissue Palpation

As with the palpation of other regions of the musculoskeletal system, the 3 markers of soft tissue impairment include an assessment of tenderness, tightness, and tone. Tightness involves an increase in myofascial density without associated hypertonicity, whereas an increase in tone (eg, splinting, guarding, bracing) is neuroreflexive in nature and points to the presence of increased tissue reactivity as discussed previously in Chapter 3. Extracapsular impairment of the TMJ (ie, myofascial pain) is common in patients suffering from TMD. It can occur in conjunction with a capsular impairment, intracapsular derangement, or be found in the presence of a normal TMJ.

The basic evaluation of the TMJ soft tissues consists of an extraoral examination (Figures 13-10a to 13-10e) of the following structures:

- Temporalis muscle (anterior, middle, and posterior fibers)
- Masseter muscle (no distinction made between superficial and deep fibers)
- Medial pterygoid muscle (deep to the gonial angle)
- The soft tissues lateral and posterior to the lateral pole of the mandibular condyle (ie, TMJ ligament, joint capsule laterally and posteriorly, lateral collateral ligament, and the periosteum) in both the closed and open mouth positions.

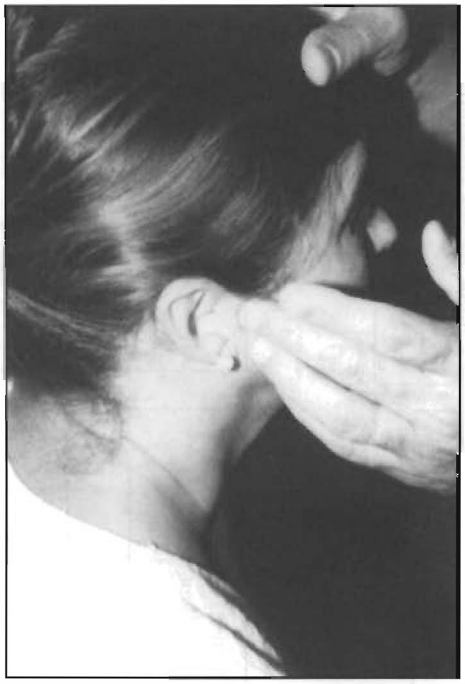

Figure 13-10d. Lateral pole in the closed mouth position (lateral structures).

Figure 13-10e. Lateral pole in the open mouth position (posterior and lateral structures).

Some examiners assess for posterior TMJ capsulitis by placing their fifth digits in the patient's ear canal (finger pads facing anteriorly) with the mouth open and then have the patient close against this anteriorly directed pressure. Although this method will detect TMJ pain/inflammation, it can be very uncomfortable for the patient and, in this author's opinion, unnecessary.

For an overview of the differential diagnosis of mechanical TMD, the reader is referred to Figure 13-11.

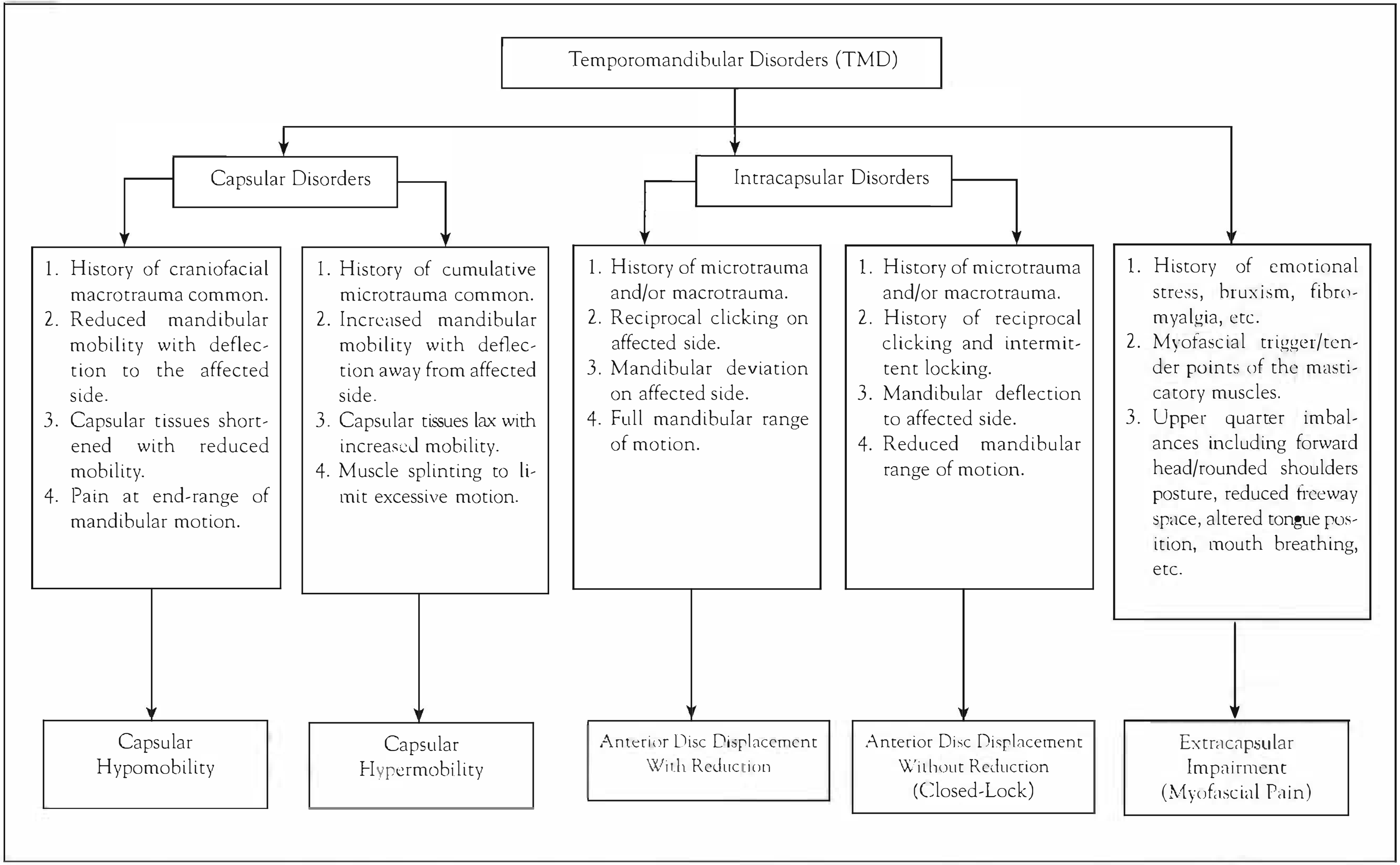

Figure 13-11. Differential diagnosis of mechanical TMD.

14

Connective Tissue Techniques for the Temporomandibular Joint/Facial Region

The utilization of direct fascial techniques for the purpose of achieving myofascial relaxation and relief of TMD symptoms is strongly recommended in conjunction with traditional physical therapy modalities (eg, ice, heat, electrical stimulation, ultrasound). This chapter will begin with the more basic extraoral approach to treatment followed by a description of intraoral direct fascial technique. When performing myofascial massage to the TMJ/facial area, the therapist must be mindful of the emotions related to a patient's facial area. For those individuals with histories of physical abuse, touching the face may evoke unpleasant memories. The key to effective facial technique is a gentle and caring touch. Once the patient grows accustomed to having the soft tissues of the jaw and face massaged, the therapist is then able to explore the release of deep tissue tension, myofascial trigger points, and muscle-holding states. For a review of the principles of direct fascial technique, the reader is referred to Chapter 5. A small amount of massage or hand cream is helpful when working in the craniofacial region.

Frontalis

The frontalis muscle, as with many of the TMJ/facial muscles, is overactive in many patients, often resulting in forehead wrinkles, frontal headache, and at times compression of the supraorbital nerve. Because the frontalis is contiguous with the occipitalis muscle (occipitofrontalis), this cutaneous muscle of the scalp will often require treatment of both components. Direct fascial technique of this epicranial muscle, along with the connecting temporoparietalis, can bring significant relief to patients suffering from chronic and episodic tension-type headache. Figure 14-1 illustrates a connective tissue technique that separates the muscle fibers of the frontalis in a medial to lateral direction. This approach can be extended posteriorly to include the occipitalis and laterally to include the temporoparietalis.

Corrugator, Orbicularis Oculi, and Procerus

The corrugator supercilii muscle (Figure 14-2) runs from the medial end of the superciliary arch to the deep surface of the skin above the middle of the orbital arch. It can be a source of pain at the medial and inferior aspect of the eyebrows and is overworked in patients who are habitual frowners. The orbicularis oculi is the closing muscle of the eye and is often tender and tight in patients who habitually squint. Proper eyeglasses and sunglasses will often remedy this problem. Direct fascial technique of these muscles must be performed with sensitivity, especially when releasing the taut fibers of the orbicularis oculi as they insert into the orbit and frontal bone above the eye. While in the upper nasal region, the procerus muscle should also be treated with gentle direct fascial technique, if necessary. It arises from either side of the nasal bone and runs upward to insert into the skin over the lower part of the forehead between the eyebrows. When the procerus muscles contract, the skin of the nose is pulled upward as the lower forehead is

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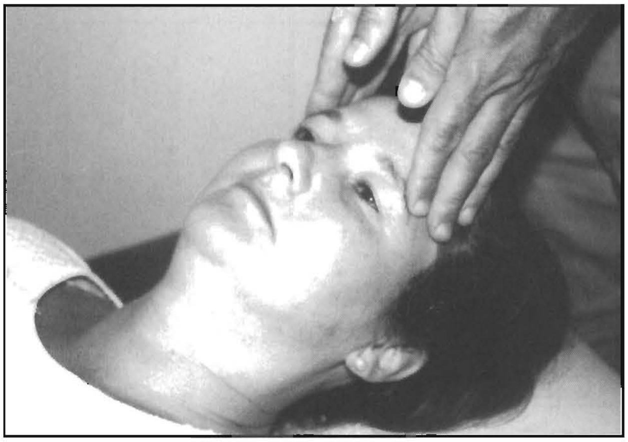

Figure 14-1. Frontalis.

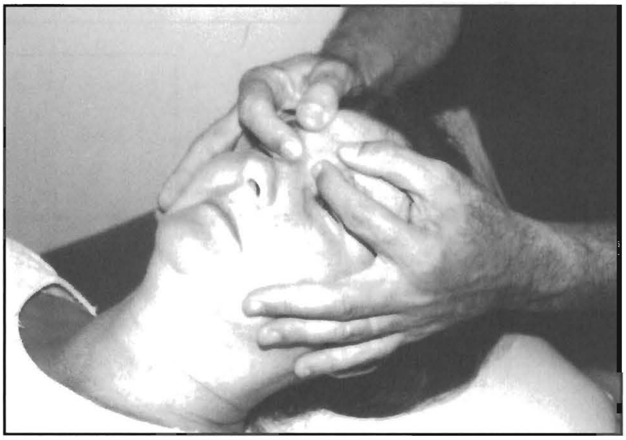

Figure 14-2. Corrugator, orbicularis oculi, procerus.

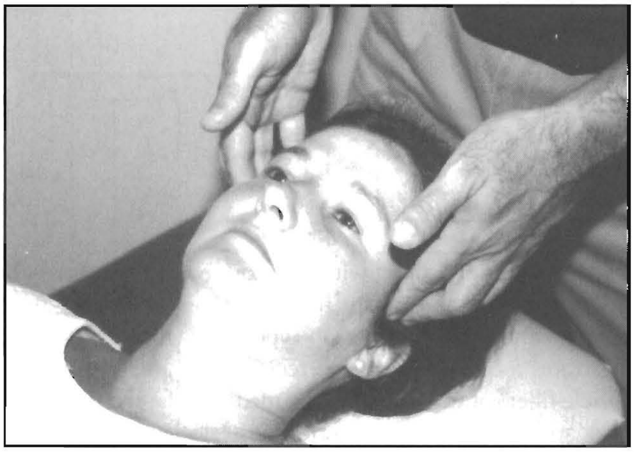

Figure 14-3. Temporalis.

Figure 14-4. Masseter.

pulled down, forming horizontal wrinkles between the eyebrows and over the bridge of the nose. It is the facial muscle responsible for the expression of distaste.

Temporalis

The temporalis muscle (Figure 14-3) is commonly involved in patients with TMD, especially in those suffering from extracapsular/myofascial impairment associated with bruxism and emotional stress. It is also a source of symptoms in patients suffering from tension-type headache. In addition, there appears to be a correlation between FHP and increased temporalis activity in which the mandible is displaced posterior and superior, thus reducing the interocclusal freeway space.[7] Consequently, treatment of the temporalis muscle with direct fascial technique is beneficial in a variety of musculoskeletal conditions related to the TMJ/facial region.

The therapist works in a direction perpendicular to the fibers and addresses all aspects of the muscle, including the anterior, middle, and posterior fibers. As with all soft tissue mobilizations, the goal is to soften, relax, and improve local circulation to the area in hopes of relieving painful symptoms and restoring normal function. It is also important that the therapist identify and correct all related impairments (eg, posture, jaw parafunction, stress) so that temporalis tone can return to a normal level.

Masseter

The masseter muscle (Figure 14-4) is a powerful elevator of the mandible and is commonly involved in the presence of restricted jaw opening. Myofascial trigger points of the superficial fibers result in facial pain. Involvement of the deep layer can be a cause of TMJ and ear symptoms. In addition to ipsilateral ear pain, the deep fibers can also be a source of tinnitus.

There is no attempt to differentiate the superficial from the deep layer when performing direct fascial technique to the masseter muscle. The therapist will find the most myofascial impairment (ie, tenderness, tightness, and tone) along the inferior aspect of the zygomatic arch and all along the angle and ramus of the mandible. Digital oscillations along these bony landmarks is quite effective in achieving the desired release of tension in this area.

Figure 14-5. Suprahyoids.

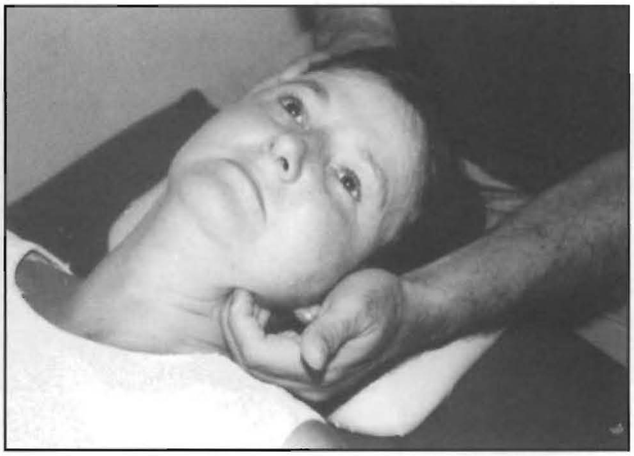

Figure 14-6. Medial pterygoid.

Suprahyoids

The suprahyoids (Figure 14-5) include the mylohyoid, stylohyoid, geniohyoid, and digastric muscles (anterior and posterior bellies). With FHP, the infrahyoids are under stretch but the suprahyoids tend to shorten as their origin and insertion approximate. This shortening will retrude the mandible and elevate the hyoid bone, both of which can adversely affect swallowing and the rest position of the mandible. Whereas myofascial trigger points in the mylohyoid muscle can refer pain to the tongue, trigger points in the stylohyoid and posterior belly of the digastric can cause head and neck pain. In addition, dentists should be aware that myofascial dysfunction of the anterior belly of the digastric can refer pain into the lower incisors.[8]

Medial Pterygoid

The medial pterygoid (Figure 14-6) along with the masseter and temporalis muscles is an elevator of the mandible. In patients who clench and grind their teeth, these muscles are prone to developing myofascial pain. Because of the proximity to the tensor palati muscle, hypertrophy of the medial pterygoid muscle may contribute to barohypoacusis (ie, ear stuffiness). Other myofascial symptoms include referred mouth, jaw, and ear pain.[8]

Palpation for examination and intervention can be accomplished either intraorally or extraorally. Palpation extraorally is accomplished by having the patient tilt his or her head to the ipsilateral side in order to slacken the tissues and permit greater access. The inner aspect of the angle of the mandible is explored with the palpating finger(s) as it presses in a superior and medial direction. The inferior fibers of the muscle's mandibular attachment are thereby accessed and treated with the appropriate direct fascial procedure. Strumming over taut fibers is especially useful in diminishing tone and restoring extensibility to the medial pterygoid muscle.

In addition to manual therapy interventions, including the connective tissue techniques described previously, other nonsurgical options such as electrotherapeutic modalities (eg, iontophoresis, low-level laser therapy), TMJ occlusal splint therapy, heat or ice, biofeedback, spray and stretch with fluorimethane spray, and acupuncture, have demonstrated effectiveness when dealing with TMD of somatic origin.[1,3,9-11] If indicated, the use of trigger point injections and short-term muscle relaxants should be discussed with the patient's dentist or physician. With regard to the role of intraoral devices, the Nociceptive Trigeminal Inhibition Tension Suppression System (NTI-TSS) has shown promise for the management of nocturnal bruxism[12] and may be an effective nonmedicinal intervention in the treatment of primary headache.[13] By disoccluding the posterior teeth, the clenching muscles are inhibited. Therefore, bruxism is also controlled, and the entire trigeminal afferent system is "defacilitated," explaining the therapeutic role of the NTI-TSS in both migraine and tension-type headaches.

Lateral Pole

There are several soft tissue attachments into the lateral pole of the mandibular condyle (Figure 14-7) that respond well to soft tissue mobilization. The author finds circular friction to be the intervention of choice in this region. The structures from superficial to deep include the TMJ ligament (outer-oblique and inner-horizontal fibers); the articular capsule, which is reinforced by the TMJ ligament; and the lateral collateral ligament, which secures the TMJ disc to the lateral pole. Circular friction around the lateral pole assists with decongestion of venous and lymphatic stasis, an increase in arterial flow, and relief of painful symptoms through the stimulation of various mechanoreceptors.

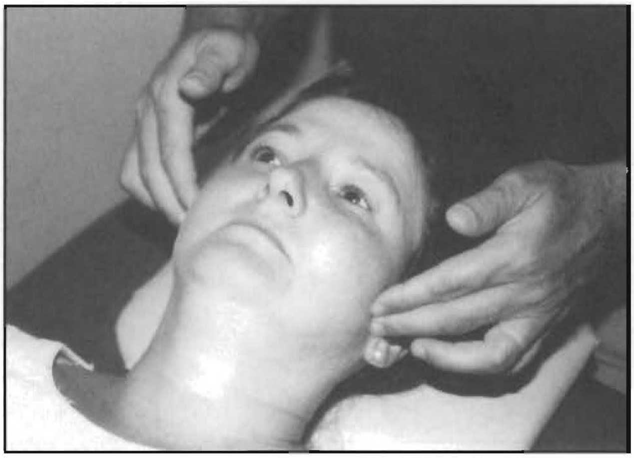

Figure 14-7. Lateral pole.

Figure 14-8. Intraoral temporalis fascial technique.

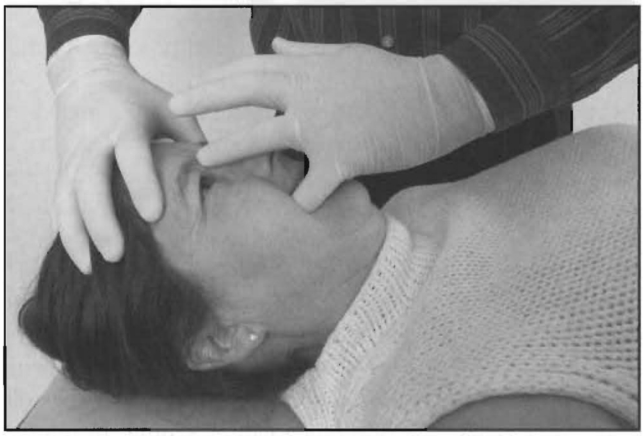

Figure 14-9. Intraoral lateral pterygoid fascial technique.

Figure 14-10. Intraoral medial pterygoid fascial technique.

Intraoral Direct Fascial Technique

If the extraoral approach fails to achieve satisfactory results, the therapist should consider working within the oral cavity to eradicate painful myofascial trigger points[8] of the masticatory muscles. The 4 intraoral techniques described in this section are for the temporalis, inferior head of the lateral pterygoid, the medial pterygoid, and masseter muscles (all intraoral procedures are performed using a sterile glove).

1. Temporalis – the patient is treated in supine with the therapist standing on the same side as the treated area. The therapist's fifth digit (palmar surface directed toward the therapist) is positioned above the maxillary teeth on the buccal surface, as far distal as possible, between the mandible and maxilla (more space is created by having the patient actively displace the mandible toward the therapist in lateral excursion). The therapist's fifth digit is then directed in a superior and lateral direction ("up and out") towards the coronoid process (Figure 14-8). This is the site of insertion of the temporalis tendon and can be extremely sensitive to touch. A gentle massaging motion is then applied to the temporalis tendon for 30 to 60 seconds. The desired response is a softening of the tissues.
2. Inferior head of the lateral pterygoid – the therapist now stands on the opposite side of the treated area as illustrated (Figure 14-9). The therapist's 5th digit is again placed above the maxillary teeth on the buccal surface, as far distal as possible, between the mandible and maxilla (active lateral excursion to the treated side creates more space for the therapist's 5th digit as above). The palmar surface of the 5th digit is then directed superiorly and medially ("up and in") against the lateral pterygoid plate of the sphenoid. This is also quite sensitive to touch and the technique must be performed slowly and gently. A massaging motion is applied to this area of myofascial insertion for 30 to 60 seconds.
3. Medial pterygoid – the therapist, standing on the same side of the treated area, places his or her index finger along the occlusal surface of the mandibular teeth and proceeds distally until the vertical mandibular ramus is encountered (Figure 14-10). The medial pterygoid is found on the medial side of the

Figure 14-11. Intraoral masseter fascial technique.

Figure 14-12. Auricular acupressure.

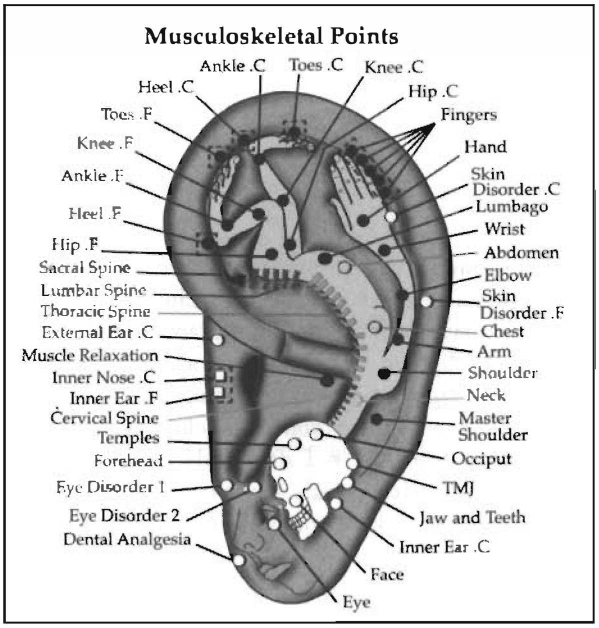

Figure 14-13. Auriculotherapy points.

ramus just above the back molars. Once again, a gentle massaging motion is applied for 30 to 60 seconds. As alluded to earlier, the medial pterygoid lies adjacent to the tensor palati and, if in spasm, it can mechanically limit the ability of the tensor palati to open the eustachian tube, resulting in ear stuffiness or fullness.[8] Consequently, myofascial treatment of the medial pterygoid can improve this troubling symptom when present.

4. Masseter muscle – the therapist uses a pincer grip to release areas of myofascial tension and eradicate painful trigger points in the superficial and deep fibers of the masseter (Figure 14-11). This technique is especially useful in patients who clench or grind their teeth (ie, bruxers).

If tolerated by the patient, any intraoral myofascial trigger point detected can also be treated with gentle specific compression. However, because of the extreme sensitivity of these intraoral structures, it is important for the therapist to monitor the patient's comfort level at all times. Patients can easily raise a hand when they have had enough!

Auricular Acupressure

The final manual soft tissue technique for the TMJ involves the use of ear acupressure, which is known as auriculotherapy. This system of therapy can be traced back to ancient China but received modern day notoriety because of the work of the French neurologist, Dr. Paul Nogier. It was Nogier who developed the Somatotopic Map of the Ear, based upon an inverted fetus orientation, which was subsequently verified in modern China in the 1960s.

Practitioners of auriculotherapy[14] claim to make use of various ear points in both diagnosis and intervention. The use of needles or pressure at specific points is used by these practitioners to manage a plethora of musculoskeletal pain conditions, including headache and TMD. Neurologically, the auricle is differentially innervated by the trigeminal, facial, glossopharyngeal, vagus, and superior cervical plexus nerves, making it a rich sensory area of the body for the purpose of relieving painful symptoms. The analgesia attained in response to auriculotherapy is not well understood, but may be a function of the spinal "gating" mechanism, descending neural inhibition, or endorphin release.

To effect the relief of pain and muscle guarding in the TMJ/facial region, the therapist applies pressure to the lowermost aspect of the helix tail at its junction with the lobe for approximately 2 minutes (Figure 14-12). It is suggested that both ears be treated simultaneously. Even though specific points are assigned to various conditions, the author has found a generalized examination of the lobe, antitragus, and inferior helix tail to reveal heightened areas of sensitivity and soft tissue density that, when treated with digital pressure, give several hours of relief of head, jaw, and face pain (Figure 14-13).

The reader is encouraged to explore this exciting modality for the temporary relief of painful symptoms in other areas of the body as well.

15

Intraoral Manipulation of the Temporomandibular Joint

As discussed previously, there are 2 conditions under which the TMJ becomes hypomobile. They are capsular impairment and a closed-lock (ie, anterior disc displacement without reduction). In this chapter, both causes of TMJ hypomobility will be addressed with intraoral manipulation. In the case of a tight and restricted capsule, the purpose will be to restore normal extensibility to the dysfunctional connective tissues through a series of joint play movements. In the case of an internal derangement, the purpose is to reduce the disc displacement and return it to a more normal and functional position. The author's experience in the management of TMD patients is that manipulation is seldom required as the vast majority of patients are hypermobile and instead require neuromuscular stabilization. In fact, a closed-lock is the terminal stage of hypermobility and it must be followed by a stabilization regimen when reduction is accomplished. In this chapter, the patient is positioned in supine. The advantage of the supine position is greater relaxation of the masticatory muscles. However, manual intraoral mobilization of the TMJ can also be performed in the sitting position if necessary.

Long Axis Distraction

The therapist's gloved thumb is placed intraorally over the mandibular arch on the posterior molar region while the other hand stabilizes the cranium (Figure 15-1). As with all manipulative procedures, the joint being mobilized should be monitored throughout the technique, in this case with the middle or index fingers at the lateral pole. As noted previously, the gloved thumb should be protected from the mandibular teeth with a sterile gauze pad.

Gentle long axis distraction involves separation of the mandibular condyle away from the mandibular fossa in a direction perpendicular to the fossa but parallel to the long axis of the mandibular ramus. Joint distraction is given a grade of 1 through 3 with 1 = joint unloading (Piccolo traction), 2 = the soft tissue slack is taken up, and 3 = the tissues are stretched beyond the slack to patient tolerance. As the therapist's thumb presses in an inferior direction, the other fingers simultaneously provide an upward force on the patient's chin.

Anterior Glide

Anterior glide or translation should be performed in conjunction with either grade 1, 2, or 3 distraction to avoid compression of the joint structures. The technique is performed as with long axis distraction with the addition of a pulling force on the mandible in an anterior direction. To enhance the stretch, the mandible can be mobilized anteriorly and slightly across midline, but only in those patients who show capsular impairment without signs of internal disc derangement.

Because the patient will be unable to speak during the intraoral procedure, he or she should indicate discomfort by raising his or her hand. The therapist should avoid excessive force on tissues that are prone to hypermobility and avoid working through pain except in rare cases of fibrous ankylosis in which some degree of discomfort is expected.

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Figure 15-1. Left TMJ long axis distraction, anterior glide, and lateral glide set-ups.

Figure 15-2. The bilateral TMJ disc reduction technique.

Lateral Glide

Lateral glide is achieved by applying a lateral force to the lingual surface of the posterior molar or to the lingual gum tissue on the side being treated. To avoid contralateral TMJ pain during this technique, the mandible should be slightly distracted and anteriorly glided.

With the above joint play gliding movements of the TMJ, graded mobilization is useful. As with other joints, grade 1 movements are used in the presence of high tissue reactivity, grade 2 and 3 movements are used in moderately reactive states, and grade 4 movements are indicated when tissue reactivity is low.

Manual Reduction of a Closed-Lock

The author has been comparing the efficacy of a unilateral manipulative technique to a bilateral approach for some time and has determined that patients with a closed-lock prefer and respond better to the bilateral approach. The muscles appear to "guard" less and the TMJ allows for more manipulative force when both joints are mobilized simultaneously. The goal in either method is to distract the TMJ enough to allow the disc to slide back over the head of the condyle so that condylar anterior translation can proceed without obstruction.

The bilateral reduction technique to unlock either the right or left TMJ will now be described (Figure 15-2). The patient must be assured that with the raising of a hand, the manual reduction will be aborted immediately. At no time is the joint to be forcefully "unlocked." Rather, it should be coaxed open in a gentle manner without anything more than mild discomfort. With both hands gloved and gauze pads wrapped around the thumbs, the therapist makes contact with the posterior mandibular molars, bilaterally. While stabilizing the cranium through the abdominal region, the therapist distracts both TMJs slowly to the end of range (ie, grade 3) by pressing down on the molars and lifting up under the chin. The mandible is then translated anteriorly from the distracted position to the end of range at which time the patient is asked to open his or her mouth as wide as possible. The mandible is slowly returned to the rest position and re-examined. If the closed-lock persists, a slightly different approach is used. The TMJs are again distracted and anteriorly translated. However, in addition to opening the mouth wide, the patient is asked to move the lower jaw from left to right several times. Again the mandible is returned to the rest position and re-examined. Although this author prefers the bilateral approach (as described above) for achieving TMJ disc reduction, there are patients who respond equally as well to the unilateral manual technique.[15]

Providing the nonreducing disc displacement has been successfully "recaptured," the patient should have cotton rolls placed between the posterior molars and go immediately to the dentist for fabrication of a TMJ intraoral appliance. A temporary splint can be used until the permanent device becomes available. In some cases, patients are able to remain reduced without the appliance, but in the majority of patients with chronic closed-lock, the reduction will not hold without it. As a general rule, the shorter the duration of the closed-lock, the better the likelihood of obtaining a manual reduction without the need for TMJ surgery. When considering a referral to a TMJ surgeon for a refractory closed-lock, the indications for either an arthroscopic or open joint procedure include intractable pain, failed conservative interventions (eg, physical therapy, TMJ occlusal splint therapy, pharmacologic measures, psychological therapy), and diagnostic confirmation of a nonreducing disc displacement with MRI, discography, etc. The author has only seen the need for surgical intervention on a few occasions and it has been the patient's intractable pain that has been the deciding factor. For those patients who choose to avoid TMJ surgery, the use of good physical therapy and dental splint therapy will enable the vast majority of patients to regain close to normal function despite the presence of a nonreducing disc displacement. The TMJ has a remarkable capacity to heal and should not be subjected to irreversible interventions unless absolutely necessary.

16

Therapeutic and Home Exercises for the Temporomandibular Joint

When considering the various nonsurgical options available for the management of TMD, therapeutic exercise has demonstrated efficacy in several studies,[9-11] including a randomized controlled trial showing statistically significant improvement in patients with anterior disc displacement with reduction.[16] The exercises described and illustrated in this chapter are based upon the prior work of Friedman and Weisberg,[17,18] Rocabado,[6,19] Kraus,[20] and Mannheimer,[21] who should all be recognized for their outstanding contribution to our current understanding of the role of physical therapy in dentistry and TMD. The exercises to follow in this chapter have been available to physical therapists and TMD patients for several years and are based upon an earlier publication by the author and his colleague, Lisa Morrone,[22] as well as a more recent publication by the latter author.[23] The feedback from across the country has been encouraging and, with only a few modifications, the exercises will be presented in their entirety at this time. With the exception of the tongue blade stretch, these exercises are geared toward the hypermobile TMJ, which represents the majority of TMDs seen clinically.

Balancing the Upper Half With RPTTLB

The concept of balance in the upper half (ie, cervical/thoracic spine, craniofacial region, shoulder girdle, and upper extremities) is crucial in achieving the optimum neuromusculoskeletal rest position for the joints and tissues in this region of the body. With regard to the mandible and the TMJ, the rest position is critical. The physiologic rest position of the mandible has traditionally been described as a postural relation of the mandible to the maxilla in which the mandibular condyles are in a neutral, unstrained position in the glenoid fossae and the mandibular musculature is in a state of minimum tonic contraction.[1-,3,6,7,20] Although this concept conveys the essence of the rest position, it is incomplete. Unless the influence of the head-neck region on mandibular position is appreciated, one can never discover the true rest or neutral position of the mandible and TMJs. Because of the neuromuscular and kinesiologic influence of head posture on the craniomandibular region, it is necessary to also place the head, neck, and back into physiologic rest or a neutral relationship at the same time.[3,4,6,7,19-21,24-27] The author instructs his students and patients to liken the TMJs to a car's transmission. There are 3 "gears" in this "transmission," in addition to neutral. In addition to achieving a physiologic rest position throughout the upper half, the use of the RPTTLB method (see list below) ensures that the mandible is placed in its neutral "idling" state. It is this position that affords the greatest opportunity for relaxation, pain relief, and recovery of function to the soft tissues of the TMJs. We will now proceed to describe each component of this method in greater detail.

- Relax: The first step in this process of balancing alignment and tension involves learning to completely relax. Patients are asked to perform a self-assessment of the muscle tension throughout the neck, shoulders, jaw and face, arms, legs, and trunk on an hourly basis. The key to "turning off" unnecessary muscle tension in the body is to understand the prin-

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Figure 16-2a. Phase 1 opening.

Figure 16-2b. Phase 2 opening.

Figure 16-2c. Phase 3 opening.

Figure 16-2d. Phase 4 opening.

previous phase and is again repeated 5 times and performed 5 times throughout the day. It, too, addresses the first gear but without the assistance of the finger-to-chin contact.

Phase 3 involves motion that includes both rotation and translation and therefore is directed toward the normalization of the second gear of the TMJ "transmission." Phase 3 begins, as does phase 1, with a rotation-only motion of the mandible. However, at the end of phase 1 opening, the patient is instructed to drop the tongue to the floor of the mouth and direct the lower jaw toward the throat. Thus, a combination of rotation/translation is introduced with the active-assisted guidance of the index finger (Figure 16-2c). Phase 2 is now replaced by phase 3 and is repeated 5 times and performed 5 times per day.

Phase 4 opening addresses the third and last gear by restoring terminal translation to the disc/condyle complex. It begins, as does phase 2, with the tongue on the hard palate in the RPTTLB state but proceeds from there with the patient continuing to open to the end of the active range with the tongue on the floor of the mouth (Figure 16-2d). The bilateral index contact on the TMJ ensures symmetrical joint motion with little if any joint sounds noted; the mirror continues to provide feedback in order to minimize deflections or deviations of the mandible. Phase 4 replaces phase 3 and is repeated 5 times and performed 5 times per day. Because phase 4 involves terminal translation, patients must not show signs of TMJ hypermobility when advanced to this final stage. The average patient takes 1 to 2 months of perfecting phases 1 to 3 before attempting phase 4. Consequently, the patient will often return to the clinic for a follow-up visit in order to advance to phase 4.

Having said that, patients are advised to resume phase 1 rotation immediately upon the first signs of an exacerbation of their TMJ symptoms (ie, phase 1 active-assisted rotation has a "reducing" effect on TMJ disc derangements as well as a "relaxing" effect on muscle splinting).

TMJ Neuromusculoskeletal Stabilization (Phases 1 to 3)

The application of isometric muscle training has become popular as a means of achieving optimal neuromuscular control in many regions of the body. In the craniomandibular region, gentle isometric contractions help to reduce

Figure 16-3a. Push to the left.

Figure 16-3b. Push to the right.

Figure 16-3c. Push upward.

Figure 16-3d. Push inward.

TMJ hypermobility as well as refine sensorimotor control of the mandible both statically and dynamically. In addition, neuromusculoskeletal stabilization training helps to alert the brain of potentially stressful postures and movements of the jaw so that the central nervous system can make the necessary adjustments to prevent injury and impairment from occurring. Although an increase in muscle strength may result from these exercises, their purpose is focused not on strength but on motor control.

As with the previous TMJ exercises, phase 1 stabilization is performed in the RPTTLB state in front of a mirror (Figures 16-3a to 16-3f). The patient is asked to apply a light pressure to the chin ("2" on a 0 to 10 scale), with his or her index finger in 6 different directions while maintaining the normal 3-mm freeway space between the upper and lower teeth. The mirror is used to ensure that the mandible remains stationary throughout the application of the gentle isometric force. The sequence is as follows:

1. Push to the left
2. Push to the right
3. Push upward
4. Push inward
5. Push diagonally in the direction of the opposite ear to the left
6. Repeat step 5 toward the right ear

Each gentle isometric force is held for 2 seconds, repeated 5 times, and performed 5 times through the day.

Phase 2 and 3 stabilizations are similar to phase 1 except for the amount of space between the teeth. In phase 2 (Figure 16-4), the patient is asked to open to 1 knuckle's

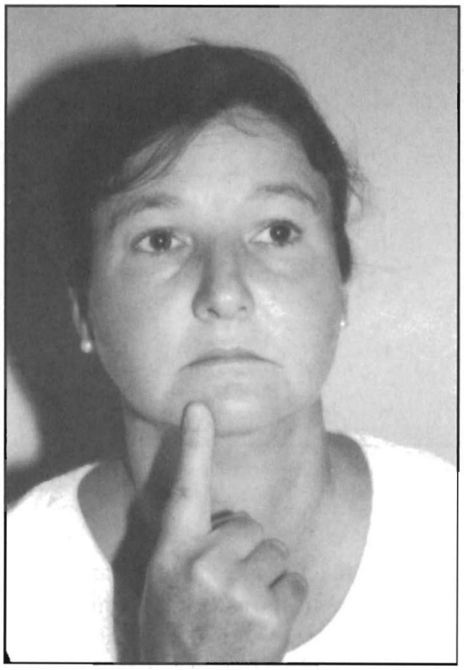

Figure 16-3e. Push to the left ear.

Figure 16-3f. Push to the right ear.

Figure 16-4. One-knuckle opening.

Figure 16-5. Two-knuckle opening.

width; in phase 3 (Figure 16-5) to 2 knuckles' width. Once the desired opening is achieved, the knuckles are removed and the stabilization exercises commenced. Unlike the TMJ rotation/translation control exercises in which the subsequent phase replaced the previous one, the subsequent phase of the stabilization program is added onto the previous one. This is because the benefit of isometric exercise occurs in the specific joint position in which it is performed. Consequently, patients are progressed through all 3 phases over a period of weeks or months, depending upon exercise mastery and the state of tissue healing.

In the management of TMJ hypermobility, it is recommended that the above exercises (ie, RPTTLB, TMJ rotation/translation control, and TMJ stabilization) be integrated with other therapies, including pain-relieving modalities, manual therapy of hypomobile spinal joints, TMJ occlusal splint therapy, stress management/biofeedback, etc.[3,9-11,13,23,34] Patient compliance, however, is crucial to the overall success of the program; for this reason, a patient's willingness to commit to the exercises must be assessed early in the process.

Tongue Blade Stretch

Until this point, all self-treatment has been geared toward the management of TMJ hypermobility. However, there is a small percentage of patients who present with true capsular impairment and thus require a home program of stretching and joint mobilization. These include postsurgical TMJ and orthognathic cases, as well as healed craniomandibular fractures, which may develop adhesions and joint contracture.

There are devices on the market such as the Therabite (Therabite Corp, Newtown Square, PA) and the Jaw Helper (MedDev Corp, Palo Alto, CA) that are used to assist patients with self-temporomandibular joint mobility. A more recent concept involves the use of continuous passive motion (CPM) to the TMJ with the E-Z Flex (Fluid Motion Biotechnologies Inc, New York, NY), which is especially useful postoperatively to prevent fibrous ankylosis.[35]

There is, however, a simple, cost-effective way (Figure 16-6) of having the patient perform self-temporomandibular joint mobilization/stretching that the author has successfully used with nonsurgical and postoperative patients alike. The patient is shown how to use tongue blades placed between the upper and lower molars as a means of achieving an effective mobilization/stretch of the TMJs and associated elevator muscles of the jaw. Because tissues that are preheated become more extensible, the patient should also be instructed in home heat application (dry or moist) prior to the self-stretch.[36] The appropriate number of tongue blades is determined by the degree of mandibular depression. Given that the anterior incisor-to-posterior molar opening is in a 3-to-1 ratio and that a tongue blade is 1 mm thick, the number of blades begins with the maximal interincisal opening (MIO) divided by 3. For example, a patient with an MIO of 30 mm should start with 10 tongue blades, which open the mouth 10 mm posteriorly and 30 mm anteriorly. The patient should maintain this position for 30 seconds and repeat this stretch 3 times every 2 hours. The therapist should progress the stretch by slowly adding tongue blades over time as determined by tissue reactivity, patient compliance, and the functional needs of the patient. When postoperative protocols are involved, there should be strict adherence to the guidelines; if questions arise, the surgeon should be consulted. In most cases, 40 mm of opening is functional; more than that puts the patient at risk for developing hypermobility or internal disc derangement.

Figure 16-6. Tongue blade stretch.

Section IV: Key Points

1. The majority of TMD patients benefit from posture correction.
2. The majority of TMD patients require a multidisciplinary approach to diagnosis and management. This consists of dentistry and physical therapy initially with possible referral to neurology for headaches and trigeminal neuralgia, rheumatology for fibromyalgia and other inflammatory conditions, oral and maxillofacial surgery for a surgical consult, and clinical psychology for stress management and cognitive-behavioral therapy.
3. TMJ surgery is required on rare occasions (eg, debilitating closed-lock).
4. The majority of TMD patients suffer with hypermobility and require stabilization, not mobilization.
5. Therapists interested in performing intraoral techniques (ie, myofascial, cranial, TMJ mobilization, and manipulative reduction of a closed-lock) should take continuing education courses and not depend exclusively on this text.

References

1. Okeson JR, ed. *Orofacial Pain: Guidelines for Assessment, Diagnosis, and Management.* Chicago, IL: Quintessence Publishing Co, Inc; 1996.
2. Neff PA. *TMJ Occlusion and Function.* Washington DC: Peter A. Neff; 1975, 1999.
3. Pertes RA, Gross SG, eds. *Clinical Management of Temporomandibular Disorders and Orofacial Pain.* Chicago, IL: Quintessence Publishing Co, Inc; 1995.
4. Makofsky HW, Sexton TR, Diamond DZ, Sexton MT. The effect of head posture on muscle contact position using the T-scan system of occlusal analysis. *J Craniomandib Pract.* 1991;9(4):316-321.
5. Darling DW, Kraus SL, Glasheen-Wray MB. Relationship of head posture and the rest position of the mandible. *J Prosthet Dent.* 1984;52:111-115.
6. Rocabado M, Iglarsh ZA. *Musculoskeletal Approach to Maxillofacial Pain.* New York, NY: JB Lippincott; 1991.
7. Kraus SL. Influences of the cervical spine on the stomatognathic system. In: Donatelli R, Wooden MJ, eds. *Orthopaedic Physical Therapy.* 3rd ed. New York, NY: Churchill Livingstone; 2001.
8. Simons DG, Travell JG, Simons LS. *Travell & Simons' Myofascial Pain and Dysfunction: The Trigger Point Manual. Vol. 1. Upper Half of Body.* Baltimore, MD: Williams & Wilkins; 1999.
9. Medlicott MS, Harris SR. A systematic review of the effectiveness of exercise, manual therapy, electrotherapy, relaxation training, and biofeedback in the management of temporomandibular disorder. *Phys Ther.* 2006;86(7):955-973.
10. McNeely ML, Olivo SA, Magee DJ. A systematic review of the effectiveness of physical therapy interventions for temporomandibular disorders. *Phys Ther.* 2006;86(5):710-725.
11. Furto ES, Cleland J, Whitman JM, Olson KA. Manual physical therapy interventions and exercise for patients with temporomandibular disorders. *J Craniomandib Pract.* 2006;24(4):283-291.
12. Glaros AG, Burton E. Parafunctional clenching, pain, and effort in temporomandibular disorders. *J Behavioral Med.* 2004;27(1):91-99.
13. Shankland WE. Migraine and tension-type headache reduction through pericranial muscular suppression: a preliminary report. *J Craniomandib Pract.* 2001;19(4):269-278.
14. Oleson TD. *Auriculotherapy Manual: Chinese and Western Systems of Ear Acupuncture.* Los Angeles, CA: Health Care Alternatives; 1992.
15. Weisberg J, Friedman MH. Displaced disc preventing mandibular condyle translation: mobilization technique. *J Orthop Sports Phys Ther.* 1981;3(2):62-66.
16. Yoda T, Sakamoto I, Imai H, et al. A randomized controlled trial of therapeutic exercise for clicking due to disk anterior displacement with reduction in the temporomandibular joint. *J Cranio Pract.* 2003;21(1):10-16.
17. Friedman MH, Weisberg J. The craniocervical connection: a retrospective analysis of 300 whiplash patients with cervical and temporomandibular disorders. *J Craniomandib Pract.* 2000;18(3):163-167.
18. Friedman MH, Weisberg J. *Temporomandibular Joint Disorders: Diagnosis and Treatment.* Chicago, IL: Quintessence Publishing Co, Inc; 1985.
19. Rocabado M. Diagnosis and treatment of abnormal craniocervical and craniomandibular mechanics. In: Solberg WK, Clark GT (eds). *Abnormal Jaw Mechanics, Diagnosis and Treatment.* Chicago, IL: Quintessence Publishing; 1984.
20. Kraus SL, ed. *TMJ Disorders: Management of the Craniomandibular Complex.* New York, NY: Churchill Livingstone; 1989.
21. Mannheimer JS. Overview of physical therapy modalities and procedures. In: Pertes RA, Gross SG, eds. *Clinical Management of Temporomandibular Disorders and Orofacial Pain.* Chicago, IL: Quintessence Publishing Co, Inc; 1995.
22. Morrone L, Makofsky HW. The TMJ home exercise program. *Clin Manag.* 1991;11(2):20-26.
23. Morrone L. *Overcoming Headaches and Migraines: Clinically Proven Cure for Chronic Pain.* Eugene, OR: Harvest House Publishers; 2008.
24. Makofsky HW. The effect of head posture on muscle contact position: the sliding cranium theory. *J Craniomandib Pract.* 1989;7(4):286-292.
25. Darnell MW. A proposed chronology of events for forward head posture. *J Craniomandib Pract.* 1983;1(4):50-54.
26. Rocabado M, Johnson BE, Blakney MG. Physical therapy and dentistry: an overview. *J Craniomandib Pract.* 1983;1:47-49.

27. Mannheimer JS, Rosenthal RM. Acute and chronic postural abnormalities as related to craniofacial pain and temporomandibular disorders. *Dent Clin North Amer.* 1991;35(1):185-208.
28. Mohl ND. The role of head posture in mandibular function. In: Solberg WK, Clark GT, eds. *Abnormal Jaw Mechanics: Diagnosis and Treatment.* Chicago, IL: Quintessence Publishing Co, Inc; 1984.
29. Conable B, Conable W. *How to Learn the Alexander Technique: A Manual for Students.* 2nd ed. Columbus, OH: Andover Road Press; 1992.
30. Brennan R. *The Alexander Technique Workbook: Your Personal System for Health, Poise, and Fitness.* London: Element Books Ltd; 2002.
31. Caplan D. *Back Trouble: A New Approach to Prevention and Recovery.* Gainesville, FL: Triad Publishing; 1987.
32. Jankelson RR. *Posture, Airway & the Tongue in Clinical Dentistry.* Kent, WA: Myotronics, Inc.; 2006.
33. Makofsky HW. Snoring and obstructive sleep apnea: does head posture play a role? *J Craniomandib Pract.* 1997;15(1):68-73.
34. Makofsky HW, August BF, Ellis JJ. A multidisciplinary approach to the evaluation and treatment of temporomandibular joint and cervical spine dysfunction. *J Craniomandib Pract.* 1989;7(3):205-213.
35. Israel HA, Syrop SB. The important role of motion in the rehabilitation of patients with mandibular hypomobility: a review of the literature. *J Craniomandib Pract.* 1997;15(1):74-83.
36. Poindexter RH, Wright EF, Murchison DF. Comparison of moist heat and dry heat penetration through orofacial tissues. *J Craniomandib Pract.* 2002;20(1):28-33.

Section

V

LUMBAR SPINE

17

Examination and Evaluation of the Lumbar Spine

According to the National Institutes of Health, back pain is one of the most common medical problems, affecting 8 out of 10 people at some point in their lives.[1] In addition, the direct costs of low back pain (LBP) in the United States are reportedly between $33 and $55 billion per year.[2] Furthermore, individuals with LBP experience health expenditures that are 60% greater than those without LBP, 37% of which are a direct result of physical therapy and allied specialist services.[3] According to Macnab,[4] most backache is spondylogenic (ie, it arises from an abnormality of the vertebrae or the spinal soft tissues). Mechanical disorders form the majority of these cases, but occasionally the underlying problem is less obvious. Prior to assuming that the patient's LBP is of a spondylogenic-biomechanical nature, it is important that the manual therapist takes the necessary time to rule out pathological and nonmechanical causes such as multiple myeloma, osteomyelitis, and viscerogenic backache to mention a few (Figure 17-1). Once this has been done, the challenge is to then classify the patient's biomechanical LBP in such a way that it directs treatment in an evidence-based manner. To this end, multiple diagnostic classification systems have been developed to guide clinicians in the management of LBP.[5-7] The pathoanatomic classification system is dependent on structural diagnosis and is largely based upon radiological signs and differential diagnostic injection blocks. Examples include spondylolisthesis, stenosis, disc herniations, and nerve root compressions.[8] The mechanism-based classification system is built on the premise that somatic impairments identified during the examination are the cause of the patient's LBP. Proper treatment of these impairments will result in the relief of painful symptoms and the restoration of function. The McKenzie classification system[9] includes the postural, dysfunction, and derangement syndromes (discussed in Chapter 2), and is based on pain patterns and pain behavior. In this classification system, treatment is syndrome specific (eg, posture correction for postural syndrome and specific repeated exercises and/or manual therapy for dysfunction and derangement syndromes). The multi-dimensional classification system takes a combination approach according to the stage of the disorder, the pathoanatomical diagnosis, signs and symptoms, and psychosocial factors. Lastly, we come to the treatment-based classification system proposed by Delitto et al.[10] This classification system relies on history, behavior of symptoms, and clinical signs in order to assign patients with nonspecific LBP into 1 of 4 subgroups, based upon the greatest likelihood of clinical success, and includes stabilization, mobilization/manipulation, specific exercises, and traction. The positive features of this system are that it is eclectic in nature, is clinically applicable, and provides a straightforward method of guiding treatment that seeks to be evidence based.[6,7,10,11]

Determining the chronicity of a patient's LBP is an important consideration for management and is critical in the classification process.[11] Whereas the International Association for the Study of Pain uses a temporal basis for defining and distinguishing acute and chronic pain—accepting 3 months as the division between the two[12]—the Quebec Task Force on Spinal Disorders takes a different approach.[13] Based on the distribution of claims of spinal disorders by duration of absence from work, stages of a patient's LBP were determined as follows: acute (less than 7 days), subacute (7 days to 7 weeks), and chronic (more than 7 weeks).

Makofsky HW
Spinal Manual Therapy, 2nd ed. (pp 141-152)

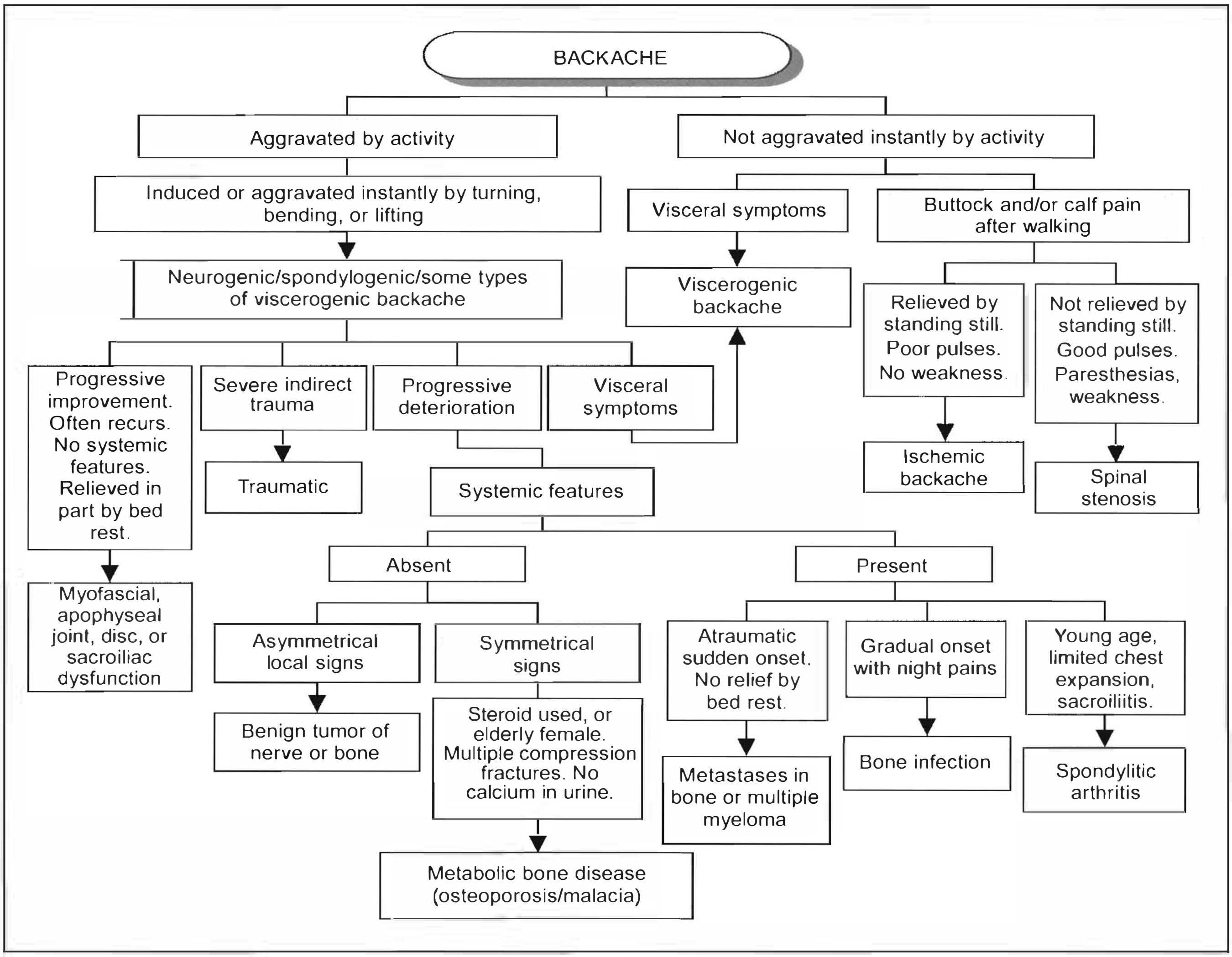

Figure 17-1. Differential diagnosis of LBP. (Reprinted with permission of GC Willis, Igaku-Shoin Ltd, Tokyo, 2008.)

Physical Examination

As alluded to in the beginning of Chapter 8, the divisions according to anatomic region in this textbook are necessary for instructional purposes. However, in the actual clinical application of the examination and intervention techniques, there is less separation and more integration. For example, the assessment of postural alignment of the entire lumbar-pelvic-hip complex can be approached as one functional unit and examined concurrently. Why then the didactic separation as per the anatomy? The author realizes that the trend in physical therapy education is toward integration, and there is no argument on that point. There is, however, disagreement as to when that integration process should begin. It is the author's philosophy that students, not unlike developing children, must "crawl before walking." Once the basic principles of examination, evaluation, and intervention are learned for each region of the body, the student is then taken to the next step of integration. The venue for this progression can occur in the classroom with patient demonstrations, case studies, etc, as well as at the clinical site with patient rounds (as is performed in the clinical education of medical students and residents).

Posture

An important aspect of postural alignment is an understanding of clinical stability. Joints move through a physiologic ROM consisting of the neutral zone, which is characterized by high flexibility, and the elastic zone, which is the region of high stiffness at the end of range. Clinical spinal instability[14-16] is the loss of the spine's ability to maintain its normal patterns of displacement under physiologic loads such that there is no incapacitating pain, major deformity, nor neurologic deficit (components of the stabilization system will be covered in further detail in Chapter 20). The assessment of lumbar spine posture provides evidence for either stability or instability. If it is determined that the lumbar joints are not positioned in their neutral lordotic

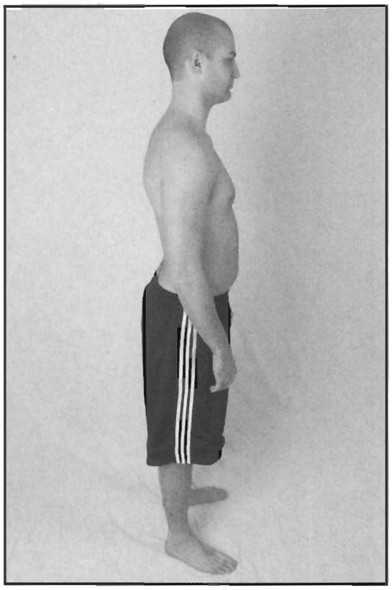

Figure 17-2. Lumbar spine lateral view.

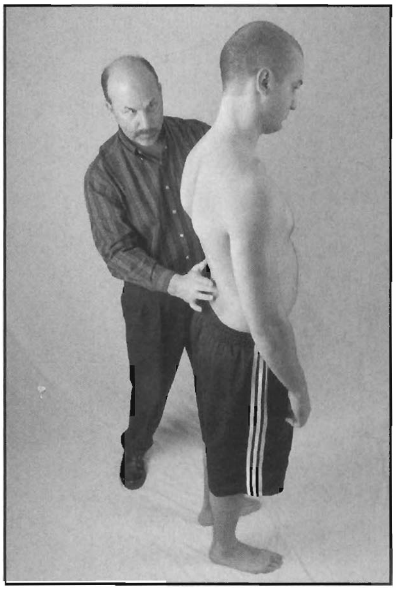

Figure 17-3. Palpating for a lumbar shelf.

curve but rather toward the end of range in the elastic zone (ie, border position), then there is reason to suspect that clinical instability is either present or developing. In severe cases of instability, the passive restraints (ie, osteoligamentous structures) may be deficient, resulting in joint laxity at the end of range. Although poor posture may suggest the possibility of clinical instability, the final determination is always based upon motion testing, which will be covered subsequently.

As with previous assessments of posture, the standing patient is viewed laterally, posteriorly, and anteriorly. The lateral view of the lumbar spine (Figure 17-2) includes an evaluation of the following structures for misalignment:

- The lumbar curve between the thoracolumbar and lumbosacral junctions should demonstrate a smooth posterior concavity (ie, lordosis) from top to bottom. As with all spinal segments, the balanced state is represented by a "tripod" consisting of the vertebral body/intervertebral disc in the front and the 2 apophyseal joints in the back. Unlike the lower cervical spine, where all 3 components bear equal weight, the typical lumbar segment bears approximately 85% of the weight anteriorly and 15% posteriorly. In states of increased lumbar extension (ie, hyperlordosis) the tripod shifts posteriorly onto the facets. When there is a decreased lordosis (ie, flat back), the tripod shifts onto the vertebral body/disc complex. Over time the hyperlordotic spine may accelerate degenerative changes in the posterior facet joints, whereas the flattened lumbar curve is often associated with discogenic conditions. If poor postural alignment persists into adulthood, impairment of mobility usually occurs with the flattened spine becoming restricted in extension and the hyperlordotic spine becoming limited in flexion. The swayback posture is sometimes mistaken for hyperlordosis but is actually quite different. Its components include forward displacement of the hips, posterior rotation of the pelvis, a flat lumbar spine, and an increased thoracic kyphosis. Relative to the concept of the lumbar-pelvic-hip complex, it is important that the clinician sees the connection between standing lumbar hyperlordosis, anterior pelvic tilt, and hip flexion; lumbar spine hypolordosis, PPT, and hip extension; swayback, PPT, and hip extension; and lumbar scoliosis, lateral pelvic tilt, and hip abduction/adduction (eg, lumbar spine convexity on the right, lateral pelvic tilt, lower on the right, and right hip abduction). Postural deviations can be quantified with an inclinometer or flexible ruler or be described as minimal, moderate, and severe. Prsala et al[17,18] demonstrated that men with LBP had a statistically greater average kyphotic-lordotic length ratio versus pain-free men (ie, men with chronic LBP had a longer lumbar lordotic curve as measured with a 24-inch long "French flexible curve"). What makes this study interesting is that the authors draw attention to the length of the lumbar curve, whereas most studies emphasize the depth of the lordosis as a predictor of impairment.
- The therapist should examine each lumbar SP for a palpable "shelf" (Figure 17-3) consistent with spondylolisthesis.[19] These shelves or "steps" are most common in the lower lumbar region and tend to become prominent in the standing position. Paris[20] suggests that a palpable "step" in stance that normalizes in prone is less stable than a step that is palpated in both standing and prone; it is the unstable spondylolisthesis that is "the most likely to progress." According to

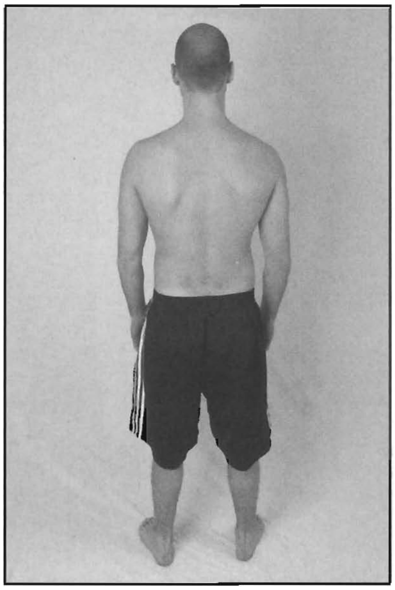

Figure 17-4. Lumbar spine posterior view.

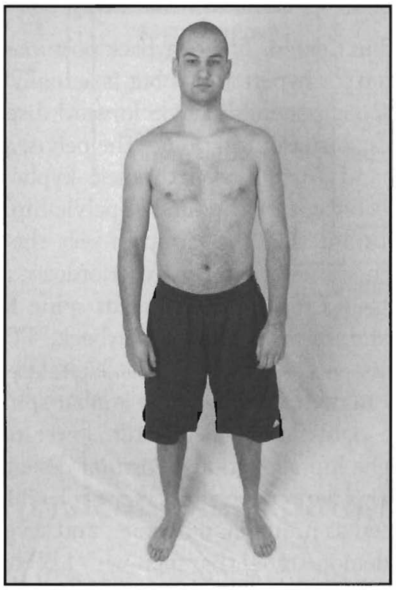

Figure 17-6. Lumbar spine anterior view.

Macnab,[4] the presence of a spondylolisthesis is more likely to produce low back symptoms in younger patients; rarely, if ever, is it the sole cause of pain in those over age 40.

- The abdominal region should be observed for excessive protrusion, which is often associated with a hyperlordotic posture (this appears to be the case with pregnant women).

The patient is now observed posteriorly (Figure 17-4). Common misalignments and abnormalities include the following:

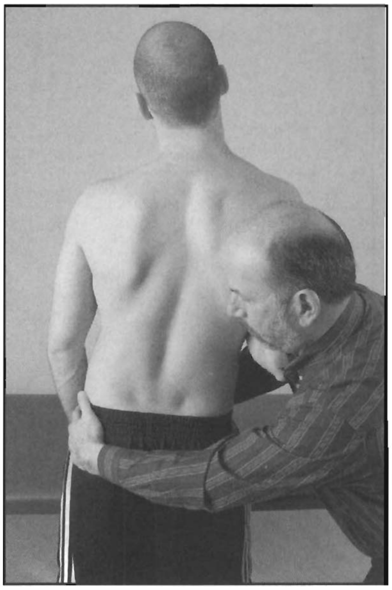

Figure 17-5. Right lateral shift.

- Asymmetrical fullness of the paravertebral muscles, which suggests spinal rotation toward the prominent side consistent with a neutral type 1 rotoscoliosis (see Chapter 1).
- Segmental hypertonicity: A hypertrophied band of musculature at one level, bilaterally or unilaterally, suggests clinical spinal instability at that level.
- Lateral trunk shift (named for the direction the shoulders move and not the hips). Some authors refer to this antalgic posture of the lumbar spine as acute sciatic or protective scoliosis (Figure 17-5). Regardless of the term used, most patients shift away from the side of the pain. When it occurs over a period of minutes to hours, it is highly suggestive of a lumbar derangement, usually at either L4,5 or L5,S1. More often than not, the lateral trunk shift is associated with an acute lumbar kyphosis. As discussed in Chapter 2, McKenzie's derangement syndrome[9] has traditionally been attributed to disc displacement. However, other possibilities include facet joint impingement, nonarticular reflex-induced muscle splinting (ie, hypertonicity from either nociceptive or hyperactive muscle spindle responses), pelvic girdle impairment (ie, sacral torsions), etc.
- Asymmetrical waist angles.
- Thoracolumbar scoliosis.
- Tell-tale skin signs of benign nerve root tumors including a port wine hemangioma, café au lait spots, a tuft of hair in an unusual place, or neurofibromata (typically there are neurological manifestations restricted to a single root level).

The patient is finally observed anteriorly (Figure 17-6). Asymmetries to note include the following:

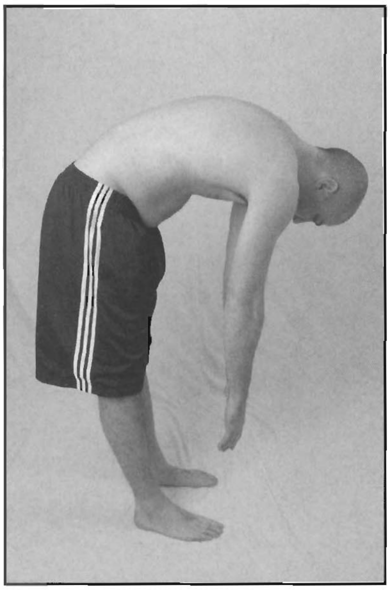

Figure 17-7a. Active lumbar forward bending.

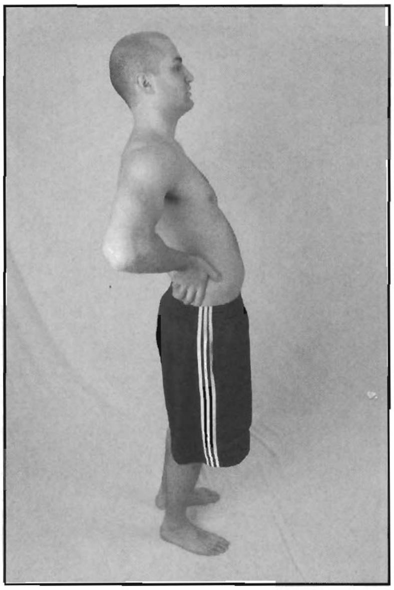

Figure 17-7b. Active lumbar backward bending.

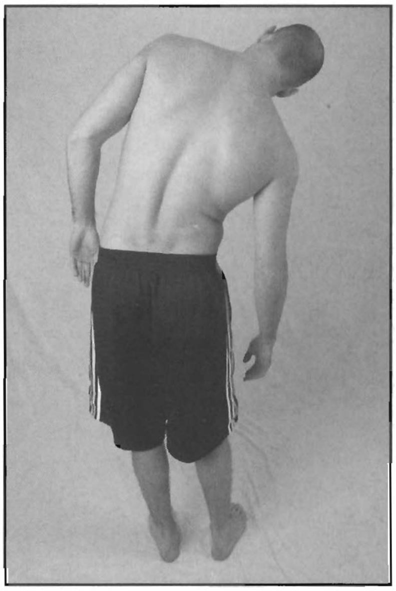

Figure 17-7c. Active lumbar side bending right.

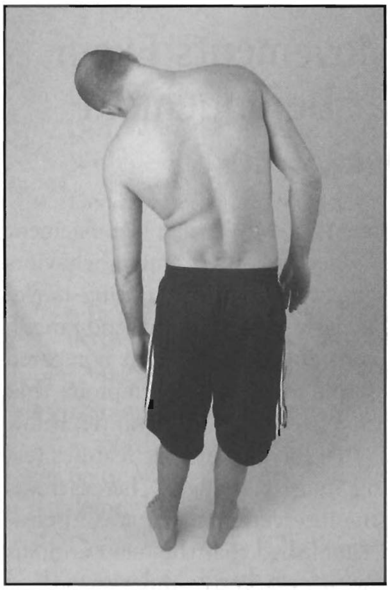

Figure 17-7d. Active lumbar side bending left.

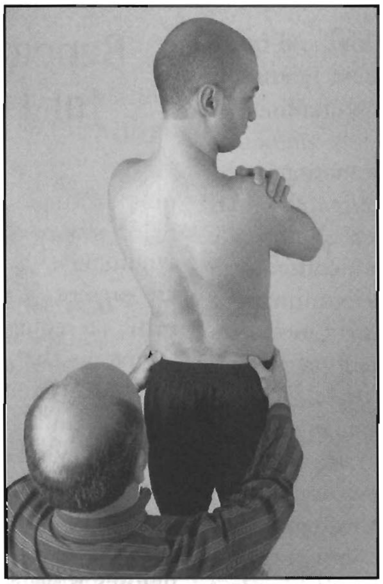

Figure 17-7e. Active lumbar rotation right.

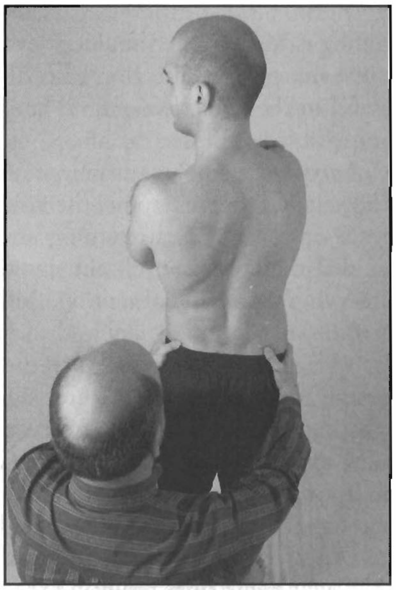

Figure 17-7f. Active lumbar rotation left.

- Abdominal scars, which through the superficial fascia, exert asymmetric stress patterns with resultant skeletal misalignment.
- Deviation of the linea alba, suggestive of a rotoscoliosis posteriorly.
- An anterior perspective of a lateral trunk shift. In addition to observing the shift from the anterior aspect, this view also allows observation of the patient's face for signs of distress.

Active Movements

The examination of active lumbar movements consists of an analysis of the same 6 spinal motions observed in the scapulothoracic region (ie, forward bending, backward bending, side bending right and left, and rotation right and left) with the addition of side gliding to the right and left, which is named by the direction of the shoulder motion and not the hips (Figures 17-7a to 17-7h). This translational movement of the trunk may necessitate an explanation as well as a few tactile cues. The key to proper trunk side

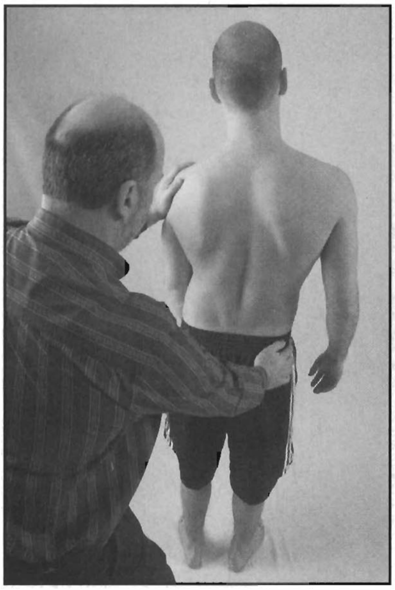

Figure 17-7g. Assessing side glide right.

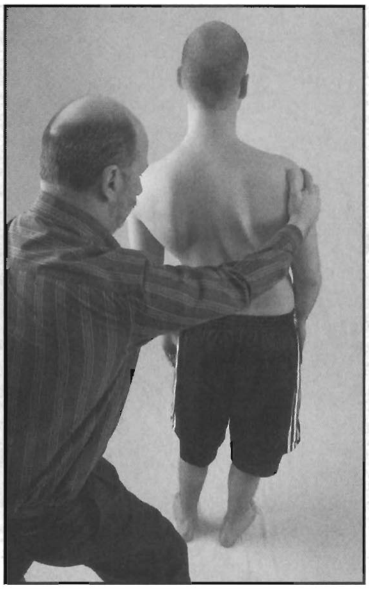

Figure 17-7h. Assessing side glide left.

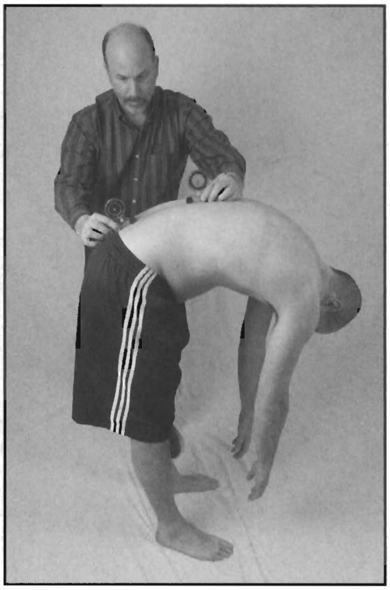

Figure 17-8. Measuring lumbar flexion with 2 inclinometers.

gliding is to keep the shoulders level as the hips and trunk move in opposite directions. Impairment of side gliding is useful in the detection of mild lateral trunk shifts that are not easily identified.

As with the previous examination of active motion in the cervical and thoracic spine, the visual estimation method is again employed. With training and experience, manually trained therapists can obtain significant information from observing active spinal motion. Although determining the quantity of motion available through the use of inclinometers (Figure 17-8), a tape measure, range of motion devices, computerized technologies, etc is certainly useful, it is no substitute for the skillful observation of human motion. How else can it be determined that a standing patient's ability to place both hands on the floor is accomplished through hamstring flexibility, while the lumbar lordosis fails to reverse its curvature due to stiffness or conversely that tight hamstrings result in excessive lumbar flexion as a means of compensation. Instructors of manual therapy need to continue to teach this and other valuable assessment techniques even though they may lean more toward the art than the science of physical therapy.

Concepts worth remembering when observing spinal motion include balance, poise, coordination, distal mobility on proximal stability, segmental recruitment, roll-gliding, hypomobility (flat regions associated with a lack of curvature), hypermobility (pivot points or fulcrums), muscle splinting, tissue reactivity, functionality, etc. According to Paris,[20] sharp angulation at one or more levels during active movement suggests hypermobility, whereas a "shaking" motion, especially in forward bending, indicates instability (eg, spondylolisthesis). The importance of this aspect of the spinal examination cannot be emphasized enough!

Repeated Movements Exam for Lumbar Derangement (Phases 1 to 3)

As discussed in Chapter 8, McKenzie's derangement syndrome[9,21] is suspected when certain symptom behaviors are present. They are symptoms occurring during movement, as compared to a dysfunction (ie, at end-range); symptoms that may be constant and severe, as compared to being intermittent and mild to moderate; symptoms that start proximal but with time become more distal (ie, below the knee); and symptoms that have neurologic features (eg, burning, tingling, shooting, sharp, piercing). Whereas it was previously believed that the intervertebral disc was insensitive to pain, it has been established that the outer annulus fibrosus is well supplied with nociceptive innervation[22,23] and is a common source of backache.[24] The majority of lumbar spine disc derangements occur at the L4,5 and L5,S1 levels and are most prevalent between the ages of 25 and 50, affecting more men than women.[4,25]

In addition, the presence of an acute deformity (ie, lumbar kyphosis with or without a lateral trunk shift) is highly suggestive of a derangement. When patients use the phrase, "My back is out," think derangement!

When a McKenzie lumbar derangement is suspected, the therapist should proceed to placing the patient in 1 of 7 categories (although McKenzie's current approach no longer utilizes these categories, this author finds them useful and will therefore continue to refer to them). They are as follows:

- Derangement 1: Central or symmetrical pain across L4,5. Rarely buttock or thigh pain. No deformity.

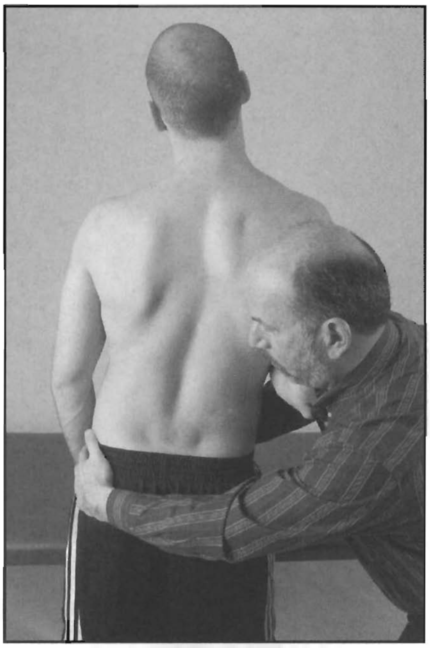

Figure 17-9. Right lateral shift correction.

- ➤ Derangement 2: Central or symmetrical pain across L4,5. With or without buttock and/or thigh pain. With deformity of lumbar kyphosis.
- ➤ Derangement 3: Unilateral or asymmetrical pain across L4,5. With or without buttock and/or thigh pain. No deformity.
- ➤ Derangement 4: Unilateral or asymmetrical pain across L4,5. With or without buttock and/or thigh pain. With deformity of lateral trunk shift.
- ➤ Derangement 5: Unilateral or asymmetrical pain across L4,5. With or without buttock and/or thigh pain. With leg pain extending below the knee. No deformity.
- ➤ Derangement 6: Unilateral or asymmetrical pain across L4,5. With or without buttock and/or thigh pain. With leg pain extending below the knee. With deformity of lateral trunk shift.
- ➤ Derangement 7: Symmetrical or asymmetrical pain across L4,5. With or without buttock and/or thigh pain. With deformity of accentuated lumbar lordosis.

McKenzie postulates that derangements 1 to 6 are progressions of the same disturbance within the intervertebral disc. The principal aim of treatment is to centralize pain and reduce deformity in order to reverse all derangements to derangement 1. Derangements 1 to 6 are generally made worse by sitting and lumbar flexion and improved by standing and walking, which tend to restore the lumbar lordosis to normal. Although the McKenzie approach to the management of derangements is explained quite well with a nuclear displacement model,[9,26-28] there are other researchers[29-31] who challenge the notion of nuclear "repositioning." Perhaps one day our understanding of the true anatomic basis of lumbar derangement will become clearer. In the meantime, our focus as clinicians should be on the bedrock principles of intervention, which include reduction of the derangement based on signs and symptoms; stabilization of the reduction; recovery of function, and prevention of recurrence. As emphasized so often to students, "Treat the patient's signs and symptoms and not the diagnosis." If at any time the derangement patient presents with frank neurologic signs (eg, muscle atrophy, weakness, hyporeflexia, and sensory loss), the patient is not a candidate for mechanical therapy and should be referred to his or her physician for consultation. Furthermore, if at any time the patient reports a loss of bowel or bladder control (eg, urinary retention), the patient requires an immediate referral to a spine surgeon.

The next step in the evaluation process is to determine the patient's response to the repeated movements examination using extension and flexion. However, for patients presenting with a lateral trunk shift (ie, derangements 4 and 6), the lateral shift correction (Figure 17-9) should be performed prior to the initiation of these test movements. Depending on the severity of the shift and the associated symptoms, the technique should be performed slowly and gently, avoiding the excessive use of force. The patient's hips are rhythmically pulled under the trunk in an "on/off" fashion. Because the patient's legs may "give way," the patient should be positioned in front of a treatment table for support if needed. In addition to the improvement in trunk alignment, the patient's symptoms should be monitored throughout the lateral shift correction. As with cervical derangements, this is done by identifying the distal-most symptom and giving it a number from 0 to 10, with 0 being the absence of discomfort and 10 being the worst pain the patient has ever experienced. As the deformity improves, the symptoms should centralize in toward the center of the low back and eventually diminish in intensity.

For derangements 1 through 6, the patient will be examined with an extension regimen; for derangement 7, repeated lumbar flexion is recommended. The main advantage of McKenzie over other manually oriented systems, in the author's opinion, is the use of repeated movements. Derangements cannot be "forced into submission" but require a coaxing or "milking" force that often takes between 20 to 100 repetitions to respond. Although the reduction of a derangement is more of an art than a science, there are guidelines that may be helpful. The author uses a 3-phase approach to guide the mechanical reduction of derangements 1 through 6. The patient begins with phase 1 and is only progressed to phases 2 and 3 if necessary. Because derangement 7 is so rare, it will not be covered in this text.

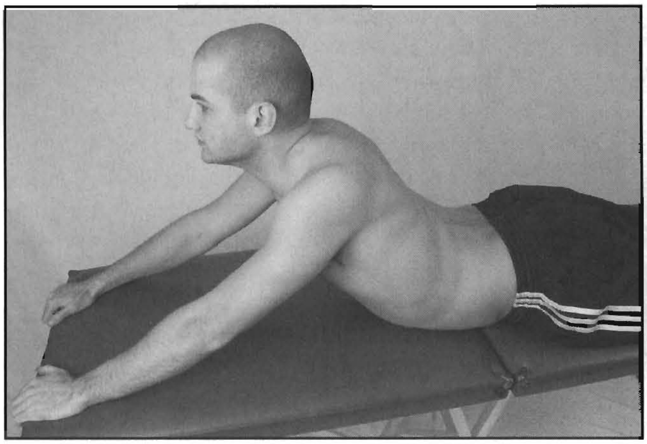

Figure 17-10. Phase 1 self-exam prone press-up.

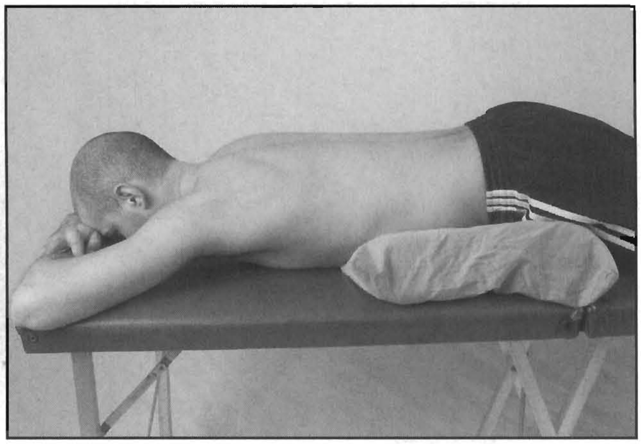

Figure 17-11. Management of derangement 2 with one or more pillows.

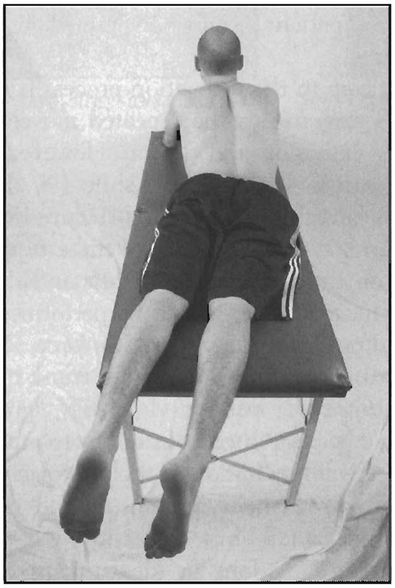

Figure 17-12. Phase 2 self-exam shifted-hips right press-up.

Phase 1: Self-Examination Prone Press-Ups

The patient must be instructed in the proper execution of the press-up (Figure 17-10). Simple instructions include the following:

1. Place the hands out in front far enough to permit the elbows to attain full extension without causing strain to the lower back upon pressing up.
2. The back muscles should not be working when performing the press-up. The work is done solely through the arms.
3. The hips are not permitted to leave the table (this ensures lumbar rather that hip extension).
4. When the arms are fully extended, there should be a slight pause before returning to the start position.
5. After each set of 10 repetitions, the distal-most symptom should be reassessed regarding its position and intensity. If there is no change or centralization has occurred, the patient should continue with phase 1. If peripheralization has occurred or the pain becomes intolerable, the prone press-ups should be stopped.
6. When an increase in the extension effort is indicated by a good response, this can be achieved by bringing the hands closer to the shoulders prior to pressing upward. The goal is still to achieve full extension of the elbows so that the back muscles remain relaxed. Once the elbows are fully extended, lumbar extension can be further enhanced by having the patient fully exhale through pursed lips after taking a deep breath through the nose. This progression continues until full reduction of the derangement has been attained.

The phase 1 self-exam is the basis for the intervention when the desired response is achieved. It is the starting point for derangements 1, 3, and 5. For derangements 4 and 6, the lateral trunk shift must be corrected prior to initiating phase 1 as mentioned above. Regarding derangement 2, the patient may require a prone-lying progression commencing with 1 or 2 pillows placed under the abdomen to accommodate the acute lumbar kyphosis (Figure 17-11). After a few minutes, the pillow should be withdrawn and the patient should remain in the prone-lying position for another few minutes, or longer if necessary, before commencing the phase 1 prone press-up.

Phase 2: Self-Examination Shifted-Hips Press-Ups

Patients are begun on shifted-hips press-ups when an additional lateral force is needed to reduce the derangement. Many posterolateral disc derangements respond well to straight press-ups, but others require a "wedge effect" in order to obtain complete reduction. The patient is instructed to shift his or her hips away from the side of pain and press-up from this position (Figure 17-12). Again, the patient's distal symptom and its intensity are reassessed, following 10 repetitions, for signs of centralization. As long

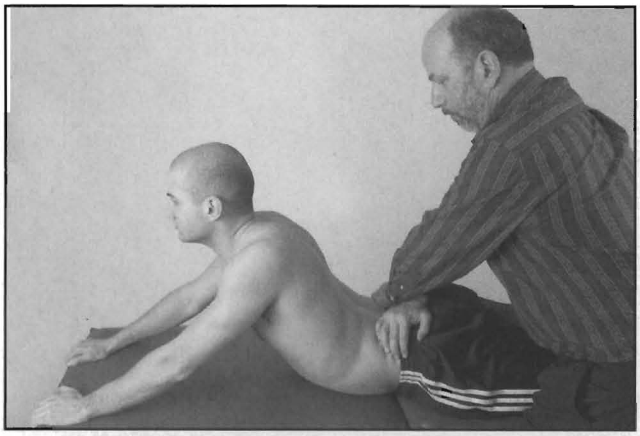

Figure 17-13a. Phase 3 press-up with manual stabilization of inferior segment.

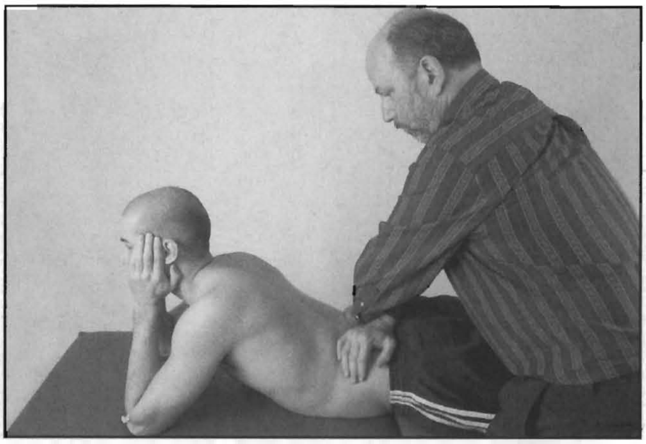

Figure 17-13b. Phase 3 Manual mobilization for reduction of a lumbar derangement.

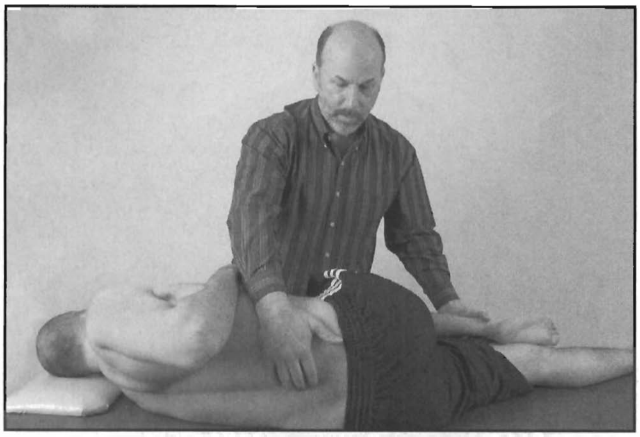

Figure 17-14a. Assessment of L1-L5 forward bending.

as the desired response is obtained, the patient continues with the shifted press-ups in sets of 10. If at any time the patient's symptoms peripheralize, the exercises should be stopped. When the symptoms have settled in the center of the patient's low back, phase 1 press-ups may be resumed in hopes of obtaining a full reduction of the derangement.

Phase 3: Therapist-Assisted Reduction

One of the main strengths of the McKenzie approach is the emphasis on self-treatment. However, there comes a point when therapist-assisted intervention is necessary. If the derangement persists following the application of phase 1 and 2 attempts at reduction, the patient is progressed to phase 3. Those who have poor success with the extension principle usually do so not because extension is inappropriate, but because the amount of extension at the deranged segment is inadequate. If the patient is unable to "close down" with sufficient force independently, it is up to the therapist to ensure that the necessary extension takes place. When the extension force at the deranged segment is increased, the previously recalcitrant derangement often responds favorably.

Phase 3 intervention includes the following measures:

1. Manual stabilization of the inferior component (Figure 17-13a) of the involved segment—ie, sacrum for L5,S1 derangement, over the transverse processes (TP) of L5 for an L4,5 derangement.
2. PA mobilizations over the TP of the lumbar vertebrae (unilaterally or bilaterally) and sacral base in neutral, prone on elbows (Figure 17-13b), and in the prone press-up position.
3. Manual stabilization of the hips and pelvis away from the side of pain during the performance of the prone press-up (not illustrated).

Phase 3 intervention is the therapist's final attempt at coaxing a stubborn derangement "back into place" before determining that it is nonreducible. It is a manual skill that can be improved with practice.

Passive Physiologic Intervertebral Movements

As discussed in Chapter 4, PPIVMs[32] are a means of evaluating physiologic motion in the spine, segment by segment, as it occurs during active movement with the exception of muscle contraction. As in the thoracic spine, the quality, quantity, and end-feel for each motion segment is assessed. Gonnella et al[33] demonstrated dependable intratherapist reliability using the 0 to 6 mobility scale, whereas intertherapist reliability was not dependable. The motions of forward and backward bending, side bending right and left, and rotation right and left will be assessed as the palpating finger (usually the index or middle finger) examines motion in the interspinous space from L1 to L5. Although these motions can be induced and assessed in both weightbearing and nonweightbearing positions, the recumbent position will be utilized for the basic, introductory approach (Figures 17-14a to 17-14f).

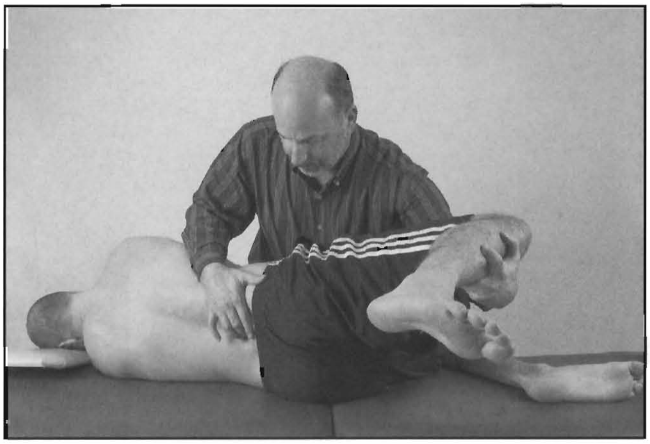

Figure 17-14b. Assessment of L1-L5 backward bending.

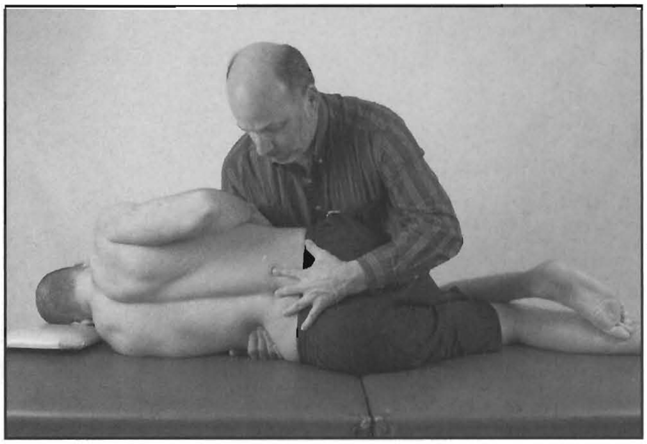

Figure 17-14c. Alternative assessment of backward bending.

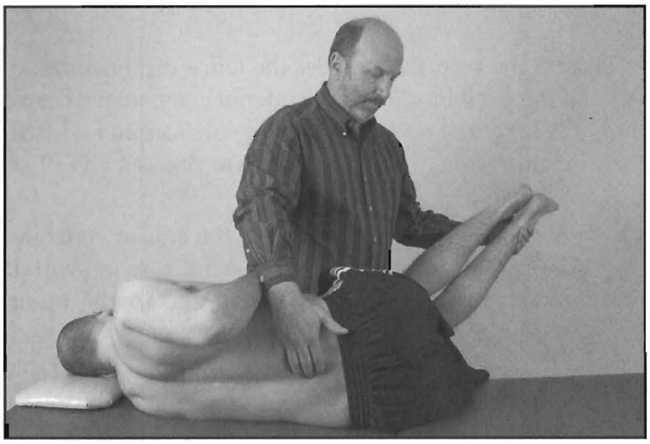

Figure 17-14d. Assessment of L1-L5 side bending right.

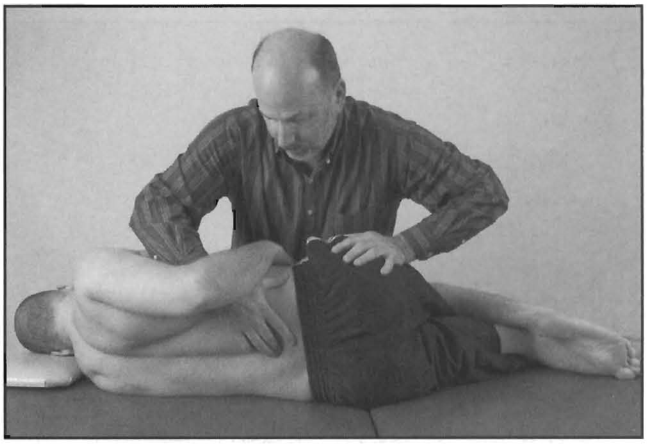

Figure 17-14e. Alternative assessment L1-L5 side bending right.

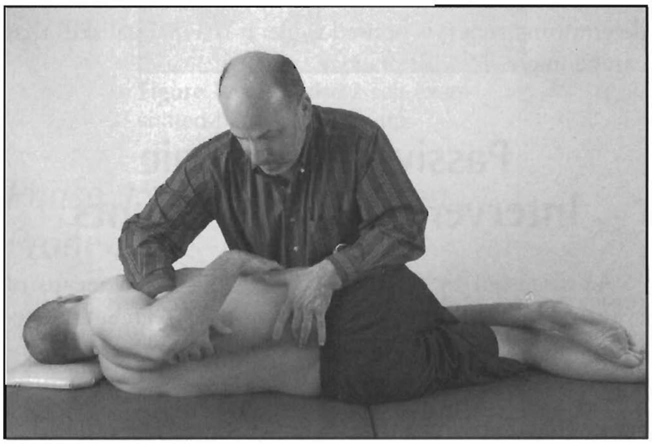

Figure 17-14f. Assessment of L1-L5 rotation right.

For forward bending, the palpating finger assesses the separation of the SPs starting at L5,S1, whereas for backward bending, the approximation of the SPs is examined. When assessing side bending, the ipsilateral aspect of the interspinous space is palpated. However, for rotation the contralateral aspect of the interspinous space is preferable. To determine segmental levels in the lumbar spine, the L4,5 interspinous space is usually at the level of the iliac crest. However, it is better to identify the last mobile segment (L5,S1) in extension and work up from there.

Soft Tissue Palpation

As with other regions of the musculoskeletal system, the examiner is seeking to identify areas of tenderness (myofascial trigger points and/or tender points), tightness, and increased tone. The important structures of the lumbar region amenable to palpation include the following:

- Abdominal muscles: Consisting of the rectus abdominis, external and internal obliques, and transversus abdominis.
- Psoas muscle: Palpated anteriorly, approximately 2 inches lateral to the umbilicus at the lateral border of the rectus abdominis.
- Skin and superficial fascia: Note temperature changes, erythema, moist or dry areas, edema, scar tissue/adherences, skin lesions, nodules, trigger points, etc. A tuft of hair ("faun's beard") may indicate a spina

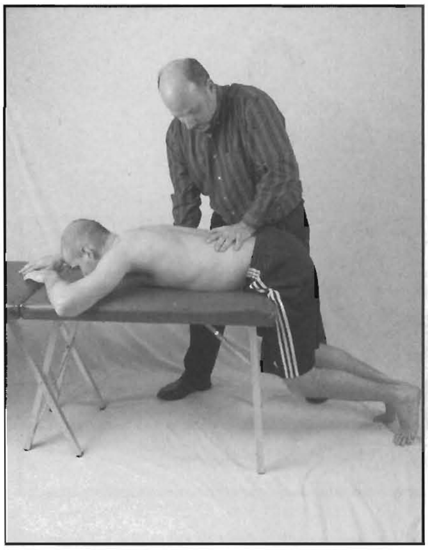

Figure 17-15a. Prone instability test part 1.

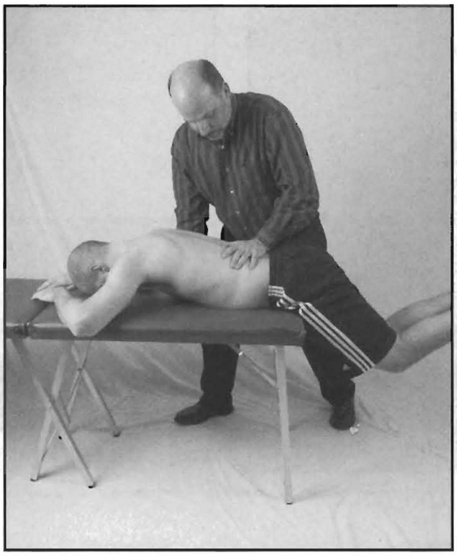

Figure 17-15b. Prone instability test part 2.

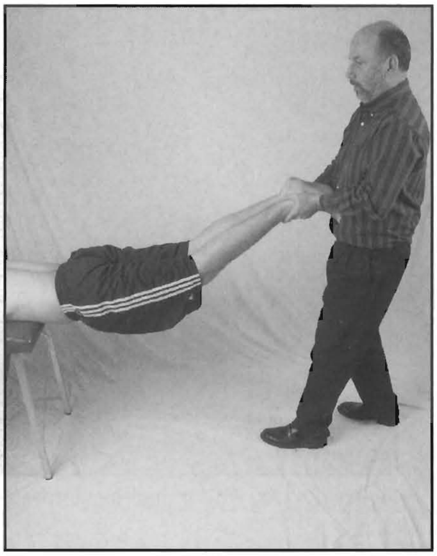

Figure 17-16. Passive lumbar extension test.

bifida occulta or diastematomyelia; café au lait spots may indicate neurofibromatosis or collagen disease.

- Supraspinous ligament: Felt in the interspinous spaces.
- Thoracolumbar fascia: Anterior, middle, and posterior layers (posterior layer is reinforced by the latissimus dorsi superficially).
- Erector spinae muscles: Spinalis, longissimus, and iliocostalis. Palpate for signs of somatic impairment (ie, ropey, stringy, or boggy feel to the tissues).
- Quadratus lumborum: Between the rib cage and the iliac crest. By having the patient lift the pelvis toward the thorax, the muscle can be felt to contract.
- Transversospinalis muscles: Semispinalis, multifidi, and rotatores (deep to the erector spinae between the spinous and transverse processes).

In addition to the inspection for tenderness, tightness, and tone, the myofascial tissues of the lumbar spine and abdominal region can also be examined for extensibility and length. If findings of a medical nature emerge during the soft tissue examination (eg, masses, large palpable pulsations, painful nodes, abdominal rigidity, suspicious skin lesions), the patient's physician should be notified immediately.

Special Tests

This section will be organized into 3 categories. They are neurologic, orthopedic, and physician-based special tests. For more information on special tests, the reader is referred to other textbooks on the topic.[34-38]

Under the neurologic tests, the following examination procedures should be included:

- Myotomes (L1 to S2).
- Dermatomes (L1 to S2): Light touch, pin prick, etc.
- Deep tendon reflexes (knee and ankle jerk).
- Neurodynamic testing (straight leg raise test, well-leg raise, Braggard's test, bowstring sign, femoral stretch test, slump sit test, and variations of the straight leg raise for the proximal sciatic, tibial, common fibular [peroneal], and sural nerves). These tests are performed to assess the mechanical movement of neural tissues and to test their sensitivity to mechanical stress and/or compression.
- Upper motor neuron lesion (upper, middle, and lower abdominal skin reflexes, and Babinski's sign).
- Valsalva's test (used to test for intra- or extrathecal pathology, such as tumor or disc herniation).
- Waddell's signs (nonphysiologic pain symptoms).

The following orthopedic test procedures are recommended:

- Kemp's compression or quadrant test (seated or standing)
- Spondylolysis test (extension in one-leg standing)
- Osteopathic hip drop test
- Schober test (range of lumbar flexion)
- Johnson's lumbar stability tests (vertical compression test, elbow flexion test, lumbar protective mechanism-flexion, and lumbar protective mechanism-extension)
- Prone instability test (Figures 17-15a and 17-15b)
- Passive lumbar extension test[39] (Figure 17-16)
- Nine-point Beighton scale for generalized hypermobility
- Hoover test (assists in identifying the malingering patient)

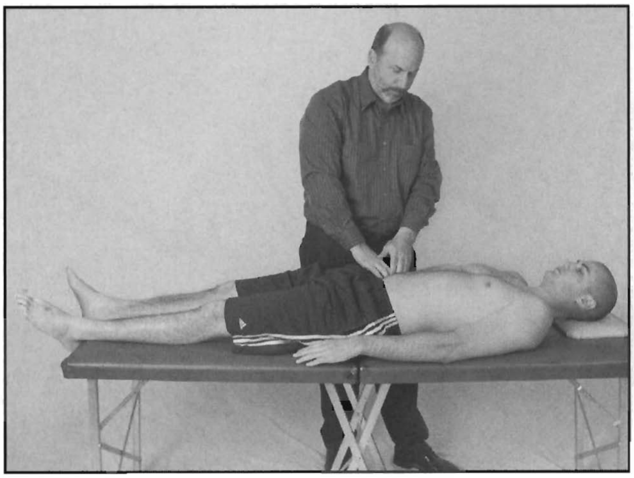

Figure 17-17a. Abdominal tension test stage 1.

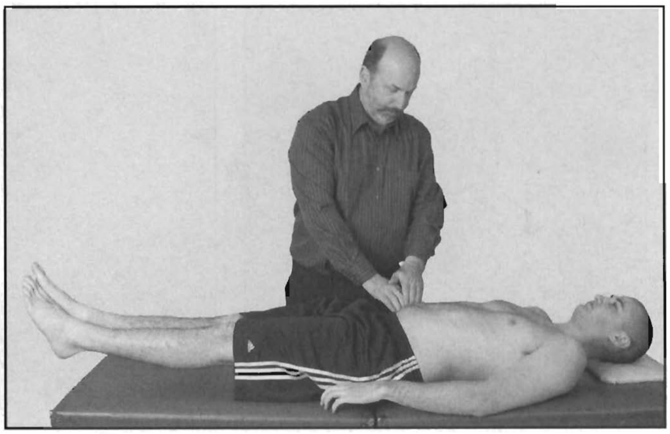

Figure 17-17b. Abdominal tension test stage 2.

- Abdominal tension test (Figures 17-17a and 17-17b), also called Carnett's sign (abdominal wall pain versus pain of abdominal or pelvic visceral origin)

Questionnaires/scales

- Fear avoidance questionnaire, Roland Morris Disability Questionnaire, Zung Depression Inventory (ZDI), Global Rating of Change (GRC) Scale, McGill Pain Questionnaire, Physical Function Scale of the Swiss Spinal Stenosis Questionnaire, Modified Somatic Perception Questionnaire (MSPQ), Oswestry Disability Index, Dallas Pain Questionnaire, Hendler 10-Minute Screening Test for Chronic Back Pain Patients, Functional Capacity Evaluation, etc.

Regarding physician-based special tests, the following tests and procedures are performed as indicated:

- Examination (S3 and S4 sensation and anal sphincter control should be tested by the physician when cauda equina syndrome is suspected; the cremasteric reflex, along with the other superficial reflexes, are performed to rule out upper motor neuron disease)
- Radiologic (x-rays, CT scan, MRI, myelogram, discography, bone scan, etc)
- Electrodiagnosis (EMG, conduction velocity, etc)
- Lab work (CBC, ESR, rheumatoid factors, HLA-B27 antigen, Lyme, Epstein-Barr virus, antinuclear antibodies, etc)
- Tissue biopsy
- Sleep studies (sleep apnea, fibromyalgia/chronic fatigue, etc)
- Psychiatric/psychological evaluation

Regarding the use of MRI and CT in the diagnosis of herniated lumbar intervertebral discs, the following findings in 98 asymptomatic subjects[40] must be kept in mind:

- Only 36% were shown to have normal discs at all 5 lumbar levels
- About half of these pain-free subjects had a bulge of at least one intervertebral disc and about a quarter had at least one disc protrusion
- Annular defects were present 14% of the time; all of these discs had a decreased signal on the T (sub 2) weighted image

In addition, it is estimated that a herniated disc is seen in 20% to 40% of CT scans and myelograms among normal persons.[41] Consequently, a patient's clinical presentation must be carefully evaluated in conjunction with the results of imaging studies as disc bulges and protrusions in patients with LBP and even radiculopathy may be coincidental.[40]

18

Connective Tissue Techniques and Stretching Procedures for the Lumbar Spine

Thoracolumbar Junction Release

This release (Figure 18-1) is an important myofascial release technique for both the thoracolumbar junction and the posterior diaphragm. The therapist's hands are placed lightly on either side of the prone patient's thoracolumbar junction with the thumbs close to the SPs and the fingers pointing cephalward. The "shear-clock" method is again utilized to identify impairment in mobility of the skin and superficial fascia (indirectly, the deep fascia as well through its connection to the basement membrane of the dermis). Once the AGR is located, the therapist uses either indirect or direct treatment technique to obtain the desired "release" (the 4 Ms procedure described in Chapter 5 is applicable to either approach).

Ward[42] describes a direct myofascial release technique in which he employs compression, traction, and twist to the tissues between the right and left hands for 10 to 30 seconds. As softening and elongation occur (ie, myofascial "release"), the therapist follows behind in search of new motion barriers. Compression is achieved by applying a light posterior to anterior force through both hands; traction involves a perpendicular stretch of the paraspinal tissues, and twist is achieved through a clockwise/counterclockwise rotation of the hands. These 3 "pre-release" forces cause a "winding up" of the tissues, which sets the stage for myofascial unwinding. The Ward approach is actually a combination technique that begins direct and ends indirect.

Quadratus Lumborum Release

This connective tissue technique (Figure 18-2) employs a combination of muscle stretching, hold-relax or postisometric relaxation, direct fascial technique, and myofascial release. With the patient in the side-lying position, the therapist prestretches the soft tissues by separating the iliac crest from the rib cage while simultaneously grasping and lifting the soft tissues upward. This is followed by a longitudinal stretch of the quadratus lumborum (QL) muscle. To enhance the stretch, several hold-relax contractions and relaxations are added. Direct fascial technique can be integrated at any time during the stretch (eg, perpendicular strumming, muscle play, myotherapy, progressive pressure technique).

The clinical importance of eradicating trigger points and restoring length and myofascial extensibility to the QL muscle cannot be emphasized enough. The quadratus has been identified as a source of backache and lumbar myalgia. It is also a source of referred pain into the sacroiliac region, hip, buttock, greater trochanter, abdominal region, and groin.[43]

Having said that, the QL is also an important stabilizer of the spine and must never be stretched to the point of undermining this function.[44]

Iliopsoas Fascial Technique

Understanding the actions of the iliopsoas muscle serves as a useful guide in both the evaluation and intervention

Makofsky HW
Spinal Manual Therapy, 2nd ed. (pp 153-158)

Figure 18-1. Thoracolumbar release.

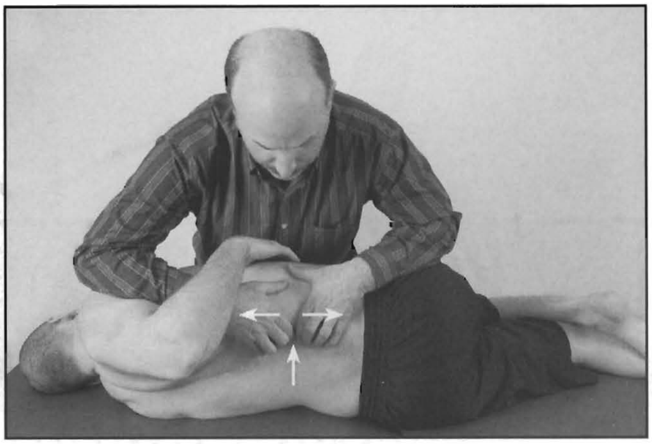

Figure 18-2. Quadratus lumborum fascial technique.

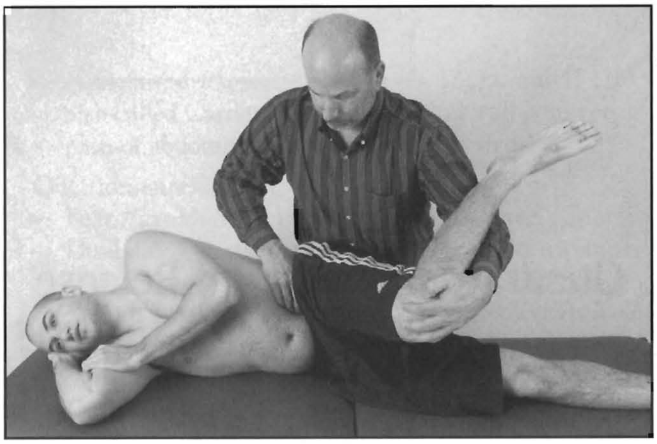

Figure 18-3a. Iliopsoas fascial technique phase 1.

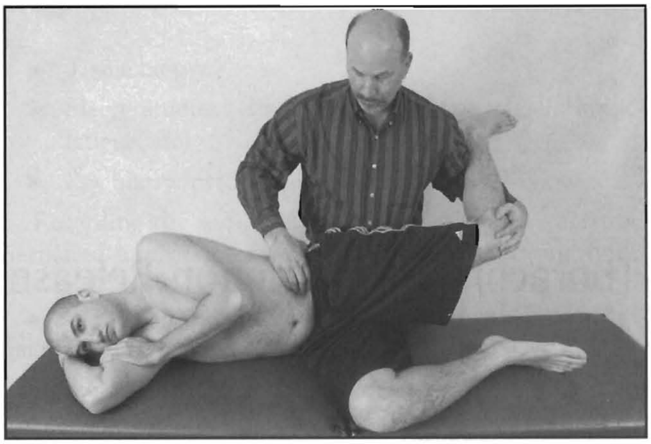

Figure 18-3b. Iliopsoas fascial technique phase 2.

of patients with lumbar-pelvic-hip impairment. The actions are as follows:

- Hip flexion with a secondary role in external rotation and abduction.
- In erect posture, upper lumbar extension with lower lumbar flexion (ie, exaggerated lumbar lordosis with anterior pelvic tilt).
- In the forward bent position, the iliopsoas contributes to lumbar flexion.
- A unilateral contraction laterally flexes the lumbar spine to the ipsilateral side and, by compression, contributes to spinal stability.
- Unilateral tightness may also contribute to a type-2 impairment such that the inferior component of the motion segment is laterally translated to the ipsilateral side. For example, left-sided tightness at L3,4 causes lateral translation of L4 to the left with overturning of L3 into flexion, rotation, and side bending to the right (ie, FRS right at L3,4).

In the author's opinion, it is imperative that the status of the iliopsoas muscle be assessed in all low back patients. In the pelvic girdle section of this textbook (Section VI), the length of the iliopsoas will be assessed and treated as one component of the tensor fascia latae, rectus femoris, and iliopsoas (TRI) muscle stretch. At this point, however, our focus will be on the application of a direct fascial technique with the muscle on slack and under stretch.

As illustrated, the patient is positioned in side lying with the involved side up. The initial phase of this technique (Figure 18-3a), involves passively placing the upper-most hip in flexion and external rotation in order to relax the iliopsoas for greater access. While adjusting the lower limb for maximal relaxation, the other hand monitors the psoas approximately 2 inches lateral to the umbilicus. In this position, an isometric contraction of the hip flexors ensures that the iliopsoas has been located. Following deep tissue massage in the muscle's slackened state, the iliopsoas is then placed under stretch by extending the lower limb at the hip (Figure 18-3b). In this position, the therapist again applies deep tissue massage in conjunction with postisometric relaxation, which helps to decrease the resting tone of the iliopsoas muscle (the therapist may have the patient hold the bottom leg in hip and knee flexion for enhanced counter stability of the pelvis).

Treating the muscle in both positions (ie, slackened or stretched) allows either an indirect or direct approach to intervention, depending on the state of tissue reactivity

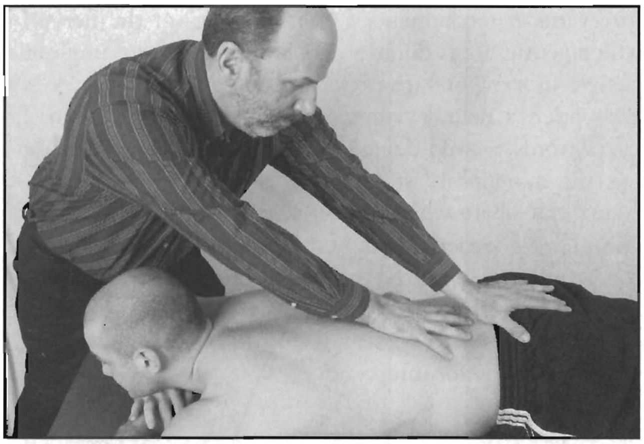

Figure 18-4. Lumbosacral junction release.

present (see Chapter 3 for a description of direct and indirect treatment methods).

According to Bogduk et al,[45] the psoas major at maximum contraction exerts "severe compression forces on the lumbar segments," which is consistent with recent thinking on the role of the psoas in spinal stabilization.[44]

Lumbosacral Junction Release

In order to disengage the lumbosacral junction and release abnormal soft tissue tension (Figure 18-4), the therapist's cephalic hand is placed over the lower lumbar spine, while the caudal hand is placed over the sacrum with the heel of the hand on the sacral base. The therapist can approximate the 2 hands to perform an indirect myofascial "unwind" or place the tissues of the lumbosacral junction on maximal stretch and perform a direct technique against the restrictive barrier(s). A pillow can be placed under the patient's abdomen to further decompress the region.

Sacrospinalis Stretch

Patients with McKenzie's flexion dysfunction respond well to the sacrospinalis stretch (Figure 18-5). However, it is contraindicated in patients with posterior derangement of the lumbar spine.

With the patient placed in the "knees to chest" position, the therapist places his or her hand under the patient's sacrum with the fingers on the base and the heel of the hand over the apex. The stretch is accomplished by directing the lumbar spine into further flexion through the lower limbs as the sacrum is counternutated. A gentle postisometric relaxation often enhances the technique's efficacy, providing that the patient's symptoms are not exacerbated by this procedure.

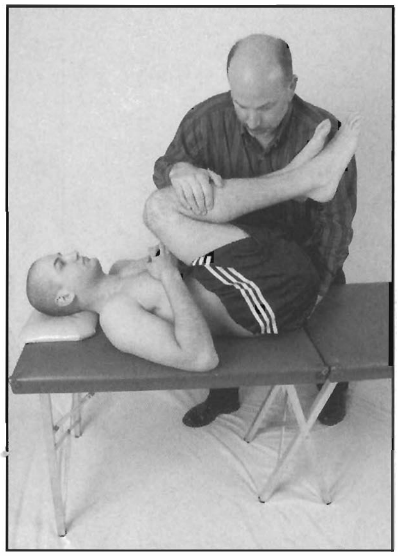

Figure 18-5. Sacrospinalis stretch.

Neural Mobilization

Butler[46,47] recommends that neural mobilization be viewed as another form of manual therapy similar to joint mobilization. In this regard, the treatment of signs and symptoms based on the severity, irritability, and nature of the impairment must be kept in mind at all times. The danger in presenting this material outside the context of the entire art and science of neural mobilization is that it be seen as a technique rather than as a comprehensive system involving clinical reasoning, problem solving, and a thorough understanding of the anatomy, physiology, and pathophysiology of neurobiologic structures. Having said that, we will proceed to using manual methods in order to restore the mechanical function of impaired neural tissues (intra- and extraneural impairment) in the lumbar-pelvic-lower limb complex. As with all manual therapy procedures, the goal remains the same (ie, "to restore maximal pain-free movement within postural balance"). Contraindications include irritable conditions, inflammation, spinal cord signs, malignancy, nerve root compression, peripheral neuropathy, and complex regional pain syndromes I and II. Regarding outcomes data, there is only limited evidence to support the use of neural mobilization at the present time.[48]

Proximal Sciatic Nerve

The sciatic nerve is the largest nerve in the body but actually consists of 2 nerves—the common fibular (peroneal) and tibial—that are tightly bound together by connective tissue. The common fibular nerve is a posterior branch of the sacral plexus originating from the lumbosacral trunk (L4 to S2); the tibial nerve is an anterior branch of the sacral plexus originating from the ventral rami of L4 to S3.

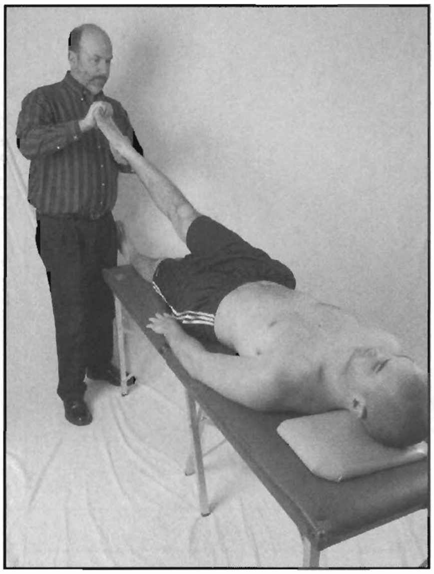

Figure 18-6. Proximal sciatic nerve stretch.

Sites of potential proximal compression include the lower lumbar spine (eg, intervertebral disc, spinal canal, lateral recess, intervertebral foramina), the piriformis muscle, and hamstrings. Because the sciatic nerve runs posterior to the hip and knee joints, the optimal means of inducing longitudinal tension is through the straight leg raise (SLR) first described in 1864 by Leseague.[46] The leg is lifted upward, as a solid lever, while maintaining extension at the knee. To induce dural motion through the sciatic nerve, the leg must be raised past 35 degrees in order to take up slack in the nerve. Since the sciatic nerve is completely stretched at 70 degrees, pain beyond that point is usually of hip, sacroiliac, or lumbar spine origin. The unilateral SLR causes traction on the sciatic nerve, lumbosacral nerve roots, and dura mater. Adverse neural tension produces symptoms from the low back area extending into the sciatic nerve distribution of the affected lower limb. To introduce additional traction (ie, sensitization) into the proximal aspect of the sciatic nerve, hip adduction is added to the SLR (Figure 18-6). This is because the sciatic tract is lateral to the ischial tuberosity; therefore, adduction causes further tensing of its proximal aspect.

Prior to commencing neural mobilization, McKenzie's derangement syndrome must be ruled out. Stretching nerve roots that are reacting to local compression is only indicated for examination purposes. Cyriax[49] described the straight leg raise "painful arc" sign, which usually appears from 45 to 60 degrees. This sign, in which there is no pain above and below the point of adverse neural tension, implies that the nerve root momentarily "catches" against a small protrusion and then slips over it. In the presence of this finding or other indications of disc herniation, neural mobilization should not be performed. The purpose of neural mobilization is to restore normal function to impaired neural structures that were previously compressed, irritated, and inflamed. The intervention recommended is a "flossing" of the nerve in which gentle, short duration (1 second) and large amplitude passive movements are performed at the "feather edge" of the patient's neural symptoms in an "on/off" fashion. In other words, a mild degree of discomfort is permitted during the momentary stretch (ie, "on" phase), which must completely abate when the tension is withdrawn (ie, "off" phase). The patient's symptoms must be monitored at all times, and it is suggested that the patient be initially undertreated until the irritability of the impairment becomes apparent. Thirty to 60 seconds of on/off mobilization is a useful guideline for intervention.

Femoral Nerve

The femoral nerve is a branch of the lumbar plexus, which is formed by the ventral primary rami of L1, L2, L3, part of L4, and possibly T12. The femoral nerve continues medial to the knee as the saphenous nerve. The femoral nerve stretch was first described by Wasserman[46] in 1919, who proposed it as a physical sign to explain anterior thigh and shin pain in soldiers. In 1946, O'Connell recommended the inclusion of hip extension.[46]

As with other nerve-stretching maneuvers, the femoral nerve stretch (prone knee bend or Nachla's test) can be used for both examination and intervention. There are 2 components to the nerve stretch:

1. The uppermost part of the thigh is passively extended just short of producing lumbar spine extension. By creating tension in the iliopsoas, the upper lumbar nerve roots are put under traction.
2. The knee is then progressively flexed to increase femoral nerve tension by stretching the quadriceps femoris muscle. Pain in the anterior thigh may be of muscular or nerve origin. A careful history should help to delineate the problem.

Again, the neural flossing technique in an on/off fashion is recommended for adverse neural tension (Figure 18-7). The pelvis should be properly stabilized to prevent stress from being placed on the sacroiliac joint and lumbar spine (Yeoman's test). The lateral femoral cutaneous nerve can be stretched by adding hip adduction to the extended hip and flexed knee (Figure 18-8). The saphenous nerve is stretched by placing the hip in extension, abduction, and lateral rotation while extending the knee and dorsiflexing/everting the ankle (Figure 18-9).

Common Fibular Nerve

The common fibular (peroneal) nerve (L4,5; S1,2) lies directly posterior to the proximal fibular head and, therefore, can be injured with posterior fibular head displacement or fracture of the fibula. Since supination of the ankle

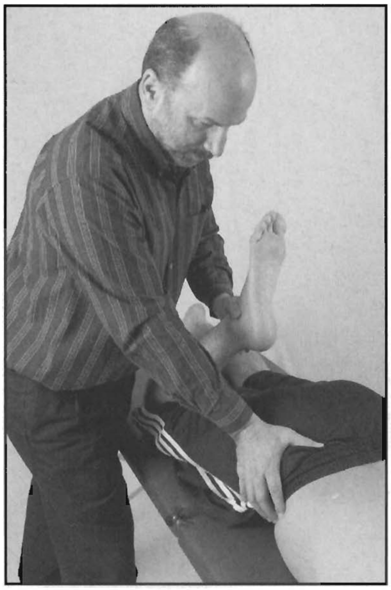

Figure 18-7. Femoral nerve stretch.

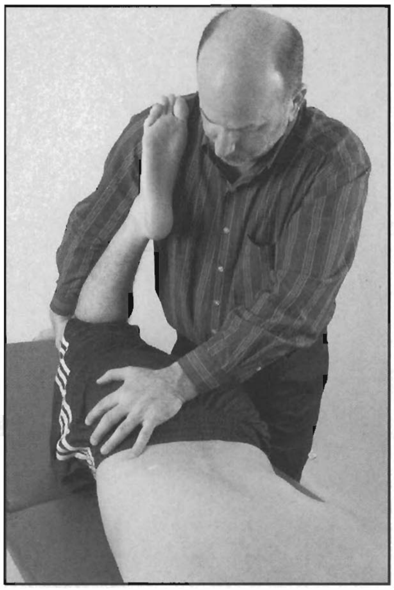

Figure 18-8. Lateral femoral cutaneous nerve stretch.

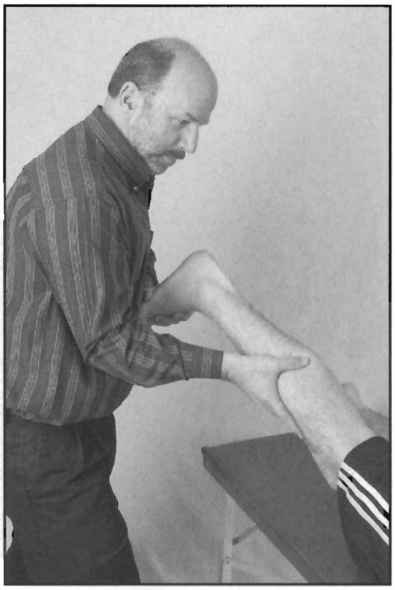

Figure 18-9. Saphenous nerve stretch.

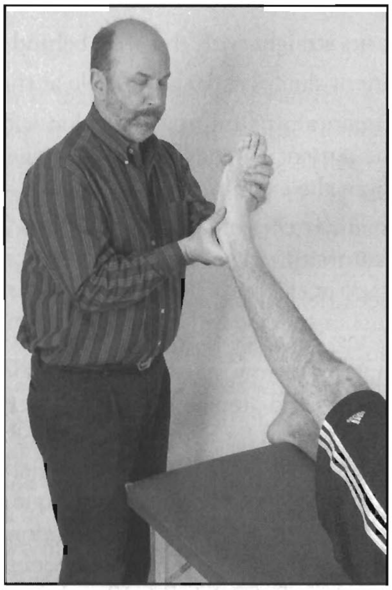

Figure 18-10. Common fibular nerve stretch.

causes a posterior glide of the fibular head, a lateral ankle sprain can be a contributing factor to injury of the nerve.

To place the common peroneal nerve under tension, the hip is flexed and medially rotated, the knee is extended, and the ankle is plantar flexed and inverted (Figure 18-10). According to Butler,[46] plantar flexion/inversion may be added before the SLR or at the completion of the SLR. Once again, the management of adverse neural tension involves a gentle on/off stretch of large amplitude at the onset of symptoms. The goal is to achieve functional gliding of the common fibular nerve along its complete course from proximal to distal.

Tibial Nerve

The tibial nerve (L4,5; S1 to S3) is brought under tension with the addition of ankle dorsiflexion. This is because its terminal branches, the medial and lateral plantar nerves, course along the plantar surface of the foot and are therefore stretched by dorsiflexing the ankle. In addition to hip flexion, knee extension, and ankle dorsiflexion, the tibial tract can be further sensitized by everting the ankle, extending the toes, and stretching the plantar fascia (Figure 18-11). Butler states that ankle dorsiflexion may be added first and then the limb lifted, or performed at the limit of the SLR. The tibial nerve forms the largest component of the sciatic nerve in the thigh. Inferiorly, it descends through the popliteal space, passing between the heads of the gastrocnemius muscle to the dorsum of the leg, as the posterior tibial nerve, and into the ankle and foot. As the posterior tibial nerve traverses under the flexor retinaculum at the tarsal tunnel, it is subject to possible compression (ie, tarsal tunnel syndrome). As mentioned above, it then divides into the medial and lateral plantar nerves, which supply sensation to the sole of the foot and toes as well as supplying sensation to the foot joints and efferent fibers to the small muscles of the foot. When adverse neural tension is present, neural mobilization is gently performed for 30 to 60 seconds.

Sural Nerve

The medial sural cutaneous nerve, a branch of the tibial nerve, joins the lateral sural cutaneous nerve, a branch of the common fibular nerve, to form the sural nerve (L5, S1,

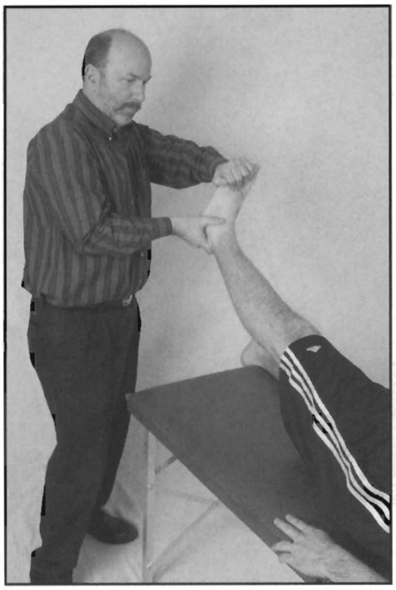

Figure 18-11. Tibial nerve stretch.

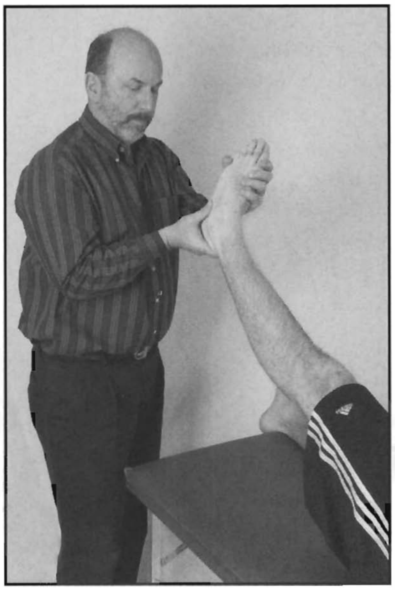

Figure 18-12. Sural nerve stretch.

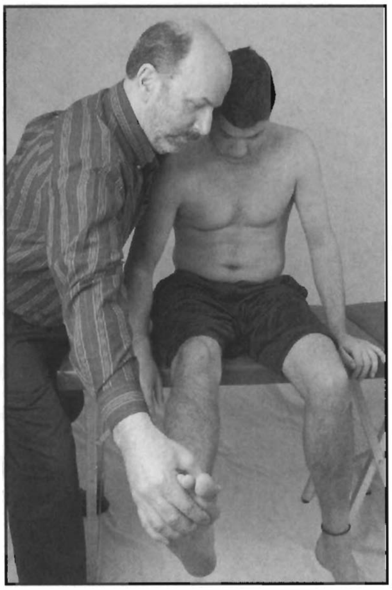

Figure 18-13. Slump sit test and neurodynamic mobilization.

S2), which supplies the skin of the posterolateral part of the leg and the lateral side of the foot. According to Butler,[46] "The sural nerve is a forgotten nerve and is responsible for far more symptoms than it is given credit for." With practice, the sural nerve can be palpated along the lateral aspect of the foot, behind the lateral malleolus, and lateral to the Achilles' tendon. The position of maximal sural nerve tension consists of hip flexion, knee extension, and ankle dorsiflexion followed by ankle inversion (Figure 18-12). Butler refers to this combination of movements as the sural nerve tension test. As with the other nerve tension tests, the same limb position is then transformed into a neural mobilization in the presence of impairment.

To further sensitize the tibial, common fibular, and sural nerves, additional loading is made possible by adding cervical flexion, lumbar and thoracic side bending to the contralateral side, hip adduction, and medial rotation.

Slump Sit Test/Mobilization

The Slump sit test described by Butler[46,47] and Cook and Hegedus[38] has a sensitivity of 83% and a specificity of 55% for lumbar radiculopathy and involves the following steps:

1. Patient sits straight with the arms behind the back.
2. The patient slumps as far as possible at the trunk.
3. While maintaining full spinal flexion with overpressure, the patient extends his or her knee with assistance from the examiner.
4. The examiner then moves the foot into dorsiflexion while maintaining knee extension.
5. Head-neck flexion is then added.

A positive test consists of reproduction of the patient's radicular symptoms at any stage of the test. Herrington,[50] using the slider and tensioner techniques originally described by Butler et al for neural mobilization,[51] reported that in normal females both the tensioner (ie, nerve tract elongation/stretching) and the slider techniques (ie, sliding of the nerve along its bed without elongation) or neural "flossing" had a positive and significant effect on improving knee extension range of motion in the slump-sit position (P=.003) and (P<.001), respectively. This could potentially decrease sensitivity of the sciatic nerve and neuromeningeal structures to mechanical load in a symptomatic population, but further investigation is required (Figure 18-13).

19

Lumbar Spine Mobilization/Manipulation

As with manual therapy of the thoracic spine (see Chapter 6), PPIVMs in the lumbar spine can be easily transformed into sitting mobilization/manipulation techniques. This is done by identifying the AGR and then proceeding with either the "hold one/move one" approach (ie, stabilize the bottom and mobilize the top vertebra of the motion segment into its restricted range) or the "roll-glide" technique. The specific grade (1 to 4) is, of course, determined by the level of reactivity present.

Having said that, this chapter will not cover manipulation of the lumbar spine in each of 6 possible directions as with the thoracic spine (eg, flexion, extension, side bending right and left, and rotation right and left). Instead, an apophyseal distraction (ie, gapping) maneuver will be described as a means of introducing a simple, efficient, and effective way of mobilizing any of the impaired 10 lumbar spine facet joints (5 left, 5 right) as necessary. In addition to using PPIVMS from L1 to L5, as described in Chapter 17, to identify the most restricted lumbar facet, the therapist can also assess joint distraction relative to the quality, quantity, end-feel, and tissue reactivity of each of the 10 lumbar facets by following the instructions for gapping a hypomobile lumbar facet in this chapter. To further corroborate lumbar facet impairment, the active movement examination performed in Chapter 17 will likely reveal the presence of the facet capsular pattern of restriction as follows: 1) limited contralateral side bending/ipsilateral rotation, 2) trunk flexion associated with deviation to the affected side (eg, restricted facet opening at L3,4 on the left side "steers" the vertebral column to the side of impairment, namely to the left, followed by a return of the trunk to the midline providing that the other facets compensate for the left L3,4 restriction), and 3) extension associated with deviation of the trunk to the nonaffected side (eg, restricted facet closing at L3,4 on the left side "steers" the vertebral column to the side opposite the impairment, namely to the right, followed by return to the midline, again, if there is compensation by the other lumbar facet joints).

As with use of facet distraction in the cervical spine (Chapter 10), this author will use Kaltenborn's grades[52] 1 to 3: grade 1 (support of the joint to neutralize negative pressure and loosen the capsuloligamentous tissues), grade 2 (passive movement taken to the end of the tissue slack or first stop), and grade 3 (beyond the tissue slack against the restrictive barrier to patient tolerance). The manipulative technique to follow is not recommended when McKenzie's derangement syndrome[9,21] is present, lest further injury to the intervertebral disc result, and is absolutely contraindicated in the presence of nerve root compression resulting in peripheral neuropathy or symptoms of cauda equina syndrome (eg, acute urinary retention, saddle anesthesia, diminished anal sphincter tone).

Apophyseal Joint Gapping

A manipulative distraction of the right L4,5 facet joint will be described. The patient is placed in the left lateral decubitus position (ie, side lying on the left side) with the affected joint uppermost. In order to "gap" the right L4,5 facet, a combination of flexion, left side bending, and right rotation is introduced. In this position, the inferior articular process of L4 separates from the superior articular process of L5 in a perpendicular direction (Figure 19-1). In females, the width of

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Figure 19-1. Lumbar spine facet gapping technique.

Figure 19-2. Right L4,5 facet gapping technique.

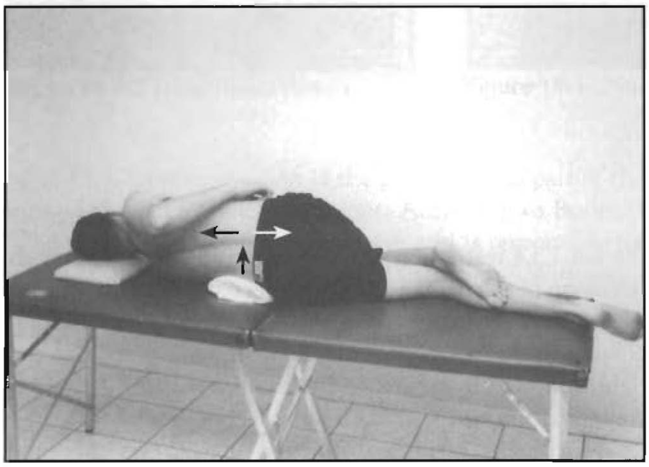

Figure 19-3. Lumbar spine positional distraction right.

the pelvis may necessitate placing a towel roll under the waist in order to enhance lumbar side bending to the left.

The components of proper manipulative technique include localization, balance, and control. The therapist must properly localize the forces of X-axis flexion, Y-axis rotation, and Z-axis side bending to the "feather edge" of the restrictive barrier prior to performing the graded distraction. The L4,5 level can be located by first identifying the last mobile segment (ie, L5, S1) in extension and then coming up one level. The L4,5 interspinous space is usually at the level of the iliac crest as well. With the right middle finger between L4 and L5, the patient's right lower limb is flexed at the knee and hip with the therapist's left hand until flexion first arrives at L4,5. The patient's right foot is then placed on top of his or her left knee and kept there.

At this point, the movement of right rotation of the trunk is introduced from above until motion is first detected at L4,5 with the left middle finger. This can be achieved by pulling the left upper arm up and forward (above the elbow) with the "lawn mower" maneuver. The patient's right arm is then placed on his or her flank with the elbow flexed.

The final phase of the set-up involves the localization of L4,5 side-bending left. The therapist does this by separating the patient's pelvis from his or her shoulder girdle with a pushing force in opposite directions. When the side-bending left motion is felt to arrive at L4,5, the position is maintained.

Now that the motions of flexion, right rotation, and left side bending have been localized to the restrictive barrier at the right L4,5 apophyseal joint, the manipulative force can be introduced (grades 1 through 3). As with any mobilization performed thus far, a postisometric relaxation technique can be utilized to reduce muscle hypertonus.[53-56] Following 2 to 3 cycles, the patient can be relocalized to the new barrier in preparation for graded distraction.

The manual distraction of the right L4,5 apophyseal joint involves a "gapping" of the joint such that the facet surfaces are separated in a perpendicular fashion. The best way to accomplish this without causing undue stress on the intervertebral disc is by emphasizing additional left side bending by gently pushing the patient's pelvis and shoulder girdle in opposite directions. However, in order to maintain the right rotation component, the therapist's right thumb maintains contact on the right side of the L4 SP as the therapist's left middle finger provides pressure on the L5 SP from below (Figure 19-2). The specific grade selected (ie, 1 through 3) is dependent upon the reactivity and the degree of facet impairment present. In addition to the effects of mobilization on the articular tissues, this manipulative distraction will also widen the intervertebral foramina between L4 and L5 on the right and consequently is useful when there is nerve root compression (ie, pinched nerve) at the foramina or the lateral recess. Because the female pelvis is wider, as mentioned previously, it may be necessary to place a towel roll under the female patient's waist in order to achieve the desired side bending on the inferior side. A towel roll, placed under the patient's waist in side lying, can also be used as a means of achieving positional distraction of the uppermost facets, which can be taught to the patient for home use. It is an excellent way of decompressing irritated nerve roots in the neuroforamina, especially as a consequence of lateral spinal stenosis, in the lumbar spine in a safe and effective manner (Figure 19-3).

As mentioned previously, the "gapping" maneuver described in this chapter can be used for any of the 10 facet joints in the lumbar spine. For those who are trained in the correction of FRS impairment, it is useful to first distract the affected facet prior to closing it.

20

Therapeutic and Home Exercises for the Lumbar Spine

As with all regions of the vertebral column, there needs to be a balance between mobility and stability. In states of impairment, the thoracic spine tends toward hypomobility, whereas the cervical and lumbar regions, especially in the lower segments, tend toward hypermobility. The Panjabi model[14-16] refers to this balance as clinical stability, and the loss of balance in the direction of hypermobility as clinical spinal instability. More specifically, clinical spinal instability is a significant decrease in the capacity of the stabilizing system of the spine to maintain the intervertebral neutral zone within physiologic limits, resulting in pain and disability.[57] Furthermore, clinical spinal instability is thought to damage position sensors in the lumbar spinal tissues resulting in altered reporting to the CNS. As a consequence of impaired position-sense information (ie, proprioception), there is faulty control of deep spinal-stabilizing muscle activation.[44,57,58] In turn, decreased control of the local spinal stabilizers (eg, multifidus) leads to further damage of the position-sensors of the spine. This ultimately results in progressive worsening with the potential for developing incapacitating LBP, major deformity, and neurologic deficit.[11,14-16,39,44,57,58] According to Panjabi,[14-16] the 3 components of this spinal stabilization system are the passive, active, and neural-control subsystems. The passive spinal subsystem consists of the osseous, articular, and ligamentous structures of the vertebral column; the active spinal subsystem consists of the musculofascial structures that promote stability of the spine through the force-generating abilities of individual muscles; and the neural-control subsystem consists of the sensorimotor control process that monitors (afferent component) and adjusts (efferent component) muscular forces acting on the spinal joints. Under normal conditions, the 3 subsystems work in harmony to provide the needed mechanical stability. The purpose of spinal or core stabilization training, therefore, is to restore an optimal neutral zone whereby all 3 subsystems work together to prevent segmental hypermobility (ie, clinical spinal instability) and to consequently reduce the problems associated with this condition (eg, repetitive microtrauma, impaired neural regulation of the active spinal subsystem, degenerative changes, chronic low back pain and radiculopathy).

In the lumbar spine specifically, Richardson et al[58] have introduced the concept of a deep local muscle system that is ideally suited for the control of neutral zone motion, including shear forces between spinal vertebrae. The deep muscles of this local system, being closer to the center of rotation with short muscle lengths, are ideal for controlling intersegmental motion. According to these Australian researchers, the functional unit of local stabilization consists of the respiratory diaphragm, the pelvic floor, the lumbar multifidus, and the transversus abdominis (Figure 20-1). Under normal conditions, the isolated action of "drawing the navel in toward the spine" not only causes a deep contraction of the transversus abdominis, but also causes a co-contraction of the other components of the system. Consequently, this deep, local system co-contraction acts as an inner corset of musculofascial support that provides static and dynamic stability to each of the lumbar segments. In addition, it has been demonstrated that under normal conditions, the transversus abdominis[58,59] and respiratory diaphragm[60] have an anticipatory or feedforward function whereby they contract to stabilize the spine prior to movement of the limbs. However, in people with chronic LBP,

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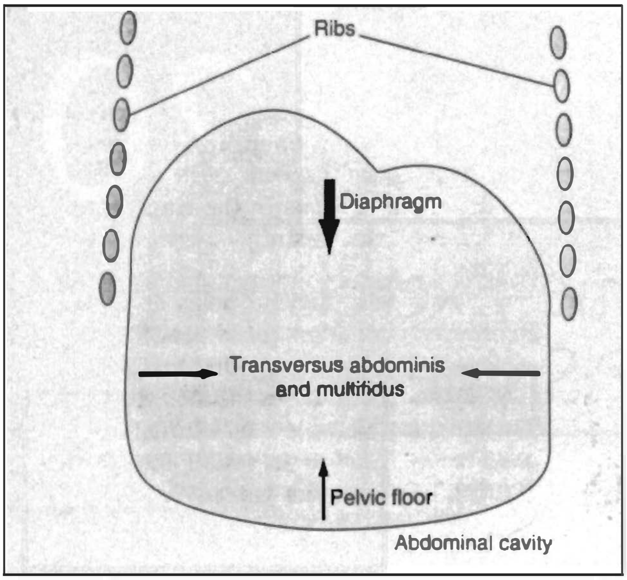


Figure 20-1. The functional unit of core stability. (Reprinted from Richardson C, Jull G, Hodges P, Hides J. *Therapeutic Exercise for Spinal Segmental Stabilization in Low Back Pain: Scientific Basis and Clinical Approach.* Edinburgh, UK: Churchill Livingstone; 1999, by permission of the publisher Churchill Livingstone.)

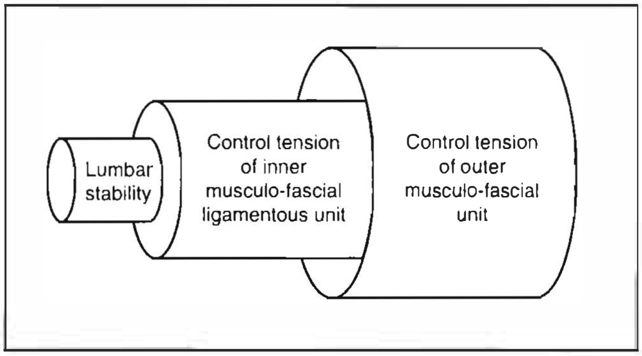


Figure 20-2. The inner "corset" concept of the core stabilizers. (Reprinted from Richardson C, Jull G, Hodges P, Hides J. *Therapeutic Exercise for Spinal Segmental Stabilization in Low Back Pain: Scientific Basis and Clinical Approach.* Edinburgh, UK: Churchill Livingstone; 1999, by permission of the publisher Churchill Livingstone.)

it has been shown that this protective response is impaired and that these important core muscles demonstrate delayed response times, leaving the spine vulnerable to suffering microtrauma.[58,60]

To better understand the mechanism for spinal support, one needs to appreciate the concept of intra-abdominal pressure (IAP) and LaPlace's law, which states that $T = Pr$, where T is circumferential tension, P is pressure, and r is radius. Theoretically, bilateral contraction of the transversus abdominis raises IAP by increasing abdominal wall tension (T) as well as by decreasing abdominal radius (r). This increase in IAP helps to convert the abdomen into a semirigid cylinder, which results in a stiffer and more stable structure. In addition, an increase in IAP has been shown to generate a small but consistent extension moment of the spine,[61] which is thought to reduce the demand for back extensor activity and decrease the compressive load on the lumbar spine.[58] Furthermore, contraction of the transversus abdominis muscle, through its attachment to the lateral raphe, exerts lateral tension on the middle and posterior layers of the thoracolumbar fascia and therefore may contribute to the control of intersegmental motion by restricting vertebral displacement.[58]

The "global" muscles are those torque-producing muscles that attach the pelvis to the thoracic cage (eg, rectus abdominis and external oblique). Unlike the local system, which provides core stability, the global muscles provide a more general lumbopelvic stability function. In normal function, the local and global systems work together to provide trunk mobility on "core stability" (Figure 20-2). However, in patients with chronic LBP, the global system appears to overpower the local one, which has become inhibited and ineffective. This loss of core stability is what causes many of the problems found in LBP patients and what practitioners of spinal or core stabilization therapy[62] and Pilates[63] attempt to retrain. Hides et al[64,65] point out that multifidus dysfunction (ie, muscle atrophy and pathologic changes) and the associated loss of segmental lumbar stability are correlated to poor functional outcomes and recurrence of LBP after disc surgery. They suggest that impaired function of the multifidus muscle may be a factor in the high recurrence of pain (ie, 60% to 80%) in the year following an individual's first episode of acute LBP. Furthermore, Hides et al[65] demonstrated that multifidus recovery does not occur automatically with resolution of pain and disability but requires specific exercises that focus on activating the multifidus at the affected segmental level. Grenier and McGill[66] challenge the Australian Stabilization approach[58] by contending that transversus abdominis activation via the abdominal "draw-in" or "hollow" maneuver does little to provide mechanical spinal stability. Conversely, they recommend abdominal "bracing" as a more effective way to address mechanical LBP related to clinical spinal instability.

In this chapter, we will address the issue of core stability, but first we must deal with the self-treatment of common lumbar derangements and dysfunctions in the lumbar region.

Management of Lumbar Derangement (Phases 1 to 3)

The patient's response to the repeated movements exam provides the foundation upon which the self-treatment model for lumbar disc derangement is based. Because

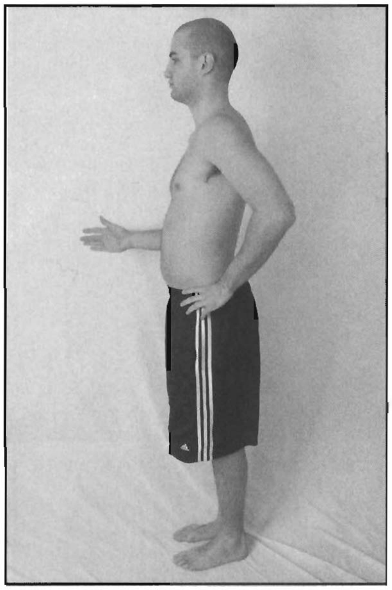


Figure 20-3. Self-correction for right lateral shift.

the techniques used for intervention are the same as for the examination, the reader is advised to review phases 1 through 3 in Chapter 17. Having said that, the patient must be taught one additional maneuver not yet covered: the McKenzie self-correction of a lateral shift for derangements 4 and 6. For example, a patient with a right lateral shift (ie, shoulders shifted to the right) stands with his or her right side against the wall (Figure 20-3). The right elbow is then flexed to 90 degrees with the right forearm placed against the lower ribs. With the feet approximately 12 inches from the wall, the patient is advised to gently and rhythmically shift the hips to the right in an on/off fashion several times with the left hand. The patient is properly instructed in McKenzie's principles of derangement reduction (ie, centralization phenomenon) and advised to perform this self-treatment procedure as often as necessary. Once the lateral shift has been corrected, the patient can proceed with phases 1 through 3, as indicated, to obtain complete reduction of the posterior derangement (the clinician should keep in mind that phase 1 intervention is always the preferred phase provided that it yields the desired outcome).

Regarding the McKenzie management of derangement syndrome, the following guidelines must be kept in mind:

1. The therapeutic movement is the one that yields the "most for the least" (ie, the least force for the most centralization).
2. Self-treatment is preferred to therapist-assisted technique because it empowers the patient to become independent.
3. The myth of not performing lumbar extension must be overcome. For those therapists trained in "flexion only," lumbar extension feels like the unpardonable sin! These therapists must remember that the McKenzie approach is not synonymous with extension. The approach taken is based upon what works for the patient. There are situations when flexion is indeed necessary (eg, derangement 7, flexion dysfunction). However, the efficacy of lumbar extension exercises for the management of certain posterior and posterolateral disc derangements is undisputed. As long as the patient possesses an intact nociceptive afferent system, there is minimal risk. When the intervention is working, the centralization phenomenon will be observed[67]; when the lesion is not responding or becomes worse, the peripheralization phenomenon will reveal this and the exercise is stopped. Regarding the role of extension exercises in cases of clinical instability, the therapist is attempting to reduce the derangement that may be placing the patient in an unstable position. If, as just stated, the intervention is working, the patient's symptoms will centralize and improve, but if the patient is being made more unstable by extension, then the symptoms will worsen and the intervention will be stopped. This is why McKenzie stresses ongoing feedback from the patient at all times. In the presence of a stable spondylolisthesis (grade 1 or 2), extension is not a contraindication but a precaution. Having said that, any worsening of the patient's condition should be noted and extension exercises should be immediately stopped. However, in the presence of either a stable or unstable grade 3 or 4 spondylolisthesis, in this author's opinion, lumbar extension should be avoided because of anterior shearing of the inferior vertebra of the motion segment making matters worse.
4. The patient is only escalated to the next phase when necessary (eg, prone press-ups before shifted-hips press ups, self-treatment before manual stabilization and mobilization).
5. It must be stressed to the patient that derangements require constant vigilance. If the patient is not committed to performing the derangement-reducing movements a minimum of 3 sets of 10 every 2 hours, the results, in most cases, will be limited.
6. Following the repeated movement component of the McKenzie approach, the patient must be instructed in the proper maintenance of a neutral lumbar lordosis. In the case of derangements 1 through 6, lumbar flexion must be avoided for at least 3 to 5 days to allow stabilization of the derangement to occur. The patient should avoid sitting, if possible, because of greater intradiscal pressure in this position.[8,68] However, if this is not possible, then the chair should be positioned so that the patient's knees are lower than the hips. There are several commercially available lumbar support pillows that are also helpful. In addition to their role in managing disc derangements, lumbar supports are useful in managing backache associated with McKenzie's postural and dysfunction syndromes. They are also used to prevent the postural problems

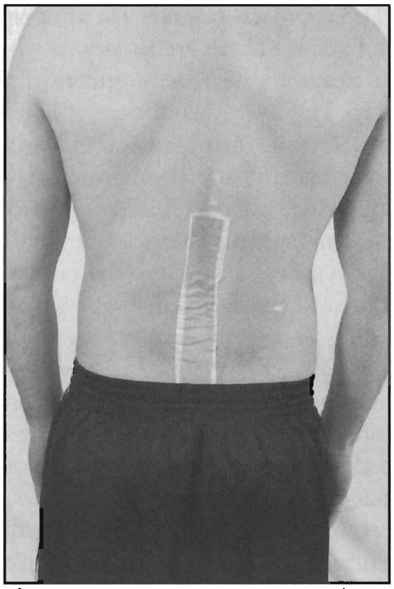


Figure 20-4. Taping to maintain lumbar lordosis.

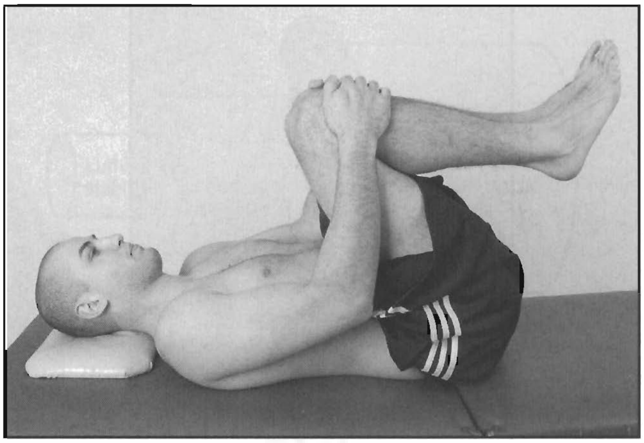


Figure 20-5. Lumbar flexion with both knees to chest.

associated with slump sitting (eg, forward head, rounded shoulders). To maintain the lordosis, a beach towel around the waist at night is recommended. The author has also had good success with taping. Leukotape P (over Biersdorf tape) is applied over the SPs, vertically spanning the thoracolumbar and lumbosacral junctions with the patient in standing position while maintaining a neutral lordosis (Figure 20-4). The tape pulls on the patient's skin each time he or she flexes the lumbar spine and acts as a reminder to maintain the position of optimal healing.

Once the derangement has been reduced and properly stabilized, the final goals are to recover lost function and prevent recurrence. For derangements 1 and 2, the recovery of lost function involves the use of lumbar flexion (ie, knees to chest); for derangements 3 through 6, it involves the use of combined flexion and contralateral side bending; and for derangement 7, the use of extension. The derangement must be healed and stable prior to the use of forces that stretch the tightened area. It is wise to follow all flexion stretches with a prophylactic set of 10 prone press-ups to ensure that the discal tissue remains in proper alignment.

Regarding the prevention of recurrence of the derangement, the patient must be aware of proper body mechanics (ie, the 5 Ls of lifting), ergonomic factors at home and in the workplace, the effect of emotional stress and tension, etc. Any persistent musculoskeletal impairments or imbalances must be addressed by the therapist (eg, impaired neurodynamic function, tight postural muscles, weak phasic muscles, poor postural alignment). If chronic pain (ie, symptoms lasting longer than 3 months) becomes an issue, a referral to a chronic pain clinic may be necessary.

As mentioned in the cervical section (Section III), there are times when disc derangements are not amenable to conservative measures. Spine surgeons are appreciative of patient referrals in whom nonsurgical interventions have been exhausted. This removes one of the criteria in their consideration of surgery as the next option. The remaining 3 criteria include intractable pain, neurologic signs of nerve root compression, and diagnostic confirmation of disc herniation with imaging. Some surgeons may add 1 or 2 additional criteria, but these 4 form the basis of whether to operate in most cases. The decision regarding the type of surgical intervention most appropriate for the patient lies within the realm of spinal surgery. A good working relationship between the surgeon and the therapist helps to reduce the incidence of unnecessary surgery but ensures that surgery is performed when indicated. It also increases the likelihood of appropriate postoperative rehabilitation, which is often overlooked.

Self-Mobilization of the Lumbar Spine

The indications for flexion exercises include lumbar flexion dysfunction, healed posterior derangement, and anterior derangement (derangement 7). Although the supine "both knees to chest" exercise (Figure 20-5) is used for all 3 conditions, the manner in which the exercise is performed is dependent upon the type of underlying impairment. The self-mobilization of lumbar flexion in the presence of McKenzie's flexion dysfunction syndrome (see Chapter 2) can be performed as with other stretches (ie, 5- to 10-second stretch to begin, working up to a 30-second stretch, repeated 3 times every 2 hours if possible). However, in the case of a status post-healed posterior derangement, the knees-to-chest maneuver should be performed gingerly in an on/off manner. If it is introduced following a recently healed posterior derangement, it may be wise to begin with the "single

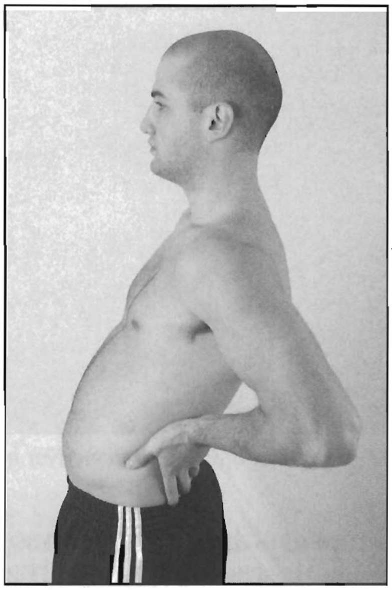


Figure 20-6. Standing lumbar extension.

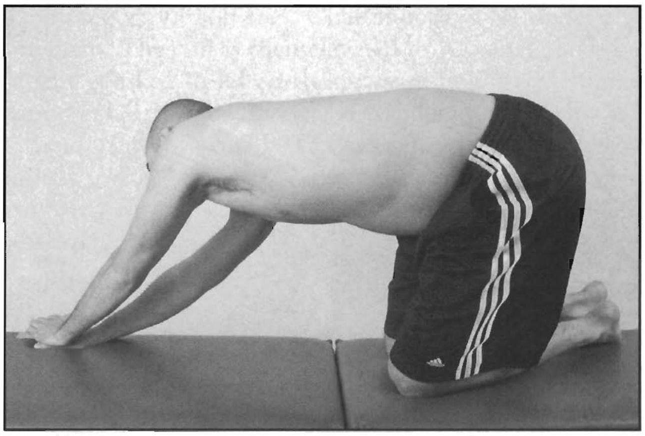


Figure 20-7. Quadruped lumbar side-bending stretch.

knee to chest" maneuver until it can be demonstrated that lumbar flexion is tolerated by the patient. Regardless of the reason for performing flexion exercises, the patient should always end each session with prophylactic prone press-ups to guard against the possibility of developing a posterior derangement.

The indications for extension exercises include lumbar extension dysfunction, healed anterior derangement, prophylactic extension to follow all flexion exercises, and, as discussed previously, posterior derangements 1 through 6. As with management of posterior derangements, prone press-ups are the method of choice. However, standing extension (Figure 20-6) is extremely useful because it is so easily performed in comparison to lying extension, which requires a carpeted floor, mat, or table.

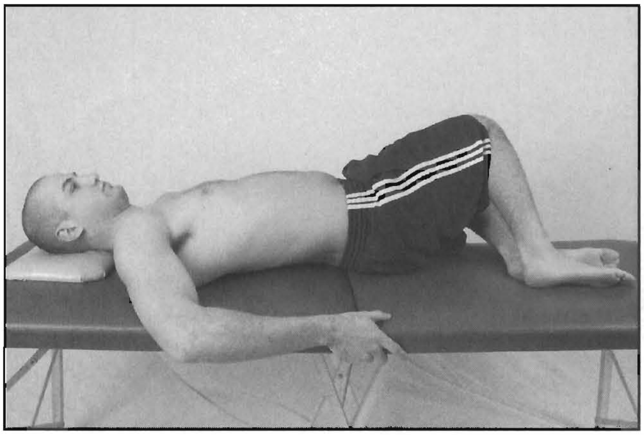
It is imperative that patients be given instruction in the proper performance of extension exercises. For prone press-ups, the arms and not the back extensors should perform the movement. The spine should be sequentially extended from the thoracic region down to the lumbosacral junction with the hips on the table at all times. The hands should be positioned so that the elbows are able to fully extend; as extension range improves, the patient's hands should be moved closer to the trunk. As with the management of cervical derangements, at least 3 sets of 10 repetitions are recommended every 2 hours. Standing extension should be performed with the hands on the hips with the thumbs forward. This should be done 10 times whenever rising from the sitting position. The patient must not "cheat" by extending at the hips!

Lumbar side bending is achieved in quadruped as shown for the thoracic spine in Chapter 7. It has utility for stretching the QL and sacrospinalis muscles unilaterally, as well as self-mobilizing the lumbar spine in the presence of side-bending restriction. In addition, it is carefully used to recover lost function following a healed posterolateral disc derangement.

The patient is instructed to place his or her hands and feet away from the side of the stretch without tilting the shoulders or hips. By simply leaning into the convexity, the desired stretch is achieved (Figure 20-7). The patient must stop at the point of the initial stretch and, if possible, hold for 30 seconds. If this is uncomfortable for the patient, he or she should begin with a 5- to 10-second hold and escalate upward from there. As range improves, the hands and feet can be moved further apart to enhance the efficacy of the stretch. To mitigate any untoward effects of the stretch, the patient should perform 10 prophylactic prone press-ups before standing. If at any time symptoms peripheralize, the stretch should be stopped immediately.

The final lumbar spine self-mobilization is rotation. It can be argued that lumbar rotation should be omitted from the list of therapeutic exercises for 2 reasons. First, there is minimal rotation in the lumbar spine because of the sagittal orientation of the apophyseal joints, and second, unstable disc derangements may respond poorly to rotation, which places added mechanical stress on disc structures. Nevertheless, rotation is a physiologic movement of the lumbar spine that can be limited in states of impairment, which should be enhanced when possible. However, because of the shear forces placed on the disc during rotation, it should be avoided in the presence of acute derangements.

Unlike quadruped rotation in the thoracic spine, which occurs from top to bottom, lumbar rotation is performed in the hook-lying position (Figure 20-8) and occurs from below upward. With practice, the patient can be trained to move segmentally upward from L5 to L1 on either side (ie, when moving the bent knees to the left, the motion involves L5 rotation to the left under L4, followed by L4 under L3, and L3 under L2, etc). The benefit of this type of movement is not only to improve the quantity of motion but the quality as well. This self-mobilization/stretch should be monitored for possible peripheralization and followed by prophylactic prone press-ups.



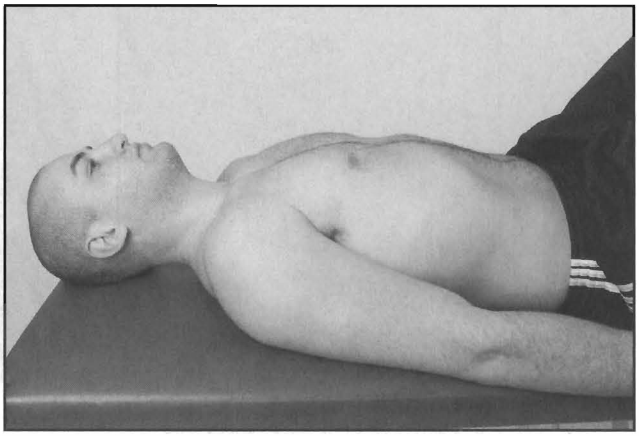


Figure 20-9. Abdominal drawing-in maneuver in hook lying.

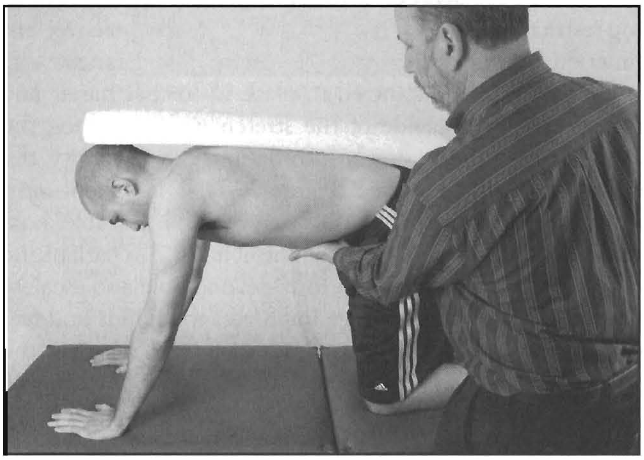


Figure 20-10. Drawing-in maneuver in quadruped with neutral spine.

Core Stability

The use of the core in this context refers to the lower trunk and encompasses the lumbar-pelvic-hip region. As way of introduction, Bruce Lee is quoted as saying, "My strength comes from my abdomen. It's the center of gravity and the source of real power."[62] Regarding the difference between strength and stability, Gray Cook[62] states, "Strength is the ability to produce force, whereas stability is the act of controlling force." He goes on to say that, "Core training will lay the foundation for strength, power, speed, and agility training. The core is the 'powerhouse' of the body." According to William Hanney, physical therapist, most movements of the locomotor system are initiated from or translate through the core.[57] Our discussion of core stability will commence with proper transversus abdominis activation, one of the essential muscles of the core system.

There are 2 ways of effectively training patients to achieve an isolated contraction of the transversus abdominis muscle.[58] The first method involves placing the patient in the hook-lying position with the index and middle fingers placed over the anterior superior iliac spines (ASISs). The patient is asked to draw the navel in toward the spine without moving the pelvis (Figure 20-9). If the patient performs a PPT, then the global abdominal muscles are substituting for the local system. The patient may also inhale as an incorrect means of drawing the abdomen inward. Consequently, the drawing in of the navel must be performed without lumbopelvic motion and during exhalation. It is the motor control of an isolated transversus abdominis contraction that is crucial in obtaining core stability. Otherwise, the torque-producing superficial muscles (ie, the external obliques, the rectus abdominis, and all but the posterior fibers of the internal obliques, which insert into the lateral raphe in most people) will dominate and inhibit one's ability to isolate the deeper core system.

The second way of achieving an isolated contraction of the transversus abdominis muscle is with the patient in the quadruped position. The therapist instructs the patient to relax the abdominal wall into the therapist's hand. The patient is then advised to lift the abdominal wall off the therapist's hand while exhaling through pursed lips. Again, the patient's trunk should be motionless throughout, indicating an isolated contraction of the transversus abdominis without global muscle substitution (Figure 20-10).

Once the patient has mastered the art of isolating the transversus abdominis, he or she is ready to activate the pelvic floor muscles (ie, the levator ani consisting of the puborectalis, pubococcygeus, levator prostatae or pubovaginalis, iliococcygeus muscles, and the coccygeus muscle, posteriorly). There is thought to be a synergistic relationship between the transversus abdominis and the pelvic floor muscles, especially the pubococcygeus muscle (located in the urogenital triangle). Patients with stress incontinence have reported improvement following training of the transversus abdominis, while patients with low back pain have reported improvement with the use of pelvic floor exercises.[58] It is also believed that the core stabilizing function of the transversus abdominis is contingent upon the simultaneous contraction of the pelvic floor, respiratory diaphragm, and multifidus (Lisa Morrone's "symphony of

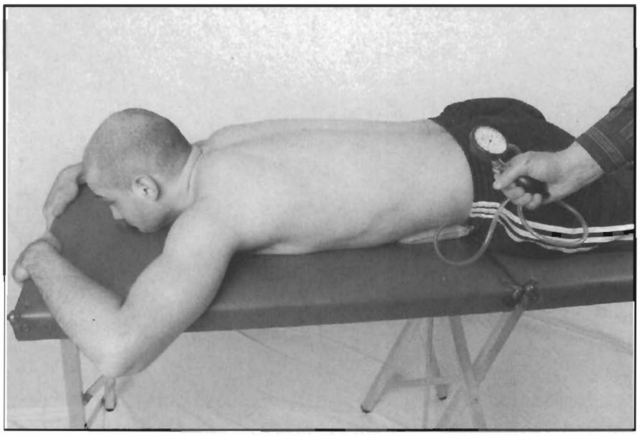


Figure 20-11. Drawing-in maneuver with Stabilizer.

stability" concept[69]). In this way, a transversus abdominis contraction is able to generate an increase in IAP.

To train the pelvic floor, males are instructed to "pull your scrotum upward" (pretending to stop the flow of urine is just as effective), while females are instructed to "pretend stopping the flow of urine" or "imagine placing firm pressure around a tampon" (eg, Kegel exercises). The Kegelmaster (Kegelmaster, Inc, Ocklawaha, FL) is an effective modality for pubococcygeus training as it provides a means of resistance. Pelvic floor training can be performed in isolation or in conjunction with a contraction of the transversus abdominis. In either case, the contraction should be slow, gentle, and of low effort. Hanney[57] suggests that patients pretend, "Your pelvic floor is an elevator; contract slightly to the first floor, a little harder for the second floor, and even harder to get to the third floor." Because of the synergistic relationship of the core musculature, pelvic floor activation assists in the facilitation of the multifidus as well as the transversus abdominis; therefore, it should be recruited first. As important as contraction of the pelvic floor is for core stability, the relaxation of these same muscles is equally as important. Tonic holding, especially of the coccygeus muscle (located in the anal triangle), often results in "crampy" pain in the groin or tail bone region. In addition, tonic holding will weaken the urogenital diaphragm and may result in stress incontinence during coughing, sneezing, and laughing.

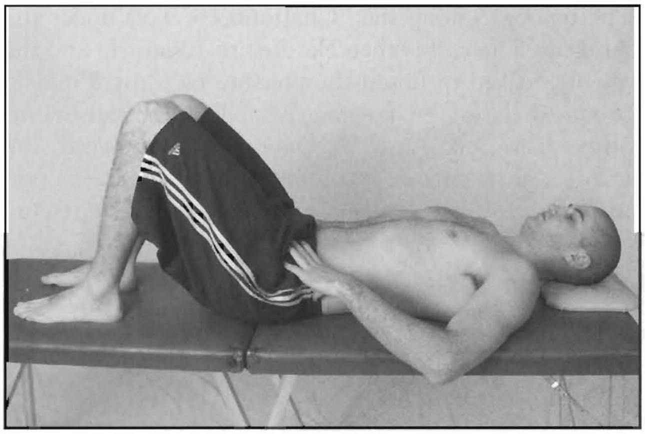
Once the motor control aspect of core muscle activation is achieved, the patient is then instructed in functional neutral/lower abdominal exercises. However, throughout the performance of these exercises, the patient must maintain a low-level, tonic contraction of the local system. The most common mistake made in abdominal training is neglecting to recruit the core muscles first. If this core recruitment does not occur, the patient is only training the global torque producers and does not attain optimal improvement in clinical lumbar stability.

A quantitative way of training the transversus abdominis and other core components is by placing the patient prone with the Stabilizer pressure biofeedback device (Chattanooga Group Inc., Chattanooga, TN) under the umbilicus. The cuff is then elevated to 70 mmHg and the patient is asked to lessen the pressure by 6 to 10 mmHg during exhalation by drawing the abdominal wall inward without performing a PPT. Once this is achieved, the patient is instructed to maintain this pressure reduction for 10 to 30 seconds while breathing normally (Figure 20-11).

Functional Neutral/Lower Abdominal Training

The interest in spinal stabilization therapy peaked in the early 1990s with a wave of enthusiasm that spread quickly from the west to the east coast. A new lexicon of words, including *neutral*, *functional range*, *abdominal bracing*, *lower abdominals*, *instability*, *spinal stabilization*, etc became the jargon of the times; new forms of exercise involving Swiss balls, foam rollers, rocker boards, and sophisticated medical exercise equipment suddenly appeared in clinics across the country and around the globe. Some of the pioneers involved with this form of therapy include Biondi, Drinkwater-Kolk, Johnson, Saliba-Johnson, Parker, Morgan,[70] Moore, Christensen, Irion, Liebenson, Posner-Mayer,[71] Paris, Sahrmann,[72] Holten, Grimsby, Rogers, Svendsen, Janda, Saunders,[73] Bookhout,[54] McGill,[74] Ellis, Sarver, etc. The basic principles, however, can be traced back to the work of the Kendalls, the Bobaths, Knott, Voss, Pilates, Daniels, Worthingham, and others, to mention a few.

Training low back patients in this way begins by identifying the functional neutral range or what Panjabi[14-16] refers to as the neutral zone. This is the optimal position or range of position within which the lumbar spine is stable, the least symptomatic, and within which it functions the most efficiently. To borrow a term from Kaltenborn,[52] it is the *loose-packed* or *resting position* of the lumbar region. The author also uses the terms *osteocentric* and *somatocentric* when describing the neutral position, zone, or range. To find the neutral position, the hook-lying patient is instructed to explore the extremes of an anterior pelvic tilt (ie, hyperlordosis) and posterior pelvic tilt (ie, lumbar kyphosis). The neutral position is approximately half-way between the 2 extremes of sagittal motion where the patient experiences maximal ease or comfort. It is what osteopathic physicians refer to as dynamic or functional neutral. Some patients prefer a slight flexion bias, while others incorporate a slight bias toward extension in their neutral position. The basic philosophy of functional neutral/lower abdominal training is that patients can be made more stable and less symptomatic if they learn to function in the neutral range (sitting, standing, recumbent lying, etc.) where their tissues are under less mechanical stress and strain. This is consistent with Panjabi's concept of training the active and neural



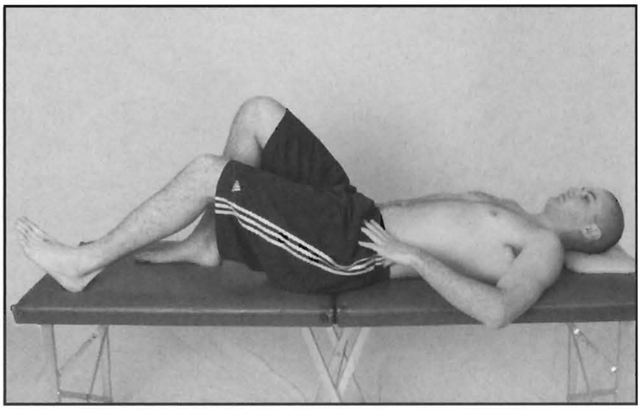


Figure 20-13. Exercise 1: heel slides.

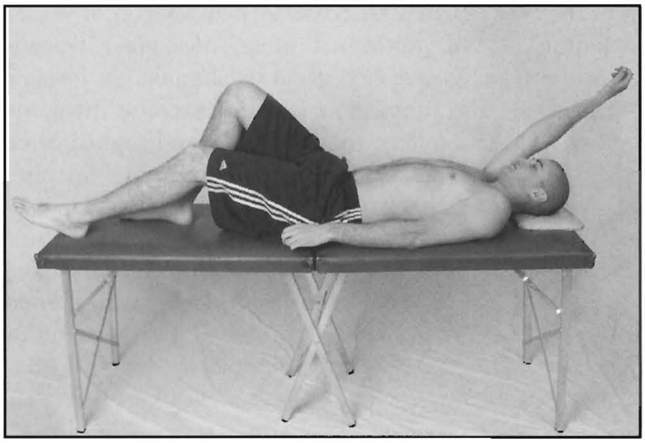


Figure 20-14. Exercise 2: heel slide with opposite arm elevation.

control subsystems to enhance neutral zone function, while discouraging movements into the hypermobile and symptomatic elastic zone (ie, border positions) where the passive subsystem controls motion.

In retrospect, one shortcoming of the spinal stabilization revolution was the lack of recognition of the deep local system of core stability. By failing to first activate the core system, the torque-producing global system was strengthened instead. With the discovery of the role of the deep local muscle system in the late 1990s came the realization that the approach to functional neutral training needed modification. Consequently, the exercises covered in this text will integrate our newer understanding of core stability (Australian approach) into many of the traditional spinal stabilization exercises in which abdominal bracing, advocated by Grenier and McGill,[66] are utilized. In this way, patients will benefit from local as well as global trunk stabilization training. In Chapter 25, closed-chain stabilization exercises, using the PostureJac, will be illustrated and described, emphasizing the importance of one-joint muscle recruitment.[58]

In the specific exercises to follow, the patient is progressed from stable to unstable positions and from less to more difficult exercise procedures requiring increased levels of strength, endurance, and motor control.

Exercise 1: Heel Slides

1. The patient is placed on a mat or table in the hook-lying position with the index/middle fingers on the ASIS, bilaterally.
2. The patient finds his or her neutral lumbopelvic position (Figure 20-12).
3. The deep local muscle system is activated by contracting the pelvic floor muscles (ie, "scrotum pulled upward" for males and "stop the flow of urine" or "squeeze the tampon" for females) first, followed by drawing in the abdominal wall (ie, navel to spine on exhalation without performing a PPT).
4. Once the core muscles are set, the patient is now instructed to slide the left heel along the table in order to straighten the left knee (Figure 20-13). This is done during exhalation, counting backward slowly from 5 to 1, while maintaining both the neutral position and the activation of the core muscles. Once the leg is straight, the patient can relax completely and repeat this sequence 10 times. The entire procedure is then performed on the right side. In order to progress the patient to the next level of difficulty, he or she must be able to maintain a decent core contraction (ie, abdominal drawing in) and not allow the lumbar spine to hyperextend as the legs are lowered.

Exercise 2: Heel Slide With Opposite Arm Elevation

1. Steps 1 to 3 are repeated as in exercise 1.
2. While maintaining a core contraction in the neutral position, the patient performs the heel slide but now simultaneously raises the opposite arm overhead to a slow count of 5 to 1 on exhalation (Figure 20-14).

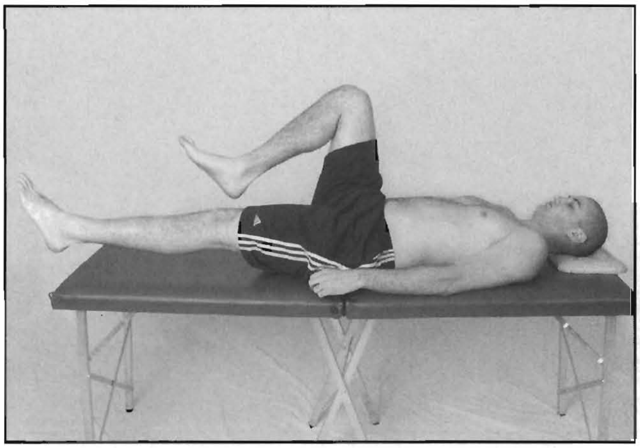


Figure 20-15. Exercise 3: unilateral leg lowering.

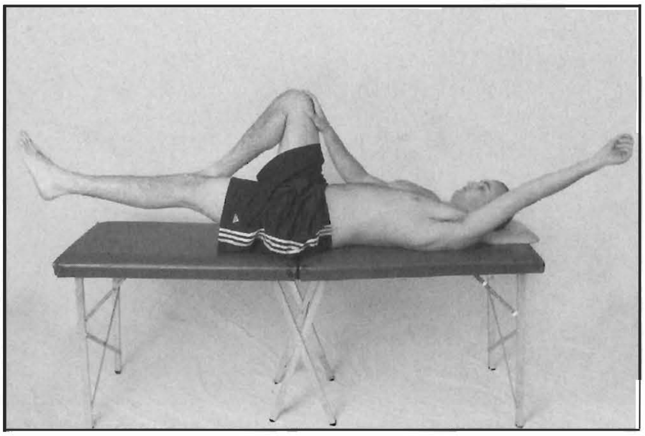


Figure 20-16. Exercise 4: hand to ipsilateral knee.

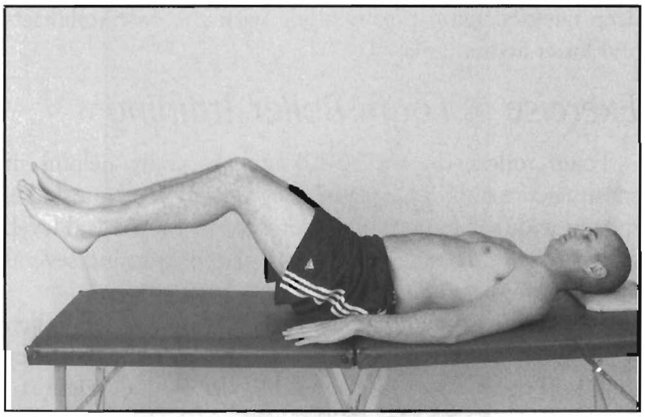


Figure 20-17. Exercise 5: bilateral leg lowering.

3. This sequence is repeated 10 times with the left heel slide/right arm combination and 10 times with the right heel slide/left arm combination. As with exercise 1, the patient must be able to maintain a decent contraction of the transversus abdominis and not allow the lumbar spine to hyperextend as the legs are lowered before moving on to exercise 3.

Exercise 3: Unilateral Leg Lowering

1. The patient is now progressed to raising both feet off the table in supine.
2. Following core muscle setting in a neutral position, the patient lowers one leg at a time without touching either foot to the table or mat (Figure 20-15).
3. Ten repetitions per side is performed to a slow count of 5 to 1 on exhalation.
4. The leg must not be lowered beyond the point at which the pelvis anteriorly tilts, the lumbar spine hyperextends, and the local core muscles fail to maintain drawing in of the abdominal wall.

Exercise 4: Hand to Ipsilateral Knee

1. The patient again begins by raising both feet off the table in supine as in exercise 3 with bilateral arm support.
2. Once the core muscles are set in the neutral lumbopelvic position, the patient brings one hand to the ipsilateral knee while the opposite arm and leg move away from each other (Figure 20-16). This maneuver is then repeated in an alternating fashion on the contralateral side for a total of 10 repetitions on each side.
3. It is essential that a neutral core contraction be maintained at all times while the patient inhales and exhales normally.

Exercise 5: Bilateral Leg Lowering

1. The supine patient begins by raising both feet off the table.
2. Once the pelvic floor and other core muscles are set by drawing the abdominal wall inward in the neutral lumbopelvic position, the patient proceeds by lowering both legs simultaneously (Figure 20-17).
3. The objective of this more challenging exercise is to maintain core stability as the weight of the descending lower limbs are inducing an anterior pelvic tilt/lumbar spine hyperextension. It is the lower abdominals (primarily the external oblique muscles and rectus abdominis) that work with the deeper local muscle system to prevent this from occurring.
4. The patient must be proficient with exercises 1 through 4 before attempting this more challenging maneuver; the degree of leg lowering must be commensurate with the patient's ability to maintain a neutral lumbopelvic position.
5. Ten repetitions are performed to a slow count of 5 to 1 on exhalation.

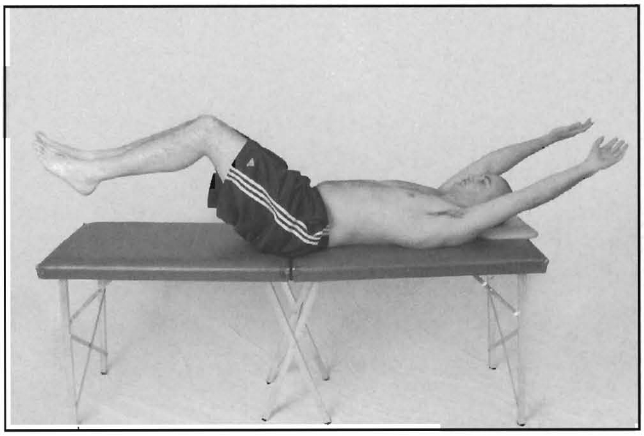


Figure 20-18. Exercise 6: bilateral leg lowering with bilateral arm elevation.

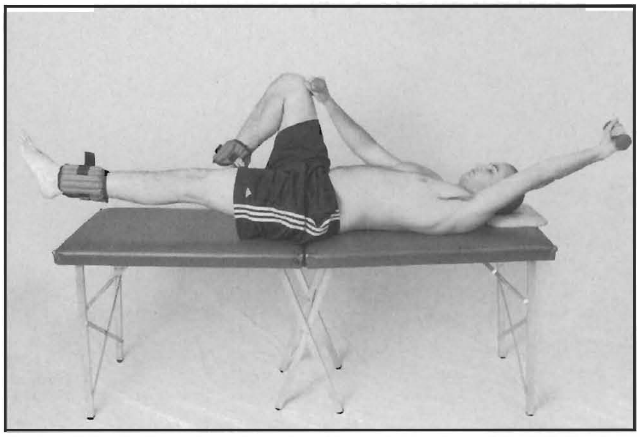


Figure 20-19. Exercises 1 to 6 with weights for added difficulty.

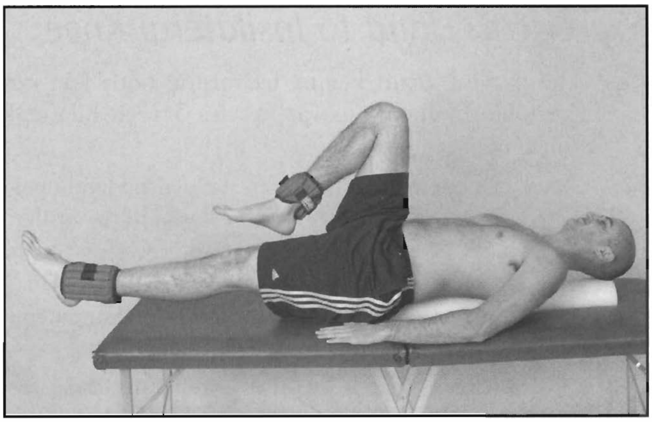


Figure 20-20. Foam roller training with ankle weights.

Exercise 6: Bilateral Leg Lowering With Bilateral Arm Elevation

This is the most advanced of the exercises thus far. For those who master exercise 5, this next maneuver is attempted.

1. As the legs are lowered, the arms are simultaneously elevated on exhalation (Figure 20-18). The patient must not proceed to the point where the pelvis begins to anteriorly rotate and the lumbar spine hyperextend.
2. The core system must also be able to maintain the drawing-in of the abdominal wall at all times.
3. Each of the 10 repetitions should be performed to a 5 count; the degree of leg lowering/arm elevation is based upon the patient's mastery in the early ranges of motion.

At any point in the performance of exercises 1 through 6, ankle weights or dumbbells can be added to enhance muscular effort (Figure 20-19). In order to maintain cervical spine stability, a chin-tuck is performed with the occiput either making contact with the mat/table, or elevated less than 1 inch off the surface for maximum recruitment of the deep neck/occipital flexors along with the core stabilizers and lower abdominals.

Exercise 7: Foam Roller Training

Foam rollers (Figure 20-20) are especially helpful in training core stability. Because they are inherently unstable, they provide sensorimotor challenges on a subcortical level, which is an efficient and effective way of training several muscle groups simultaneously.

Exercises 1, 2, 3, and 5 are well-suited to the foam roller, whereas exercises 4 and 6 are not because of the tendency to fall off the roller without arm support. Ankle weights or dumbbells can be added at the appropriate time. In addition, there are many more foam roller applications for a variety of patient conditions.[75]

Exercise 8: Quadruped Training

Patients with lumbar hypermobility/instability must be taught to maintain a stable, neutral core while involved with limb movements that threaten to undermine their spinal stability. Quadruped training enhances the concept of "distal mobility on proximal stability," which is hopefully carried over into a patient's activities of daily living.

1. In the quadruped position, the patient "sets" the core system in the neutral lumbopelvic region (Figure 20-21).
2. The patient starts by raising all 4 limbs, one at a time, while maintaining a neutral and stable core throughout.
3. To progress the patient, diagonal raises are performed such that the right arm and left leg are raised simultaneously, followed by the left arm and right leg (Figure 20-22). Care must be taken not to permit lumbar hyperextension during the raises (a dowel is used to assist with maintaining a neutral spine). This exercise, like the others, is more about motor control than the generation of brute force. There are many low back patients with "great looking abs." The key is not the appearance, but the functionality!

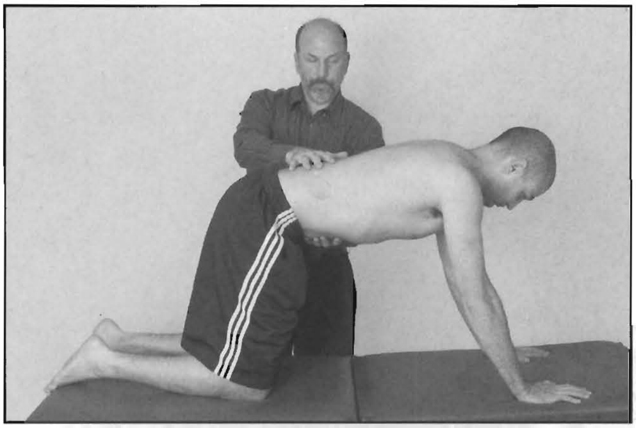


Figure 20-21. Abdominal drawing-in with neutral spine in quadruped.

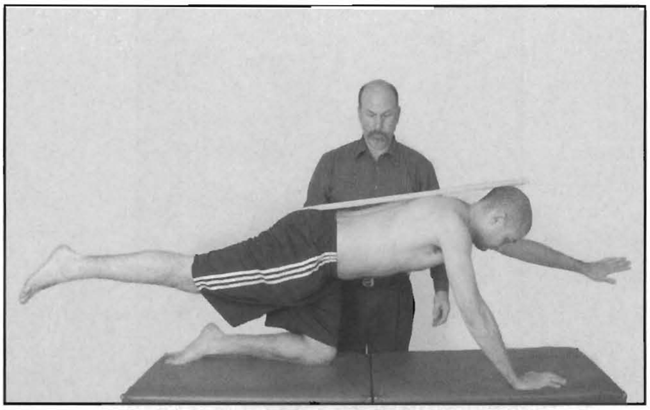


Figure 20-22. Functional neutral quadruped training with dowei.

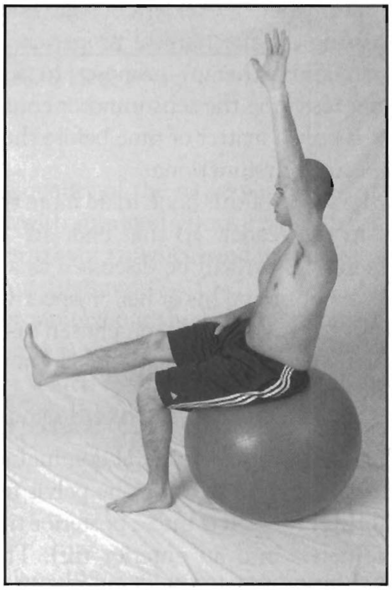


Figure 20-23. Stabilization training on the Swiss ball.

4. As with the other lower abdominal exercises, ankle weights and the use of dumbbells can be added when the patient has mastered the maneuver without weights.

Exercise 9: Sitting Swiss Ball Training

The "Swiss ball" (Figure 20-23) originated in 1963, when an Italian manufacturer started manufacturing toys made of vinyl instead of rubber.[71] Some of the people responsible for the use of the Swiss ball in physical therapy clinics today include Kong, Quinton, Bobath, Klein-Vogelbach, Kucera, Carriere, Hanson, Schorn, Posner-Mayer, Corning-Creager, Irion, Christensen, Morgan, Johnson, Saliba-Johnson, Biondi, and Rocabado. The Swiss ball, also known as the Physio, Gymnic, Yoga, Opti, or Gym ball, is useful in promoting proper movement patterns using key muscle groups. The outcome involves safe and pain-free functional movement, which translates into reduced pain, functional limitation, and disability in one of our most challenging patient populations, namely chronic LBP sufferers.

The patient sits on the ball with knees and hips flexed to 90 degrees and the feet placed flat on the floor. To begin, the patient rocks back and forth into an anterior and posterior pelvic tilt. Once the neutral position is discovered, the core muscles are set as usual by activating the pelvic floor and drawing the abdominal wall inward on exhalation without pelvic motion. From here patients can perform such exercises as the basic bounce, the leg march, the kick out, march-arm and leg, etc, with or without ankle weights and dumbbells, while maintaining core muscle stability in the neutral lumbopelvic position. The reader is referred to Posner-Mayer's book[71] for a complete description of Swiss ball options with emphasis on mobility, strength, cardiovascular training, sensory perceptual retraining, balance, postural relearning, as well as injury prevention and fitness.

Exercise 10: Standing Wall Slides

The final exercise in our series of functional neutral/lower abdominal exercises involves the wall slide. There are many variations of this exercise, but all claim to assist with lumbar stabilization and the dissociation of the hips from lumbar motion.

1. The patient stands with his or her back to the wall.
2. With a moderate bend of the knees (approximately 45 degrees), the patient sets the core muscles in the neutral lumbopelvic position.
3. A chin-tuck is then performed to stabilize the cervical region and lengthen the spine.
4. The patient then straightens his or her knees while maintaining neutral core and cervical stability (ie, chin-tuck) to a slow count of 5 to 1.
5. Once the patient has returned to normal stance, the core contraction and chin-tuck can be released.
6. This sequence is repeated 10 times.

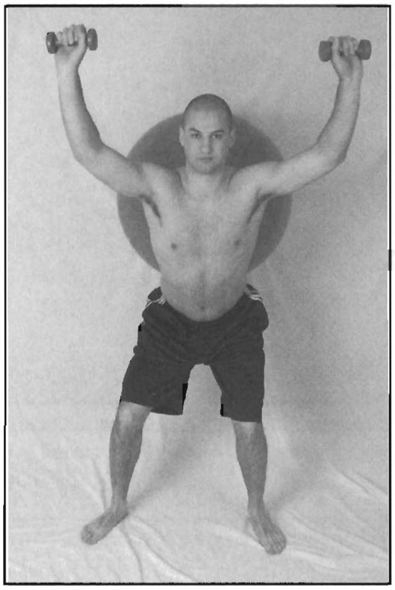


Figure 20-24. Wall slide exercises with the Swiss ball.

Variations of the wall slide include unilateral or bilateral arm elevation during the knee-bending phase as well as maintenance of the core contraction and chin-tuck in both the up and down directions. The Swiss ball can be placed between the patient and the wall to facilitate the up and down movement of the spine; dumbbells can be used for added difficulty (Figure 20-24).

Advanced methods of neutral core stabilization (not covered in this text) include dynamic core control, which challenges the core during dynamic motions of the core (eg, seated chop and lift, side support exercise on the Swiss ball), and reactive core control, which challenges the core in reaction to unexpected environmental influences (eg, shadow boxing, shadow mime, standing ball toss).[11,57,76]

Sensorimotor Training (Feldenkrais)

The Feldenkrais method[77] is based upon the work of Moshe Feldenkrais (1904 to 1984), an Israeli physicist who devoted his career to the relationship between human movement, conscious thought, and sensorimotor learning. His findings led to the discovery of a new method of neuromuscular re-education that has had profound implications in the rehabilitation of patients with movement disorders. The Feldenkrais sensorimotor learning system is based upon the sciences of biomechanics, neurophysiology, stress reduction, and accelerated learning. When applied to patients with orthopedic impairments, its purpose is to reduce or eliminate painful symptoms in the musculoskeletal system as a consequence of the rediscovery of the ease and flexibility of movement. In computer terminology, manual therapy is to the "hardware" what the Feldenkrais method is to the "software." Restoring the mechanical properties of human motion is what manual therapy proposes to accomplish. However, without restoring the sensorimotor control aspect of movement, it is only a matter of time before the mechanics once again become dysfunctional.

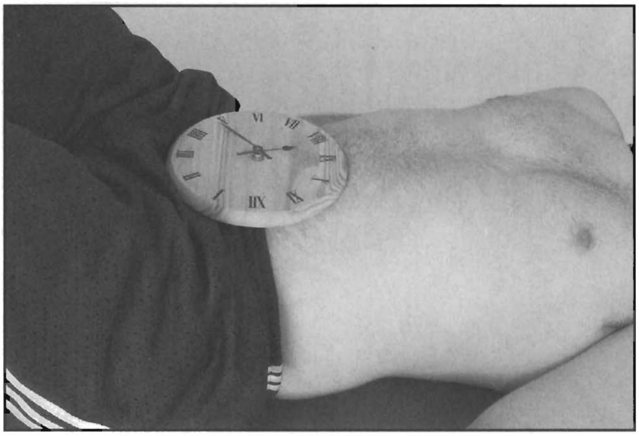


Figure 20-25. Pelvic clock exercise in hook lying.

It is beyond the scope of this book to do more than introduce the topic to the reader. To that end, an "awareness through movement" lesson will be discussed as a means of introducing the therapist and his or her prospective patients to the Feldenkrais method. The lesson chosen here because of its great utility with low back patients is known as the "pelvic clock."

The hook-lying patient (Figure 20-25) is asked to imagine a large "clock" placed over the lower abdominal region. To begin, the patient is advised to move the pelvis from 12:00 to 6:00 (12:00 brings the pelvis into a posterior tilt whereas 6:00 brings the pelvis into an anterior tilt). The patient then proceeds in diagonal patterns of movement from 1:00 to 7:00, 2:00 to 8:00, 4:00 to 10:00, and 5:00 to 11:00. The patient also explores the horizontal 3:00-to-9:00 movement as well. Other options for gaining greater sensorimotor control of lumbopelvic movement include moving around the "clock" in a clockwise as well as counterclockwise fashion. When Feldenkrais practitioners are teaching new movements to a student, they often place their hands on the body to provide a manual assist with the acquisition of a new motor skill. This is referred to as functional integration.

Once the patient becomes more adept with these movements in hook lying, the patient can then integrate them into a variety of other positions, including supine, prone, quadruped, kneeling, half-kneeling, standing, etc. To further enhance new motor skill acquisition,[57] the patient is encouraged to perform the pelvic clock with slow versus fast motions, on stable versus unstable surfaces, with the eyes open versus eyes closed, under cognitive distraction versus no cognitive distraction, and with perturbations versus no perturbations.

Some of the foundational principles that are essential to a successful Feldenkrais experience include paying atten-

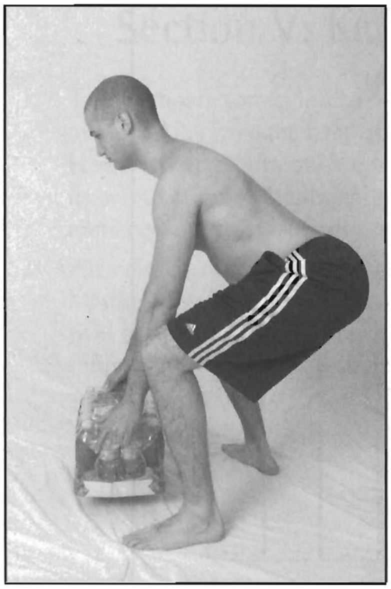


Figure 20-26. 5 Ls of lifting.

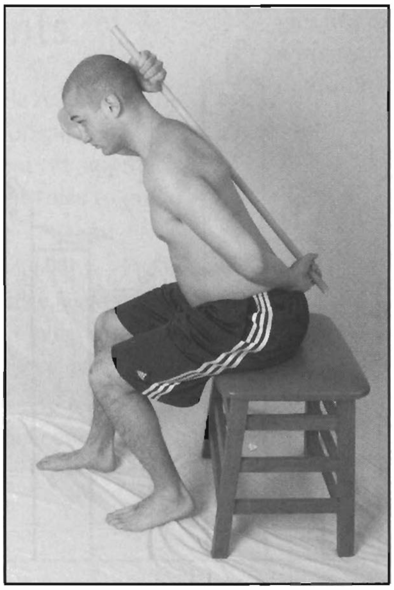


Figure 20-27a. Sit to stand with a dowel: stage 1.

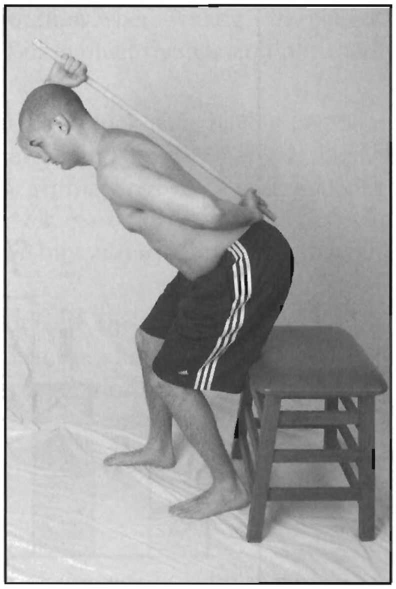


Figure 20-27b. Sit to stand with a dowel: stage 2.

tion to the quality of the movement, doing the movements slowly and with minimal effort, resting frequently between movements to avoid physical and mental fatigue, and avoiding pain and discomfort.

To learn more about the Feldenkrais method, the reader is encouraged to contact the Feldenkrais Guild of North America or search for additional publications, course information, etc, on the Internet.

5 Ls of Lifting

In the late 1980s, physical therapists at the Southside Health Institute in Bay Shore, NY, developed a useful education tool for the instruction of proper lumbar spine lifting mechanics. What came to be known as the 5 Ls of Lifting technique (Figure 20-26) was ultimately published in the *Physical Therapy Forum*.[78] However, because this publication was discontinued a few years later, the information was no longer available. Consequently, it is now being made available a second time with a few minor revisions. In addition to lifting, this technique can be adapted for bending, pushing/pulling loads, etc.

To serve as a memory jogger, each of the 5 instructions begins with the letter L, representing the 5 lumbar vertebrae as follows:

- L1: Load
- L2: Lever
- L3: Legs
- L4: Lordosis
- L5: Lungs

A brief explanation of the 5 Ls will serve to educate the patient in the theory behind the technique.

Patients should always check the **load** prior to lifting in the event that additional help or the use of a mechanical device is indicated. He or she may also decide not to attempt such a lift depending on the load involved.

The **lever** arm should always be kept as short as possible. Because Torque = Force x Lever Arm, the one aspect of the equation that is controllable is the distance from the object being lifted to the patient's center of rotation (ie, the torque or lever arm). Therefore, the patient should always get as close as possible to the item being lifted. It is also important to realize that a patient's torso has its own weight. Consequently, the mere act of bending can be potentially stressful to the lower back.

The next instruction relates to the use of the **legs** rather than the use of the back muscles. This is perhaps the most crucial component of a correct lift. Good lifting technique is contingent upon flexible and stable ankles, strong knee extensors, flexible hips, strong gluteal muscles, and good balance. Consequently, the patient is instructed to bend at the hips and knees and not at the waist. A lesson in "hip-hinging" with a stable trunk is often necessary for patients who are accustomed to bending at the waist. Patients should feel the load of the lift in their legs, not in their backs. The sit-to-stand transfer with a dowel helps to perfect the art of "hip-hinging" while maintaining a neutral spine (Figures 20-27a and 20-27b).

Regarding the lumbar **lordosis**, there is much controversy. Some advocate lumbar flexion, while others recommend functional neutral or hyperextension for lifting. Although the concept of a neutral spine makes the most sense, the author's experience is that patients mitigate their risk of injury by accentuating their lumbar lordosis. This position, if tolerated, loads the lumbar facets and "locks" the lumbar spine in its "close-packed" position. Consequently,

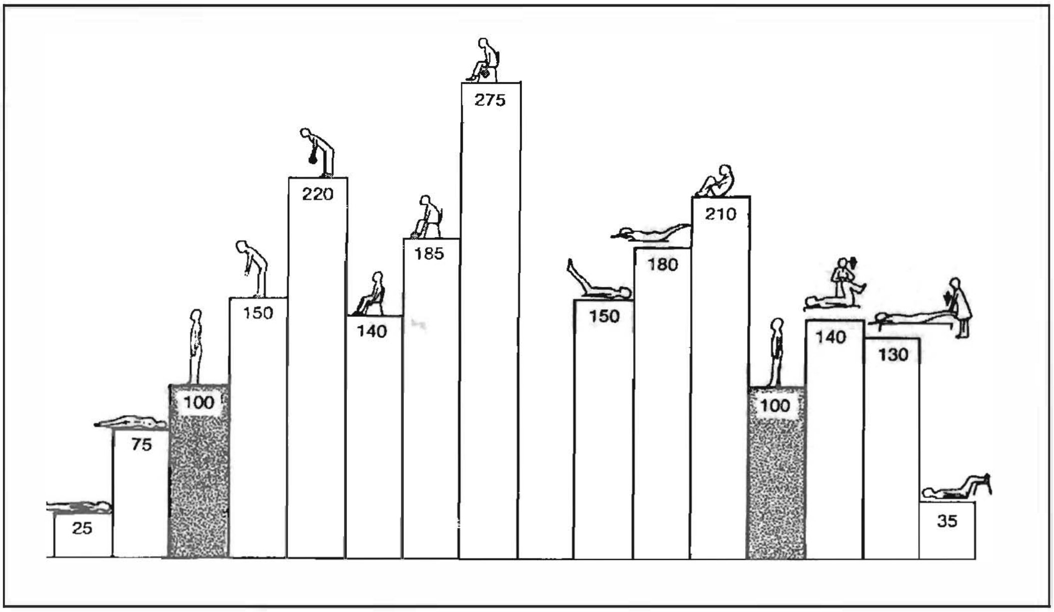


Figure 20-28. Lumbar intradiscal pressure in various positions and during exercise in kilograms per square centimeter. (Adapted from Nachemson.[8,68])

unless unable to do so, which is rarely the case, patients are instructed to "hollow" their lower back and to maintain this position throughout the lift.

The final instruction is a safeguard especially related to heavy loads or lifts to which the patient is unable to get close and thereby minimize the lever arm (eg, working over a car, lifting a patient out of bed). As observed with weight lifters, a deep inhalation followed by pursed lip exhalation increases IAP, which in turn stabilizes the trunk. Consequently, the final but very useful instruction in the 5 Ls of lifting technique involves the use of the **lungs** as a means of adding additional protection to the lumbar spine.

Though not one of the original 5 Ls, added protection to the low back can certainly be obtained by activating the core stabilizers for the duration of the lift. There is one final recommendation to consider regarding lifting. Patients should avoid twisting "like the plague." There's only one thing worse than lifting with the back forward bent at the waist, and that is to lift with the back forward bent and twisted at the waist. In addition to the 5 Ls of lifting technique, Morrone does an excellent job of illustrating and describing the right and wrong way of performing many activities of daily living (ADLs) for neck and back pain sufferers in her very useful self-help book.[69] With regard to proper sitting and standing postures, optimal body mechanics for bending, reaching, lifting, etc, patients benefit greatly by reviewing Nachemson and Morris' study[68] on lumbar spine intradiscal pressure (Figure 20-28). This helps to explain the correlation between poor static and dynamic lumbar spine alignment and discogenic symptoms.

Our discussion of the self-management of LBP would not be complete without addressing the role of fear-avoidance behaviors. Research has confirmed that psychosocial factors, such as fear-avoidance beliefs, serve as predictors of future chronic disability following an episode of acute LBP.[79,80] In this regard, Godges et al demonstrated that education and counseling regarding pain management, physical activity, and exercise can reduce the number of days off work in people with fear-avoidance beliefs and acute LBP.[81]

Conclusion

The approach to nonspecific LBP taken in this text tends toward a mechanism-based classification system. However, this author sees definite value in the increasingly popular treatment-based model (see Chapters 3 and 8), which consists of subtypes of patients classified by specific signs and symptoms identified during the examination.[6,11] What is becoming increasingly clear is that the pathoanatomical classification system, which attempts to identify the nociceptive source of a patient's symptoms based on diagnostic imaging and injections, fails to provide manual therapists with clinically useful information.[6,8,11] This is based on several studies in which asymptomatic subjects have demonstrated a number of common pathologies of the lumbar spine with various imaging studies[40,41,82] (eg, MRI, CT). Based upon these new ways of looking at an old subject, manual therapists must be just as skilled in clinical reasoning (eg, lateral thinking, inductive or forward reasoning, self-reflection) as they are in any of the manipulative techniques used in the clinic today.[83]

Section V: Key Points

1. The pathoanatomic model is of little value in the manual therapy management of nonspecific LBP. However, a treatment-based classification approach in conjunction with clinical prediction rules to guide management yields superior outcomes.
2. An eclectic approach consisting of McKenzie, Paris/Maitland/osteopathic, and core stability yields positive clinical outcomes when dealing with patients suffering from acute and chronic low back pain.
3. The biopsychosocial approach, addressing fear avoidance behavior, and utilizing pain management strategies are excellent options when dealing with patients who are refractory to manual therapy and not candidates for spinal surgery.
4. Many patients with low back pain have a combination of clinical instability (Panjabi) of the lumbar spine and hip joint stiffness/tightness. Consequently, a useful approach for many low back patients is to stretch/mobilize the hips and stabilize/strengthen the lower trunk.
5. Thrust manipulation of the lumbar spine has the potential to undermine disc integrity (especially when incorporating rotation) and is therefore not recommended.

References

1. Back pain. National Institutes of Health. National Institute of Arthritis and Musculoskeletal and Skin Diseases. www.nlm.nih.gov/medlineplus/backpain.html. Accessed July 13, 2008.
2. Bhargava A, Gelb D, Ludwig S, DePalma MJ. Physical therapy for low back pain. *Curr Opin Orthop.* 2006;17:207-211.
3. Maniadakis N, Gray A. The economic burden of back pain in the UK. *Pain.* 2000;84:95-103.
4. Macnab I. *Backache.* Baltimore, MD: Williams & Wilkins; 1977.
5. Riddle DL. Classification and low back pain: a review of the literature and critical analysis of selected systems. *Phys Ther.* 1998;78
6. Pinto D, Cleland J, Palmer J, Eberhart SL. Management of low back pain: a case series illustrating the pragmatic combination of treatment- and mechanism-based classification systems. *J Manual Manipulative Ther.* 2007;15(2):111-122.
7. Jam B. *When the Back Goes Out...Where Does It Really Go?* Thornhill, Ontario: APTEI; 2006.
8. Nachemson AL. The lumbar spine, an orthopaedic challenge. *Spine.* 1976;1(1):59-71.
9. McKenzie R, May S. *The Lumbar Spine, Mechanical Diagnosis & Therapy. Volume 1.* Waikanae, New Zealand: Spinal Publications New Zealand Ltd; 2003.
10. Delitto A, Erhard RE, Bowling RW. A treatment-based classification approach to low back pain syndrome: identifying and staging patients for conservative treatment. *Phys Ther.* 1995;75:470-485.
11. Wilmarth MA, ed. *Current Concepts in Orthopaedic Physical Therapy.* La Crosse, WI: APTA; 2006.
12. Bogduk N, McGuirk B. *Management of Acute and Chronic Neck Pain: An Evidence-Based Approach.* Edinburgh: Elsevier; 2006.
13. Spitzer WO, LeBlanc FE, Dupuis M, et al, eds. Diagnosis of the problem. In: Scientific Approach to the Assessment and Management of Activity-related Spinal Disorders. A Monograph for Clinicians, Report of the Quebec Task Force on Spinal Disorders. *Spine* (European Edition, Suppl 1). 987;12(7S):S16-S21.
14. Panjabi M. The stabilizing system of the spine. Part I: function, dysfunction, adaptation and enhancement. *J Spinal Disord.* 1989;5:383-389.
15. Panjabi M. The stabilizing system of the spine. Part II: neutral zone and stability hypothesis. *J Spinal Disord.* 1992;5:393-397.
16. Panjabi MM. Clinical spinal instability and low back pain. *J Electromyography and Kinesiology.* 2003;13:371-379.
17. Prsala J, Belzer EG, Simpson C, Stalker RL. The ratio of the length of the thoracic-kyphotic curve to that of the lumbar-lordotic curve in relation to low-back pain. *The Nova Scotia Medical Journal.* 1993;185-87.
18. Prsala J. *Help Yourself to a Healthy Back.* 2nd ed. Halifax, Nova Scotia: Dalhousie University Printing Center; 2001.
19. Collaer JW, McKeough DM, Boissonnault WG. Lumbar isthmic spondylolisthesis detection with palpation: interrater reliability and concurrent criterion-related validity. *J Manual Manipulative Ther.* 2006;14(1):22-29.
20. Paris SV. Physical signs of instability. *Spine.* 1985;10(3):277-279.
21. McKenzie R, May S. *The Lumbar Spine, Mechanical Diagnosis & Therapy. Volume 2.* Waikanae, New Zealand: Spinal Publications New Zealand Ltd.; 2003
22. Paris SV. Anatomy as related to function and pain. *Orthop Clin North Am.* 1983;14(3):475-489.
23. Adams MA, May S, Freeman BJC, et al. Effects of backward bending on lumbar discs: relevance to physical therapy treatments for low back pain. *Spine.* 2000;25(4):431-437.
24. Kuslich SD, Ulstrom CL, Michael CJ. The tissue origin of low back pain and sciatica. *Orthop Clin North Am.* 1991;22(2):181-187.
25. Kramer J. *Intervertebral Disk Diseases: Causes, Diagnosis, Treatment, and Prophylaxis.* 2nd ed. New York, NY: Thieme Medical Publishers; 1990.
26. Beattie PF, Brooks WM, Rothstein JM, et al. Effect of lordosis on the position of the nucleus pulposus in supine subjects. *Spine.* 1994;19:2096-2102.

27. Fennell AJ, Jones AP, Hukins DWL. Migration of the nucleus pulposus within the intervertebral disc during flexion and extension of the spine. *Spine*. 1996;21:2753-2757.
28. Alexander LA, Hancock E, Agouris I, Smith FW, MacSween A. The response of the nucleus pulposus of the lumbar intervertebral discs to functionally loaded positions. *Spine*. 2007;32(14):1508-1512.
29. Vleeming A, Mooney V, Dorman T, Snijders C, Stoeckart R. *Movement, Stability & Low Back Pain: The Essential Role of the Pelvis*. New York, NY: Churchill Livingstone; 1997.
30. Adams M, Bogduk N, Burton K, Dolan P. *The Biomechanics of Back Pain*. Edinburgh: Churchill Livingstone; 2002.
31. Twomey LT, Taylor JR. Age changes in lumbar vertebrae and intervertebral discs. *Clin Orthop Relat Res*. 1987;224:97-104.
32. Haxby Abbott J, McCane B, Herbison P, Moginie G, Chapple C, Hogarty T. Lumbar segmental instability: a criterion-related validity study of manual therapy assessment. *BMC Musculoskel Disorders*. 2005;6:56.
33. Gonnella C, Paris SV, Kutner M. Reliability in evaluating passive intervertebral motion. *Phys Ther*. 1982;62(4):436-444.
34. Magee DJ. *Orthopedic Physical Assessment*. 5th ed. St. Louis, MO: Saunders Elsevier; 2008.
35. Konin JG, Wiksten DL, Isear JA, Brader H. *Special Tests in Orthopedic Examination*. 3rd ed. Thorofare, NJ: SLACK Incorporated; 2006.
36. Flynn TW, Cleland JA, Whitman JM. *User's Guide to the Musculoskeletal Examination: Fundamentals for the Evidence-Based Clinician*. Buckner, KY: Evidence in Motion; 2008.
37. Dutton M. *Orthopaedic Examination, Evaluation, and Intervention*. 2nd ed. New York, NY: McGraw–Hill Companies, Inc; 2008.
38. Cook CE, Hegedus EJ. *Orthopedic Physical Examination Tests: An Evidence-Based Approach*. Upper Saddle River, NJ: Pearson Prentice Hall; 2008.
39. Kasai Y, Morishita K, Kawakita E, Kondo T, Uchida A. A new evaluation method for lumbar spinal instability: Passive lumbar extension test. *Phys Ther*. 2006;86(12):1661-1667.
40. Jensen MC, Brant-Zawadzki MN, Obuchowski N, Modic MT, Malkasian D, Ross JS. Magnetic resonance imaging of the lumbar spine in people without back pain. *NE J Med*. 1994;331(2):69-73.
41. Weinstein JN, Rydevik BL, Sonntag VKH, eds. *Essentials of the Spine*. New York, NY: Raven Press; 1995.
42. Ward RC. *The Myofascial Release Concept*. East Lansing, MI: Michigan State University, College of Osteopathic Medicine; 1986.
43. Travell JG, Simons DG. *Myofascial Pain and Dysfunction: The Trigger Point Manual. Vol. 2, The Lower Extremities*. Baltimore, MD: Williams & Wilkins; 1992.
44. Jemmet R. *Spinal Stabilization. The New Science of Back Pain*. 2nd ed. Canada: Libris Hubris Publishing; 2007.
45. Bogduk N, Pearcy M, Hadfield G. Anatomy and biomechanics of psoas major. *Clin Biomechanics*. 1992a;7:109-119.
46. Butler DS. *Mobilization of the Nervous System*. Melbourne, Australia: Churchill Livingstone; 1991.
47. Butler DS. *The Sensitive Nervous System*. Adelaide, Australia: Noigroup Publications; 2000.
48. Ellis RF, Hing WA. Neural mobilization: a systematic review of randomized controlled trials with an analysis of therapeutic efficacy. *J Manual Manipulative Ther*. 2008;16(1):8-22.
49. Cyriax J. *Textbook of Orthopaedic Medicine, Vol. One: Diagnosis of Soft Tissue Lesions*. London: Bailliere Tindall; 1978.
50. Herrington L. Effect of different neurodynamic mobilization techniques on knee extension range of motion in the slump position. *J Manual ManipulativeTher*. 2006;14(2):101-107.
51. Butler D, Shacklock M, Slater H. Treatment of altered nervous system mechanics
Manual Therapy. 2nd ed. London: Churchill Livingstone; 1994.
52. Kaltenborn FM. *Manual Mobilization of the Joints. Vol. 1. The Extremities*. Oslo, Norway: Norli; 2007.
53. Greenman PE. *Principles of Manual Medicine*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2003.
54. Isaacs ER, Bookhout MR. *Bourdillon's Spinal Manipulation*. 6th ed. Boston, MA: Butterworth-Heinemann; 2002.
55. Hammer WI. *Functional Soft Tissue Examination and Treatment Methods: New Perspectives*. 2nd ed. Gaithersburg, MD: Aspen Publishers, Inc; 1999.
56. Bookhout M. Back and bilateral leg pain in a 63-year old woman. In: Jones MA, Rivett DA. *Clinical Reasoning for Manual Therapists*. Edinburgh: Butterworth-Heinmann; 2004.
57. Hanney W. *Facilitation and Training Techniques for Core Stability*. Brentwood, TN: William Hanney & Cross Country Education, Inc; 2005.
58. Richardson C, Hodges P, Hides J. *Therapeutic Exercise for Lumbopelvic Stabilization: A Motor Control Approach for the Treatment and Prevention of Low Back Pain*. Edinburgh: Churchill Livingstone; 2004.
59. Cresswell AG, Oddson L, Thorstensson A. The influence of sudden perturbations on trunk muscle activity and intra-abdominal pressure while standing. *Exp Brain Res*. 1994;98(2):336-341.
60. Hodges PW, Butler JE, McKenzie DK, Gandevia SC. Contraction of the human diaphragm during rapid postural adjustments. *J Physiology*. 1997;505(2):539-548.
61. Hodges PW, Cresswell AG, Daggfeldt K, Thorstensson A. In vivo measurement of the effect of intra-abdominal pressure on the human spine. *J Biomechanics*. 2001;34:347-353.
62. Cook G. *Athletic Body in Balance: Optimal Movement Skills and Conditioning for Performance*. Champaign, IL: Human Kinetics; 2003.
63. Kelly E. *Emily Kelly's Common Sense Pilates*. London: Lorenz Books; 2000.
64. Hides JA, Stokes MJ, Saide M, Jull GA, Cooper DH. Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. *Spine*. 1994;19(2):165-172.
65. Hides JA, Richardson CA, Jull GA. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. *Spine*. 1996;21(23):2763-2769.
66. Grenier SG, McGill SM. Quantification of lumbar stability by using 2 different abdominal activation strategies. *Arch Phys Med Rehabil*. 2007;88:54-62.
67. Razmjou H, Kramer JF, Yamada R. Intertester reliability of the McKenzie evaluation in assessing patients with mechanical low-back pain. *J Orthop Sports Phys Ther*. 2000;30(7):368-389.
68. Nachemson A, Morris JM. In vivo measurements of intradiscal pressure. Discometry, a method for the determination of pressure in the lower lumbar discs. *J Bone Joint Surg*. 1964;46-A(5):1077-1092.
69. Morrone L. *Overcoming Back and Neck Pain: a Proven Program for Recovery and Prevention*. Eugene, OR: Harvest House Publishers; 2008.
70. Morgan D. Concepts in functional training and postural stabilization for the low-back-injured. *Topics in Acute Care and Trauma Rehabilitation*. 1988;8-17.
71. Posner-Mayer J. *Swiss Ball Applications for Orthopedic and Sports Medicine: A Guide for Home Exercise Programs Utilizing the Swiss Ball*. Denver, CO: Ball Dynamics International Inc; 1995.
72. Sahrmann SA. *Diagnosis and Treatment of Movement Impairment Syndromes*. St. Louis, MO: Mosby-Year Book; 2002.
73. Saunders HD, Saunders R. *Evaluation, Treatment, and Prevention of Musculoskeletal Disorders. Vol. 1, Spine* Chaska, MN: The Saunders Group; 1995.
74. McGill SM, Cholewicki J. Biomechanical basis for stability: an explanation to enhance clinical utility. *J Orthop Sports Phys Ther.*. In: Boyling J, Pa
2001;31(2):96-100.

75. Corning-Creager C. *Therapeutic Exercises Using Foam Rollers*. Berthoud, CO: Executive Physical Therapy; 1998.
76. Clark MA. *Integrated Training for the New Millennium*. Thousand Oaks, CA: National Academy of Sports Medicine. 2001.
77. Jackson-Wyatt O. Feldenkrais method and rehabilitation. In: Davis CM, ed. *Complementary Therapies in Rehabilitation*. Thorofare, NJ: SLACK Incorporated; 1997.
78. O'Sullivan JJ, Ellis JJ, Makofsky HW. The 5 Ls of lifting. *Physical Therapy Forum*. 1991;3-6.
79. Fritz JM, George SZ, Delitto A. The role of fear-avoidance beliefs in acute low back pain: relationship with current and future disability and work status. *Pain*. 2001;94:7-15.
80. Fritz JM, George SZ. Identifying psychosocial variables in patients with acute work-related low back pain: the importance of fear-avoidance beliefs. *Phys Ther*. 2002;82:973-983.
81. Godges JJ, Anger MA, Zimmerman G, Delitto A. Effects of education on return-to-work status for people with fear-avoidance beliefs and acute low back pain. *Phys Ther*. 2008;88(2):231-239.
82. Jarvik JJ, Hollingworth W, Heagerty P, Haynor DR, Deyo RA. The longitudinal assessment of imaging and disability of the back (LAIDBack) study. *Spine*. 2001;26(10):1158-1165.
83. Rivett DA, Jones MA. Improving clinical reasoning in manual therapy. In: Jones MA, Rivett DA, eds. *Clinical Reasoning for Manual Therapists*. Edinburgh: Butterworth-Heinemann; 2004

Section VI

PELVIC GIRDLE

21

Examination and Evaluation of the Pelvic Girdle

Although motion of the sacroiliac (SI) joint appears limited to minute amounts of rotation and translation,[1] the SI joint retains its mobility throughout life.[2] It is also well established that the SI joints can be a source of painful symptoms, especially when affected by inflammatory diseases such as ankylosing spondylitis but also in conditions of mechanical impairment as occurs at the other synovial joints of the body.[3,4] As with all painful symptoms of musculoskeletal origin, a distinction needs to be made between peripheral nociceptive pain and the pain of altered central processing seen in chronic pain, referred to as central sensitization.[5,6] Otherwise, management is misdirected and ineffective. With regard to the importance of the pelvis, Greenman[4] says the following, "The osseous pelvis has a significant contribution to the functional capacity of the musculoskeletal system and warrants appropriate investigation and management in all patients." This author is not convinced of its role in the management of "all patients," but clearly as a component of the lumbar-pelvic-hip complex, its role in low back and pelvic pain needs to be appreciated. Furthermore, in light of regional interdependence[7,8] in the body, clinical instability[2,4,8-12] of the pelvic joints (ie, hypermobility of the SI joint and/or pubic symphysis) has the potential to destabilize the lower limb resulting in lower extremity symptoms of biomechanical origin.[8,13,14]

Because the ilium is capable of motion on the sacrum that is distinct from motion of the sacrum between the paired ilia,[4,15] the SI joint can be functionally separated into the iliosacral and SI joints. It is believed that iliac motion is related to function of the lower extremity, whereas sacral motion is more related to the lumbar spine. The author believes that the complex mechanics of sacral motion (ie, SI), with its 5 "imaginary" axes,[4,15] dictate that the subject of sacral examination/evaluation and intervention are best covered in an advanced course.

Consequently, this textbook will deal exclusively with the examination/evaluation and intervention of iliosacral and pubic symphysis impairments, which will enable the novice practitioner to manage a significant majority of patients with mechanical disorders of the pelvis. If a disorder of the SI complex is suspected and has not responded to manual correction of the lumbosacral junction nor iliosacral complex, then referral to a practitioner with advanced knowledge and skill in this area is warranted.

In the pelvic girdle, there are 2 systems that contribute to mechanical stability—the osteoarticularligamentous and the myofascial. Vleeming et al[3] and Lee[9] refer to these 2 systems as "form" and "force closure," respectively. According to Schamberger,[16] form closure of the SI joint is based on the following features:

- The triangular shape of the sacrum, which fits between the 2 ilia like a keystone in a Roman arch.
- The interlocking of sacral and iliac articular surfaces, helping to counter vertical and anterior-posterior translation.
- The anteriorly widening sacrum restricts movement between the innominates by causing wedging in an anterior-posterior direction.
- The ligaments of the SI joint—anterior, interosseous, posterior, and pelvic floor ligaments.

From a myofascial perspective, Schamberger[16] attributes force closure to the contraction of the "inner" and "outer"

Makofsky HW
Spinal Manual Therapy, 2nd ed. (pp 183-194)

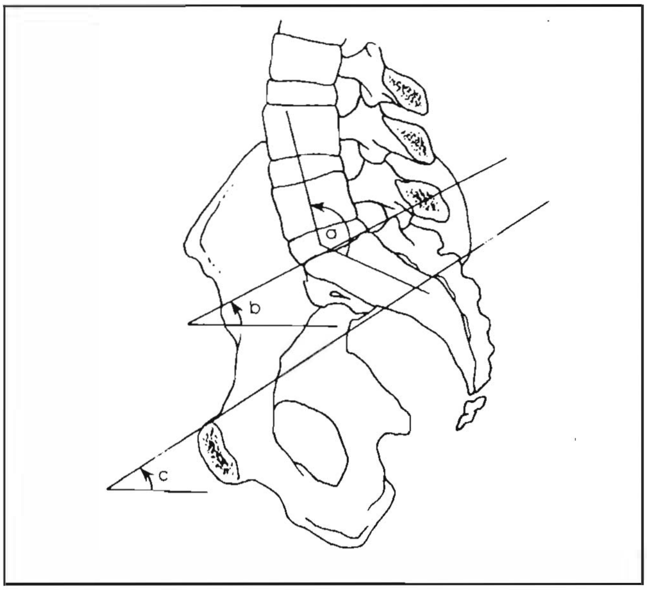


Figure 21-1. Normal angles of the spine, sacrum, and pelvis. a = lumbosacral angle (140 degrees), b = sacral angle (30 degrees), and c = pelvic angle (30 degrees). (Reprinted with permission from Magee DJ. *Orthopedic Physical Assessment.* 3rd ed. Philadelphia, PA: WB Saunders; 1997.)

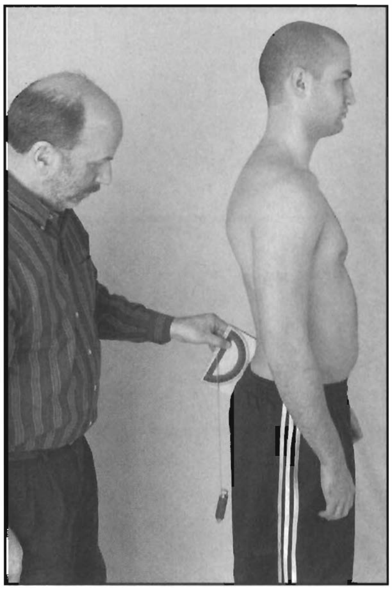


Figure 21-2. Measuring sacral inclination.

myofascial units. The inner unit consists of the multifidi, respiratory diaphragm, transversus abdominis, and pelvic floor muscles. The outer unit consists of the following:

- Posterior oblique system: Latissimus dorsi → thoracolumbar fascia → contralateral gluteus maximus causing compression of the SI joint on the side of the gluteus maximus;
- Anterior oblique system: External/internal abdominal obliques → anterior abdominal fascia → contralateral adductors of the thigh;
- Deep longitudinal system: Erector spinae → deep lamina of the thoracolumbar fascia → contralateral sacrotuberous ligament and biceps femoris causing SI joint compression upon contraction, and
- Lateral system: Gluteus medius/minimus → contralateral adductors of the thigh.

Instability is defined by Lee as, "A loss of the functional integrity of a system which provides stability."[9] The manual examination of the pelvis in this chapter focuses on signs of instability, which include the presence of subluxations (ie, positional faults or misalignments) that often develop as a result of the underlying hypermobility. Through the process of inspecting pelvic asymmetry (A), range of motion (R), and tissue texture abnormality (T), the most common iliosacral (ie, anterior iliac rotation, posterior iliac rotation, and superior iliac shear or upslip) and pubic symphysis subluxations (ie, superior and inferior shears) can be identified so that the proper manual intervention is rendered. In this way, form closure is addressed and stability improved. Force closure, as in other regions of the body, is restored through normalization of myofascial function. This will be accomplished by stretching and "releasing" what is tight and strengthening and "retraining" what is weak.

Structural Exam (With Emphasis on Asymmetries)

As with the other examination procedures covered thus far, the patient's pelvic girdle will be observed laterally, posteriorly, and anteriorly. In addition to observing alignment, key pelvic landmarks will also be palpated for positional asymmetry.

Lateral Postural Examination

A lateral radiographic analysis of the lumbopelvic region provides normative values for 3 important angles. They are the lumbosacral angle (140 degrees), the sacral angle (30 degrees), and the pelvic angle (30 degrees) as illustrated in Figure 21-1. From a clinical point of view, a simple sacral goniometer is useful in assessing anterior sacral inclination in the sagittal plane (Figure 21-2). Prsala[17] describes 20 degrees of anterior inclination as an approximation of normal in most people. Movement of the sacral base anterior and inferior is referred to as sacral flexion, nutation, or anterior nutation, whereas movement of the sacral base in a posterior and superior direction is referred to as sacral extension, counternutation, or posterior nutation. There is a tendency to avoid the use of flexion and extension in this regard because of the way in which sacral motion is described in the craniosacral literature.[4] In craniosacral terms, sacral flexion is equivalent to counternutation, whereas sacral extension is equivalent to nutation. Consequently, the terms *nutation*

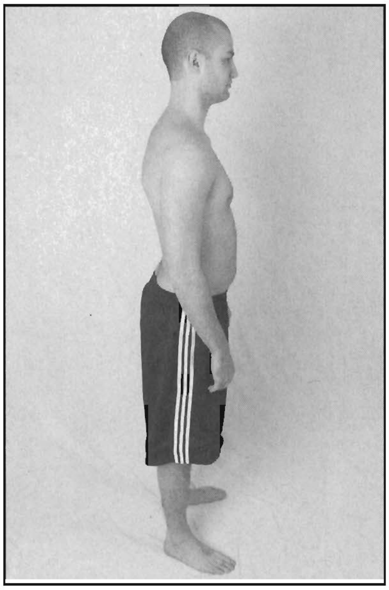


Figure 21-3. Lateral view of the pelvic girdle and lower half.

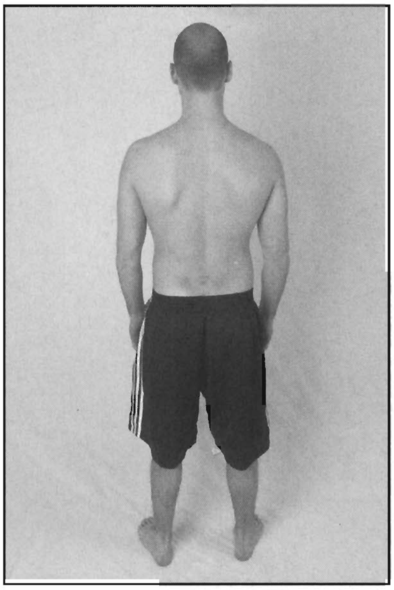


Figure 21-4. Posterior view of the pelvic girdle and lower half.

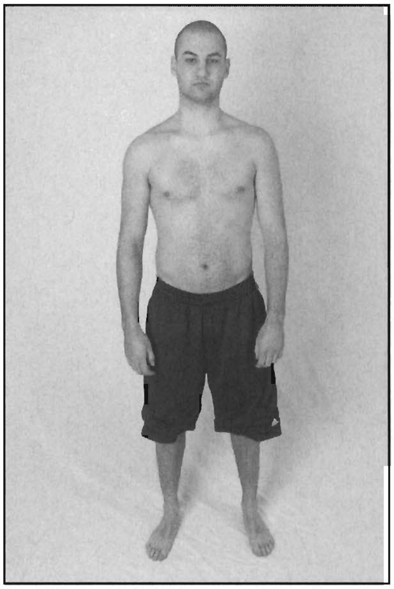


Figure 21-5. Anterior view of the pelvic girdle and lower half.

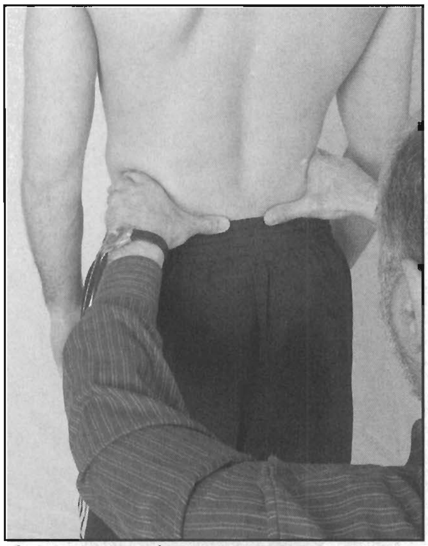


Figure 21-6a. Iliac crest comparison in standing.

and *counternutation* serve us better. Although by definition sacral nutation and counternutation refer to movement of the sacrum relative to fixed iliac bones, the sacrum can also be said to nutate and counternutate relative to the L5 vertebra (ie, the lumbosacral junction).

Regarding the normal inclination of the innominates in stance, the posterior superior iliac spine (PSIS) should be slightly superior to the anterior superior iliac spine (ASIS) at approximately a 30-degree angle to the horizontal plane. According to Kendall et al,[18] the pelvis is considered to be in neutral when the ASISs are level and in the same plane as the pubic symphysis.

In addition to a lateral analysis of the pelvic girdle (Figure 21-3), the therapist is encouraged to begin the process of integrating the entire lower half of the body into the examination process (ie, from T6 to the feet). This includes a description of the mid/lower thoracic region, lumbar lordosis, pelvic tilt, hips, knees, ankles, and feet. Because of the interdependence of the lumbopelvic region and the lower limb, special attention should be given throughout the postural examination to such biomechanical and structural relationships as true versus functional leg length disparity; hip joint alignment; genu valgum, varum, and recurvatum; the quadriceps or Q-angle; tibial varum; tibial torsion; rearfoot/forefoot varus and valgus; compensatory rearfoot pronation; Feiss line; first ray position; hallux rigidus/limitus; hallux abductovalgus, etc.[3,19-22]

Posterior Postural Examination

From a posterior perspective, the therapist should assess for a lateral shift of the trunk, signs of pelvic obliquity in the frontal plane (ie, lateral pelvic tilt), unilateral pelvic rotation in the sagittal plane, contour of the buttock region, and lower limb position as mentioned above (Figure 21-4).

Anterior Postural Examination

In addition to assessing for the above-mentioned asymmetries, the anterior perspective offers the optimal view of hip joint position as well (Figure 21-5).

Palpation of Bony Landmarks

The second aspect of the examination for pelvic girdle asymmetry includes the palpation of key bony landmarks. The pelvic/hip landmarks used for this purpose include the iliac crest, PSIS, ischial tuberosity, greater trochanter, ASIS, and the pubic tubercle.

The patient is first examined in the standing position. Posteriorly, the therapist palpates the iliac crests (Figure 21-6a), the PSISs at their inferior aspect (Figure 21-6b),

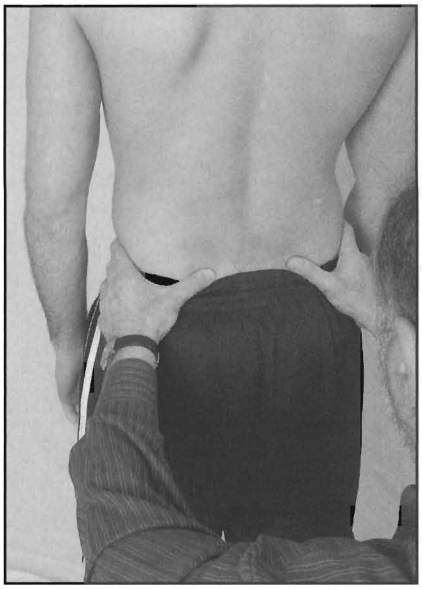


Figure 21-6b. PSIS comparison in standing.

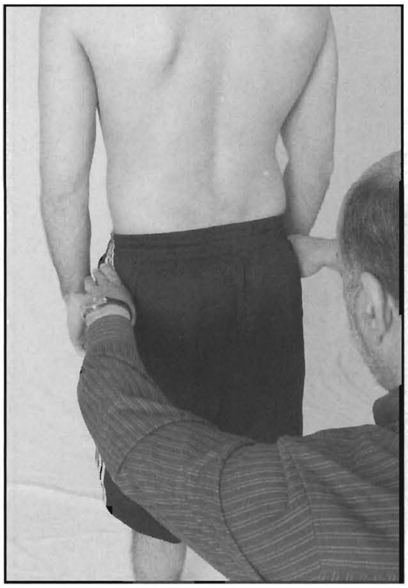


Figure 21-6c. Greater trochanter comparison in standing.

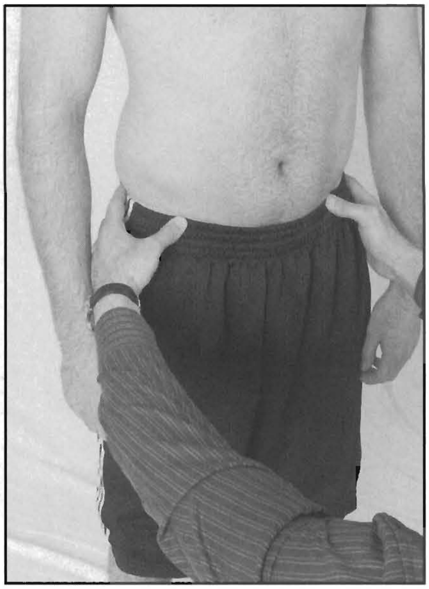


Figure 21-6d. ASIS comparison in standing.

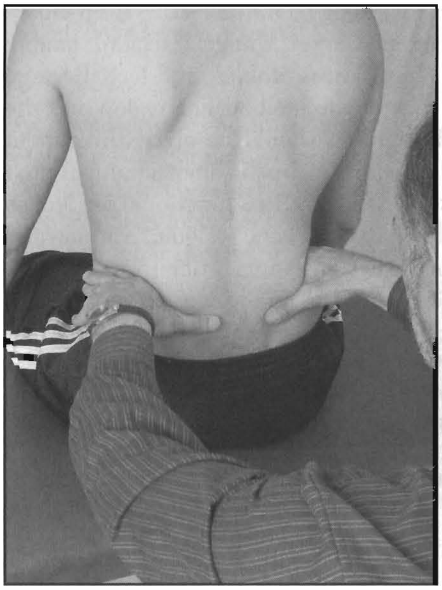


Figure 21-6e. Iliac crest comparison in sitting.

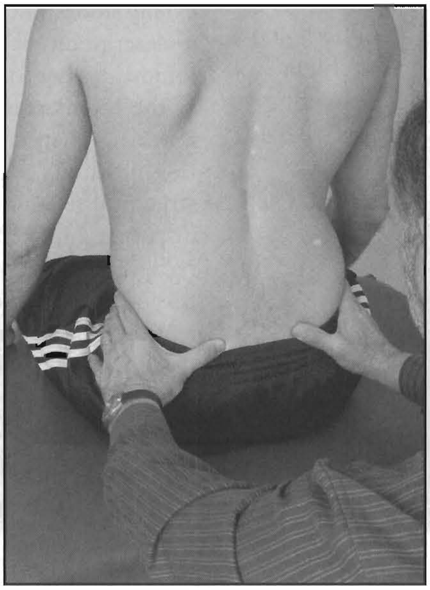


Figure 21-5f. PSIS comparison in sitting.

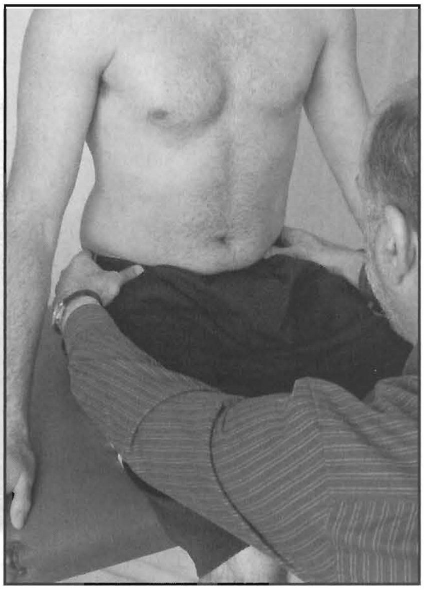


Figure 21-6g. ASIS comparison in sitting.

and the greater trochanters at their superior aspect (Figure 21-6c). Whereas the iliac crests and greater trochanters are compared for asymmetry in height, the PSISs are assessed for differences in height and posterior prominence. When comparing for structural differences in height, osteopathic physicians suggest placing one's dominant eye in the midline of the patient's body. Anteriorly, the standing patient's ASISs are palpated at their inferior aspects and assessed for asymmetry in height and anterior prominence (Figure 21-6d).

The following landmarks are assessed in sitting: the iliac crests, PSISs, and ASISs (Figures 21-6e to 21-6g).

Prior to the examination for bony asymmetry in supine, the patient's pelvis should be balanced. This involves bridging (stage 1) followed by lower limb traction (stage 2) as illustrated (Figures 21-6h and 21-6i). The following landmarks are then evaluated for asymmetries: ASISs (Figure 21-6j), and the pubic tubercles (Figures 21-6k). Because of the sensitive nature of the pubic region, it is suggested that the examiner ask the patient for permission to assess these bony landmarks. A picture of the bony anatomy is sometimes helpful in allaying the patient's apprehension. It is also recommended that the patient assist the therapist by finding his or her own pubic symphysis first and then from a

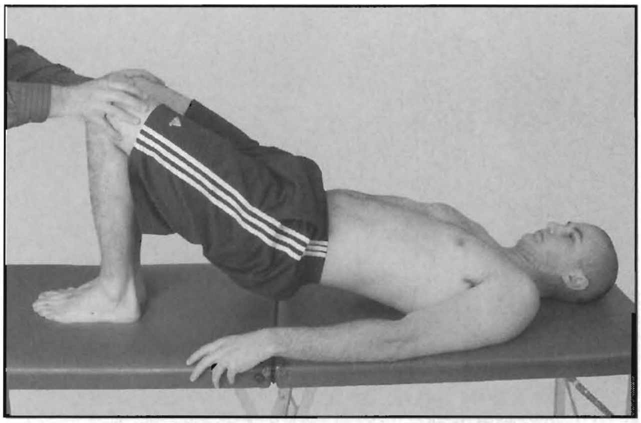


Figure 21-6h. Balancing the pelvis stage 1.

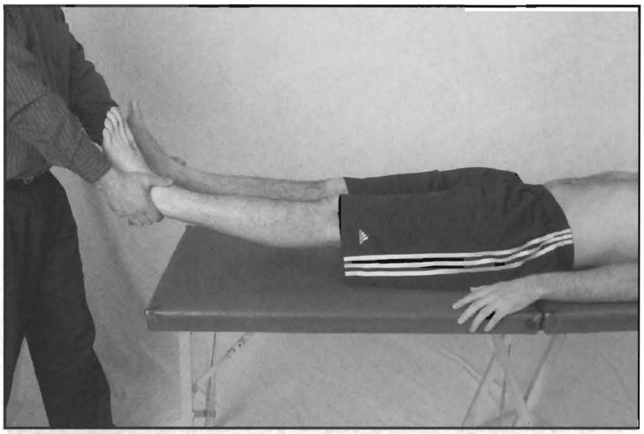


Figure 21-6i. Balancing the pelvis stage 2.

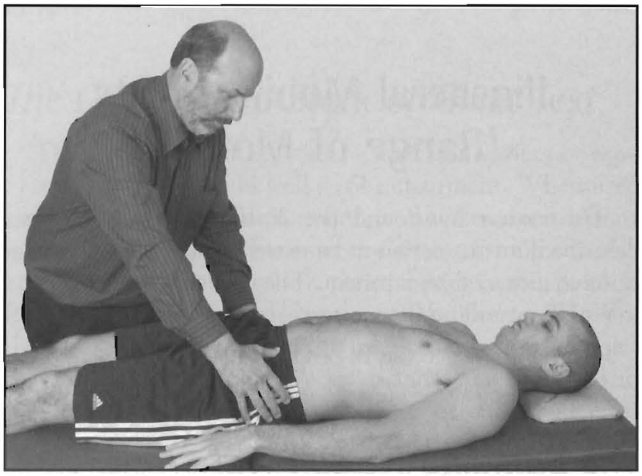


Figure 21-6j. Supine ASIS comparison.

Figure 21-6k. Pubic tubercle comparison.

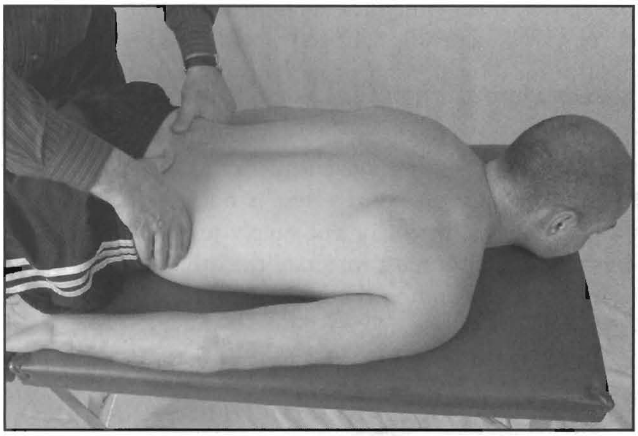


Figure 21-6l. Iliac crest comparison prone.

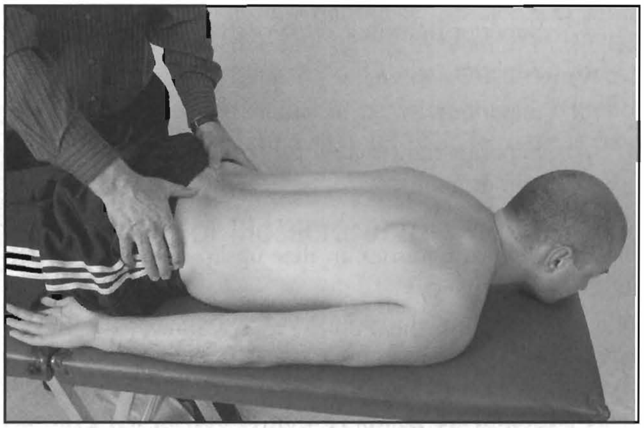


Figure 21-6m. PSIS comparison prone.

superior direction, the therapist palpates the patient's pubic tubercles (approximately 2 cm lateral to the pubic symphysis) for asymmetry in height and anterior prominence.

The final position for the comparison of bony landmarks in the osseous pelvis is prone lying (slight traction through the legs helps to balance the gross alignment of the pelvic girdle). With the patient in a prone position, the therapist palpates the iliac crests (Figure 21-6l), PSISs (Figure 21-6m), and the ischial tuberosities (Figure 21-6n). Because we are dealing with iliosacral impairments only, there is no need at this point to palpate sacral landmarks (ie, the sacral base and inferior lateral angle) for asymmetry. This, however, would be a component of the advanced examination involving SI impairment. Because the ilium rotates and translates in the

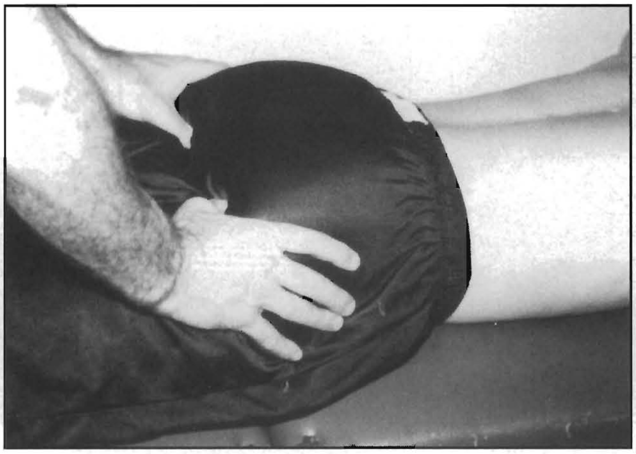


Figure 21-6n. Ischial tuberosity assessment in prone.

same direction, the PSIS may become more prominent with posterior rotation and less prominent with anterior rotation. Conversely, the ASIS may become more prominent with anterior iliac rotation and less prominent with posterior rotation.

The expected asymmetry in pelvic landmarks associated with iliosacral and pubic symphysis subluxation is as follows in all 5 of the following impairments:

1. Anterior iliac rotation on the right
   a. Superior right PSIS versus the left
   b. Inferior right ASIS versus the left
2. Posterior iliac rotation on the left
   a. Inferior left PSIS versus the right
   b. Superior left ASIS versus the right
3. Upslip of the right ilium (superior shear)
   a. Superior right iliac crest vs the left
   b. Superior right PSIS versus the left
   c. Superior right ASIS versus the left
   d. Superior right ischial tuberosity versus the left (6 mm or more)
   e. Level greater trochanters in standing (this finding distinguishes an iliac upslip from a leg length discrepancy)
4. Inferior pubic shear on the right ("down pube")
   a. Inferior right pubic tubercle versus the left
5. Superior pubic shear on the left ("up pube")
   a. Superior left pubic tubercle versus the right

Although it is useful for the therapist to begin considering the cause of these asymmetries early in the examination process, the therapist must avoid the temptation to make a diagnosis of iliosacral impairment prior to the completion of the remaining aspects of the exam, namely iliosacral mobility, soft tissue findings, and special tests, including provocation maneuvers.

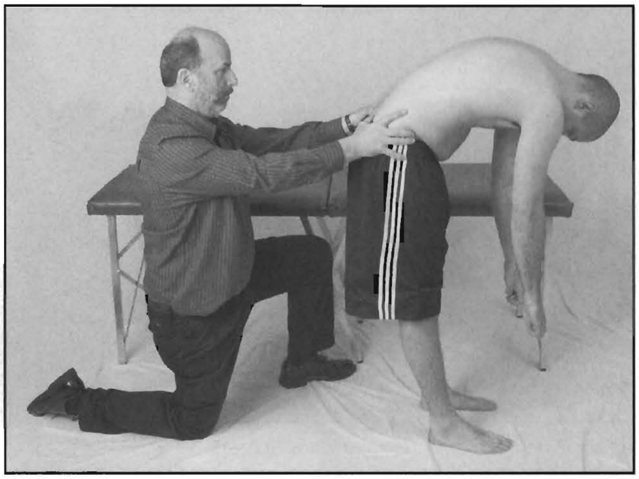


Figure 21-7. Standing flexion test.

Iliosacral Mobility Tests (Range of Motion)

The author has found the 3 iliosacral mobility tests described in this section to be extremely useful in the diagnosis of iliosacral impairment. They are as follows:

- The standing flexion test[4,15,22]
- The one-legged stork or Gillet test[4,15,23]
- The long sitting test[24]

The Standing Flexion Test

The standing patient is asked to forward bend as the therapist's thumbs monitor motion at the inferior aspect of the PSIS, bilaterally. The test (Figure 21-7) is considered positive for iliosacral impairment on the side in which the PSIS moves first and/or more superior. This represents a fixation, whereby the ilium becomes "bound" to the sacrum, resulting in premature movement or greater excursion on the affected side. A positive test is not specific as to the nature of the impairment but simply reveals that there is one. The therapist must integrate the other findings of the examination, including the history, to determine the specific impairment present.

There are at least 4 reasons for a false-positive (ie, poor specificity) result with the standing flexion test. They are as follows:

- A tight hamstring on the contralateral side
- Iliac posterior rotation hypermobility on the contralateral side
- A short leg on the contralateral side
- Osseous (structural) asymmetry of the PSISs

If a leg length discrepancy is suspected, a lift should be used under the short leg prior to the test. If this is not done to balance pelvic alignment, the test is invalid.

Figure 21-8a. Right upper SI joint posterior rotation assessment.

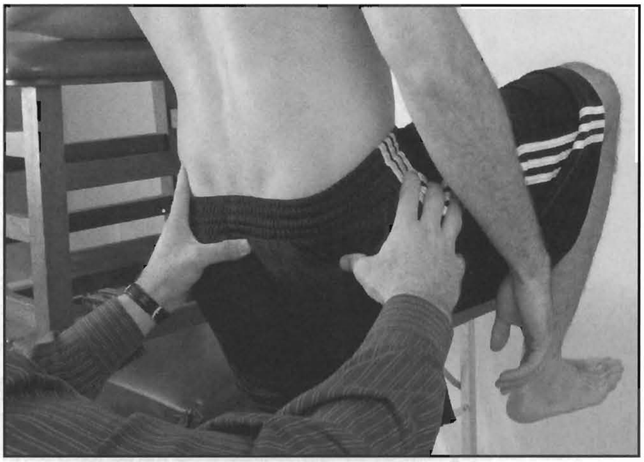


Figure 21-8b. Right lower SI joint posterior rotation assessment.

The One-Legged Stork or Gillet Test

The stork or Gillet test can be used in a variety of ways to test both iliosacral as well as SI impairment. When used as a test for iliosacral impairment, it can detect restrictions in both posterior as well as anterior iliac rotation. It can also be applied separately to the superior aspect (upper pole) of the iliosacral joint, which consists of the shorter "arm" of the L-shaped surface and to the inferior aspect (lower pole) that consists of the longer "arm." Schafer and Faye[23] liken the iliosacral joint surface to a "boot," whereby the superior articular surface (S1 segment) is above the "ankle" and the inferior surface (S2 and S3 segments) makes up the "foot" of the "boot."

Consequently, in the diagnosis of iliosacral impairment, the one-legged stork or Gillet test will be used to test motion loss in 4 different ways. They are as follows:

1. Superior iliosacral joint, posterior iliac rotation, right and left
2. Inferior iliosacral joint, posterior iliac rotation, right and left
3. Superior iliosacral joint, anterior iliac rotation, right and left
4. Inferior iliosacral joint, anterior iliac rotation, right and left

Superior Iliosacral Joint, Posterior Iliac Rotation

The patient is asked to raise his or her right knee to his or her chest while holding the table for support. The therapist examines motion of the right ilium by palpating the inferior aspect of the right PSIS with the right thumb, while simultaneously palpating the S2 segment of the sacrum at the median sacral crest with the left thumb (Figure 21-8a). With normal iliosacral posterior rotation, the PSIS moves slightly inferior and lateral relative to the S2 segment. Restricted posterior rotation at the superior iliosacral joint (ie, the upper pole) is consistent with an anterior iliac rotation subluxation (misalignment) or an iliac shear lesion. In some patients, the PSIS actually moves superiorly, which suggests marked restriction. The left side is then tested accordingly.

Inferior Iliosacral Joint, Posterior Iliac Rotation

The patient performs the same knee-to-chest motion maneuver as in the previous exercise. However, in order to test right inferior or lower pole motion, the therapist places his or her left thumb over the sacral apex at the hiatus, while the right thumb is placed at the same level on the posterior/inferior aspect of the right ilium (Figure 21-8b). With normal motion, the right iliac contact will move slightly anterior, inferior, and lateral in relation to the left thumb. Restricted motion is consistent with a right-sided, anterior iliac rotation or iliac shear misalignment (inferior shear or downslip is extremely rare). The left side is then tested accordingly.

Superior Iliosacral Joint, Anterior Iliac Rotation

As mentioned previously, the one-legged stork or Gillet test can also be used to test for restricted anterior iliac rotation (ie, "reverse stork"). For those who use the term *marcher's test* instead of *stork* or *Gillet*, the following exam procedure is referred to as the "reverse marcher's test."

To assess upper pole anterior iliac rotation on the left side, the standing patient is instructed to bring his or her right knee to his or her chest, while the therapist maintains contact at the S2 segment (in the midline) with the right thumb and the left PSIS with the left thumb (Figure 21-8c). As the left ilium rotates posteriorly and forces the sacrum into counternutation, a relative "anterior rotation" of the left ilium is induced. This is appreciated by the left PSIS "moving" superior and lateral relative to the sacrum. This is an example of

Figure 21-8c. Left upper SI joint anterior rotation assessment.

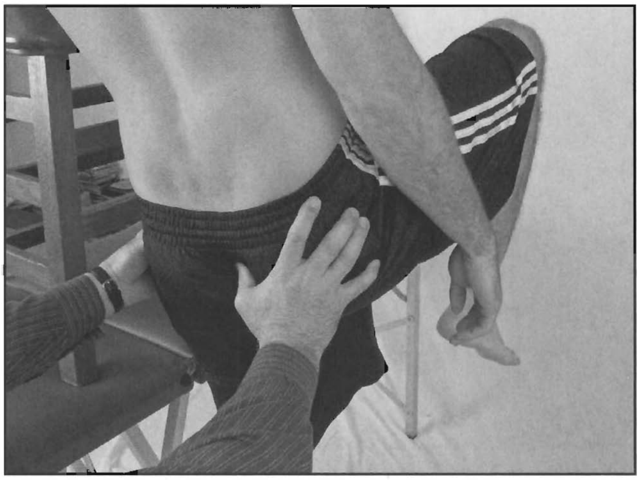


Figure 21-8d. Left lower SI joint anterior rotation assessment.

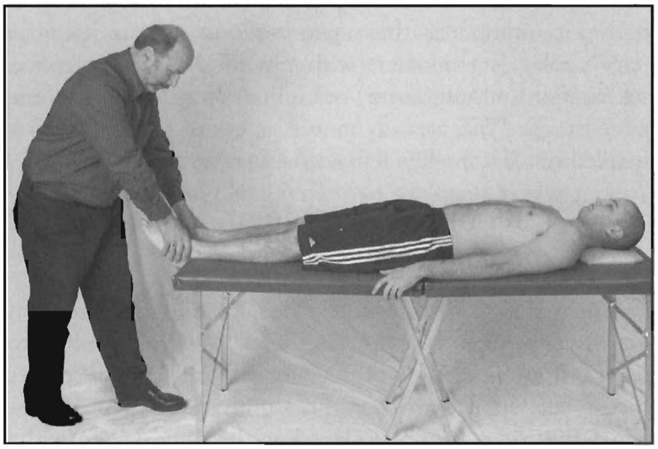


Figure 21-9a. Long sitting test stage 1.

"relative motion," whereby the change in iliac position, relative to the moving sacrum, is what the therapist is assessing. For example, a moving car passing a stationary car in the opposite direction can be said to cause relative "motion" of the stationary car when in fact it has not moved.

Consequently, the patient must bring the right knee high enough to his or her chest in order for the right ilium to recruit the sacrum and counternutate it. Should the left PSIS fail to "move" in a superior and lateral direction, it is said to be restricted. Restricted anterior iliosacral rotation implies that the ilium is "stuck" in either posterior rotation or a sheared position at the upper pole. The right side is then tested, accordingly, by having the patient raise his or her left knee to the chest.

Inferior Iliosacral Joint, Anterior Iliac Rotation

The final of the 4 variations of the stork test involves an examination of anterior iliac rotation at the inferior aspect of the iliosacral joint ("reverse stork," lower pole). This, again, applies the principle of "relative motion" as described above. However, unlike the procedure demonstrated in Figure 21-8c, the examiner's thumb contacts now monitor motion at the lower pole. The examiner places his or her right thumb over the sacral apex at the hiatus, while the left thumb makes contact with the posterior/inferior aspect of the left ilium at the same level (Figure 21-8d). To test the left side, the patient raises the right knee high to the chest in order to force the sacrum into counternutation. A normal response is observed when the therapist's left thumb moves superior and lateral relative to the right thumb. Motion restriction in conjunction with the expected asymmetry in iliac landmarks points to either a posteriorly rotated right iliac bone with impairment of motion at the inferior aspect or lower pole of the left iliosacral joint or to a left iliac shear (superior much more likely than inferior). The definitive diagnosis, however, cannot be made until the examination of tissue texture abnormality and special tests are completed. The right side is tested, similarly, by reversing the thumb contacts and having the patient raise his or her left knee to the chest.

With all 4 variations of the stork test, an assessment of end-feel provides additional diagnostic information about impaired joint function (ie, normal joints have a small degree of end-play present).

The Long Sitting Test

The long sitting test is also commonly used as an indicator of iliosacral impairment. The patient performs a bridging maneuver in order to obtain neutral alignment of the pelvis. The therapist then compares the length of the medial malleoli with the legs flat on the table (Figure 21-9a). This is followed by a similar comparison with the patient in the long sitting position (Figure 21-9b). A posterior iliac rotation misalignment is suspected when a short ipsilateral medial malleolus in supine becomes longer than the contralateral side in the long sitting position. An anterior iliac rotation is suspected when the ipsilateral medial malleolus changes from long in supine to short in long sitting vs the contralateral leg (Figure 21-10).

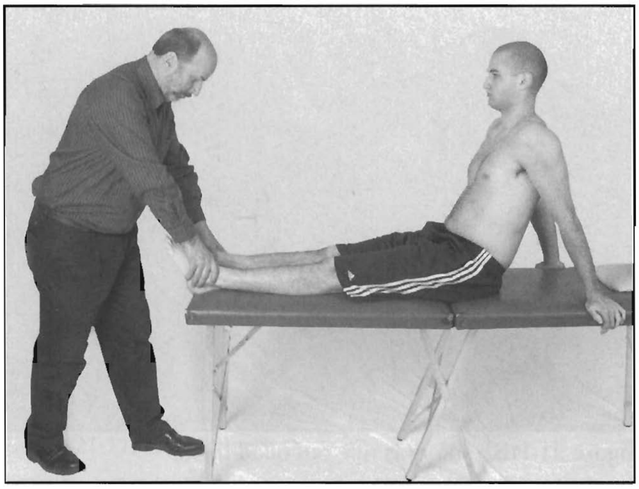


Figure 21-9b. Long sitting test stage 2.

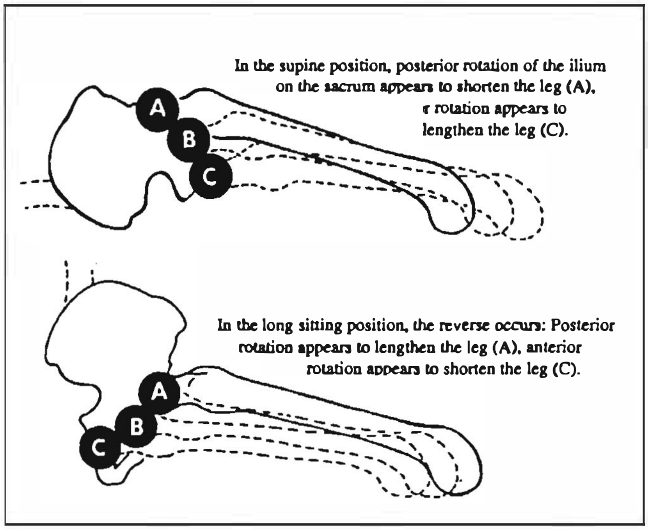


Figure 21-10. Mechanical explanation of the long sitting test. (Reprinted from Saunders HD, Saunders R. *Evaluation, Treatment, and Prevention of Musculoskeletal Disorders*. 3rd ed. Chaska, MN: The Saunders Group; 1993. Used with permission from the Saunders Group, Inc. © 1993.)

Soft Tissue Palpation (Tissue Texture Abnormality)

The inspection for signs of tissue texture abnormality in the pelvis is crucial in the diagnosis of somatic impairment. As with the other regions of the musculoskeletal system, the therapist is looking for the presence of the following associated indicators of a mechanical disorder: tenderness, tightness, hypertonia, fibrosis, swelling, and alterations in tissue texture, including a ropy, stringy, or boggy feel to the tissues. In the pelvic region, fibrositic nodules are common, which, like myofascial trigger points, can be latent (asymptomatic) or active (symptomatic). This author suggests the use of electrotherapeutic modalities to reduce their tissue reactivity but prefers to treat the underlying somatic impairment that causes them to become symptomatic. In addition to these aspects of the soft tissue examination, the therapist must also include the assessment of myofascial extensibility and muscle length of the entire lumbar-pelvic-hip region.

The soft tissue structures amenable to examination in the pelvic region include the following:

- Baer's SI point[22]: 2 inches from the umbilicus on a line from the umbilicus to the ASIS (tenderness is often associated with a SI impairment).
- Iliopsoas muscle: Medial to the sartorius muscle, medial and deep to the ASIS.
- Pubic symphysis, inguinal ligament at its medial attachment, and the rectus abdominis at its distal attachment.
- QL muscle.
- Iliolumbar ligament: Running from the transverse processes of L4 and L5 to the anterior surface of the iliac crest.
- Posterior SI ligaments: Consisting of a deep layer of short interosseous ligaments running from the intermediate and lateral sacral crest to the rough sacropelvic surface of the ilium; the long interosseous ligaments extending from the median and lateral sacral crest, diagonally in a superior direction across the sacral sulcus, and attaching to the PSIS of the ilium. Particularly prominent is the long dorsal SI ligament,[25] which is a thickened band extending from the PSIS to the lateral sacral crest (it resists sacral counternutation and is thought to cause the all-too-common tenderness at the PSIS, when it comes under tension from below). The posterior, together with the anterior, SI ligaments are referred to as the intrinsic ligaments of the SI joint.
- Gluteus maximus, medius, and minimus muscles.
- Piriformis muscle: Palpable in the posterior buttock, deep to the gluteus maximus muscle, at the intersection of 2 lines. One line extends from the ASIS to the ischial tuberosity, while the other line runs from the PSIS to the greater trochanter.
- Short lateral rotators of the hip (superior/inferior gemellus, obturator internus and externus, and the quadratus femoris): Deep to the gluteus maximus, anterior to the sciatic nerve, coming off the upper end of the greater trochanter. The obturator internus, lying between the 2 gemelli, is partly an intrapelvic muscle and partly a hip muscle.
- Tensor fascia latae: The therapist performs myofascial inspection from its proximal attachment
rior iliac crest and ASIS to its distal insertion into the lateral patellar retinaculum, anterolaterally, and into Gerdy's tubercle at the lateral proximal tibia via the iliotibial tract, posterolaterally.

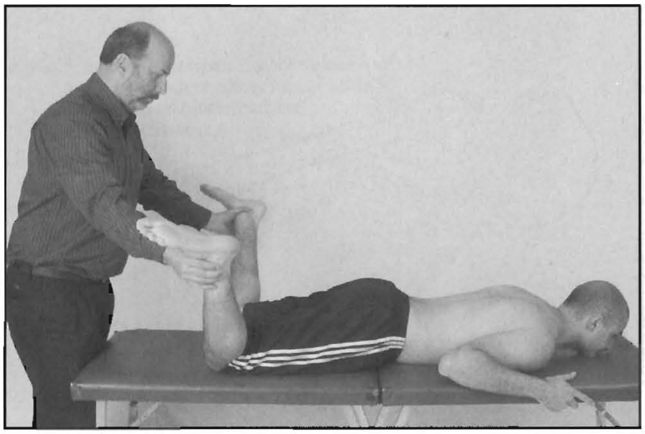


Figure 21-11a. Piriformis rule out prone.

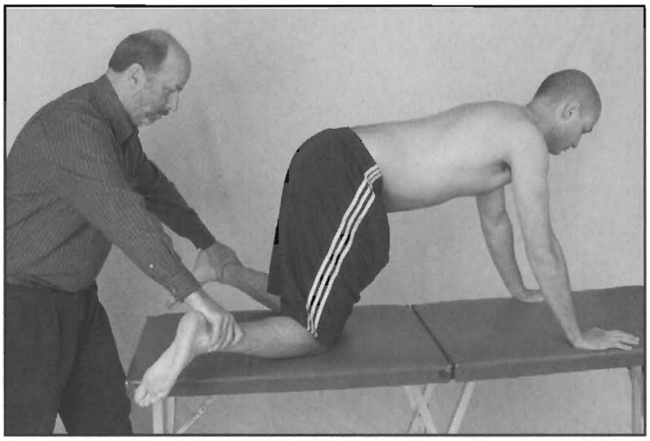


Figure 21-11b. Piriformis rule out quadruped.

- Pelvic floor muscles: Although optimal access to these intrapelvic muscles requires either a rectal or vaginal approach, the coccygeus, iliococcygeus, and pubococcygeus muscles can be partly accessed medial to the ischial tuberosity in the perineum, proceeding from the anal triangle to the urogenital triangle, with the patient in the hook-lying position.
- Adductor longus, brevis, magnus, gracilis, and pectineus muscles at the lower borders of the pelvis, pubic ramus, ischial ramus, and ischial tuberosity.
- Hamstring muscles at their attachment into the ischial tuberosity.
- Sacrotuberous ligament is a triangular-shaped structure connecting the PSIS, SI joint capsule, coccyx, and the ischial tuberosity with connecting fibers from the biceps femoris muscle a common finding. The tendons of the deepest laminae of the multifidus often extend into the sacrotuberous ligament. Its role is to resist sacral nutation and posterior iliac rotation. Consequently, the sacrotuberous ligament becomes palpably tender and taut in the presence of a posterior iliac misalignment, but slacked with an anterior innominate rotation. Furthermore, through tautening of the sacrotuberous ligament, posterior iliac rotation misaligment can deviate the coccyx toward the dysfunctional side; this asymmetry of coccyx position should be detectable on palpation.
- Sacrospinous ligament is a triangular-shaped structure that lies under the sacrotuberous ligament, extending from the inferior lateral angle of the sacrum to the ischial spine. The sacrospinous ligament separates the greater from the lesser sciatic foramen. Like the sacrotuberous ligament, it resists sacral nutation and posterior iliac rotation. The iliolumbar, sacrotuberous, and sacrospinous ligaments are collectively referred to as the extrinsic SI ligaments.

Following the direct palpatory examination of the aforementioned tissues, the therapist should evaluate the length of all the postural muscles of the pelvis and hip that are prone to tightness (eg, rectus abdominis, sacrospinalis, quadratus lumborum, piriformis, hamstrings, adductors, tensor fascia latae, iliopsoas). Hip flexor length can be tested with the Thomas test or modified Thomas test (ie, TRI muscle test covered in the following chapter); the tensor fascia latae is tested with Ober's test. A tight piriformis is distinguished from a tight hip capsule by the range of hip internal rotation in prone vs quadruped (Figures 21-11a and 21-11b). Because the piriformis is an external rotator of the hip in neutral and an internal rotator above 60 degrees of hip flexion,[26,27] a restriction of internal rotation in neutral prone lying that normalizes in quadruped points to muscle tightness. However, restricted hip joint internal rotation in both positions points to stiffness of the hip joint capsule.

Manual muscle testing of the weak phasic muscles of the pelvis and hip (ie, the oblique abdominals, gluteus maximus, medius, and minimus, etc) can be performed at this point or in the special test section to follow.

Special Tests

Tests that mechanically stress the SI joint structures in order to reproduce the patient's symptoms are called provocation tests.[3,28,29] These tests do not assess for asymmetries, range of motion deficits, nor tissue texture abnormality, but rather help to determine whether the SI joint is the anatomic source of the pain regardless of whether the underlying problem is due to disease or mechanical impairment. The provocation tests described in this section are based upon the research of Laslett, Aprill, and colleagues[28] and do not discriminate between iliac misalignment on the sacrum (ie, iliosacral impairment) and sacral misalignment within the paired ilia (ie, SI impairment). They include the following:

- Distraction or "gapping" test
- Compression test
- Posterior shear or "thigh thrust" test
- Pelvic torsion or Gaenslen's test for the right side

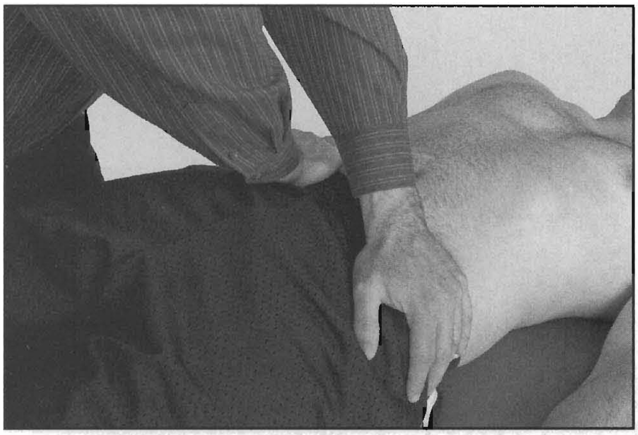


Figure 21-12. Distraction test.

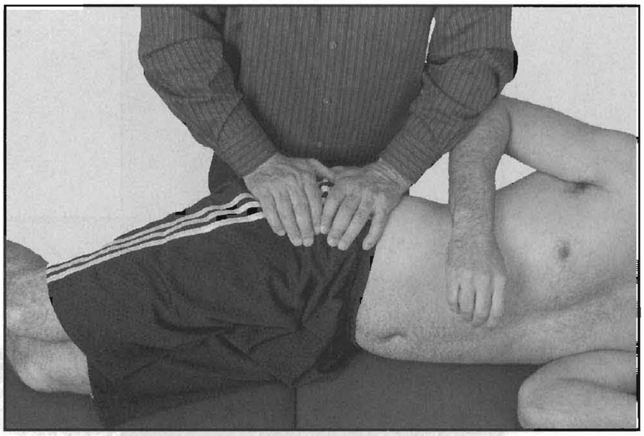


Figure 21-13. Compression test.

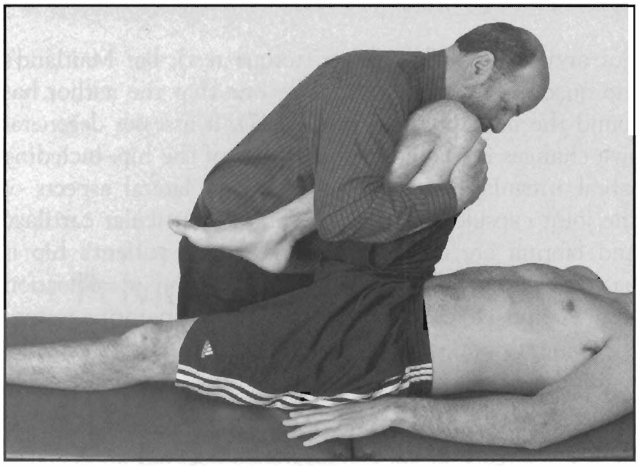


Figure 21-14. Posterior shear test.

- Pelvic torsion or Gaenslen's test for the left side
- Sacral thrust test

Laslett et al[28] found the above tests to have the best predictive power of a positive intra-articular SI joint block (accepted criterion standard) when either 2 of 4 tests, namely distraction, compression, posterior shear (thigh thrust), and sacral thrust were positive, or 3 or more of the above 6 SI joint provocation tests were positive. When 3 or more of the above tests were positive, sensitivity and specificity were 94% and 78%, respectively; when all tests are negative, the SI joint can be ruled out as a source of the patient's pain.

The Distraction or "Gapping" Test

In this test (Figure 21-12), the therapist applies a posterior and lateral force to both ASISs in order to distract the anterior aspect of the SI joints and stretch the anterior SI ligaments. Reproduction or exacerbation of the patient's pelvic pain constitutes a positive response.

The Compression Test

In this test (Figure 21-13), the therapist applies downward pressure to the uppermost iliac crest directed toward the opposite iliac crest with the patient in side lying. This test purports to stretch the posterior SI joint ligaments and compress the anterior aspect of the SI joint. The test is positive if the patient's symptoms are either reproduced or worsened.

The Posterior Shear or "Thigh Thrust" Test

In this test (Figure 21-14), the therapist imparts a posterior shearing stress on the SI joint through downward pressure on the supine patient's flexed femur. For optimal application, the therapist blocks motion of the sacrum with one hand while applying downward force through the femur with the other hand. Excessive hip adduction should be avoided lest the test becomes overly stressful to the joint and produces false-positive results. The hip quadrant or scour test does, however, involve compression with adduction, but this is a provocation test for the hip joint and will be mentioned subsequently.[24] Again, provocation of the patient's pelvic pain is considered a positive test response.

The Pelvic Torsion Test or Gaenslen's Test

With the patient in supine and the left knee pulled to the patient's chest, the therapist applies overpressure to the left leg (Figure 21-15), causing end-range left posterior iliac rotation. In the meantime, the right hip is held in extension with the leg off the end of the table. It is expected that a left posterior iliac rotation subluxation will react to this end-range stress. However, the test is not specific for this given misalignment but introduces sufficient stress to the left SI joint to provoke symptoms in a variety of positional faults, including, but not limited to, posterior iliac rotation. The pelvic torsion test is then repeated on the right side by simply reversing all manual contacts.

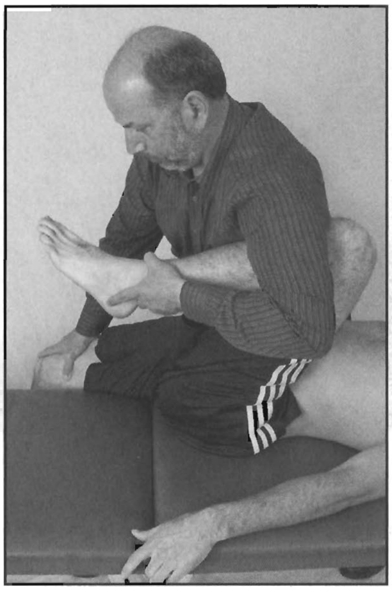


Figure 21-15. Pelvic torsion test.

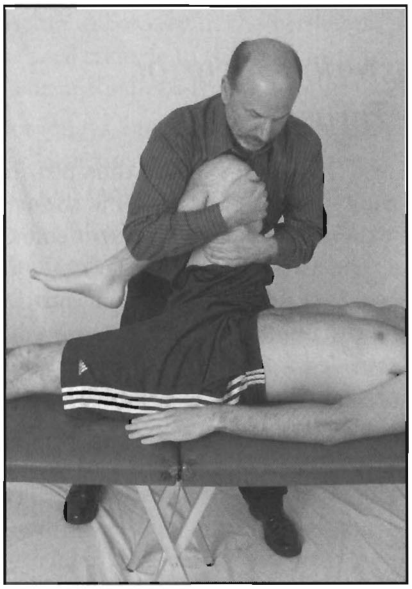


Figure 21-17. Maitland's hip quadrant test.

The Sacral Thrust Test

Manual pressure is applied to the entire sacrum in an anterior direction with the patient in the prone-lying position (Figure 21-16).

When attempting to determine whether the patient's symptoms are pelvic in origin, it is necessary to perform a clarifying exam of the hip joints. There are many orthopedic provocation tests of the hip[24,29] (eg, Patrick's test, ante-

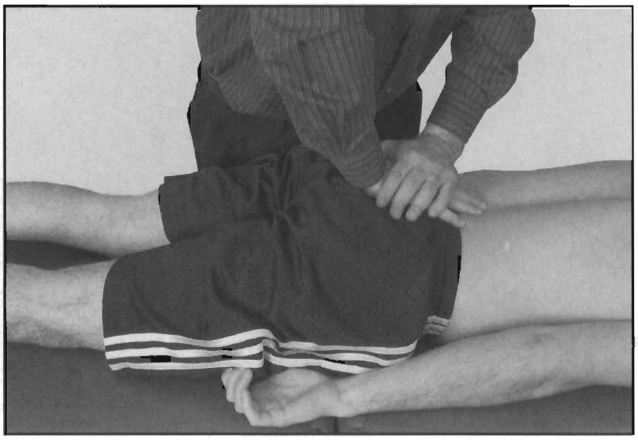


Figure 21-16. Sacral thrust test.

rior and posterior labral test, torque test), but Maitland's hip quadrant or scour test is the one that the author has found the most useful (Figure 21-17). It assesses degenerative changes in the articular surfaces of the hip, including labral irregularities. The posterior and lateral aspects of the joint capsule are stretched, and the articular cartilage and labrum are compressed. The supine patient's hip is passively moved through an arc of flexion in adduction from 90 and 140 degrees where the knee is pointing toward the patient's opposite shoulder. Compression of the hip is maintained through the femur at all times as demonstrated. Throughout the arc, the femur should lie midway between medial and lateral rotation. A positive response for a hip disorder includes pain, guarding, apprehension, crepitus, etc. A small abnormality is often felt as a "bump" along the smooth arc of this circle. In addition to the hip quadrant test, this section would not be complete without mentioning the goniometric assessment of hip joint range of motion, including an inspection of flexion, extension, abduction/adduction, medial, and lateral rotation. As noted in the soft tissue section of the examination, hip muscle length and strength must also be included in every examination of the pelvic girdle. Because of the significant influence of the hip postural (eg, hamstrings, iliopsoas, adductors, tensor fascia latae) and phasic (eg, gluteus maximus, gluteus medius) muscles on the alignment and function of the iliosacral joints, an approach that seeks to balance these influences by stretching what is tight and strengthening what is weak will certainly help many individuals. It is the author's philosophy, however, that combining stretching and strengthening procedures together with myofascial and articular mobilization/manipulation yields the best outcomes possible. To that end, let us proceed to the chapters on manual therapy intervention.

22

Connective Tissue Techniques and Stretching Procedures for the Pelvic Girdle

Pelvic Diaphragm Release

Myofascial pain and dysfunction of the muscles of the pelvic floor causes pain to be felt in the perineum, urogenital structures, the posterior pelvic floor, the sacrococcygeal region, the vagina, the anococcygeal region, and the posterior thigh. The levator ani muscle is the most widely recognized source of referred pain in the perineal region. Referred pain from the levator ani may be felt in the sacrum, coccyx, rectum, perirectal area, vagina, or low back and be aggravated by lying on the back and by defecation. Terms used to describe pelvic pain of levator ani muscle origin include *levator spasm syndrome*, *levator ani spasm syndrome*, *levator syndrome*, and *pelvic floor syndrome*. Though not a likely occurrence, there is the potential for entrapment of the pudendal nerve and the internal pudendal vessels by the obturator internus muscle in the lesser sciatic foramen. Should this occur, perineal pain or dysesthesia can result.[30]

The pelvic diaphragm release (Figure 22-1) is a three-dimensional/transverse fascial plane technique not unlike the thoracic inlet and respiratory diaphragm releases described in Chapter 5. As with any three-dimensional/transverse fascial plane release, the "4 Ms" procedure (also described in Chapter 5) is an excellent way of performing either a direct or indirect myofascial release technique of the pelvic floor. Manheim[31] reports that the reflex relaxation of the pelvic diaphragm achieved through myofascial release therapy has proven useful in easing the pain of endometriosis and premenstrual cramps and may help relieve chronic LBP and deep hip joint pain.

Using the 4 Ms procedure, the therapist molds, melds, monitors, and moves the tissues between the bottom hand placed under the sacrum and the top hand placed lightly over the pubic symphysis. The hands should be perpendicular to the patient's body and parallel to each other. The choice of whether to proceed with a direct fascial stretch or an indirect method, whereby tissue ease is sought, is dependent upon the patient's symptoms and level of tissue reactivity as discussed in Chapter 3. Because of the sensitivity of this region, the author recommends beginning with a gentle indirect approach in which myofascial relaxation or "unwinding" is achieved. The use of direct myofascial stretching is performed when the tissues require it. A slight degree of tissue compression, prior to either indirect or direct technique, is often useful in enhancing the release of tissue tension. As always, the purpose of this 3-dimensional/transverse plane myofascial intervention is to relax, soften, and restore normal elasticity/pliability to the tissues between the therapist's hands. Before proceeding, an explanation, along with the appropriate anatomy pictures, serves to allay the patient's apprehension. If the therapist is of the opposite gender of the patient, it is wise to have another person of the same gender as the patient in the treatment room during the application of the technique.

Pelvic Floor Fascial Technique

The remainder of the manual connective tissue techniques in this section fall into the category of direct fascial techniques, otherwise known as myofascial manipulation, soft tissue mobilization, deep tissue massage, etc. As with the

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Spinal Manual Therapy, 2nd ed. (pp 195-202)

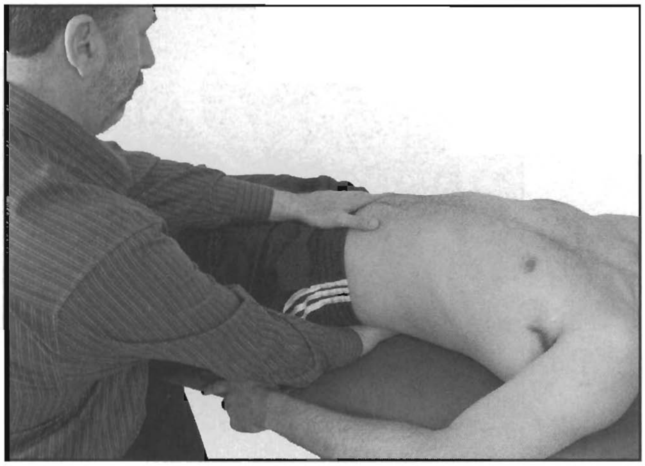

Figure 22-1. Pelvic diaphragm release.

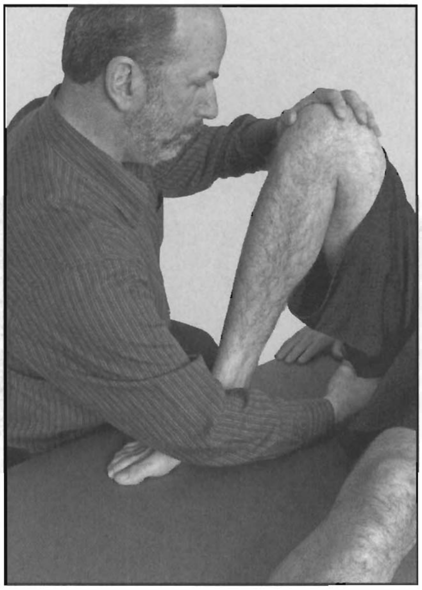

Figure 22-2. Pelvic floor fascial technique.

examination of this area in the previous chapter, the patient is placed in the hook-lying position on the treatment table as demonstrated (Figure 22-2). Because of the proximity of the external genitalia and anus to the myofascial point of entry, this manual intervention requires the utmost respect for the patient's dignity and self-respect. It is strongly recommended that a third person be present in the treatment room and that this person be of the same gender as the patient. When children are involved, it is necessary for a parent or guardian to be present. An appropriate anatomic illustration of the pelvic floor musculature should be shown to the patient, parent, etc, prior to the application of the technique, including an explanation of the clinical purpose for the use of this procedure (in many clinics, a written, informed consent is required for this and perhaps all therapeutic procedures as an added measure of legal protection for the therapist and the facility). The patient must understand his or her right to refuse such treatment at any time. In addition to the above considerations, proper draping technique of the patient's perineal area should be a priority.

With fingernails that are appropriately trimmed, the therapist applies light digital pressure with 2 or 3 fingers to the area medial and anterior to the ischial tuberosity while abducting the ipsilateral thigh for optimal access. It is the musculature of the pelvic diaphragm located between the ischial tuberosity laterally, the coccyx posteriorly, the anus medially, and the transversus perinei superficialis superiorly that are amenable to gentle direct fascial technique. Though direct skin contact is ideal, this procedure can be performed over a pair of shorts, sweatpants, or a towel. The objective is to release tension and tightness in this region of the pelvis through the application of gentle, direct manual pressure. The attachment sites of the muscles of the pelvic diaphragm into the entire ischial region of the innominate bone are likely areas of soft tissue impairment. For those patients who suffer with chronic pelvic floor pain syndromes related to myofascial involvement, there is nothing that is more useful than the direct manual release of this area.

Piriformis Fascial Technique

The piriformis muscle is thick and bulky in most individuals but occasionally it is thin and sometimes absent. The Belgian anatomist Adrian Spigelius coined its name, which in Latin means "pear shaped." Travell and Simons[30] report that in approximately 85% of cadavers, the sciatic nerve passes anterior to the piriformis and between its fibers and the rim of the greater sciatic foramen. In approximately 10% of cadavers, the fibular (peroneal) portion of the nerve passes through the piriformis and the tibial portion travels anterior to it. In 2% to 3%, the fibular (peroneal) portion loops above and then posterior to the muscle, while the tibial portion passes anterior to it; both portions lie between the muscle and the rim of the greater sciatic foramen. In less than 1% of cadavers, an undivided sciatic nerve pierces through the piriformis muscle. When the piriformis is sufficiently enlarged to fill the foramen, entrapment of the superior and inferior gluteal nerves and blood vessels, the sciatic nerve, the pudendal nerve and vessels, the posterior femoral cutaneous nerve, and the nerves supplying the gemelli, obturator internus, and quadratus femoris muscles is a possibility. When sciatic nerve entrapment is present, there are usually signs of L5 and S1 nerve root involvement.

In the presence of SI or iliosacral misalignment, contraction of the piriformis loads the joint and thus can mimic sciatic pain. Piriformis tightness may also subject the sacrum to abnormal rotary stress and produce or exacerbate a pelvic dysfunction. Myofascial pain of the piriformis may cause symptoms to develop proximal to the gluteal cleft and at the posterior, superior, and medial borders of the hip joint with possible referral down the buttock and into the posterior thigh. Symptoms tend to be aggravated by sitting; by a prolonged combination of hip flexion, adduction, and

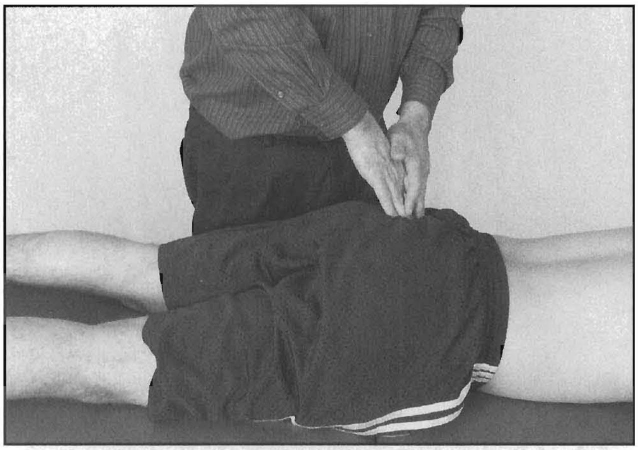

Figure 22-3a. Piriformis fascial technique in neutral rotation.

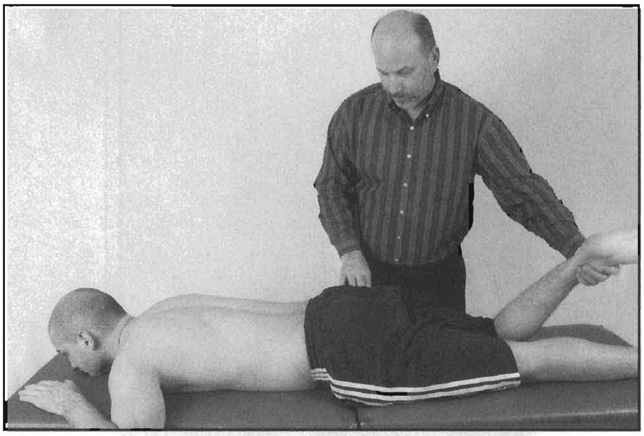

Figure 22-3b. Piriformis fascial technique in shortened range.

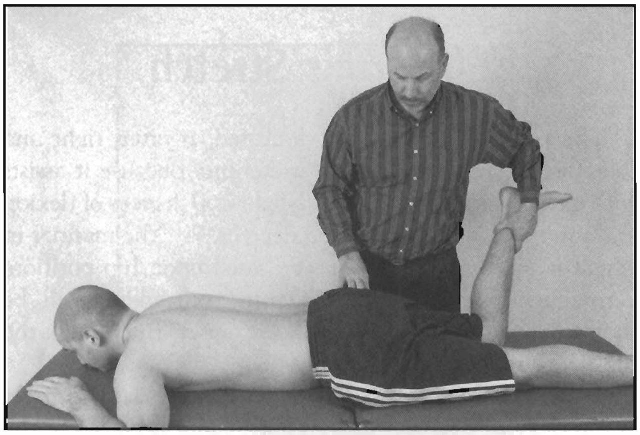

Figure 22-3c. Piriformis fascial technique under stretch.

medial rotation; or by activity. However, before the painful symptoms can truly be considered to be of piriformis origin, the clinician must first clear the lumbar spine, sacroiliac region, and hip joint as discussed in previous sections. Compensatory hypertonicity (ie, muscle substitution) of the piriformis is often seen in the presence of gluteus maximus and medius weakness.

To access the piriformis muscle in the prone-lying patient, the point of intersection of 2 imaginary lines, as described during the examination, is used as a guide. One line runs from the ASIS to the ischial tuberosity; the other from the PSIS to the greater trochanter. As illustrated in Figure 22-3a, the direct fascial technique known as "strumming" is an excellent way of freeing the piriformis muscle and its fascial attachments. Other methods include muscle play, circular friction with the thumb or elbow (used when more force is required), "steamrolling," etc. In the presence of nociceptively driven hypertonicity, a fascial technique of the piriformis can be applied in the shortened range (hip lateral rotation) to reduce myospasm (Figure 22-3b). Other indirect treatment options for local muscle hypertonicity include functional technique and counterstrain therapy.[4,15] However, in the presence of "contracture" as opposed to "contraction," direct fascial technique in the lengthened range of the piriformis (hip medial rotation) is recommended to release adherences and restore full myofascial extensibility (Figure 22-3c). As with all connective tissue techniques, the goal is to relax, soften, lengthen, and mobilize tense, restricted, and painful myofascial tissues. A small amount of soft tissue massage cream—ie, Deep Prep II—is recommended.

Tensor Fascia Latae/Iliotibial Band Fascial Technique

The term *pseudotrochanteric bursitis* refers to the pain and tenderness caused by myofascial impairment of the tensor fascia latae/iliotibial band (TFL/ITB). Patients with this disorder describe painful symptoms in the lateral hip extending down the anterolateral aspect of the thigh; they are often misdiagnosed as having trochanteric bursitis. These patients usually have difficulty lying on the involved side because of pressure on the tender region, and they often cannot lie on the contralateral side without a pillow between their knees because of the tight ITB.

The TFL assists with flexion, abduction, and medial rotation of the hip. The tendinous fibers of the posterolateral half of the TFL join the longitudinal middle layer to form the ITB, which has 2 components at the knee: the iliopatellar band and the iliotibial tract. The iliotibial band courses distally to its insertion at Gerdy's tubercle at the lateral proximal tibia; the iliopatellar band reinforces the lateral retinaculum (tightness of its deep fibers can cause tilting of the patella, resulting in increased patellofemoral stress), which adds stability to the lateral aspect of the knee. The iliopatellar band is connected to the iliotibial tract through the patellotibial ligament. There are several other iliotibial tract attachments, including the lateral intermuscular septum, the lateral femoral condyle, the lateral capsular ligament, the biceps femoris tendon, and the fibula. The ITB is influenced by both the gluteus maximus and the TFL

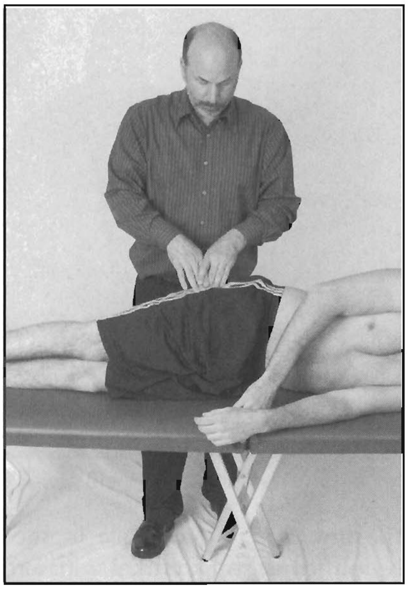

Figure 22-4a. TFL/ITB direct fascial technique.

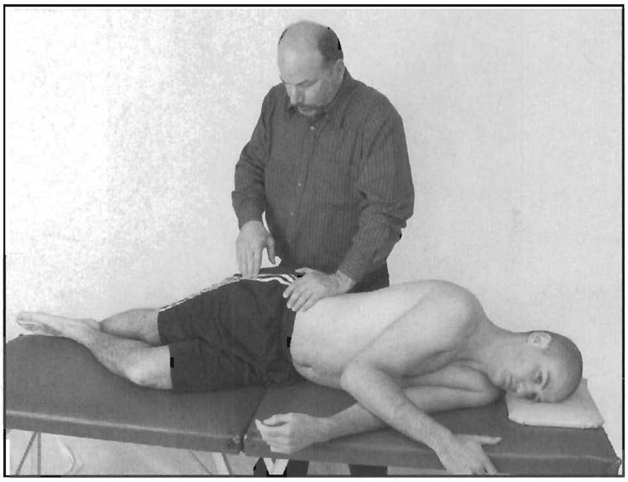

Figure 22-4b. TFL-ITB direct fascial technique under stretch.

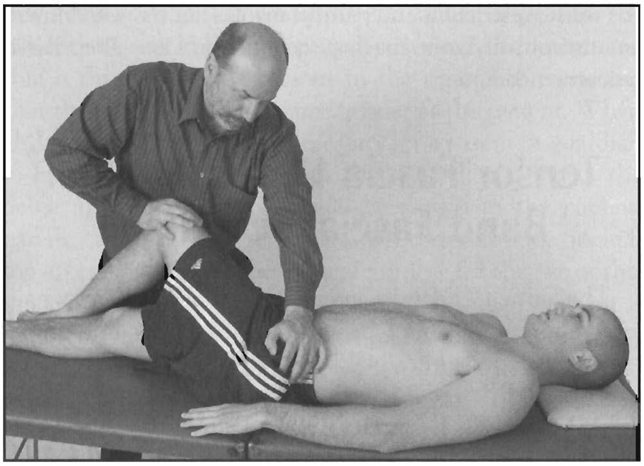

Figure 22-5a. Piriformis stretch less than 60 degrees flexion.

from which it arises. At the knee, the ITB is an extensor from 0 to 30 degrees and a flexor when the knee is flexed 30 degrees or more.[32] This information is helpful when performing stretches of the TFL/ITB, incorporating both hip and knee motions. Compensatory hypertonicity of the TFL is often seen in the presence of gluteus medius weakness.

Direct fascial technique of the TFL is performed in side lying with the affected side up. All previous manual methods, including "strumming," "steamrolling," "sculpting," etc, are applied to the bony attachments; fibers of the TFL and ITB; junction of the TFL and ITB and gluteus maximus/ITB; anterior and posterior edges of the ITB; and distally at the lateral femoral condyle, lateral retinaculum, patella, Gerdy's tubercle, and the fibula. These direct fascial techniques can be applied in the rest position (Figure 22-4a) as well as under stretch (Figure 22-4b).

Piriformis Stretch

The piriformis muscle, as discussed, is often tight and therefore in need of effective stretching. Because it assists with external rotation of the hip below 60 degrees of flexion and internal rotation above 60 degrees,[26,27] the manner in which it is stretched must differ based upon hip position. Consequently, 2 different stretching procedures will be shown to ensure full flexibility of the muscle throughout the hip joint's range of motion.

To stretch the left piriformis muscle below 60 degrees of hip flexion, the supine patient's left foot is placed to the right of the right lower leg with the foot flat on the table. Standing on the patient's right side, the therapist directs the patient's left distal femur toward the right into adduction and internal rotation (Figure 22-5a). At the barrier of motion, the postisometric relaxation (PIR) technique can be applied to enhance the stretch by having the patient perform a submaximal, isometric contraction of the abductors/external rotators for 6 seconds, followed by a stretch into the new range. After 3 cycles of the PIR/stretch technique, the patient's limb is returned slowly to the rest position (the left ASIS can be held down during the stretch to enhance control).

To stretch the left piriformis with the hip in more than 60 degrees of hip flexion (Figure 22-5b), the supine patient's left hip is passively moved into a combination of flexion, adduction, and external rotation (ie, left knee to the right shoulder). The therapist again stands on the side opposite the stretch and controls the PIR/stretch procedure by placing his or her hands on the patient's left knee. For those patients who feel pain in the anterior hip area, the amount of hip adduction should be decreased and the external rotation increased. To perform these 2 stretches to the right piriformis muscle, all directions and contacts are simply reversed.

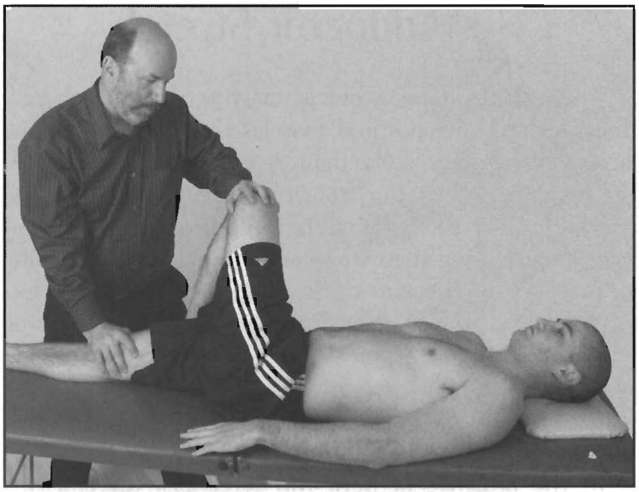

Figure 22-5b. Piriformis stretch greater than 60 degrees flexion.

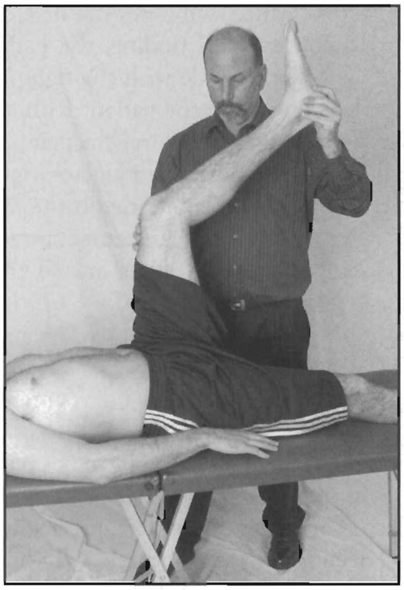

Figure 22-7. Hamstring stretch.

Tensor, Rectus, Iliopsoas Muscle Stretch

A modification of the Thomas test position is to have the patient lie at the end of the table with one knee brought to the chest and the other leg dangling off the end with the knee relaxed in flexion (Figure 22-6). This modified Thomas position was first referred to as the TRI muscle position by Ellis[33] since it assesses and treats 3 related muscles simultaneously (ie, the TFL, rectus femoris, and iliopsoas). The utility of this position involves its ability to evaluate and treat tightness in these 3 postural muscles quickly and easily. For evaluation purposes, the patient's lumbar spine ideally should remain flat on the table at all times, while the suspended limb should be in the midline with the posterior thigh flat on the table

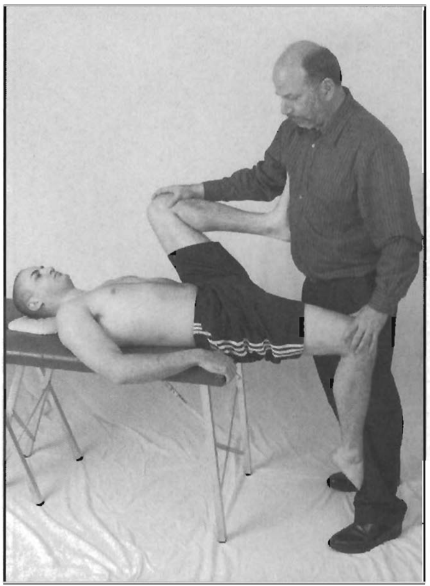

Figure 22-6. TRI muscle stretch.

surface and the knee flexed to 90 degrees. By contrast, tightness of the TRI muscles will cause deviation of limb position (ie, hip abduction, internal rotation, and flexion with TFL tightness; knee extension and/or hip flexion with tightness of the rectus femoris; and hip flexion/external rotation with tightness of the iliopsoas). In addition to observing for the effect of muscle tightness on the position of the femur, end-feel is noted and passive overpressure is applied to hip extension and knee flexion. By stabilizing the ASIS inferiorly, iliacus tightness[34] can be distinguished from psoas tightness (ie, the "iliacus test"). Tightness of the iliacus can also induce anterior rotation of the ipsilateral iliac bone when the femur is fixed (ie, iliacus contraction in reverse action).

As an intervention, the same position is used while the therapist performs postisometric stretching of the TRI muscles. In addition, the therapist can add direct fascial technique in the stretched position of the targeted muscle, especially of the TFL and rectus femoris. In the presence of iliosacral hypermobility, the patient's pelvis should be stabilized with a strap or SI belt while the TRI muscles are being stretched.

Hamstring Stretch

The importance of stretching tight hamstrings (Figure 22-7) cannot be over emphasized. It is a muscle that directly affects the lumbopelvic region, hip joint, and knee and indirectly affects the entire kinetic system, including the cervicothoracic area, by virtue of its tendency to displace the center of gravity posteriorly. When bilaterally tight, compensatory forward head carriage may ensue. In the presence of unilateral tightness, there is the tendency for posterior iliosacral rotation to occur on the ipsilateral side.

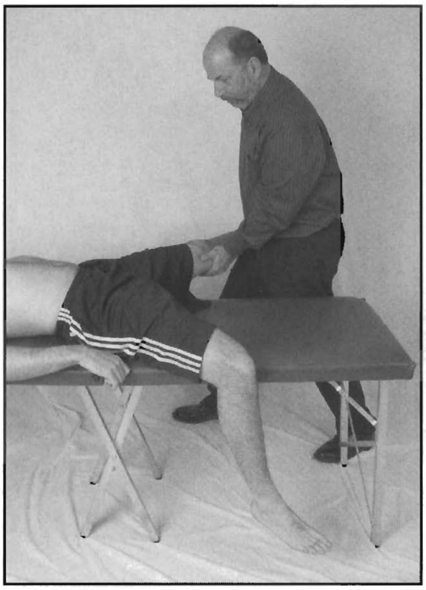

Figure 22-8. Left hip adductor stretch.

In addition, the biceps femoris is believed to be a myofascial "link" between the foot, ankle, and pelvic girdle through its connection to the fibular head. For example, rearfoot pronation displaces the fibular head anteriorly, which places the lateral hamstring under tension. This in turn introduces tension into the sacrotuberous ligament, which has the potential to affect the alignment and function of both the sacrum and ilium.

Hamstring tightness predisposes athletes and dancers to recurrent injuries. This is especially true when it is substituting for weakness of the ipsilateral gluteus maximus muscle. The astute clinician must also be mindful that adverse sciatic nerve tension, secondary to a lumbar disc derangement, will facilitate hamstring hypertonicity and predispose the hamstring to recurrent muscle strains. Consequently, the lumbar spine must always be examined before a definitive diagnosis of a hamstring "pull" is made. Conversely, in patients with a documented herniated lower lumbar disc, the hamstring must at some point be treated with connective tissue techniques and stretching because of its tendency to tighten in response to L5 and S1 nerve root compression.

Direct fascial technique to the hamstrings will not be described here. However, the therapist should consider using the same soft tissue techniques to "free up" the hamstrings as used elsewhere. The manual stretch is performed on the supine patient in the 90/90 (degrees) position. As with all stretches, the PIR component is an extremely useful addition. If iliosacral hypermobility is a concern, the patient's pelvis should be stabilized with a strap or SI belt for the duration of the stretch. Otherwise, the hamstring stretch may displace the ilium in posterior rotation.

Hip Adductor Stretch

The adductor longus, brevis, magnus, gracilis, and pectineus muscles are postural muscles that tend to become facilitated, hypertonic, and tight. According to Sahrmann,[35] the combination of hip adduction, medial rotation, and anterior pelvic tilt in standing lengthens the piriformis muscle, subjecting it to stress and strain, possibly leading to sciatic nerve entrapment. In the presence of hip adductor tightness, the TFL may also come under strain, causing pseudotrochanteric bursitis. In addition, tightness of the hip adductors may cause weakness of the gluteus medius muscle, which would undermine its important role in pelvic stability during ambulation.

In the presence of tight hip adductors, the standing patient may appear to have a longer leg on that side by virtue of a higher iliac crest.[35] This is in contrast to tightness of the hip abductors, which will lower the iliac crest on the affected side. To confirm this finding, the patient's pelvis will become level when the side with the tight adductors is adducted slightly. Conversely, the patient with tight abductors need only abduct slightly to level the pelvis. Regarding an additional effect of unilateral adductor tightness, the pubic ramus may be sheared inferiorly on the affected side in response to a strong isometric adductor contraction. Such would be the case if a soccer player missed the ball and struck the nonyielding ground instead.

"Rider's strain"[36] is characterized by the combination of painful isometric adduction and tenderness at either the musculotendinous or tenoperiosteal junction (a note should be made that painful isometric adduction is also present with fracture or neoblastic invasion of the os pubis).

Referred pain from myofascial injury and impairment of the hip adductors includes discomfort just below the inguinal ligament, deep groin pain, and referred symptoms into the hip, anteromedial thigh, and as far downward as the knee and shin.

To stretch the hip adductors, the therapist stands on the affected side, facing the supine patient's feet. In order to provide pelvic stability for a more effective stretch, the contralateral hip is slightly abducted with the lower leg off the side of the table. The adductor stretch is then performed as follows (Figure 22-8):

1. The therapist abducts the patient's tight side..
2. At the barrier of abduction, the therapist performs 3 repetitions of PIR (ie, 6-second submaximal isometric contractions of the adductors followed by relocalization to the new barrier of abduction).
3. If, in addition to the adductor tightness, the therapist detects inferior femoral glide restriction, a graded mobilization can be added with the hip in the abducted position. Through the arthrokinetic reflex mechanism,[37] the improved capsular mobility will

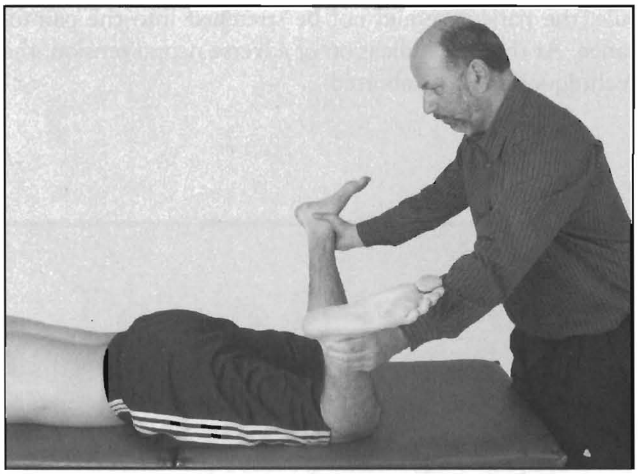

Figure 22-9a. Bilateral hip external rotator stretch.

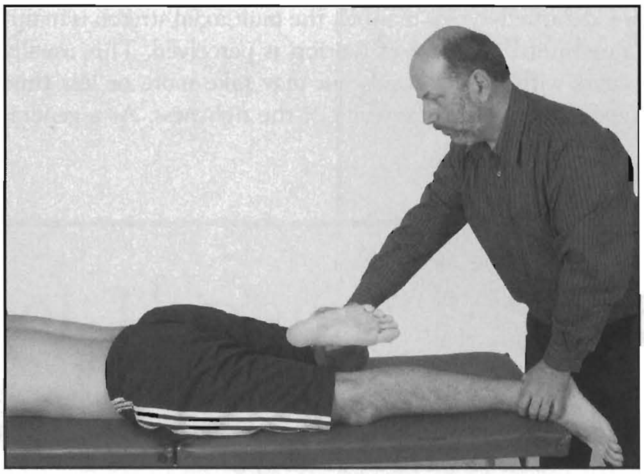

Figure 22-9b. Unilateral hip internal rotator stretch.

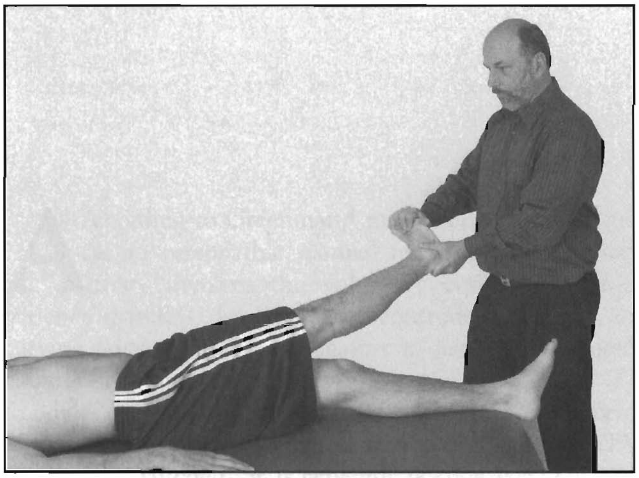

Figure 22-10. Myofascial leg pull.

theoretically inhibit adductor tone while facilitating tone and strength of the gluteus medius muscle.

Hip Rotator Stretch

The prone position is used to both assess and treat myofascial tightness of the external and internal rotators of the hip. As illustrated (Figure 22-9a), the external rotators of the hip (ie, the obturator externus/internus, quadratus femoris, piriformis, gemellus superior/inferior, gluteus maximus, posterior fibers of the gluteus medius, sartorius, and biceps femoris muscles) can be stretched bilaterally using PIR. The hip internal rotators (ie, the gluteus minimus, anterior fibers of the gluteus medius, tensor fascia latae, semitendinosus, and semimembranosus muscles) can also be stretched using PIR in prone lying but performed one limb at a time (Figure 22-9b).

Myofascial Leg Pull

The myofascial leg pull (Figure 22-10) is performed with the patient in supine. There are 2 variations of this technique that the author finds extremely useful. One is the indirect approach and the other is the direct approach. The indirect approach requires an appreciation of inherent tissue motion and seeks to relax myofascial tissues by passively moving them in the direction of tissue ease. Consequently, the myofascial tissues of the lower extremity are "unwound" the way a tangled telephone cord (when telephones had cords) would be if allowed to follow its "path of least resistance." The indirect technique is useful when dealing with increased muscle tone of peripheral origin (ie, nociceptively-mediated hypertonicity).

The direct myofascial leg pull is a "shotgun" type of approach that enables the therapist to stretch and mobilize several myofascial structures simultaneously. It imparts a vigorous stretch to the tissues and should, therefore, only be used in the presence of low reactive myofascial tightness. The myofascial leg pull applies longitudinal traction to the leg with the patient supine. The ankle is passively dorsiflexed and the leg is moved successively into abduction and external rotation, followed by adduction and internal rotation. The patient is then rolled onto his or her other side as hip adduction/internal rotation is progressed, while also maintaining strong ankle dorsiflexion. The knee remains in the extended position throughout all phases of the leg pull. Unlike the indirect approach, which involves moving into tissue "ease," the direct technique intentionally moves into tissue "bind," where restrictive barriers are challenged and mobilized. It is an efficient treatment method that enables the therapist to stretch several areas of tightness in a short period of time. It can also be used, diagnostically, to determine the exact locus of myofascial tightness using multiaxial or combined motions. When areas of restriction are identified in this manner, the multiaxial stretch is maintained until a release of tension is perceived. This usually occurs within 30 seconds but may take more or less time depending upon the severity of the tightness. As a general rule, the patient should not be stretched into the painful range. At the first indication of adverse neural tension, the technique should be aborted.

23

Pelvic Girdle Manual Therapy

According to Greenman,[4] muscle energy technique is an osteopathic manual medicine intervention that "involves the voluntary contraction of a patient's muscle(s) in a precisely controlled direction, at varying levels of intensity, against a distinctly executed counterforce applied by the operator." Though Dr. TJ Ruddy's "resistive duction" was probably the earliest form of what became known as muscle energy technique (MET), Dr. Fred L. Mitchell, Sr is generally acknowledged as the "father" of the system.

Purposes of MET include lengthening a shortened, contractured, or spastic muscle; strengthening a physiologically weakened muscle or group of muscles; reducing localized edema and congestion; and mobilizing an articulation with restricted mobility.

In the pelvic girdle, some have suggested that MET works by contracting a muscle in "reverse action," thus providing a "neuromuscular mobilization" for the purpose of realigning a subluxation or positional fault. Regardless of the theoretical mechanism of action, these treatment techniques have become the mainstay of manipulative intervention of the pelvis for years and will be applied to 4 of the 5 most common impairments affecting the iliosacral joint and pubic symphysis (ie, anterior and posterior iliac rotation and superior and inferior pubic shears). Because the iliac upslip requires additional force to correct, a manual thrust rather than MET will be described subsequently.

Because of the potential for confusion regarding the sequencing of the various lumbopelvic interventions covered thus far, guidelines will be provided at the end of this section to address this issue. In addition, more information will be provided on the subject of the effect of lower limb alignment on the pelvis and vice versa.

Anterior Iliac Rotation: Muscle Energy/Manipulation

- Lesion: Left anterior iliac rotation
- Motion restriction: Left posterior iliac rotation
- C (Chief Complaint):
  - Diffuse left posterior lumbosacral and SI pain reported, with referral into the left buttock and posterior thigh (pain on the unaffected side can and does occur)
  - Sitting usually more comfortable than standing or ambulating
- H (History):
  - Injury from golf or baseball swing common
  - Bowling
  - Direct blow to the posterior SI joint, creating hyperextension of the hip
- A (Asymmetry of Bony Landmarks):
  - Left PSIS is higher than the right in all positions (possibly anterior also)
  - Left ASIS is lower than the right in all positions (possibly anterior also)
  - Left medial malleolus is longer than the right in supine

Makofsky HW
Spinal Manual Therapy, 2nd ed. (pp 203-210)

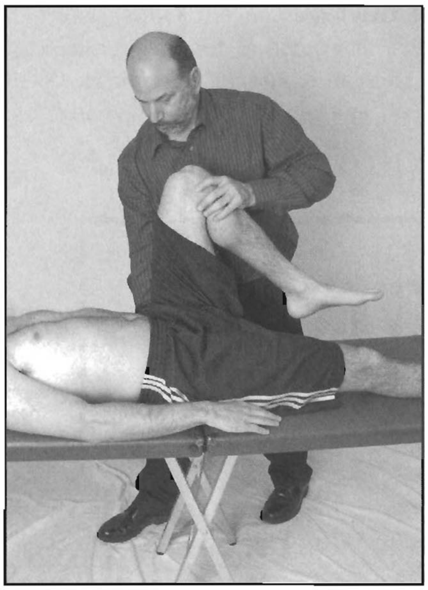

Figure 23-1. MET for left anterior iliac rotation.

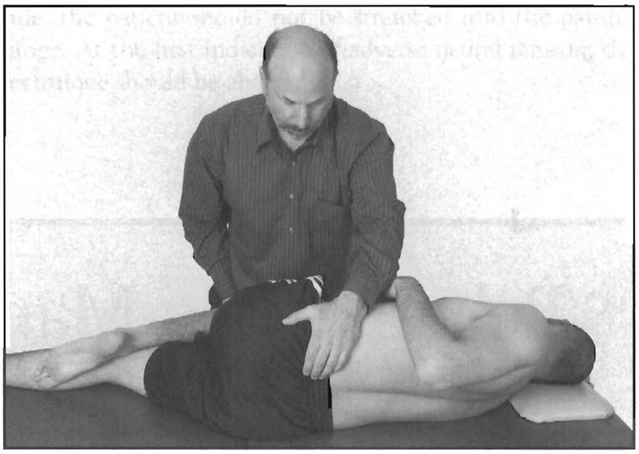

Figure 23-2. MET for left anterior iliac rotation in side lying.

- R (Range of Motion):
  - Standing flexion test is positive on the left
  - Stork test is positive on the left at the upper and/or lower pole
  - Long sitting test is positive with the left medial malleolus changing from long to short as the patient moves from supine to long sitting
- Restricted left hip external rotation
- T (Tissue Texture Abnormality):
  - Left sacrotuberous ligament is lax
  - Left hip flexors may be hypertonic, tight, and tender (TRI muscles)
  - Baer's SI point tender on the left
- S (Special Tests):
  - Some, if not all, SI provocation tests will reproduce or exacerbate the patient's chief complaint (ie, compression, distraction, posterior shear, pelvic torsion, and sacral thrust tests)
  - Normal neurological exam
  - Shortened swing phase on the left side
  - Pain with extremes of left hip extension

Two muscle energy techniques and one joint manipulation will be described and illustrated for correction of a left-sided anterior iliac rotation misalignment. To correct an anterior iliac rotation on the right side (more common of the two), all contacts and directions would be reversed.

Supine MET for Anterior Iliac Rotation on the Left

The therapist stands on the patient's left side with his or her fingers of the right hand medial to the patient's left PSIS in the sacral sulcus (Figure 23-1). Meanwhile, the patient's left hip is flexed to the first motion barrier at the iliosacral joint in posterior iliac rotation (ie, the "feather edge" of the restrictive barrier). The patient is asked to resist further left hip flexion to a count of 6 seconds. Following a 3-second relaxation phase, the left ilium is repositioned against the new motion barrier in further posterior iliac rotation. The process is repeated 2 additional times for a total of 3 repetitions. Following the MET, the patient is reassessed for signs of improvement.

There are at least 2 mechanisms to explain the realignment of the bony pelvis following the application of muscle energy. The first is related to the neurophysiologic effect of hip extensor contraction (ie, the gluteus maximus and hamstrings). Through reciprocal inhibition, contraction of the hip extensors reduces tone in the hip flexors. This reduction in tone of the TRI muscles allows the iliac bone to "derotate" in a posterior direction and resume its normal anatomic relationship with the sacrum. The second mechanism involves the kinesiologic effect of working muscles in "reverse action." In this case, an isometric contraction of the hip extensors with fixation of the distal insertion will cause movement at the proximal origin. This therapeutic movement will theoretically realign the iliac bone from an anteriorly rotated position into its normal relationship with the sacrum.

Side Lying MET for Anterior Iliac Rotation on the Left

The patient lies on the unaffected right side. The therapist localizes left posterior iliac rotation through the left lower limb to the restrictive barrier by placing the fingers of his or her left hand in the sacral sulcus just medial to the left PSIS (Figure 23-2). When the "feather edge" of the restrictive barrier in the direction of posterior iliac rotation is reached, the patient's left foot is placed over his or her right knee and kept there (slight adduction of the left thigh is helpful in decompressing the left iliosacral joint posteriorly). The lumbar spine is then "locked" by rotating

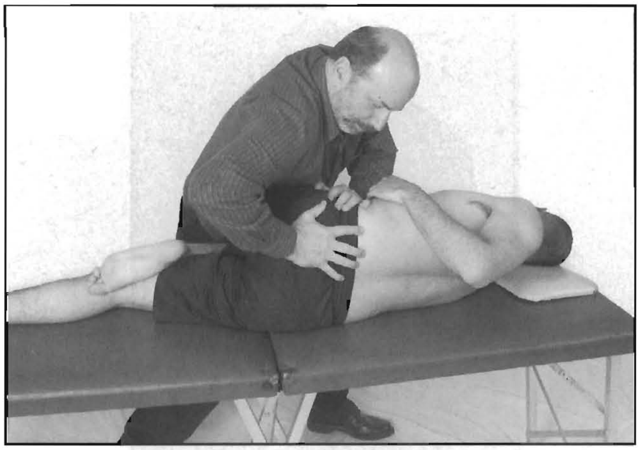

Figure 23-3a. Manipulation for anterior iliac rotation left upper SI joint.

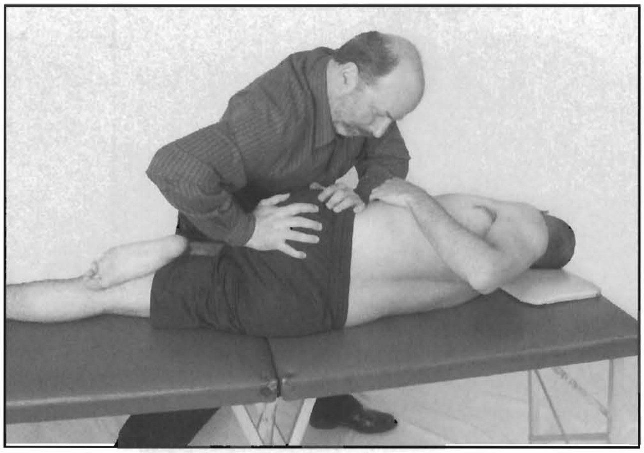

Figure 23-3b. Manipulation for anterior iliac rotation left lower SIJ.

it left via the right arm from above down, including the lumbosacral junction.

The MET is performed by having the patient perform a submaximal isometric contraction of the left hip extensors against the therapist's right hand. The appropriate instruction is, "Don't let me move your left knee up." Following a 6-second contraction, the ilium is relocalized through further hip flexion against the new barrier as determined through palpation at the sacral sulcus. This process is repeated a total of 3 times, and the patient is immediately reassessed.

Side Lying Manipulation for Left Anterior Iliac Rotation

If additional force is required to reduce the anterior iliac subluxation, the patient is then manipulated in the side-lying position as follows:

1. The patient remains in the same side-lying position (Figure 23-3a) as for the MET above (ie, lumbar spine is "locked" through ligamentous tension in rotation left with the left iliac bone up against its restrictive barrier in posterior rotation). However, the amount of left hip flexion will vary slightly depending on which pole is being mobilized.
2. For a left upper pole impairment of posterior iliac rotation, the left PSIS is engaged with the therapist's right pisiform contact; for a lower pole impairment of posterior iliac rotation, the patient's left ischial tuberosity is engaged with the therapist's right pisiform contact (Figure 23-3b). The left hand makes contact with the ASIS regardless of which pole is being mobilized.
3. The manipulation involves a simultaneous "push" with both hands, causing posterior iliac rotation to occur, as if "turning a wheel," at the restrictive barrier. The posterior rotation can be graded 1 through 4, as indicated, for up to 1 minute with 1 or 2 brief pauses along the way.

Posterior Iliac Rotation: Muscle Energy/Manipulation

- Lesion: Left posterior iliac rotation
- Motion restriction: Left anterior iliac rotation
- C (Chief Complaint):
  - Pain usually localized to the left sacroiliac joint and ipsilateral buttock
  - Pain described as deep, achy, sore, tight, etc
  - Pain may be referred into the left posterior thigh but not generally below the knee as with radicular pain (symptoms in the contralateral SI joint are often experienced, possibly due to compensation)
- H (History):
  - Repeated unilateral standing on the left side
  - Fall on the left buttock in trunk flexion
  - Vertical thrust through the extended left leg
  - Lifting in the forward bent position with the knees locked
  - Female intercourse strain with the hips flexed
- A (Asymmetry of Bony Landmarks):
  - Left PSIS is lower than the right in all positions (possibly posterior also)
  - Left ASIS is higher than the right in all positions (possibly posterior also)
  - Left medial malleolus is shorter than the right in supine
- R (Range of Motion):
  - Standing flexion test is positive on the left
  - "Reverse stork" is positive on the left at the upper and/or lower pole

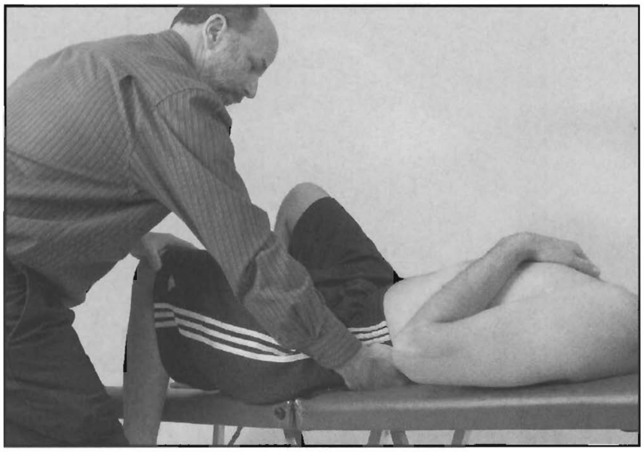

Figure 23-4. MET for left posterior iliac rotation in supine.

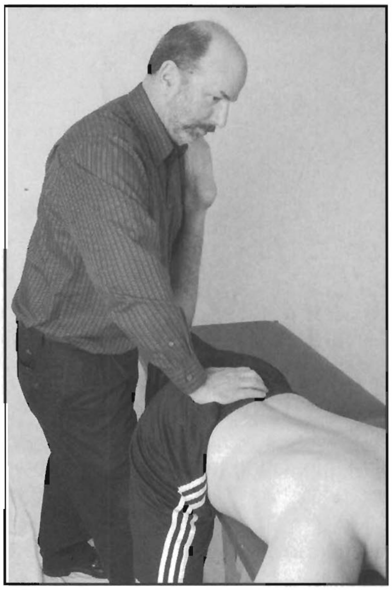

Figure 23-5. MET for left posterior iliac rotation in prone.

- » Long sitting test is positive with the left medial malleolus changing from short to long as the patient moves from supine to long sitting
- » Restricted left hip internal rotation

➤ T (Tissue Texture Abnormality):

- » Left sacrotuberous ligament is taut and tender
- » Left hamstrings may be hypertonic, tight, and tender
- » Baer's SI point tender on the left

➤ S (Special Tests):

- » SI joint provocation tests are positive on the left
- » Normal neurological exam
- » Shortened stride length on the left

Two muscle energy techniques and one joint manipulation will be described and illustrated for a left-sided posterior iliac rotation misalignment. The left side has been chosen because of the higher incidence of posterior iliac rotations on the left. To correct this impairment on the right side, all manual contacts and directions are simply reversed.

Supine MET for Posterior Iliac Rotation on the Left

The therapist stands on the patient's left side. The patient's right foot is placed flat on the treatment table with the right knee comfortably flexed (Figure 23-4). While monitoring left iliosacral motion at the sacral sulcus with the fingers of the right hand, the therapist lowers the left thigh from a flexed position until the "feather edge" of the restrictive barrier in anterior iliac rotation is reached. Three seconds after a 6-second isometric contraction of the iliopsoas, the iliac bone is relocalized to the new motion barrier by lowering the thigh in the direction of hip extension. Following 3 repetitions of MET, the pelvis is reassessed. To lessen the therapist's effort, his or her left thigh can assist with the support of the patient's left lower limb. This is especially helpful when the patient is larger than the therapist. To provide further assistance, a stool can be placed under the therapist's left foot.

The correction of posterior iliac rotation with MET can be explained both neurologically as well as mechanically. Neurologically, an isometric contraction of the iliopsoas muscle will decrease tone in the hamstrings through reciprocal inhibition, allowing the ilium to reposition itself more ideally on the sacrum. Mechanically, an isometric contraction of the iliopsoas with distal fixation will anteriorly rotate the ilium against its restrictive barrier, thus normalizing iliosacral alignment.

Prone MET for Posterior Iliac Rotation on the Left

The therapist stands on the patient's right side with the patient positioned in prone lying. To stabilize the right ilium, the patient's right foot is placed on the floor or on a stool with the right hip flexed between 75 to 90 degrees. The therapist monitors left iliosacral motion with the fingers of his or her right hand, while the patient's left thigh (flexed at the knee) is extended and slightly adducted in order to reach the restrictive barrier of left anterior iliac rotation (Figure 23-5). At the beginning of the restrictive barrier, 3 repetitions of MET are applied using the hip flexors isometrically. The following are crucial factors in performing an effective iliosacral MET:

1. Precise localization to the first barrier sensed (ie, the "feather edge"). A forceful engagement of the restrictive barrier may result in muscle hypertonicity
2. Counterstability of the contralateral ilium
3. Controlled submaximal isometric contraction for 6 seconds, which ramps up and down slowly

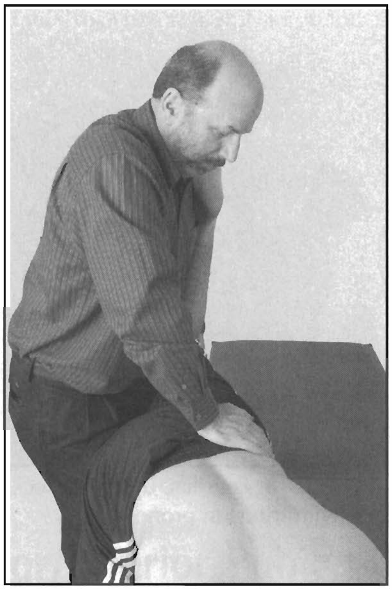

Figure 23-6a. Manipulation for posterior iliac rotation left upper SI joint.

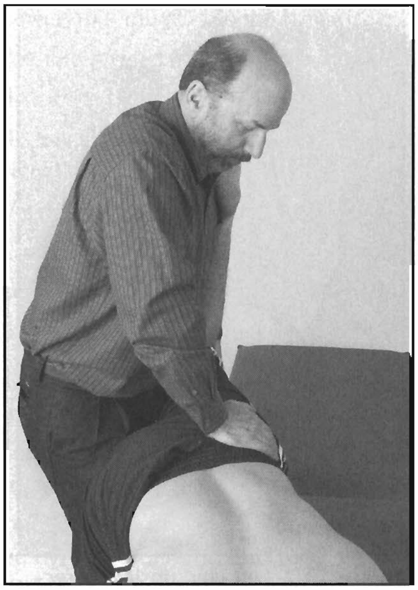

Figure 23-6b. Manipulation for posterior iliac rotation left lower SI joint.

4. Postcontraction relaxation for up to 3 seconds
5. Precise relocalization to the new motion barrier
6. Sensitivity to patient comfort at all times

Prone Manipulation for Left Posterior Iliac Rotation

When additional force is required to reduce a "stubborn" impairment, the patient is then manipulated as follows:

1. The patient remains in the same position as for the prone MET (ie, prone with the right foot on the floor). However, left hip position will differ depending on which pole is being mobilized.
2. For restriction at the upper pole of the left iliosacral joint (Figure 23-6a), the therapist's right pisiform contact performs a mobilization over the left iliac crest in an anterior, superior, and lateral direction as the left extended/adducted thigh maintains the position of the left iliac bone at the restrictive barrier. For a lower pole impairment (Figure 23-6b), the manual contact is over the left PSIS and mobilized in the same 3 directions (ie, anterior, superior, and lateral).
3. A graded mobilization is performed for up to 1 minute with 1 or 2 brief pauses along the way.

Iliac Upslip (Superior Iliac Shear): Manipulation

- Lesion: Left iliac upslip (superior shear)
- Motion restriction: Inferior iliac shear
- C (Chief Complaint):
  - Traumatically induced left SI pain localized to the SI joint or referred distally into the left buttock and thigh (pain is often experienced on the unaffected side as well)
- H (History):
  - Vertical fall on the left ischium
  - Unexpected step off a curb or "missed" step on stairs onto the left leg
- A (Asymmetry of Bony Landmarks):
  - Left PSIS is higher than the right in all positions
  - Left ASIS is higher than the right in all positions
  - Left iliac crest is higher than the right in all positions
  - Left ischial tuberosity is 6 mm higher (thumb width) than the right in prone lying
  - The greater trochanters are level in standing
- R (Range of Motion):
  - Standing flexion test is positive on the left
  - Stork test is positive on the left at the upper and/or lower pole
  - "Reverse stork" is positive on the left at the upper and/or lower pole
- T (Tissue Texture Abnormality):
  - Left sacrotuberous ligament is lax
  - Left quadratus lumborum may be hypertonic, tight, and tender
  - Left gluteus medius may become lengthened and tender.

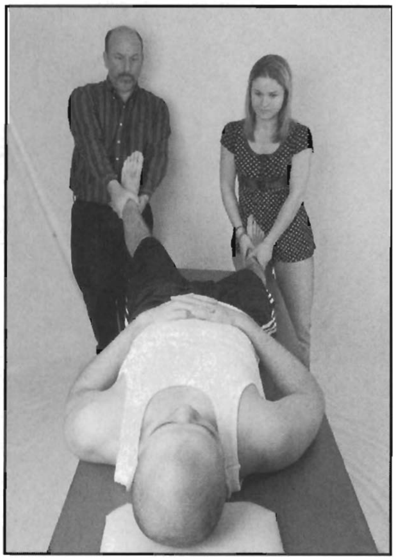

Figure 23-7. Left iliac upslip correction with thrust in supine.

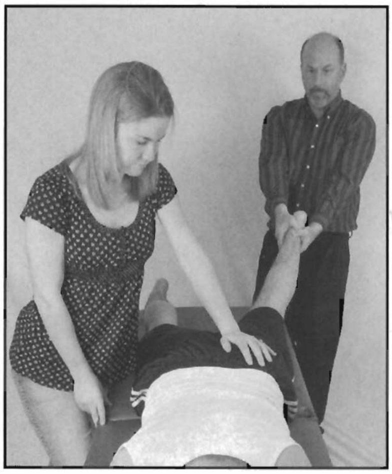

Figure 23-8. Left iliac upslip correction with thrust in prone.

➤ S (Special Tests):
  » Baer's SI point tender on the left
  » SI joint provocation tests are positive on the left
  » Normal neurological exam
  » Antalgic gait with weight bearing on the left

Unlike the rotatory impairments of the iliosacral joint, muscle energy is ineffective. This is because the underlying fixation is more articular in nature than myofascial. In fact, grade 1 to 4 mobilization is also inadequate because the ilium often becomes "locked" onto the sacrum, requiring a strong manipulative force to "unlock" it. In terms of potential injury, the risk-to-benefit ratio overwhelmingly supports the use of the long axis thrust; if taught properly, there is only minimal risk to the patient. Regarding the side selected, there is no indication that iliac upslips are more prevalent on one side than the other. The examination findings and intervention are simply reversed for an iliac upslip on the right.

Supine Manipulation of an Iliac Upslip on the Left

The efficacy of this manipulative thrust is markedly increased when an assistant is used to stabilize the contralateral ilium. The technique can be broken down as follows:

1. The therapist grasps the patient's left ankle just proximal to the malleoli.
2. The iliosacral joint is placed in its loose-packed position by abducting the left hip 10 to 15 degrees.
3. To ensure that the long axis thrust affects the iliosacral joint and not the hip, the therapist close packs the abducted hip by internally rotating it to end-range.
4. The assistant positions his or her thigh against the patient's right foot to prevent inferior movement of the right lower limb.
5. The therapist performs a grade 3 long axis traction maneuver through the left lower extremity by leaning back with extended arms.
6. Without giving up the strong long axis traction, the therapist leans forward by flexing the elbows. On the count of "3," the patient is told to cough (a cough theoretically distracts the SI joints momentarily), at which time the therapist imparts a quick thrust through the leg (Figure 23-7). If done correctly, the iliac upslip is reduced, often but not always, with an associated "thud" or "clunk." When practicing this maneuver on normal subjects, the amount of force should be kept to a minimum lest an inferior iliac shear (ie, downslip) results.

Prone Manipulation of an Iliac Upslip

If the supine thrust maneuver fails to reduce the superiorly sheared iliac bone, a thrust manipulation is attempted in the prone position. The assistant stabilizes the sacrum at its inferior lateral angle, on the affected side, while the therapist thrusts through the ipsilateral lower limb as illustrated (Figure 23-8). As with the supine technique, the affected SI joint is loose packed while the ipsilateral hip is close packed. The manipulative thrust is once again performed in conjunction with the patient's cough as with the supine procedure.

If an upslip has occurred, there is often associated laxity of the SI ligaments. Consequently, SI joint belt fixation is recommended postmanipulation to allow for proper healing and restabilization. If the SI joint remains unstable despite prolonged belt fixation, a course of prolotherapy[38] is indicated. This physician-based intervention involves the injection of proliferant solutions into the ligaments for the purpose of stimulating the proliferation of collagen, thereby enhancing joint stability.

Prior to performing the manipulative thrust described above, the therapist should review the contraindications to spinal manual therapy listed in Chapter 3. Regarding the pregnant patient with mechanical impairment of the pelvic girdle, the author recommends that novice practitioners not perform the thrust maneuver described previously. However, gentle MET can be safely and effectively applied to rotatory impairments throughout a woman's pregnancy.

Though presented as separate and distinct impairments, it is important to appreciate that anterior and posterior iliac rotations are sometimes superimposed on an iliac upslip. When this is the case, the upslip should always be managed first.

Superior/Inferior Pubic Shears: "Shotgun" Technique

- Lesion 1: Right inferior pubic shear ("Down pube")
- Motion restriction: Superior pubic shear on the right
- C (Chief Complaint):
  - Pain and tenderness over the medial attachment of the right inguinal ligament
  - Diffuse and variable pain reference over the right SI joint, anterior groin, and thigh
- H (History):
  - During a soccer kick, the foot hits the ground rather than the ball, shearing the pubic symphysis inferior on the ipsilateral side
  - Common in postpartum females
- A (Asymmetry of Bony Landmarks):
  - Right pubic tubercle is inferior versus the left
- R (Range of Motion):
  - Standing flexion test is positive on the right
- T (Tissue Texture Abnormality):
  - Tenderness at the medial attachment of the right inguinal ligament
  - Right hip adductors may be hypertonic, tight, and tender
- S (Special Tests):
  - Normal neurological examination
  - Antalgic gait with weightbearing on the right
- Lesion 2: Left superior pubic shear ("Up pube")
- Motion restriction: Inferior pubic shear on the left
- C (Chief Complaint):
  - Pain and tenderness over the medial attachment of the left inguinal ligament
  - Lower abdominal discomfort on the left
  - Possible secondary symptoms at the left SI joint
- H (History):
  - Rectus abdominis asymmetry (ie, tight left, weak right)
  - Vertical fall on the left ischium
- A (Asymmetry of Bony Landmarks):
  - Left pubic tubercle is superior versus the right
- R (Range of Motion):
  - Standing flexion test is positive on the left
- T (Tissue Texture Abnormality):
  - Tenderness at the medial attachment of the left inguinal ligament
  - Left distal insertion of the rectus abdominis may be hypertonic, tight, and tender
- S (Special Tests):
  - Normal neurological examination
  - Antalgic gait with weight bearing on the left
  - Resisted trunk flexion provokes local pain at the left symphysis pubis

Experienced manual therapists usually agree on common patterns of impairment in the pelvis (eg, more right anterior iliac rotations than left; more posterior iliac rotations on the left; more inferior pubic shears on the right) Why we see these patterns in the pelvis is unclear, but this author is of the opinion that the asymmetric demands of driving a car with an automatic transmission have something to do with it.

Though specific techniques for each of the pubic shear dysfunctions are available, there is one technique that has the ability to correct the alignment of the pubic symphysis in either case. This procedure is referred to as the "shotgun" or "blunderbuss" technique.[15]

"Shotgun" Technique for Superior/Inferior Pubic Shears

There are 2 phases to this muscle energy technique. The first phase involves a contraction of the hip abductor muscles, while the second uses an isometric contraction of the adductors. Although it is the adductor contraction that realigns the pubic symphysis (sometimes with an associated "click" or "pop"), the abductor contraction beforehand markedly enhances the efficacy of this technique. Theoretically, through reciprocal inhibition, an isometric contraction of the abductors relaxes and resets the tone in the adductors for a more optimal contraction. By applying distal resistance at the knees, the therapist forces the adductor muscles to pull the inferior pubic rami laterally, causing

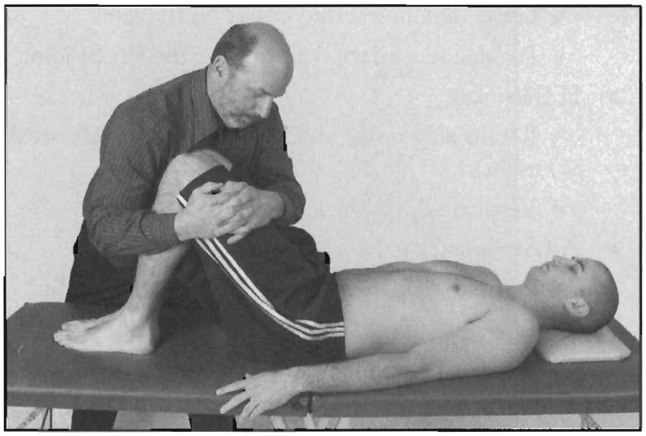

Figure 23-9a. Shotgun technique stage 1.

a momentary distraction of the pubic symphysis. It is during this distraction that superior and inferior pubic shears often correct. An audible "pop" is not necessary for a correction to occur, but patients often see this as a confirmation that the problem has been "fixed."

To perform the "shotgun" technique, the supine patient's hips and knees are flexed with both feet on the table. The therapist then grasps both knees in the adducted position and resists hip abduction 3 times for a count of 6 seconds each (Figure 23-9a). The therapist then places his or her forearm between the patient's knees and asks for a strong bilateral adductor contraction for 3 to 6 seconds (Figure 23-9b). One contraction is often sufficient, but a second one can be attempted if the therapist chooses. The patient is immediately reassessed for signs of improvement.

Before proceeding to the final chapter on the pelvic girdle (ie, therapeutic and home exercises), 2 essential concepts need to be addressed. The first is related to the issue of treatment sequencing in the patient with somatic impairment of the lumbar-pelvic-hip complex, while the second deals briefly with the interrelationship between the lumbopelvic region and the lower extremity.

The recommended manual therapy treatment sequence for a lumbar-pelvic-hip impairment is as follows:

1. Direct fascial technique and stretching of the tight postural muscles of the hip joints.
2. Mobilization/manipulation of the hips (not covered in this text.
3. Myofascial release/direct fascial technique of the lower thoracic/lumbar spine.
4. Mobilization/manipulation of the lower thoracic/lumbar spine (type 2, non-neutral impairment not covered in this text).
5. Myofascial release/direct fascial technique of the pelvic diaphragm.

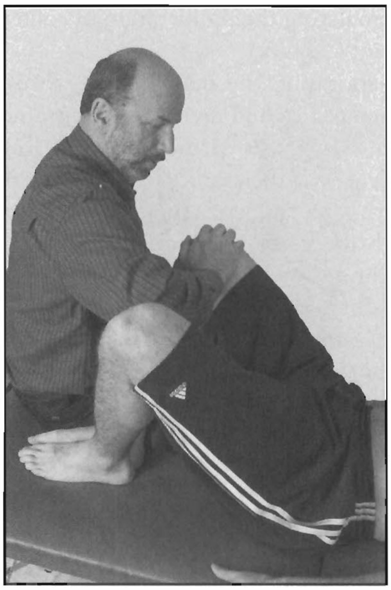

Figure 23-9b. Shotgun technique stage 2.

6. Correction of superior/inferior pubic shears.
7. Correction of superior/inferior iliac shears (inferior is rare).
8. Correction of SI impairment (not covered in this text: unilateral shears, forward/backward torsions, and bilateral nutation/counternutation).
9. Correction of anterior and posterior iliac rotations.
10. Recovery of core stability.

Priority is always given to a McKenzie derangement, as it can be the most disabling and the most serious of the mechanical afflictions of the lumbopelvic region.

Regarding the interrelationship between the lumbopelvic region and the lower extremity, it must be appreciated that the pelvis is the mechanical link between the lower limb and the trunk. Whether analyzing function or impairment of the hip, knee, foot, and ankle, one must always consider the role of the lumbopelvic region; when analyzing function or impairment of the lumbopelvic region, one must always consider the role of the muscles and joints of the lower extremity. To examine and treat each part independent of the whole is to deprive the patient of proper care. In fact, the author would say that it is irresponsible. Specializing in the foot and ankle or in the spine and pelvis is not the problem. The problem is when one fails to recognize the "unity of the body" and its total interdependence. The author has seen many low back patients helped when attention was paid to their lower extremity impairment and vice versa. It all comes back to the philosophy that finding and managing the source of the problem (ie, the AGR) will yield great dividends in the end!

24

Therapeutic and Home Exercises for the Pelvic Girdle

In order to maintain proper alignment of the pelvic joints following manual therapy, the patient is expected to be diligent with his or her home program. As with the other areas covered thus far, successful outcomes are dependent upon the patient's willingness to commit to the home program. The home exercises described and illustrated are simple to instruct and perform. However, not unlike taking medicine, the patient must strictly adhere to the regimen.

Pubic Shear Dysfunction: Self-Correction

To maintain proper alignment of the pubic symphysis, the patient performs a modification of the "shotgun" technique in the sitting position (Figure 24-1a). Following three 6-second isometric abductor contractions, the patient contracts the hip adductors against the unyielding resistance of his or her arm for 3 to 6 seconds (Figure 24-1b). This cycle can be repeated 2 to 3 times and performed every few hours as needed to maintain normal alignment until stability has returned to the region.

Anterior Iliac Rotation: Self-Correction

To self-correct the right side (Figure 24-2), the supine patient is instructed to make use of 2 muscle groups. By performing simultaneous isometric contractions of the right hip extensors and the left hip adductors, the corrective force is imparted to the right iliosacral joint (the hip adductors should not be used in the presence of inferior pubic shear dysfunction). The patient is instructed to bring the right knee to his or her chest and interlock his or her fingers behind the posterior thigh while the left foot engages the left side of the table. As the patient pushes his or her right thigh into the interlocked fingers, the left foot is pushed into the side of the table for a count of 6 seconds and repeated 2 to 3 times every few hours until stability returns to the region. The purpose of the left-sided hip adductor contraction is to provide counterstability as the right ilium is being mobilized.

Posterior Iliac Rotation: Self-Correction

To self-correct impairment on the left side, the patient activates the left hip flexors isometrically. This is done in prone lying with the right foot on the floor as illustrated (Figure 24-3). A bed or portable treatment table can be used for this purpose. The left hip flexors will theoretically decrease muscle tone of the left hamstrings, mechanically derotate the left innominate from its posteriorly rotated position into neutral alignment, and maintain correct alignment to allow healing to occur. The contraction is performed for 6 seconds and repeated 2 to 3 times every few hours.

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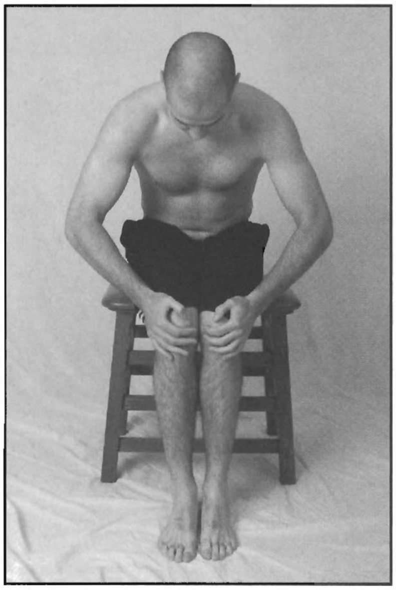
Figure 24-1a. Self-shotgun stage 1.

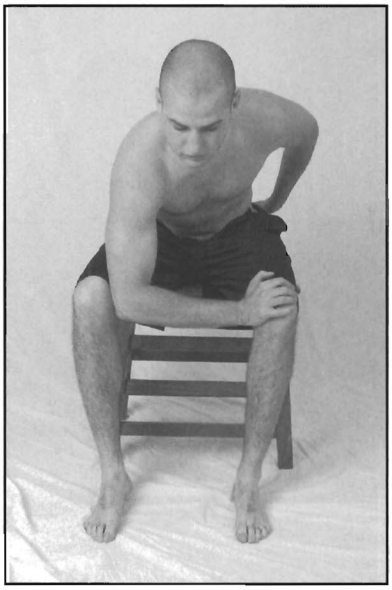
Figure 24-1b. Self-shotgun stage 2.

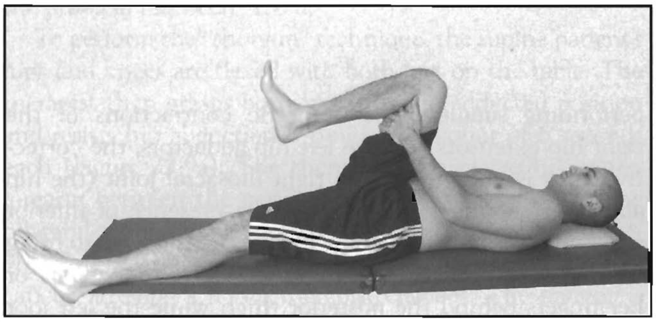
Figure 24-2. Self-correction right anterior iliac rotation.

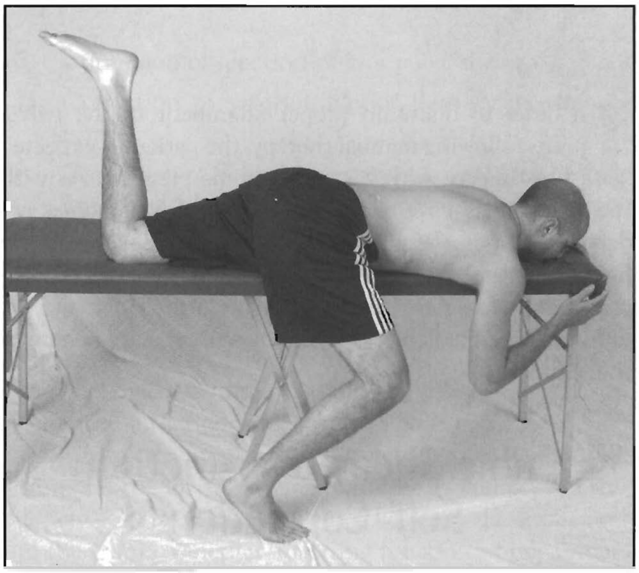
Figure 24-3. Self-correction left posterior iliac rotation.

Piriformis Self-Stretch

In keeping with the changing mechanics of the piriformis muscle below and above 60 degrees of hip flexion,[26,27] the patient is instructed to stretch the muscle in 2 positions as illustrated (Figure 24-4a and 24-4b). Below 60 degrees of flexion, the supine patient stretches the left side by placing his or her left foot on the outside of the right knee with the foot flat. The left knee is then pulled across the body so that the left hip is flexed, adducted, and internally rotated (see Figure 24-4a). The patient holds this position for a count of 30 seconds and repeats the stretch 3 times, several times per day.

To stretch the left piriformis muscle above 60 degrees of flexion, the supine-lying patient places his or her left foot above the right knee. The patient's interlocked fingers then pull the right posterior thigh toward the chest while the supported left hip is flexed, adducted, and externally rotated (see Figure 24-4b). The stretch is held for 30 seconds and repeated 3 times, several times per day. The patient must not feel discomfort in the groin and must be careful to stop the stretch should radicular symptoms be perceived in the lower extremity. If the stretch is performed too vigorously, compression of the sciatic nerve can occur. These stretches can be repeated on the right side as needed.

Iliopsoas Self-Stretch

The patient is positioned in the half-kneeling position with the right knee flexed and the right foot flat (Figure 24-5). To stretch the left iliopsoas muscle, the patient repositions the lumbar spine in PPT and the left hip in internal rotation and extension. The stretch is achieved by having the patient "lean into" the left anterior hip and thigh without arching the low back. For those patients who desire a more aggressive stretch, trunk side bending to the right can

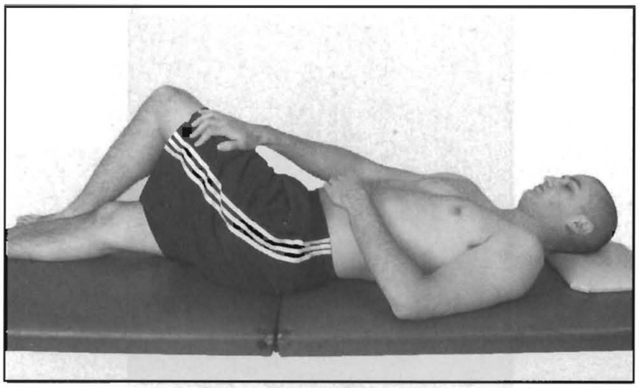

Figure 24-4a. Left piriformis self-stretch below 60 degrees.

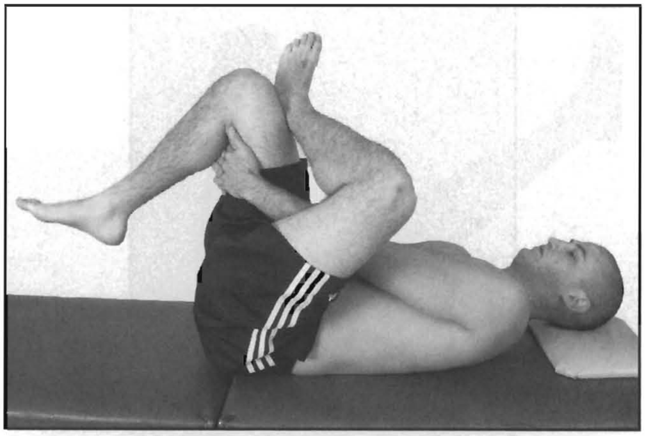

Figure 24-4b. Left piriformis self-stretch above 60 degrees.

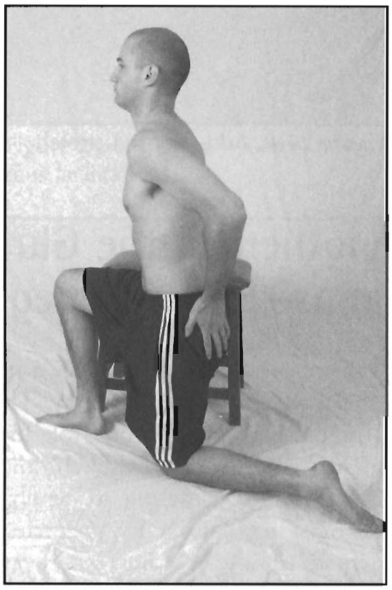

Figure 24-5. Left iliopsoas self-stretch.

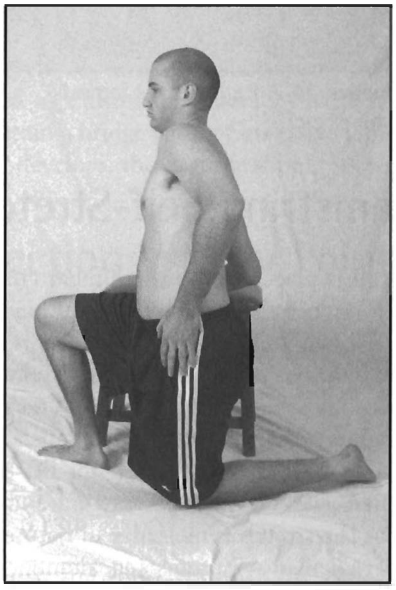

Figure 24-6. Left TFL self-stretch.

be added with the left arm overhead. The stretch is held for up to 30 seconds and repeated 3 times, several times per day. The patient can switch sides and repeat on the right. For patients who have difficulty with kneeling because of knee pain, a pillow can be placed under the affected knee. If this is still problematic, the patient can perform the TRI muscle stretch over the end of a bed.

Tensor Fascia Latae Self-Stretch

The half-kneeling position is again utilized, but the prepositioning of the hip is different than for the iliopsoas. To stretch the left TFL muscle, the left hip is positioned in external rotation and extension while the lower abdominals and gluteus maximus maintain a PPT (Figure 24-6). The stretch is felt in the anterolateral aspect of the left hip as the patient translates his or her hips from right to left. Isaacs and Bookhout[15] suggest that the patient place his or her right hand on a chair for support. The stretch is brought to the point of perceived tightness, but not to the point of pain, and held for 30 seconds. Each stretch is repeated 3 times and performed several times per day. The same stretch is performed on the right side as necessary.

Rectus Femoris Self-Stretch

The right rectus femoris is effectively stretched by having the patient place his or her right foot on a surface as illustrated and then performing a PPT until a stretch is felt in the anterior thigh (Figure 24-7). The stretch can be made stronger by simply lowering the body by means of increased left knee flexion. To maximize the stretch, the lumbar spine should not be allowed to extend (ie, maintain a neutral spine). The stretch is held for 30 seconds and repeated 3 times. The left rectus femoris can be stretched similarly as needed.

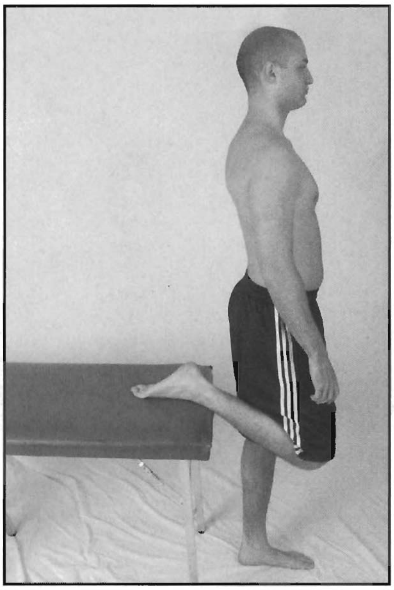

Figure 24-7. Right rectus femoris self-stretch.

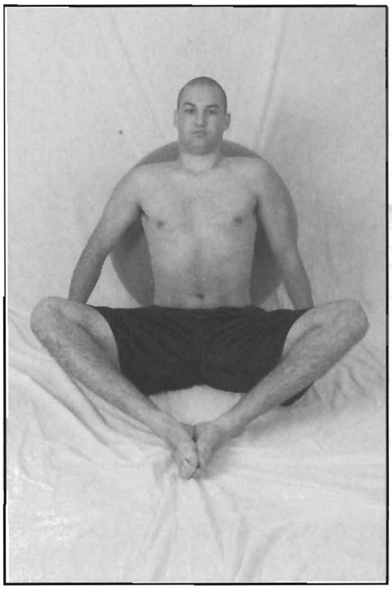

Figure 24-8. Adductor self-stretch.

Hamstring Self-Stretch

A towel roll is placed under the lumbar curve to maintain a neutral lordosis. To stretch the right hamstrings (not illustrated), the supine patient places the right hip and knee in the 90/90 position with fingers interlocked behind the right knee and the left leg straight. The patient then actively straightens the right leg until a satisfactory stretch is experienced. The stretch is held for 30 seconds and repeated 3 times, several times per day. Care must be taken to ensure that the stretch is muscular in nature and that at no time are radicular symptoms (eg, numbness, tingling) felt in the right leg. The left hamstrings are then stretched as indicated.

Hip Adductor Self-Stretch

This stretch (Figure 24-8) appears in the text, *Bourdillon's Spinal Manipulation, 6th Edition.*[15] The patient sits with his or her back to the wall, maintaining a neutral lordosis. The soles of the feet are brought together as the hips are abducted and externally rotated. The patient's hands are placed on the floor behind his or her hips to assist in lifting and anteriorly rotating the pelvis. The patient slowly and carefully performs an anterior pelvic tilt and immediately feels the stretch in the groin area, bilaterally. The stretch is held for 30 seconds and repeated 3 times, several times a day. Care must be taken not to overstretch!

Strengthening the Gluteus Maximus—Bridging Regimen

The final therapeutic exercise involves a strengthening regimen of the gluteus maximus muscles. These primary extensors of the hip play an important role in ambulation, running, and jumping. Patients with chronic LBP often demonstrate weakness of these important phasic muscles. Patients who have problems with recurring hamstring strains often have tight hamstrings as a consequence of substituting for weak gluteal muscles. Once the hamstrings have been manually "released" and stretched, it is important to retrain the gluteus maximus.

Yerys et al[39] have demonstrated the connection between anterior hip joint mobility and gluteus maximus strength. In keeping with the arthrokinetic reflex discussed previously in this text, the gluteus maximus muscle should not be retrained until the requisite degree of hip extension is present. Consequently, it once again behooves the therapist to mobilize impaired joint structures before strengthening inhibited and weak muscles.

Prior to commencing the bridging exercises, the patient may require a remedial session in gluteus maximus recruitment. One simple way of isolating these muscles is to have the prone patient resist bilateral isometric hip external rotation by pushing both feet together as illustrated (Figure 24-9). Once the sensorimotor connection has been made, the patient is ready to move on.

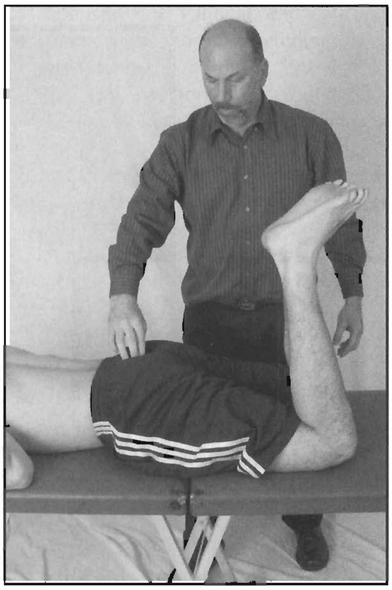

Figure 24-9. Bilateral gluteus maximus facilitation.

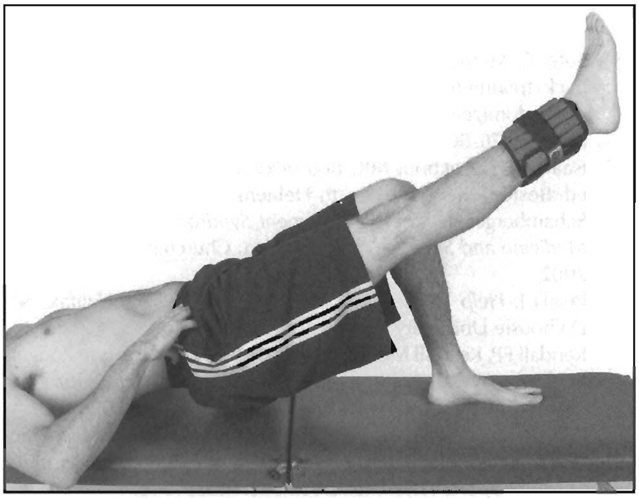

Figure 24-10b. Single-leg bridging with ankle weight.

The hook-lying patient is instructed to perform a PPT followed by a bridge maneuver involving hip and not lumbar extension. The patient is also instructed to place both hands over the ASISs so that the pelvis remains level at all times. The bridge is held for a count of 5 to 10 seconds and repeated 10 times, 3 times per day.

The more demanding phase of the bridging regimen involves straightening one leg at a time for 5 to 10 seconds while maintaining a level pelvis as per ASIS palpation (Figure 24-10a). With time and improved strength/endurance, the extended leg can be held for up to 30 seconds. This can be incorporated into motion as the patient makes the letters of the alphabet with his or her feet (A through M with one foot; N through Z with the other), while maintaining a level pelvis. For athletes, dancers, etc, an ankle weight can be added as illustrated (Figure 24-10b). If the patient cannot maintain a bridge without excessive lumbar extensor tone, or LBP develops, the exercise should be terminated.

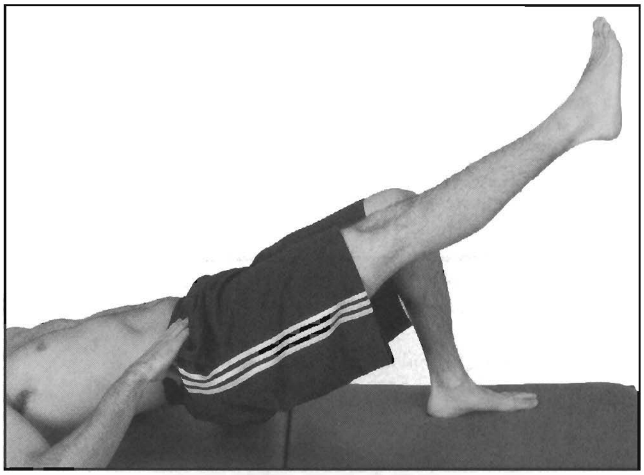

Figure 24-10a. Single-leg bridging.

Section VI: Key Points

1. The SI joint has the capacity for motion throughout the lifespan, but this motion is quite small and some authors question the clinician's ability to accurately assess it.
2. Laslett's SI provocation tests are based upon sound science and are useful in ruling out the SI joint as a source of LBP.
3. Vleeming and Lee's use of form and force closure are extremely useful concepts in the management of patients with impairment of the SI joint.
4. The osteopathic distinction between iliosacral and SI dysfunction is a clinically useful concept, but one that is more consistent with theory and not based upon scientific validation. Regarding this topic, sacral dysfunction is not covered in this text as it requires an advanced understanding of theoretical osteopathic biomechanics. This author fully endorses and recommends the learning of this valuable information for those dedicated to managing patients with LBP unresponsive to the techniques covered herein.
5. For those patients with SI hypermobility refractory to manual therapy and core training, the use of SI joint belts should be considered. If belt stabilization is unable to control the excess motion, a physician trained in the use of prolotherapy should be consulted.

References

1. Goode A, Hegedus EJ, Sizer Jr P, Brismee JM, Linberg A, Cook CE. Three-dimensional movements of the sacroiliac joint: a systematic review of the literature and assessment of clinical utility. *J Manual Manipulative Ther.* 2008;16(1):25-38.
2. Vleeming A, Wingerden JP, van Dijkstra PF, Stoeckart R, Snijders CJ, Stijnen T. Mobility in the SI-joints in old people: a kinematic and radiologic study. *J Clin Biomech.* 1992;7:170-176.
3. Vleeming A, Mooney V, Dorman TA, Snijders CJ, Stoeckart R. *Movement, Stability and Low Back Pain: The Essential Role of the Pelvis.* New York, NY: Churchill Livingstone; 1997.
4. Greenman PE. *Principles of Manual Medicine.* 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2003.
5. Butler DS, Moseley GL. *Explain Pain.* Adelaide, Australia: Noigroup Publications; 2003.
6. Hodges P. Chronic low back and coccygeal pain. In: Jones MA, Rivett DA, eds. *Clinical Reasoning for Manual Therapists.* Edinburgh: Butterworth-Heinemann; 2004.
7. Wainner RS, Whitman, JM, Cleland JA, Flynn TW. Regional interdependence: a musculoskeletal examination model whose time has come. *J Orthop Sports Phys Ther.* 2007;37(11):658-660.
8. Vaughn DW. Isolated knee pain: a case report highlighting regional interdependence. *J Orthop Sports Phys Ther.* 2008;38(10):616-623.
9. Lee D. Instability of the sacroiliac joint and the consequences to gait. *J Manual Manipulative Ther.* 1996;4(1):22-29.
10. DonTigny RL. Mechanics and treatment of the sacroiliac joint. *J Manual Manipulative Ther.* 1993;1(1):3-12.
11. Vaugh HT, Nitsch W. Ilial anterior rotation hypermobility in a female collegiate tennis player. *Phys Ther.* 2008;88(12):1578-1590.
12. Cibulka MT, Delitto A, Koldehoff RM. Changes in innominate tilt after manipulation of the sacroiliac joint in patients with low back pain, an experimental study. *Phys Ther.* 1988;68(9):1359-1370.
13. Mathews JH. A new approach to the treatment of osteoarthritis of the knee: prolotherapy of the ipsilateral sacroiliac ligaments. *AM J Pain Management.* 1995;5:91-93.
14. Suter E, McMorland G, Herzog W, Bray R. Conservative lower back treatment reduces inhibition in knee-extensor muscles: A randomized controlled trial. *J Manipulative Physiol Ther.* 2000;23:76-80.
15. Isaacs ER, Bookhout MR. *Bourdillon's Spinal Manipulation.* 6th ed. Boston, MA: Butterworth-Heinemann; 2002.
16. Schamberger W. *The Malalignment Syndrome: Implications for Medicine and Sport.* Printed in China: Churchill Livingstone; 2002.
17. Prsala J. *Help Yourself to a Healthy Back.* 2nd ed. Halifax, NS: Dalhousie University Printing Center; 2001.
18. Kendall FP, Kendall McGreary E, Provance PG, McIntyre Rodgers M, Romani WA. *Muscles: Testing and Function With Posture and Pain.* 5th ed. Baltimore, MD: Williams & Wilkins; 2005.
19. Powers CW. The influence of altered lower-extremity kinematics on patellofemoral joint dysfunction: a theoretical perspective. *J Orthop Sports Phys Ther.* 2003;33(11):639-646.
20. Dierks TA, Manal KT, Hamill J, Davis IS. Proximal and distal influences on hip and knee kinematics in runners with patellofemoral pain during a prolonged run. *J Orthop Sports Phys Ther.* 2008;38(8):448-456.
21. Tiberio D. The effect of excessive subtalar joint pronation on patellofemoral mechanics: A theoretical model. *J Orthop Sports Phys Ther.* 1987;9(4):160-165.
22. Hertling D, Kessler RM. *Management of Common Musculoskeletal Disorders: Physical Therapy Principles and Methods.* 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2006.
23. Schafer RC, Faye LJ. *Motion Palpation and Chiropractic Technique: Principles of Dynamic Chiropractic.* 2nd ed. Huntington Beach, CA: The Motion Palpation Institute; 1989.
24. Magee DJ. *Orthopedic Physical Assessment.* 5th ed. St. Louis, MO: Saunders Elsevier; 2008.
25. Vleeming A, Pool-Goudzwaard AL, Hammudoghlu D, Stoeckart R, Snijders CJ, Mens JMA. The function of the long dorsal sacroiliac ligament, its implication for understanding low back pain. *Spine.* 1996;21(5):556-562.
26. Delp SL, Hess WL, Hungerford DS, Jones LC. Variation of rotation moment arms with hip flexion. *J Biomechanics.* 1999;32:493-501.

27. Gluck NI, Liebenson CS. Clinical implications of paradoxical muscle function in muscle stretching or strengthening. *J Bodywork and Movement Therapies.* 1997;1(4):219-222.
28. Laslett M, Aprill CN, McDonald B, Young SB. Diagnosis of sacroiliac joint pain: Validity of individual provocation tests and composites of tests. *Man Ther.* 2005;10:207-218.
29. Cook CE, Hegedus EJ. *Orthopedic Physical Examination Tests: An Evidence-Based Approach.* Upper Saddle River, NJ: Pearson Prentice Hall; 2008.
30. Travell JG, Simons DG. *Myofascial Pain and Dysfunction: The Trigger Point Manual, Vol. 2 The Lower Extremities.* Baltimore, MD: Williams & Wilkins; 1992.
31. Manheim C. *The Myofascial Release Manual.* 3rd ed. Thorofare, NJ: SLACK Incorporated: 2001.
32. Tomberlin JP, Saunders HD. *Evaluation, Treatment, and Prevention of Musculoskeletal Disorders. Vol 2.* 3rd ed. Chaska, MN: The Saunders Group; 1994.
33. Ellis JJ. *LPI, Lumbo-Pelvic Integration, A Course Workbook.* Patchogue, NY: 1990.
34. Fland DC, Singleton TN, Conaster RR, et al. The "iliacus test": New information for the evaluation of hip extension dysfunction. *J Am Osteopath Assoc.* 2002;102(3):130-142.
35. Sahrmann SA. *Diagnosis and Treatment of Movement Impairment Syndromes.* St. Louis, MO: Mosby-Year Book; 2002.
36. Cyriax J. *Textbook of Orthopaedic Medicine.* London: Bailliere Tindall; 1978.
37. Makofsky H, Panicker S, Abbruzzese J, et al. Immediate effect of grade IV inferior hip joint mobilization on hip abductor torque: a pilot study. *J Manual Manipulative Ther.* 2007;15(2):103-111.
38. Dorman TA, Ravin TH. *Diagnosis and Injection Techniques in Orthopaedic Medicine.* Baltimore, MD: Williams & Williams; 1991.
39. Yerys S, Makofsky H, Byrd C, Pennachio J, Cinkay J. The effect of mobilization of the anterior hip capsule on gluteus maximus strength. *J Manual Manipulative Ther.* 2002;10(4):218-224

25

Posture, Stability, and the PostureJac

"Observations of the striking influence of postural mechanics on function and symptomatology have led to our hypothesis that posture affects and moderates every physiologic function from breathing to hormonal production. Spinal pain, headache, mood, blood pressure, pulse, and lung capacity are among the functions most easily influenced by posture. The most significant influences of posture are upon respiration, oxygenation, and sympathetic function. Ultimately, it appears that homeostasis and autonomic regulation are intimately connected with posture. The corollary of these observations is that many symptoms, including pain, may be moderated or eliminated by improved posture."

John Lennon, BM, MM; C. Norman Shealy, MD; Roger K. Cady, MD; William Matta, PhD; Richard Cox, PhD; and William F. Simpson, PhD, *American Journal of Pain Management* 1994;4(1)36.

Posture comes from the Latin verb *ponere* which means to put or place.[1] Kuchera and Kuchera[2] define posture as, "The distribution of body mass in relation to gravity over a base of support. The base of support includes all structures from the feet to the base of the skull." According to Peterson Kendall et al,[3] 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 (eg, erect, lying, squatting, or stooping) in which these structures are working or resting. Conversely, they define poor posture as a faulty relationship of the various parts of the body producing increased strain on the supporting structures in which there is less efficient balance of the body over its base of support.[3] The maintenance of normal posture is highly complex. In addition to biomechanical factors, there are neurological mechanisms that also come into play. Pettibon[4] describes 5 righting reactions involved in maintaining ideal head position. They are the labyrinthine reflex, optic reflex, neck righting reflex, body righting reflex #1, and body righting reflex #2. In addition, there are complex feed-forward[5,6] or anticipatory actions (ie, central set) and feedback control mechanisms in the nervous system[6,7] that provide for postural stability, both statically and dynamically. In the realm of bodywork, Bond[8] introduces the concept of "posture zones" of which there are 6 (eg, breathing muscles, abdomen, pelvic floor, hands, feet, and head). She states, "By learning the correct use of each posture zone, you build open stabilization within your body and open orientation to the world around you." Bond compares these posture zones to horizontally-oriented "valves" located along the body's vertical axis that, when "closed" by abnormal tension, "block" gravity's clear path through the body. Similar in thought, the author has coined the phrase, "Gravity should flow through you not to you!"

Although some authors[9,10] question a relationship between postural alignment and musculoskeletal pain, based upon a lack of basic science research, the author of this text is impressed with the number of clinical studies suggesting that such a correlation exists. In fact, the literature points to a strong relationship between forward head/rounded shoulders posture and shoulder impingement,[11-15] temporomandibular disorders,[16-18] and

Makofsky HW
Spinal Manual Therapy, 2nd ed. (pp 219-240)

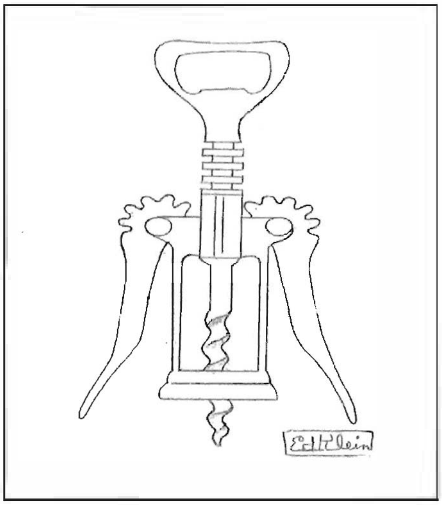

Figure 25-1. Wing-style corkscrew. (Illustration by Ed Klein.)

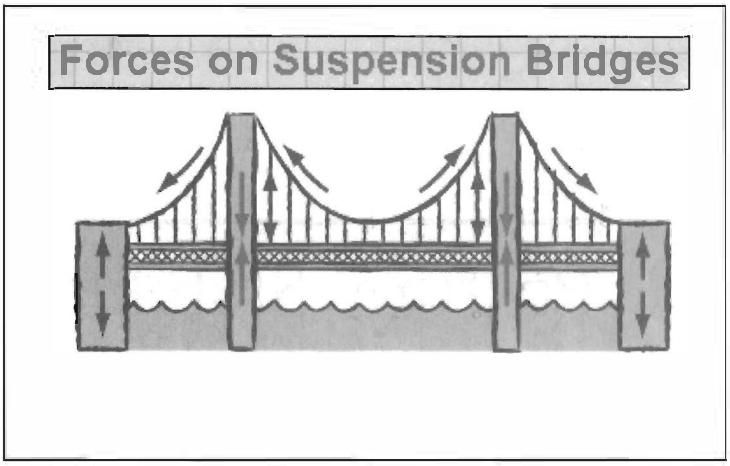

Figure 25-2. The action-reaction forces of a suspension bridge that supports the weight of the roadway.

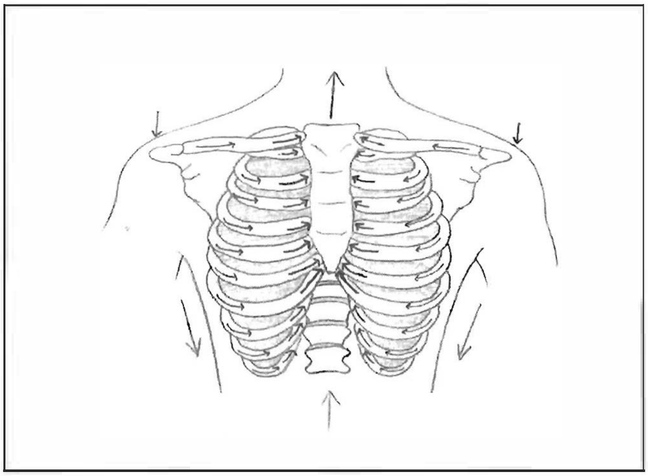

Figure 25-3a. Spinal corkscrew principle causing spinal lengthening—anterior perspective.

tension-type headaches,[19,20] as well as a likely connection between postural malalignment and breathing,[21,22] back pain,[23] balance,[24] fibromyalgia,[25] and osteoporotic spinal deformity.[26]

As mentioned previously in Chapter 3, this author suggests that ideal human alignment consists of body posture that is balanced, efficient, and vertical thus satisfying the biomechanical requirements of both static and dynamic function. According to Evcik and Aksoy,[27] muscles that function inefficiently over a prolonged period are susceptible to strain and spasm and can produce pain and poor postural relationships.

The challenge facing the manual therapist is not identifying the ideal, but achieving it. The treatment tools described in this text consist of connective tissue techniques, mobilization/manipulation, and therapeutic exercise. These manual methods coupled with electrotherapeutic modalities, pharmacologic agents, pain management (eg, trigger point injections), orthotics/appliances of various types, body work, surgery, psychotherapy, etc, performed by those qualified to render such interventions, are what modern medicine has to offer patients in pain. In addition to these therapeutic approaches currently available to healthcare professionals and the patients they treat, a new device called the PostureJac became commercially available in 2005. The remainder of this chapter will be devoted to the theory and utilization of the PostureJac as described by its inventor, the author of this text.

The Spinal "Corkscrew" Principle

What do Alexander's "upward direction" of the body,[28] the "upward rippling" motion of the Mitzvah technique, Rolf's "upward thrust," and Anusara Yoga's "root to rise" have in common? The answer is that they all recognize a crucial component of human alignment, which is the upward rise of the central column of the thorax (ie, spine and sternum) when posture is ideal. Because of the similarity of this upward rise with a wing-style corkscrew (Figure 25-1), this author has coined the term, *the spinal "corkscrew" principle*. Not unlike a suspension bridge (Figure 25-2), which drapes large steel cables over 2 concrete towers to support the roadway, the cork is propelled upwards against gravity. In human terms, the cork represents the spine (posteriorly) and the sternum (anteriorly); all that is necessary to propel them cephalward, according to Newton's third law of motion, is the equal and opposite motion of shoulder girdle depression. In terms of the biomechanical explanation for how the spine and sternum are "jacked up" through shoulder girdle depression, the thorax appears to be the most likely source of postural support. Figures 25-3a and 25-3b illustrate the anterior and posterior structures of the thorax that serve to lift the spine and sternum superiorly in response to shoulder girdle depression. In addition to the clavicles at the sternoclavicular joints and the ribs at the costovertebral, costotransverse, and costosternal

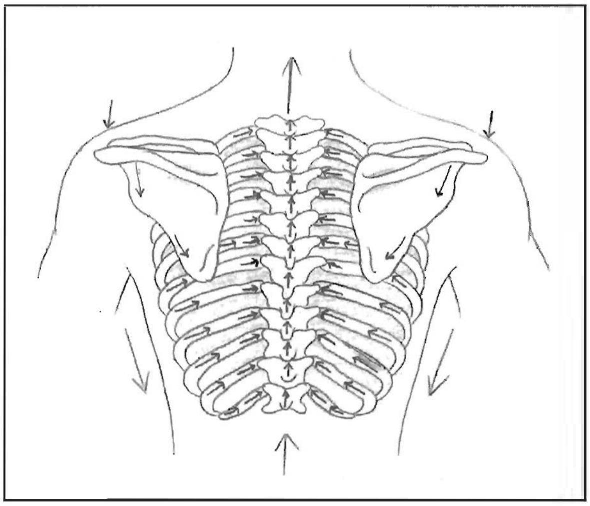

Figure 25-3b. Spinal corkscrew principle causing spinal lengthening—posterior perspective..

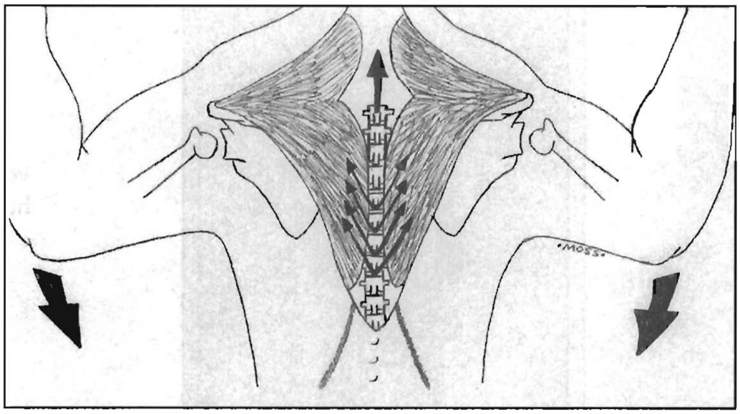

Figure 25-4. Lower trapezius vertical lifting mechanism. (Illustration by Neil Moss.)

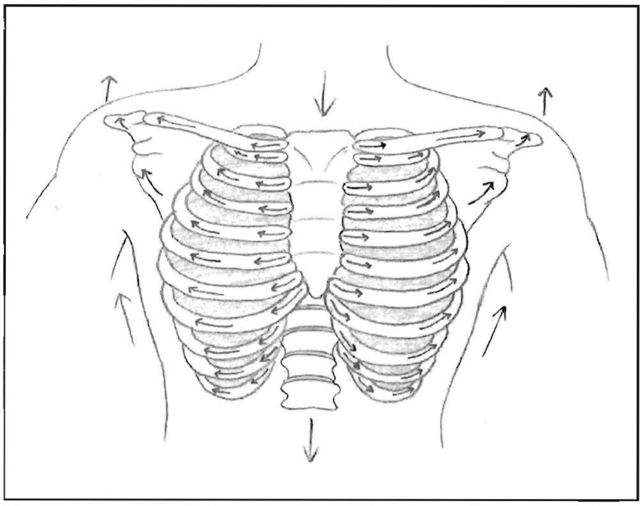

Figure 25-5a. Shoulder girdle elevation allows "collapsing" of the spine—anterior perspective.

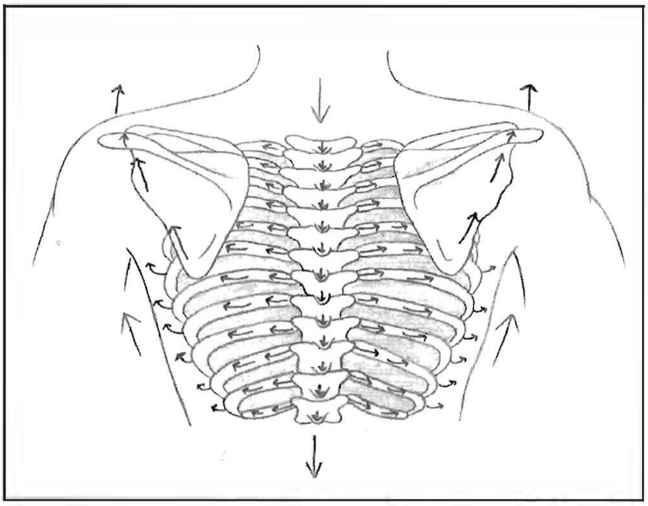

Figure 25-5b. Shoulder girdle elevation allows "collapsing" of the spine—posterior perspective.

joints, the scapular depressors, especially the lower trapezius muscles, impose a lifting force on the spine from T6 to T12 by acting in reverse action (Figure 25-4). Conversely, when the shoulder girdle is elevated, either actively or passively, the spine and sternum lose their antigravity support and "collapse" down (Figures 25-5a and 25-5b). From a theoretical perspective, when the spine is "jacked up," it is decompressed, lengthened, and stabilized; when it "collapses" down, it is compressed, shortened, and destabilized. Basically speaking, there exists a reciprocal relationship between the shoulder girdle and vertebral column (ie, they move in opposite directions). This explains how shoulder girdle elevation/protraction allows for an increased thoracic kyphosis, whereas shoulder girdle depression/retraction causes spinal lengthening with restoration of a normal, opposed to an accentuated, thoracic curve. In light of our discussion of forward head/rounded shoulders posture, the spinal "corkscrew" principle provides further insight into the art and science of posture correction. The ultimate outcome is alignment that is not only balanced, efficient, and vertical, but which provides the environment for tissue healing and restoration of function as a consequence of these components.

The PostureJac: A New Tool in Orthopedic Rehabilitation

The PostureJac is an exercise and posture-support jacket that provides the means whereby an individual can perform myofascial stretching, self-mobilization, and muscle strengthening. In addition, it "jacks" body posture upward, as per the spinal corkscrew principle, and thus the name PostureJac. Though it is true that the PostureJac works on a biomechanical basis, perhaps its most profound effects on form and function operate on a neurological level through sensorimotor learning and improved kinesthetic aware-

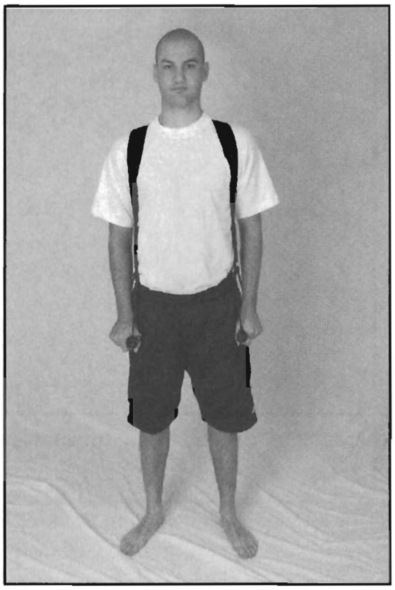

Figure 25-6. The Release.

ness. Patients are trained to recognize abnormal postures and movement patterns and exchange them for static and dynamic alignment that is balanced, efficient, and in a vertical relationship with gravity.[29] In addition to the therapeutic effects of posture correction, the PostureJac is an excellent tool for core strengthening of the local muscles of the lumbopelvic region (ie, transversus abdominis, pelvic floor, multifidi, and diaphragm) as well as the deep neck flexors (ie, rectus capitis anterior, rectus capitis lateralis, longus capitis, and longus colli); in conjunction with Dr. Jan Prsala,[30] the author has developed specific PostureJac exercises of the lumbar extensors that have proven very useful in the management of LBP.

Posture Correction Exercises

Handle adjustment is crucial before proceeding with these posture correction exercises! The handles should be approximately level with the patient's greater trochanters. With the elbows extended, there should moderate downward pressure on the shoulder area. If the elbows cannot extend, the handles need to be lengthened. If, however, the downward pressure on the shoulders is minimal, the handles need to be shortened. As mobility of the thorax improves with use, the handles may need to be shortened.

The Release

This introductory exercise with the PostureJac involves the process of releasing excess tension in the upper half of the body. The upper trapezius and sternocleidomastoid muscles are known to generate excessive and unnecessary tension,[31] the result being a tendency toward forward head/rounded shoulders posture. Most likely this tension is driven emotionally through the limbic system,[32] but other postural influences certainly play a role.[33] Ideally, the head-neck-shoulder region should remain relaxed and fluid. However, because of habitual tensing in these muscles, the head-neck may intermittently "freeze." The goal of the Release maneuver is to recognize when "freezing" occurs and to restore the head-neck region to its fluid and relaxed state.

1. In the sitting or standing position, the patient is advised to become aware of muscle tightness in the shoulders, head-neck, face, and chest. Using a mirror for visual feedback may enhance the awareness of tightness by observing poor postural alignment, including elevation of the shoulders (Figure 25-6).
2. Once aware of this excess tension, the patient is encouraged to release it by "letting go" and to enhance this release of tension by lightly pushing the PostureJac handles down toward the floor.
3. As the shoulders drop, the patient should imagine the top of the head (toward the proverbial "bald spot") floating up to the ceiling as if being "pulled" by a rope attached to a helium balloon.
4. Breathing slowly in through the nose followed by a long exhalation out through pursed lips, while gently pushing the handles down, enhances the release.
5. This can be done from 1 to 5 minutes, several times per day. Over time, the patient will become more aware of unnecessary tension in the upper body and suffer less from myofascial trigger points, tension-type headaches, etc. In addition, the patient will ultimately feel taller and less compressed.
6. If at any time the patient experiences pain, dizziness, numbness, etc, the exercise should be stopped.

The Rocket

1. In the standing or seated position (ideally in a chair without armrests so as to avoid interference), the patient pretends to be a rocket that is "blasting off." As the PostureJac handles are pushed down with moderate pressure, the patient's torso is propelled upward against gravity like a rocket until "lift-off" is experienced (Figure 25-7a). In fact, the rocket engine is an excellent example of Newton's third law of motion, which is crucial in grasping the mechanism by which the PostureJac improves body posture (ie, action and reaction). If the rocket illustration fails to communicate a sense of upward rise of one's body posture, perhaps the image of a fountain rising from the base of the spine and working its way through the vertebral column to the top of the head is preferred.
2. Initially, the joints of the thorax may not allow the unhindered upward rise of the central column of the thorax (ie, spine and sternum). However, with time and practice, this upward rise will free up and become second nature.

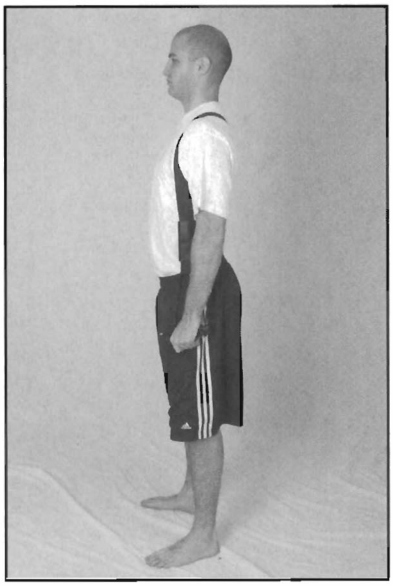

Figure 25-7a. The Rocket stage 1.

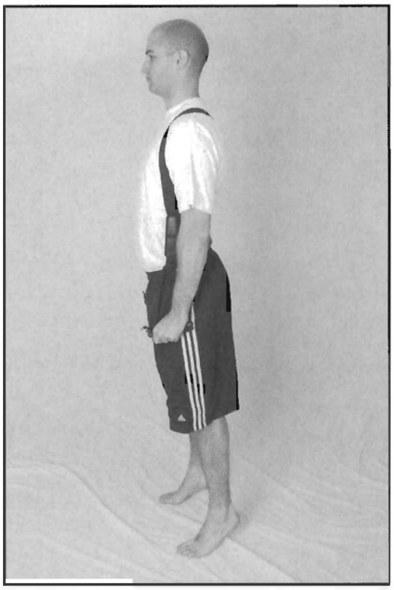

Figure 25-7b. The Rocket on toes stage 2.

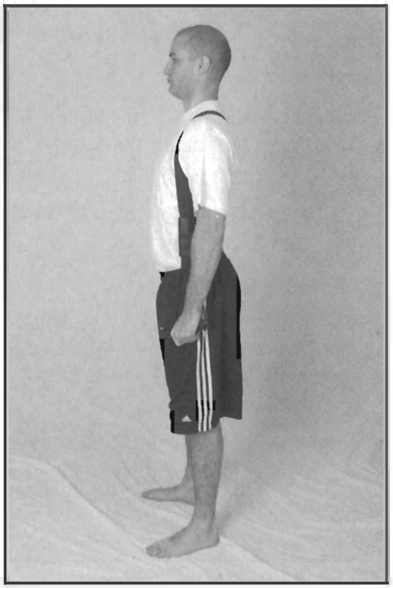

Figure 25-8a. The Piston stage 1.

3. To enhance this feeling of "lift-off," the patient can rise up on his or her toes as the spine is lengthening, provided that the requisite balance is present (Figure 25-7b).
4. As a stretching exercise, the Rocket is performed 3 times, held for up to 30 seconds, and repeated up to 6 times per day. As a strengthening exercise, it is performed 10 times, held for 5 to 10 seconds, and performed 3 times per day.
5. If at any time the patient experiences pain, dizziness, numbness, etc, the exercise should be stopped.

The Piston

1. In the standing or sitting position, the patient performs a similar motion to the Rocket, but with an alternating up-and-down motion, resembling a piston in an engine's cylinder (Figure 25-8a).
2. A helpful analogy is the "3 elevator" illustration. The patient imagines one "elevator" running up and down through the left shoulder, a second "elevator" running up and down through the right shoulder, and a third "elevator" running up and down through the center of the body (ie, the central column of the thorax).
3. Consequently, the "piston" motion involves the 2 shoulder "elevators" going down as the central "elevator" goes up; then the reverse occurs. The upward motion of the middle "elevator" is similar to the upward lift of body posture that occurs with the Rocket and provides kinesthetic awareness of good posture, whereas the downward motion of the middle "elevator" enables the patient to experience poor posture as a means of contrast.
4. To exaggerate the piston experience, the patient is advised to squat down and slouch as the shoulders rise up (Figure 25-8b) and proceed to toe standing as the shoulders are pressed down and the spine is lengthened (Figure 25-8c). As with the Rocket, the patient must have the requisite balance before attempting the toe-standing maneuver.
5. The Piston is performed up to 10 times, several times per day. The downward force on the PostureJac handles can be increased as upward mobility of the torso improves.

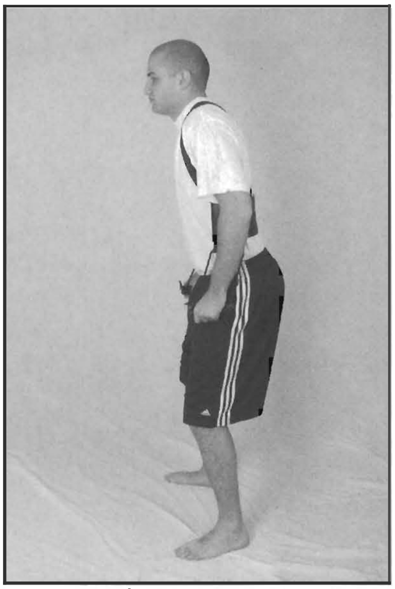

Figure 25-8b. The Piston stage 2.

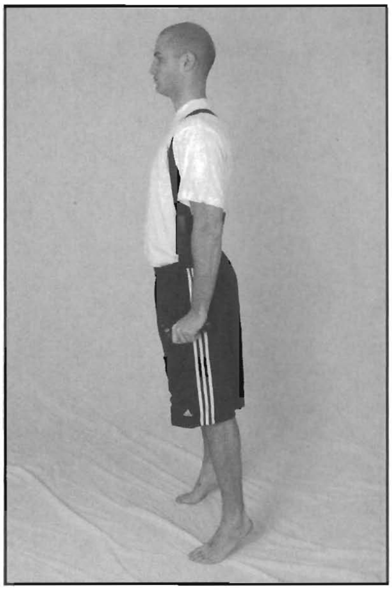

Figure 25-8c. The Piston stage 3.

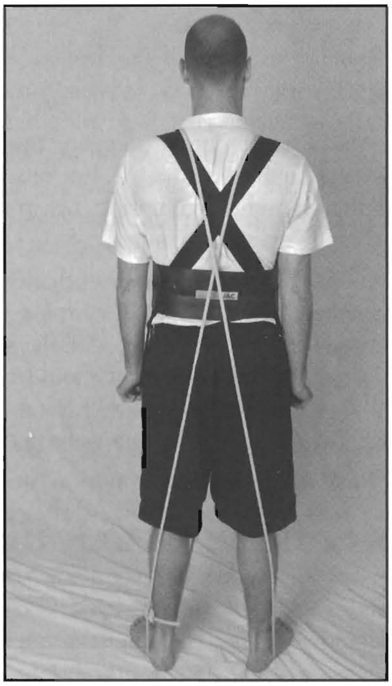

Figure 25-8d. The Piston against theratube resistance.

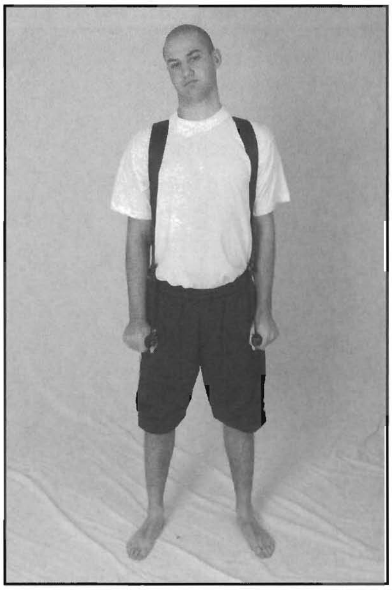

Figure 25-9. The TurtleNeck.

6. To strengthen the corkscrew mechanism, 2 loops of Thera-Band tubing (The Hygenic Corporation, Akron, OH) are used. By draping the tubing on either side of the neck and holding it down with the opposite foot, resistance to the upward rise of the central column of the thorax is provided (Figure 25-8d). The amount of resistance can be varied depending on the color of tubing used. The patient must have the requisite mobility of the central column (ie, spine and sternum) before strengthening is commenced (this is a potent form of posture therapy and should be done under therapist supervision).
7. If at any time the patient experiences pain, dizziness, numbness, etc, the exercise should be stopped.

The TurtleNeck

Similar to the Rocket and Piston, the name TurtleNeck paints a picture of spinal lengthening. In this case, of a turtle's head-neck emerging from its shell. With that in mind, the procedure is quite simple.

1. The sitting or standing patient, or nonpatient for that matter, pushes down on the "shell" as the head-neck region works its way in an upward direction (Figure 25-9).
2. The TurtleNeck allows for stretching freestyle. The patient is instructed to feel for restrictions in the head-

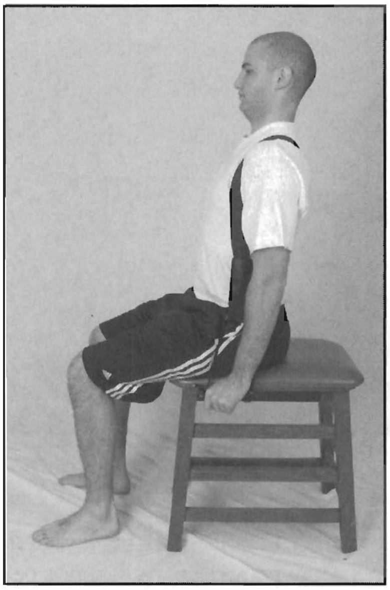

Figure 25-10a. JacRetract stage 1.

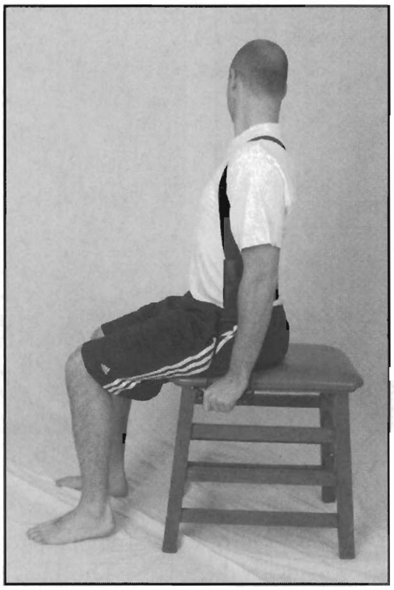

Figure 25-10b. JacRetract stage 2.

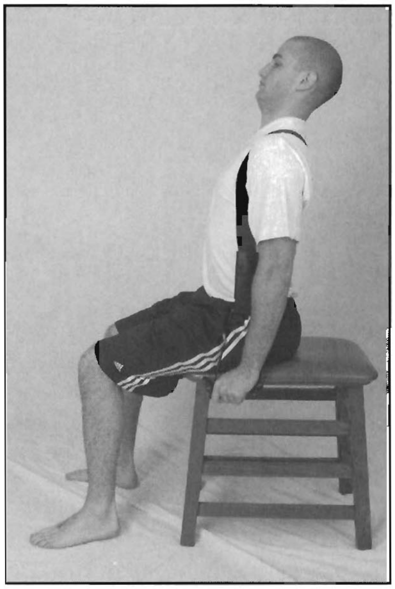

Figure 25-10c. JacRetract with extension.

neck and shoulder muscles and use the PostureJac to elongate the head, neck, and spine.

3. This freestyle stretching should be performed gently and for under 1 minute at first and then progressed in intensity and duration over the ensuing weeks.
4. The TurtleNeck exercise reinforces this concept of "shoulders down, spine up."
5. If at any time the patient experiences pain, dizziness, numbness, etc, the exercise should be stopped.

The Head Turner

Someone has said that, "Neutral is that position from which all movements are most free." What a concept! The Head Turner consists of head-neck rotation performed from the neutral or orthostatic head-neck position (illustrated on www.posturejac.com). Consequently, it allows for rotation that is more mobile than it otherwise would be. In fact, the motion is typically felt to occur into the upper thoracic spine, which is quite beneficial.

1. The sitting or standing patient neutralizes his or her posture by lightly pressing down on the PostureJac handles and adjusting head-neck position to a more vertical alignment.
2. The patient then turns slowly to the right and back to midline.
3. This movement is then repeated to the left side and back.
4. The patient turns at least 3 times to either side several times per day.
5. If at any time the patient experiences pain, dizziness, numbness, etc, the exercise should be stopped.

The JacRetract

This 2-stage exercise works very well with McKenzie's neck retraction exercises.[34] The key to successful head-neck retraction is trunk stability. Without it, the movement is only partially effective in restoring extension to the lower cervical spine.

Stage 1

1. In the sitting or standing position, the patient engages the handles in a downward direction until there is moderate pressure against the shoulders.
2. In the chin-tucked position (chin to hyoid, eyes looking straight ahead, head rotation around ear axis), the patient retracts the head-neck backward (Figure 25-10a) for 3 seconds.
3. Repeat this movement 10 times.
4. If at any time the patient experiences pain, dizziness, numbness, etc, the exercise should be stopped.

Stage 2

1. At the end of Stage 1, the patient is instructed to turn slowly to the right (Figure 25-10b) then slowly to the left, 10 times, maintaining the head-neck retraction throughout.
2. If at any time the patient experiences pain, dizziness, numbness, etc, the exercise should be stopped.
3. Under therapist supervision, some select patients may respond well to a combination of head-neck retraction/extension (Figure 25-10c). This maneuver potentially closes the facet joints from C2 down to T4 and, if done correctly, can be helpful in both McKenzie's dysfunction and derangement syndromes

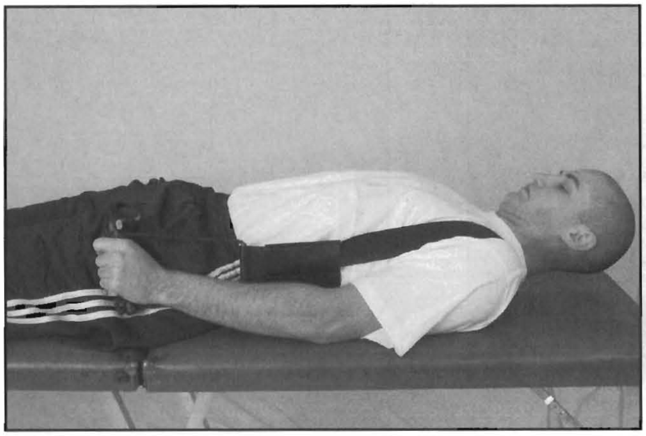

Figure 25-11a. The HeadFlex on the table.

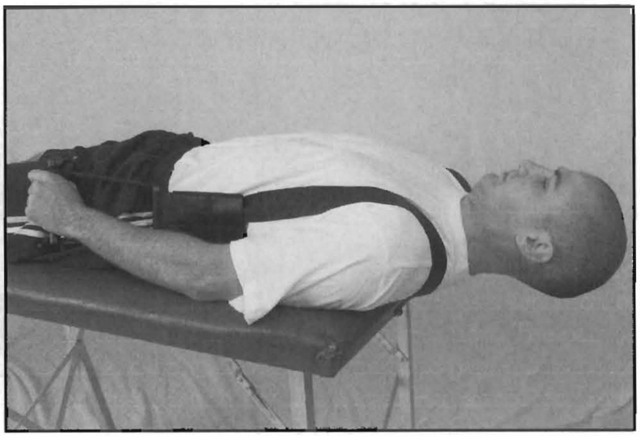

Figure 25-11b. The HeadFlex off the table.

(see Chapter 2). However, it should be done carefully as facet impingement can occur if done too quickly or too far into the range. In addition, the patient should immediately stop this exercise should pain, numbness, or dizziness result.

The HeadFlex

It is becoming well established in the scientific literature that individuals with chronic primary headaches, to a large degree, suffer from weakness and poor endurance of the deep neck flexors (ie, rectus capitis anterior, rectus capitis lateralis, longus capitis, and longus colli), which is also correlated to FHP.[35-37] By stabilizing the scapulothoracic region and lower cervical spine, the PostureJac can be used to dramatically improve function of the deep neck flexors (ie, strength and endurance). In addition, the reconditioning of this deep and local core system enables the superficial neck flexors (eg, sternocleidomastoid and scalenes) to relax, which contributes to posture correction of the head-neck region. Prior to commencing the HeadFlex exercise, the therapist should first address flexion limitation in the upper cervical spine (eg, inhibitive occipital distraction technique and occipital extensor stretching). Otherwise, the deep neck flexors will be unable to achieve their full strength and endurance potential.[38-40]

1. The supine-lying patient begins by engaging both handles down toward the feet.
2. When moderate pressure is felt under the shoulder straps, the patient performs a chin-tuck and raises the back of the head less than an inch off the surface (Figure 25-11a). It is important that the motion be confined to the upper neck as much as possible to ensure that the superficial neck flexors are kept from substituting for the deep neck flexors.
3. The goal is for the patient to maintain this position for at least 10 seconds without shaking, raising, or lowering the head. As strength and endurance show signs of improving, the amount of downward pressure applied to the PostureJac handles can be lessened. The HeadFlex can be performed up to 3 times per day.
4. An advanced form of the HeadFlex exercise consists of performing it with the head-neck off the end of the treatment table (Figure 25-11b). This should only be attempted under professional supervision.
5. If at any time the patient experiences pain, dizziness, numbness, etc, the exercise should be stopped.

The Total Core-Prone

This next exercise is called the Total Core-Prone because it trains the core muscles from the upper cervical spine down to the pelvic floor. Proposed muscle activation includes the deep neck flexors, lower cervical multifidus, lower trapezius, transversus abdominis, lumbar multifidus, oblique abdominals, rectus abdominis, pubococcygeus, gluteus maximus, etc. It is an advanced exercise that requires physiologic mobility of the spine and pelvis before it should be attempted; at no time should painful symptoms be experienced by the patient.

1. The prone-lying patient sets the lumbopelvic region in a neutral physiologic position followed by activation of the pubococcygeus (eg, "stop urine flow") and transversus abdominis (eg, drawing-in of the navel to the spine on exhalation without performing a PPT).
2. The PostureJac handles are then engaged in a downward direction while the patient retracts the head-neck region (ie, upper cervical flexion with lower cervical extension). This will flatten the cervicothoracic junction by extending it slightly.
3. The patient's upper limbs should be slightly externally rotated for optimal recruitment of the lower trapezius and posterior rotator cuff muscles (Figure 25-12).
4. The trunk should then be extended slightly, but not in the lumbar spine. Preventing an arching of the lower back will recruit the global abdominal muscles

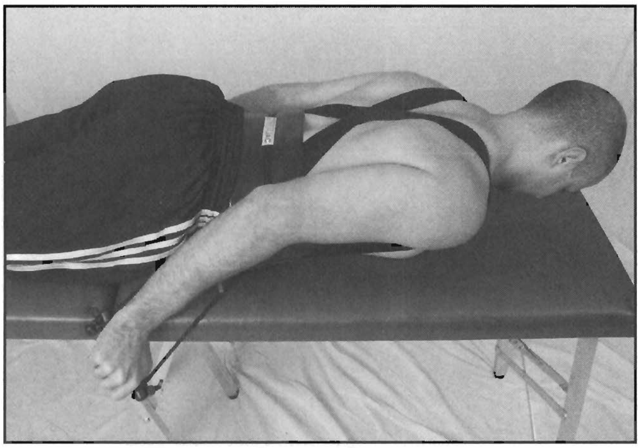

Figure 25-12. The Total Core-Prone.

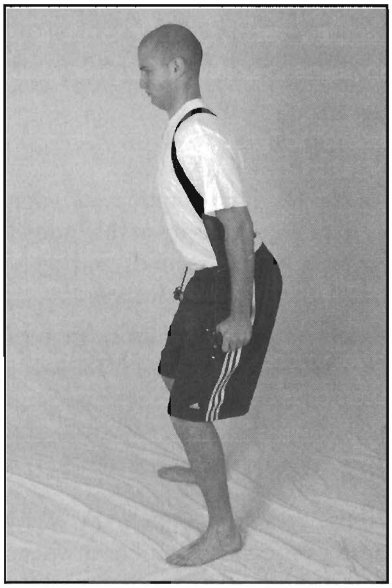

Figure 25-13b. The Total Core-Standing plus partial squats.

and gluteus maximus. The Stabilizer (Chattanooga Group, Hixson, TN) can be used to further isolate the core muscles of the lumbopelvic region.

5. The Total Core-Prone can be repeated 5 to 10 times, held for 5 to 10 seconds, and repeated 2 to 3 times per day. The patient must be reminded to breathe normally throughout.

The Total Core-Standing

This exercise activates the entire core system from the bottom up. It is a wonderful way to "awaken" the postural support muscles of the entire body.

1. The patient stands with relaxed knees, finds his or her neutral lumbopelvic position, and then activates the core muscles of the lower torso (pubococcygeus, transversus abdominis, etc).

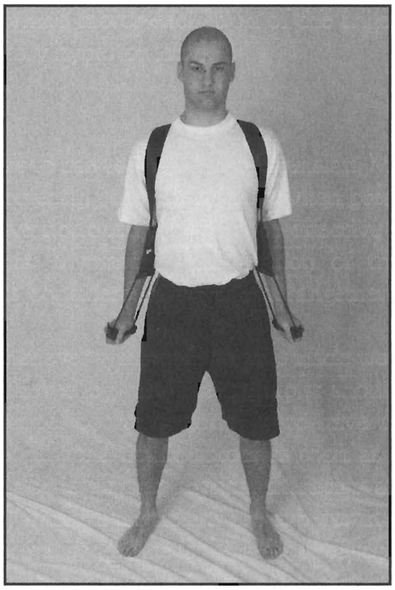

Figure 25-13a. The Total Core-Standing.

2. The posterior fibers of the gluteus medius and gluteus maximus muscles are then contracted by slightly externally rotating the hips in the standing, weight-bearing position.
3. The patient then engages the PostureJac handles toward the floor, adding slight external rotation of the shoulders (ie, lower trapezius and posterior rotator cuff activation).
4. From here the patient performs a chin-tuck/neck retraction movement to recruit the deep neck flexors and lower cervical multifidus (Figure 25-13a).
5. From this position of total core activation, the patient performs partial squats 5 to 10 times (Figure 25-13b). This not only dissociates the hips from a stable trunk, but also works the quads concentrically and eccentrically.
6. The Total Core-Standing should be done several times per day and used as a complete postural retraining tool.

The MyoPresser

Fairly recent research[41,42] has revealed that myofascial trigger points[43] have 2 interesting features: 1) Trigger points are associated with "contraction knots." These knots are thought to be caused by spontaneous electrical activity in the muscle.[41] This sustained muscle activity at the trigger point compresses local blood vessels, reducing the local supply of oxygen. As a consequence of combined impaired local circulation and increased metabolic demands of contracted muscle, the energy supply (local ATP) is depleted, result-

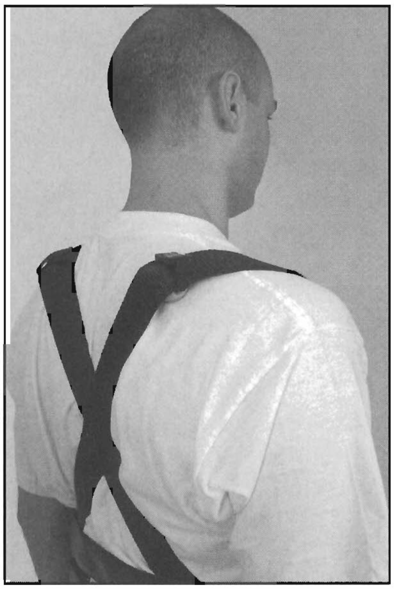

Figure 25-14. MyoPresser Press and Release.

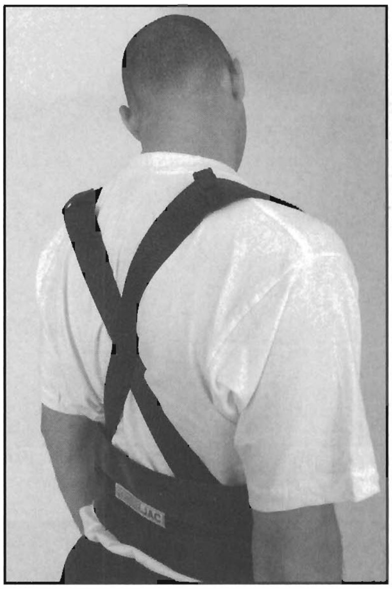

Figure 25-15. MyoPresser Press and Stretch.

ing in an "ATP energy crisis." Because ATP is necessary to restore the normal resting state of the muscle, contractile activity persists and a vicious cycle develops, resulting in pain and impairment. 2) It has also been demonstrated with microdialysis needle technique[42] that active trigger points contain significantly higher concentrations of protons, bradykinin, CGRP, substance P, serotonin, interleukin-1 beta, tumor necrosis factor-alpha, and norepinephrine than normal subjects and individuals with latent trigger points. The presence of these algogenic substances helps to explain why individuals with active trigger points have lower pressure pain thresholds and myofascial pain syndromes. Furthermore, it is theorized that active trigger points may play a role in neuronal hyperactivity (ie, central sensitization) and thereby contribute to chronic pain states (eg, fibromyalgia, migraine, chronic tension-type headache[19]).

The MyoPressers through specific compression[44] (eg, myotherapy,[43] ischemic compression,[43] etc) help to reduce contractile activity at the contraction knot[41] and disperse pain-producing biochemicals[42] from active trigger points in the upper trapezius, levator scapulae, paraspinals, and pectoralis major and minor muscles. These therapeutic effects help to explain the symptomatic relief experienced by using the combined PostureJac/MyoPresser system.

Press and Release

1. The 2 MyoPresser balls are placed under the shoulder straps over contraction knots in either the upper trapezius, levator scapulae, paraspinals, or pectoralis major/minor muscles.
2. The Press and Stretch technique involves pressing down on the PostureJac handles in order to achieve specific compression over myofascial trigger points (Figure 25-14). The duration of compression and number of repetitions is up to the individual, but most patients start with 3 seconds and repeat 3 times to assess their body's responsiveness.
3. In addition to static pressure, some patients respond well to a forward or backward rolling motion of the shoulders.

Press and Stretch

1. As with the Press and Stretch, the 2 MyoPresser balls are placed over contraction knots where they are the largest and the hardest (additional MyoPresser balls are available through the manufacturer).
2. Following downward pressure on the handles to load the trigger points, the patient is encouraged to stretch the affected muscle by directing the head-neck region to the opposite side while maintaining pressure on the knot (Figure 25-15).
3. Similar to the TurtleNeck stretch, the head-neck region searches for the most effective combination of neck movements to obtain the release.
4. If at any time the patient experiences pain, dizziness, numbness, etc, the exercise should be stopped.

Breathing Exercises

There is evidence to suggest a relationship between posture and breathing,[21,22,45] and this author believes that the PostureJac may ultimately become a useful tool in pulmonary rehabilitation. For example, when the scapulothoracic region is stabilized with the Rocket maneuver, the pectoralis

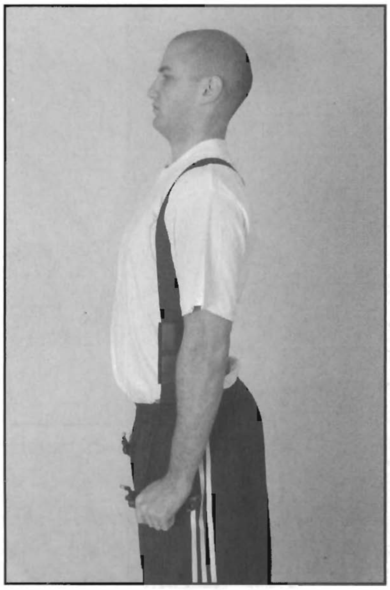

Figure 25-16a. The Posture Breath inhalation.

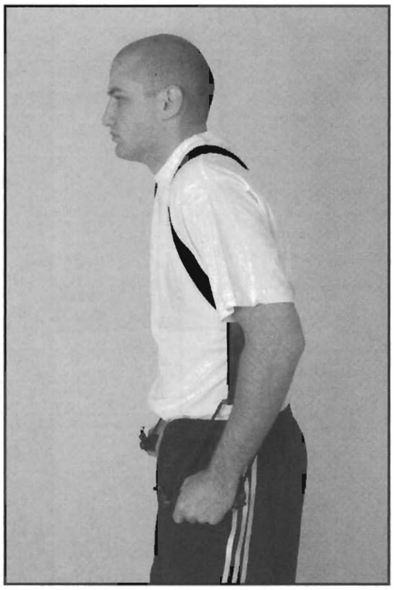

Figure 25-16b. The Posture Breath exhalation.

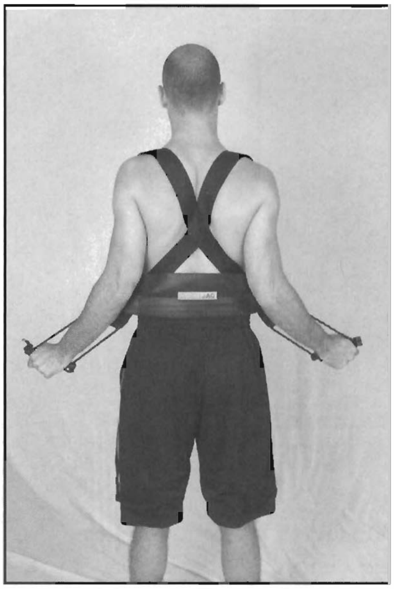

Figure 25-17. The Rotator Cuff external rotation.

major and minor, sternocleidomastoid, scaleni, and serratus anterior muscles, working in reverse action as accessory breathing muscles, have improved mechanical advantage in rib cage elevation due to enhanced proximal stability. This, in conjunction with a more vertically oriented body posture, should prove beneficial as a therapeutic tool for patients with age-related impairment of vital capacity, as well as with restrictive and obstructive lung diseases. However, this is merely speculative at this point.

The Posture Breath

1. Once "jacked up" by the PostureJac, as with the Rocket maneuver, the standing or sitting patient is instructed to breathe deeply (Figure 25-16a), recruiting the upper rib cage (ie, pump handle motion[46]). In addition to shoulder depression, slight external rotation of the shoulders, at the same time, appears to enhance anterior expansion of the rib cage during inhalation.
2. To enhance exhalation, the patient reverses course and sinks down posturally, allowing the shoulders to rise up and the central column of the thorax to collapse downward (Figure 25-16b). This exaggeration of kyphotic posture is expected to generate an upward movement of the respiratory diaphragm in order to assist with lung emptying. To further enhance exhalation, slight bilateral shoulder internal rotation appears to be beneficial.
3. This inspiratory/expiratory cycle can be repeated 3 to 5 times and performed as needed throughout the day.

A variation of the Posture Breath is for the patient to be "jacked up" during exhalation rather than inhalation. This type of breathing appears to favor diaphragmatic and lateral costal expansion (ie, bucket handle motion[46]).

Shoulder Exercises

As mentioned previously in this chapter, the scientific literature is replete with research studies demonstrating the relationship between body posture and shoulder kinematics.[11,12] Most studies point to the correlation between forward head/rounded shoulders posture and altered scapular kinematics (ie, loss of posterior tilt, upward rotation, and depression of the scapula), resulting in subacromial impingement.[13-15,47] In addition to the posture correction component of the PostureJac, the neuromuscular facilitation of the rotator cuff and lower trapezius muscle provides essential stability to the scapulothoracic and glenohumeral joints. Consequently, the PostureJac is ideally suited to become a useful tool in shoulder rehabilitation.

The Rotator Cuff

1. The standing or sitting patient first engages the handles in a downward direction with the elbows slightly flexed approximately 30 degrees.
2. Maintaining downward pressure on the handles, the patient externally rotates both shoulders to the sides, while keeping the elbows approximated to the lateral aspect of the lower ribs (Figure 25-17). The external rotation against resistance strengthens important stabilizers of the shoulder complex, including the supraspinatus, infraspinatus, teres minor, and lower trapezius.
3. To strengthen the subscapularis muscle, the patient internally rotates both shoulders and generates resis-

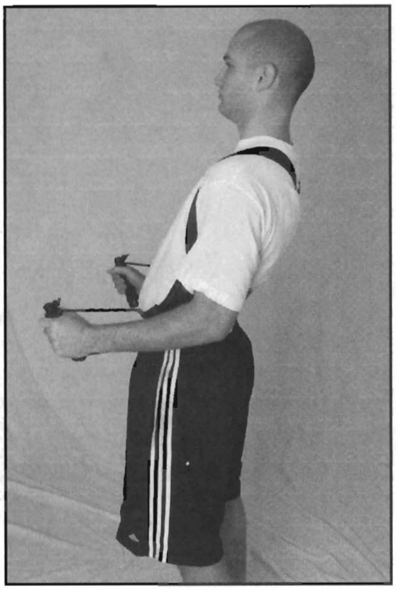
Figure 25-18. The JacBack Bend.

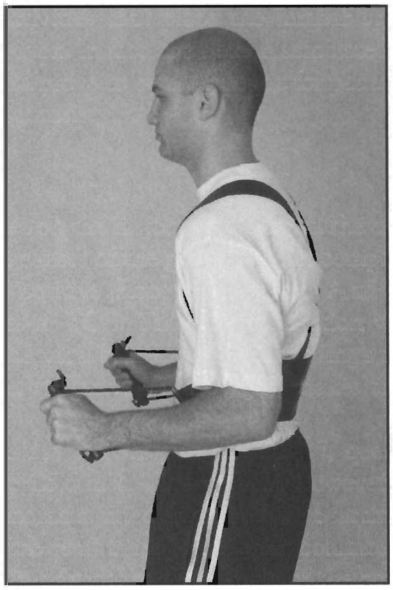
Figure 25-19. The Standing JacBack Press.

tance by pressing into the pelvis in the area of the pant pockets.

4. The patient completes 10 cycles, holding each repetition for 5 to 10 seconds and performs the Rotator Cuff 3 times per day.

If the patient is status post rotator cuff surgery, the therapist should check the postoperative protocol before commencing strengthening exercises.

Low Back Exercises/ Core Stability

The PostureJac has demonstrated utility in the training of core stability, lumbopelvic flexibility, and hip-low back dissociation training. The local system, consisting of the pelvic floor (eg, pubococcygeus), transversus abdominis, multifidus, and respiratory diaphragm, as well as the gluteus maximus and global trunk muscles (eg, erector spinae, rectus abdominis, external obliques, etc), can be easily trained with the PostureJac exercises to follow. In this regard, the PostureJac functions as "training wheels," making the performance of the exercises less difficult, more effective, and more enjoyable. For patients with a history of disc derangement, one needs to proceed cautiously with low back/core stabilization exercises; in the presence of diagnosed clinical instability,[48-51] the emphasis should be on core stability and hip mobility.

The JacBack Bend

The PostureJac can be used to enhance standing lumbar extension by providing a fulcrum over which to bend backwards. This exercise may worsen backache in patients with spondylolysis and unstable spondylolisthesis. It may also exacerbate LBP in patients with spinal stenosis and should not be used for McKenzie's anterior disc derangement.[52,53] However, it may help individuals with McKenzie's lumbar extension dysfunction and posterior derangement syndrome.[52,53] If it is used to assist with management of derangement syndrome, symptoms must demonstrate McKenzie's centralization phenomenon. If at any time symptoms peripheralize, the exercise should be stopped.

1. The standing patient leans back over the lumbar strap of the PostureJac while engaging the handles in an anterior direction (Figure 25-18).
2. The lumbar strap can be moved up or down to find the most effective spinal segment for lumbar extension. The motion should be localized to the low back and not occur in the lower limbs.
3. This exercise can be done on an as-needed basis 3 to 5 times.

The Standing JacBack Press

This exercise is excellent for patients with hyperlordosis who develop low back or leg pain when standing for prolonged periods of time (eg, spinal stenosis).

1. The standing patient adjusts the handles such that he or she engages them somewhere between 60 and 90 degrees of elbow flexion.
2. If kept relaxed, the patient's lumbar region would naturally hyperextend. However, the patient is instructed to do an abdominal "crunch" by pushing the low back into the back support of the PostureJac (Figure 25-19). The amount of force is determined by the patient pushing forward on the handles.

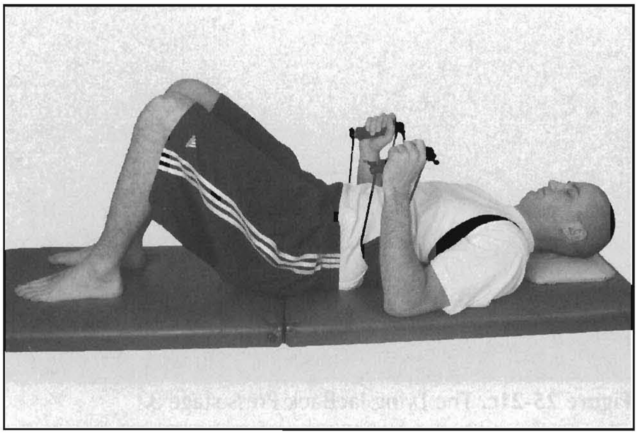

Figure 25-20. The Pelvic Tilt.

3. Therapists recognize this maneuver as a PPT. The patient should avoid a Valsalva maneuver by breathing out through pursed lips while pressing the lumbar region into the back strap.
4. This can be done on an as-needed basis or as an abdominal strengthening exercise and performed 10 times, for 5 to 10 seconds, and repeated 3 times per day. Works great for spinal stenosis!
5. A variation of this exercise is to perform a PPT (sitting or standing) into the lumbar strap of the PostureJac with downward pressure on the handles rather than with the elbows flexed 60 to 90 degrees. This unloads the lumbar facets, opens the intervertebral foramina, and stretches the back extensors. Feels great!

The Pelvic Tilt

The PPT is well known to physical therapists. However, the successful execution of the PPT by patients is another story. Part of the problem is the lack of tactile pressure on the lumbar spine, which leaves patients wondering if they are doing the maneuver correctly. The PostureJac addresses this problem by giving the patient something to "push against." In fact, the patient can adjust the amount of force used to execute the PPT by altering the pressure on the handles of the PostureJac.

1. The patient assumes the back-lying position with the knees bent and the feet flat.
2. With the elbows flexed somewhere between 60 and 90 degrees, the patient engages the handles up toward the ceiling and thereby extends the lumbar spine in a passive manner.
3. The patient, sensing pressure against the low back area, pushes the lumbar spine into flexion against the lumbar strap of the PostureJac, which posteriorly tilts the pelvis (Figure 25-20).
4. As mentioned above, the patient has the necessary control to adjust the force of the abdominal and gluteus maximus contraction.

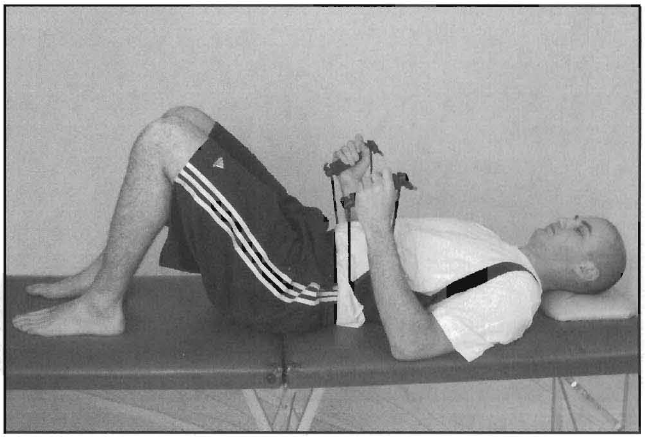

Figure 25-21a. The Lying JacBack Press stage 1.

5. The patient should perform the PPT on exhalation 10 times, for 5 to 10 seconds, and repeat 3 times per day.

It has been suggested by some individuals that the PPT performed in sitting, with PostureJac assistance, may be helpful in managing normal-transit constipation.[54,55] using it for this purpose, the patient must be reminded not to hold his or her breath during the pushing effort, but rather push on exhalation through pursed lips.

The Lying JacBack Press

This 3-stage exercise is an excellent means of training several global muscles of the abdominal and pelvic region (eg, gluteus maximus, rectus abdominis, and external/internal obliques).

Stage 1

1. The patient assumes the back-lying position with the knees bent and the feet flat.
2. The patient places his or her lumbopelvic region in neutral (ASISs in same plane as the pubic symphysis), which is where the low back region is centered, stable, and comfortable. The pelvic clock exercise can be used to fine tune lumbopelvic neutral. This is achieved by finding a position that is halfway between 12:00 and 6:00 in the sagittal plane.
3. Light pressure from the PostureJac's lumbar strap should be felt against the patient's lumbar region. As with the Standing JacBack Press, the PostureJac handles are engaged somewhere between 60 and 90 degrees of elbow flexion, which in supine is up toward the ceiling (Figure 25-21a).
4. The patient should practice finding the neutral lumbopelvic position several times.

Stage 2

1. While maintaining a neutral lumbopelvic position (stage 1), the patient performs a bicycling motion for

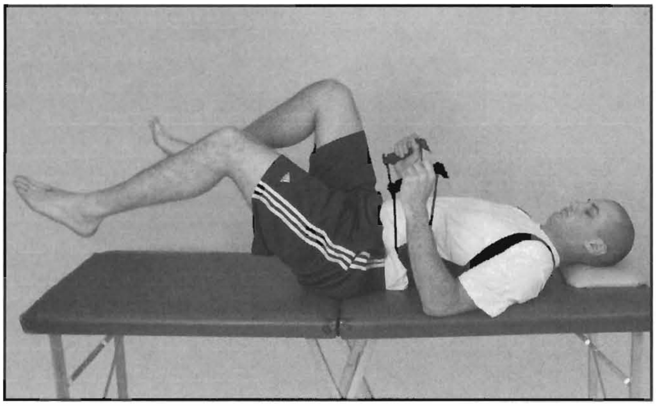

Figure 25-21b. The Lying JacBack Press stage 2.

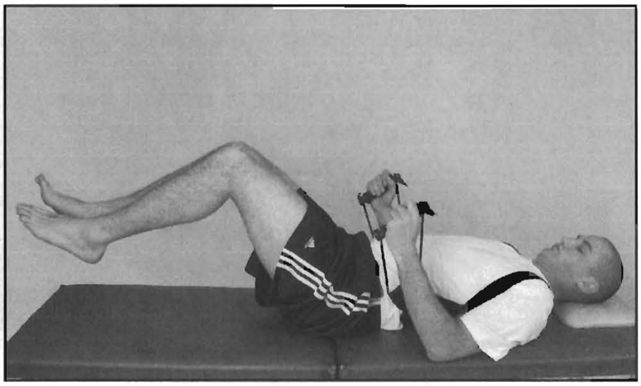

Figure 25-21c. The Lying JacBack Press stage 3.

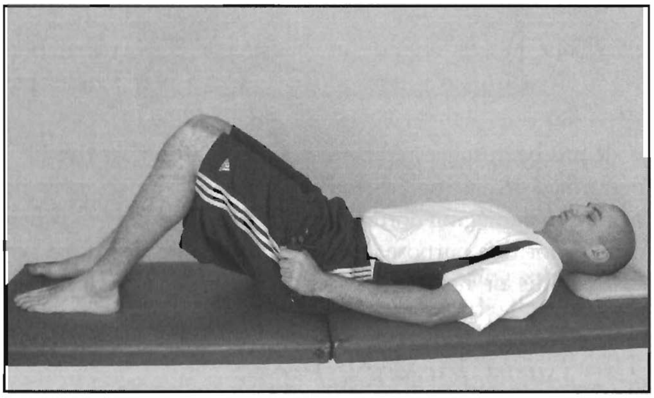

Figure 25-22a. The JacBack Stabilizer stage 1.

10 to 30 seconds (Figure 25-21b). Low back pressure needs to be maintained against the lumbar strap at all times with the handles directed up to the ceiling.

2. The bicycling motion begins with the hips and knees close to the pelvis and progresses in difficulty to a straighter leg position.
3. With mastery, the patient progresses to 30 to 60 seconds. The abdominals need to prevent hyperextension of the lumbar spine. The gentle pressure of the lumbar strap against the low back provides a tactile cue for the patient. Otherwise, it is hard to know where neutral is.

Stage 3

When the patient masters stage 2, stage 3 may commence. This exercise is very challenging for most people.

1. While maintaining the neutral lumbopelvic position (stage 1), the patient performs the bilateral leg-lowering maneuver 10 times (Figure 25-21c). When first commencing stage 3, the lever arm should be kept short by not allowing the legs to descend too far away from the pelvis; the low back region must not be allowed to hyperextend (ie, arch) at any time.
2. With improved abdominal strength/core stability, the legs can be lowered toward the straight leg position (ie, longer lever arm). If the patient shows signs of weakness (eg, shaking, anterior pelvic tilt), the legs should be brought in closer.
3. For the athlete, ankle weights can be added for a more challenging workout.

The JacBack Stabilizer

This 3-stage exercise is an excellent way of achieving core stability. It specifically targets the local muscles of the lumbopelvic region that provide direct stability to the lumbar spine and pelvis (ie, force closure). Core stabilization training with versus without the PostureJac is again comparable to teaching a child to ride a bicycle with versus without training wheels. In the author's opinion, there are fewer injuries with it!

Stage 1

1. The patient assumes the back-lying position with the knees bent and the feet flat.
2. The patient places his or her lumbopelvic region in neutral (ASISs in same plane as the pubic symphysis). Again, the pelvic clock exercise can be used to fine tune lumbopelvic neutral.
3. The PostureJac handles are then pressed down toward the feet (Figure 25-22a).
4. The pelvic floor (eg, pubococcygeus) muscles are activated by a slow and steady contraction as if "stopping the flow of urine." This maneuver can be learned by performing Kegel exercises.[56] The Kegelmaster (Wellness Partners, Orangevale, CA) is a useful tool in assisting female patients with these exercises.
5. The transversus abdominis (TA) muscle is now activated by the patient through an in-drawing of the umbilicus toward the spine on exhalation, without performing a PPT. On inhalation through the nose, the TA and pelvic floor can be relaxed to allow for normal diaphragmatic excursion. However, with each exhalation, through pursed lips, the pelvic floor and TA are again activated (this reciprocal activation/

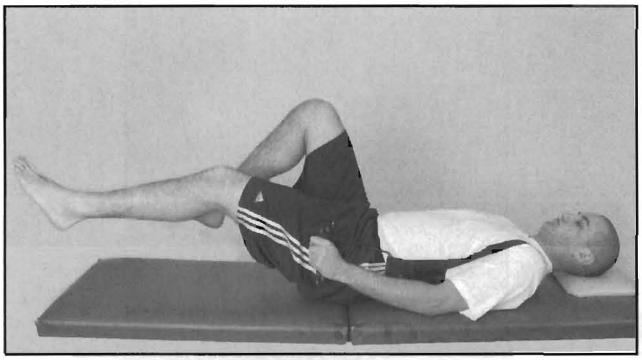

Figure 25-22b. The JacBack Stabilizer stage 2.

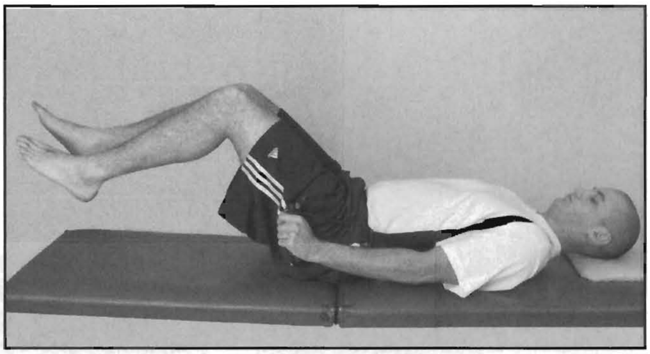

Figure 25-22c. The JacBack Stabilizer stage 3.

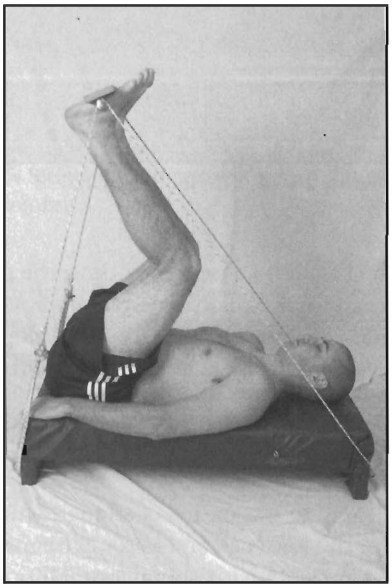

Figure 25-23a. Prsala Back Exercise Floor Unit.

deactivation of the pelvic floor and TA with breathing helps to retrain the coordination between the respiratory diaphragm and the TA).

6. Several repetitions of stage 1 are performed until all components are achieved successfully.

Stage 2

1. With all the components of stage 1 activated, the patient performs a bicycling motion with both lower limbs for 10 to 30 seconds (Figure 25-22b). As with stage 1, the pelvic floor muscles and TA are activated with exhalation and relaxed during inhalation.
2. The bicycling motion begins with the hips and knees close to the pelvis and progresses in difficulty to a straighter leg position as the pelvic floor and TA ebb and flow with breathing.
3. As core stability improves, the patient progresses the bicycling motion to 30 to 60 seconds.

Stage 3

When the patient masters stage 2, stage 3 of the JacBack Stabilizer may commence.

1. With the core system activated and synchronized with breathing as in stages 1 and 2, the patient performs 10 repetitions of bilateral leg lowering (Figure 25-22c).
2. With improved core stability, the legs can be lowered toward the straight leg position (ie, longer lever arm). If the patient shows signs of weakness (eg, shaking, anterior pelvic tilt), the legs should be brought in closer.
3. For the athlete, ankle weights can be added for a more challenging workout.

The Prsala Back Program

The exercises to follow are based upon the work of Dr. Jan Prsala,[30,57] former professor of biomechanics at Dalhousie University. Dr. Prsala's exercises are aimed at strengthening the lumbar spine extensors (eg, erector spinae, multifidi) from a lengthened position. Consequently, they are neither flexion nor extension exercises, but a combination of extensor strengthening through the full range of motion. Such strengthening can be done isometrically, concentrically, or eccentrically. These exercises are based upon the premise that most people lift with their back muscles despite being told not to. The reality is that most of our patients bend at the waist rather than at the knees regardless of what the back experts tell them. In addition, most patients slouch no matter what the experts say about sitting up straight with back support. Consequently, our low back patients are ill equipped for bending, lifting, and sitting simply because their back extensors are weak in a lengthened position. Furthermore, Dr. Prsala's concept involves a weightbearing (closed chain) exercise, which, according to Richardson and colleagues,[50] causes optimal recruitment of the one-joint slow "stabilizers." To this end, Dr. Prsala developed specialized exercise equipment, one of which is illustrated in this section and referred to as the Prsala Back Exercise Floor Unit (Figure 25-23a). The PostureJac can easily be adapted to incorporate the Prsala concept as follows:

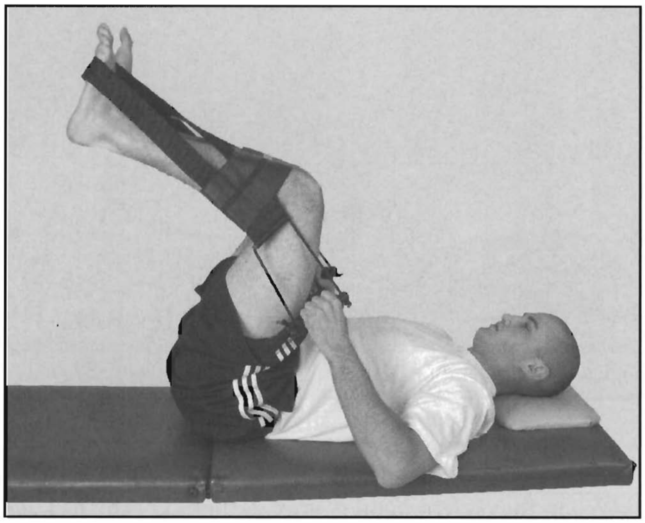

Figure 25-23b. The Prsala back exercise with the PostureJac.

1. The supine-lying patient places his or her feet into the PostureJac shoulder loops while holding the handles with both hands (the PostureJac logo, on the lumbar strap, should be facing up to the ceiling).
2. The patient starts with the hips flexed greater than 90 degrees in order to place the back extensors on stretch. Both feet are placed in the shoulder straps and the bottom edge of the lumbar strap engages the patella tendon of both knees (Figure 25-23b).
3. The motion that Dr. Prsala has found so helpful in working with low back patients is for the patient to push both feet against resistance such that the knees, hips, and lumbar spine all extend. This extension can be isometric (the pull on the PostureJac handles meets the force of lower extremity/spinal extension and no motion occurs); it can be concentric (the pull on the handles allows for the motions of knee, hip and lumbar spine extension from a position of total flexion), or eccentric (the pull on the handles overcomes the extension effort and the knee extensors, hip extensors, and lumbar extensors lengthen into flexion against resistance).
4. These exercises should be carefully used under therapist supervision and the home program should only be started after it is established that the patient is a candidate for such a program. The type and strength of the contraction as well as the number of repetitions, seconds held, and sets should be determined by the treating therapist.

Because of the amount of lumbar flexion involved with these exercises, it is extremely important that patients with McKenzie's derangement syndrome[52,53] avoid these maneuvers; if at any time symptoms appear to be radiating into the lower limb of any patient, the exercises should be stopped immediately. In addition, patients with signs of clinical instability[48-51] should be rendered more stable before attempting the Prsala exercises. Over time, the patient's low back and hip extensors will demonstrate improved strength, endurance, and flexibility; patients will develop more confidence and less fear avoidance behavior[58] with ADLs and work-related activities.

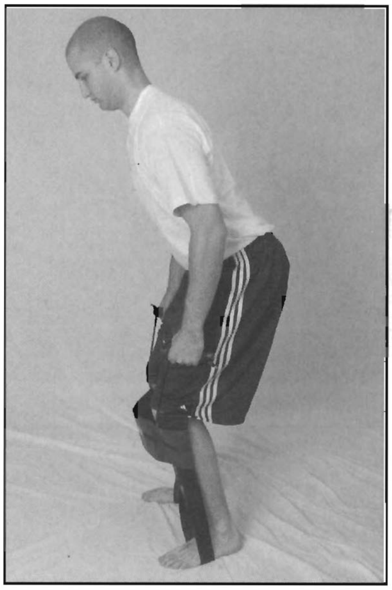

Figure 25-24. The JacBack Lift.

The JacBack Lift

This next exercise is a means of strengthening the "lifting muscles" of the low back and lower extremities. It is not to be used on patients with spinal instability[48-51] or disc derangements[52,53] because of the high forces generated in the lumbar spine. However, if performed correctly, under the direction of a physical therapist, it is an excellent way of preparing patients for the mechanical stress of lifting activities.

1. With the PostureJac inverted, the standing patient's feet are placed on the shoulder straps, hip width apart, and the handles are held by both hands (the inside of the PostureJac faces the patient).
2. The patient then bends his or her knees slightly, places the lumbopelvic region in neutral, and sets the core muscles of the lower trunk (ie, pelvic floor and transversus abdominis).
3. On exhalation the patient pulls the PostureJac handles up against the resistance of body weight, which is securing the inverted PostureJac to the floor (Figure 25-24). This isometric upward pull simulates lifting in a closed-chain environment and activates the extensors of the knee, hip, and lumbar spine.
4. Patients should start with gentle force, hold for just a few seconds, and repeat 2 to 3 times. The lumbar spine should retain its lordosis at all times. If at any time the patient feels pain in the low back or lower extremities, the JacBack Lift should be stopped immediately.

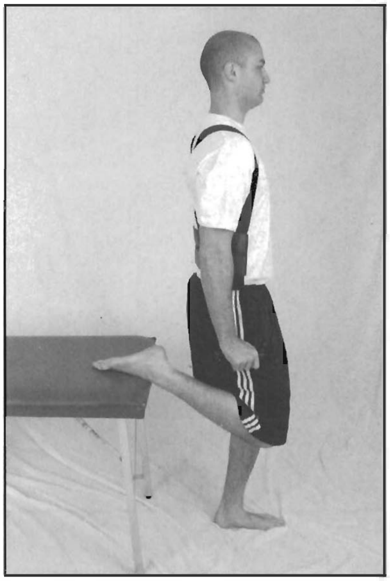

Figure 25-25. Stretching exercises—right rectus femoris.

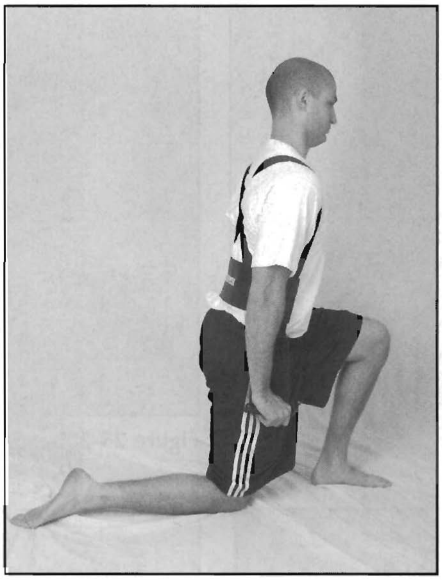

Figure 25-26. Stretching exercises—right ilipsoas.

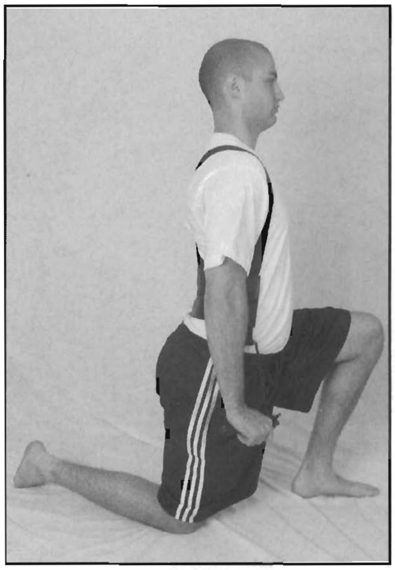

Figure 25-27. Stretching exercises—right TFL.

5. The patient should follow this maneuver with the standing JacBack bend to prevent low back strain from simulated lifting.

Stretching Exercises

Because flexible/mobile hips and a stable spine are the solution for many low back conditions, the muscles affecting the hips should be stretched periodically. Because these same muscles attach to the lumbopelvic region, they are often responsible for malalignment of the low back region when they are in a shortened state.

Rectus Femoris

1. The standing patient places the dorsum of his or her foot on a table or similar surface, bringing the ipsilateral knee into flexion.
2. The patient directs the handles down/forward and performs a PPT against the back strap of the PostureJac.
3. The patient then proceeds toward the floor by bending the opposite knee until a stretch is felt in the anterior thigh region on the stretched side (Figure 25-25). The key to a good stretch is not allowing the lower back to arch.
4. The stretch is held for 30 seconds and repeated 3 times, several times per day.

Iliopsoas

1. The half-kneeling patient places his or her stretched hip into internal rotation.
2. The patient directs the handles down/forward and performs a PPT against the back strap of the PostureJac.
3. The patient directs the top of the thigh anteriorly while preventing lumbar spine extension (Figure 25-26).
4. The stretch should be felt in the upper anterior thigh, held for 30 seconds, repeated 3 times, and performed several times a day.

Tensor Fascia Lata

1. The half-kneeling patient places his or her stretched hip into external rotation.
2. The patient directs the handles down/forward and performs a PPT against the back strap of the PostureJac.
3. The patient directs the top of the stretched thigh forward and lateral while preventing lumbar spine extension (Figure 25-27).
4. The stretch should be felt in the anterolateral upper thigh region, held for 30 seconds, repeated 3 times, and performed several times a day.

Hamstrings and Calf

1. The supine patient holds the PostureJac handles while placing the ball of the foot of the stretched side into the inverted shoulder strap.
2. The stretched leg is elevated into the 90/90 position by pulling on the handles of the inverted PostureJac.
3. The handles are then used to extend the knee until a slight stretch is felt in the hamstrings (Figure 25-28a).
4. The stretch is held for 30 seconds, repeated 3 times, and performed several times a day.
5. The calf muscles can be stretched in a similar manner, soleus with the knee flexed (Figure 25-28b) and gastrocnemius with the knee extended (Figure 25-28c).

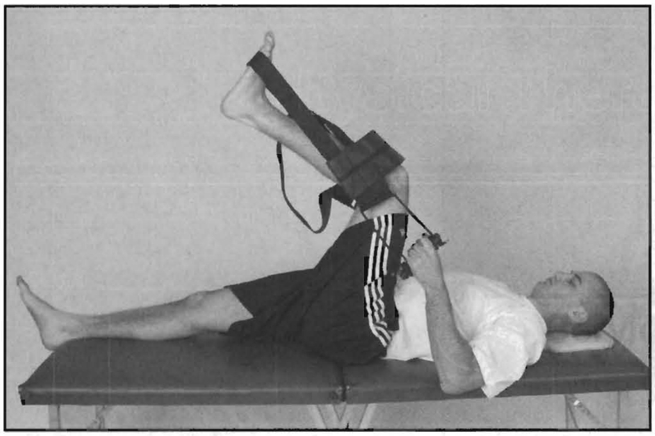

Figure 25-28a. Stretching exercises—left hamstrings.

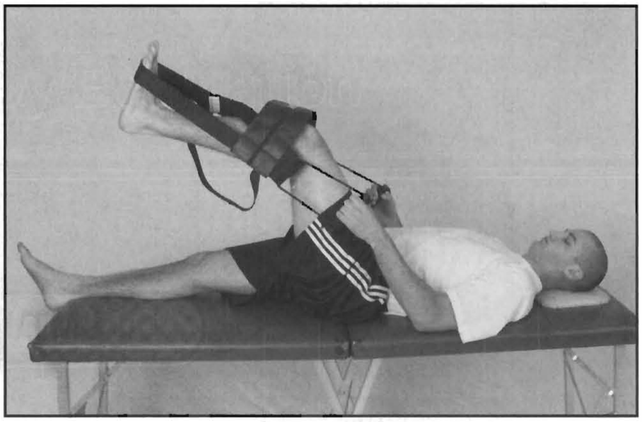

Figure 25-28b. Stretching exercises—left soleus.

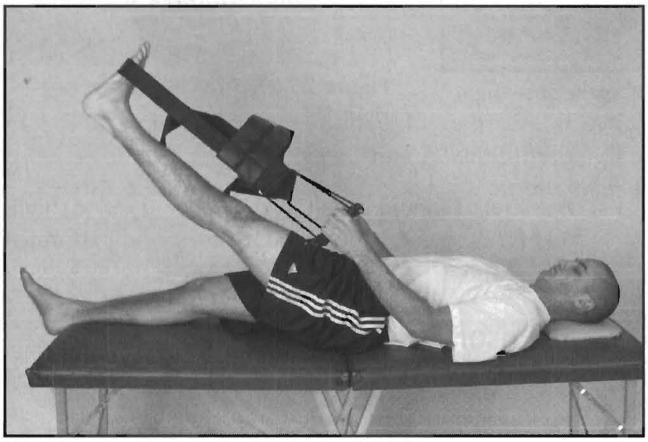

Figure 25-28c. Stretching exercises—left gastrocnemius.

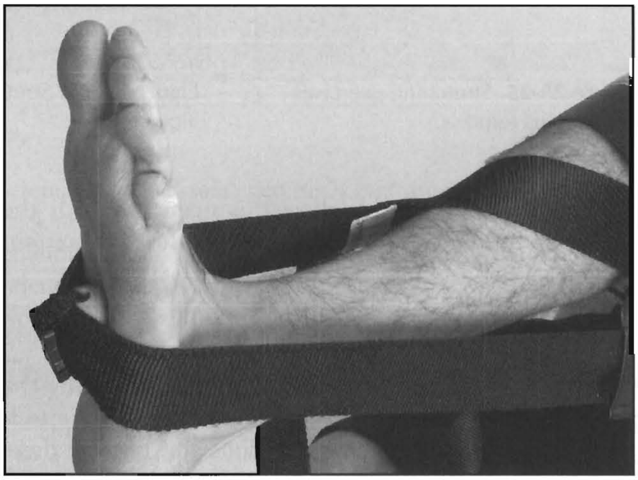

Figure 25-29. Foot massage.

6. The hamstrings and calf can also be stretched in sitting.

When persistent tightness remains in the calf and hamstrings despite regular stretching, one should suspect adverse neural tension from sciatic nerve irritation/compression.

Foot Massage

The plantar surface of the foot, including the plantar fascia, is often the site of excess tension, contraction knots, tender points, etc. The MyoPresser/PostureJac system is an excellent way of releasing such tension through specific compression therapy.

1. The sitting or supine-lying patient places his or her foot into the inverted shoulder strap of the PostureJac (MyoPresser in place) and pulls on the handles (Figure 25-29).
2. The patient finds a tender area on the bottom of the foot and proceeds to massage the foot over the MyoPresser for several seconds by pulling on the handles. After releasing the tension in one area, the patient seeks a different area of tenderness and again applies specific pressure to the area. As with any mechanical therapy, proceed cautiously in the beginning lest too much pressure be applied and posttreatment soreness results.
3. Repeat through the day on an as-needed basis.

The Hip Hinge

Dissociating hip from spinal motion is a key component of core stabilization.[28,50,59-61] Basically, the combination of stiff hips and a loose spine are a recipe for trouble. With use of the PostureJac, a patient is easily taught to reverse that trend by moving at the hip joints while the lumbar spine maintains its neutral/stable position. The benefit of this concept to LBP sufferers is enormous. In addition, it is a great way of strengthening the hip and knee extensors.

Stage 1: The Chair Rise

1. The sitting patient places his or her lumbopelvic region in a neutral position.
2. The PostureJac handles are then engaged in a downward direction.
3. The patient moves trunk on legs (ie, hip motion) forward and back 3 times and then rises to standing

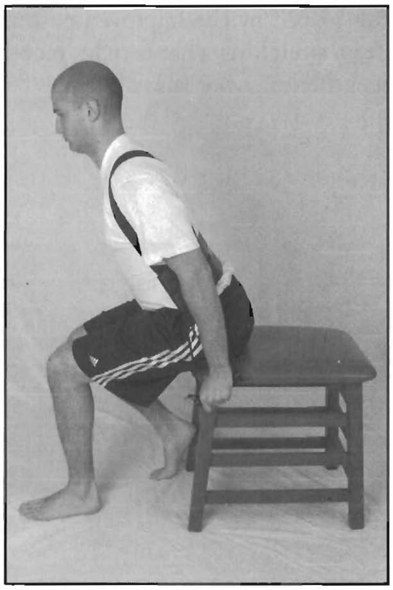

Figure 25-30. The Chair Rise.

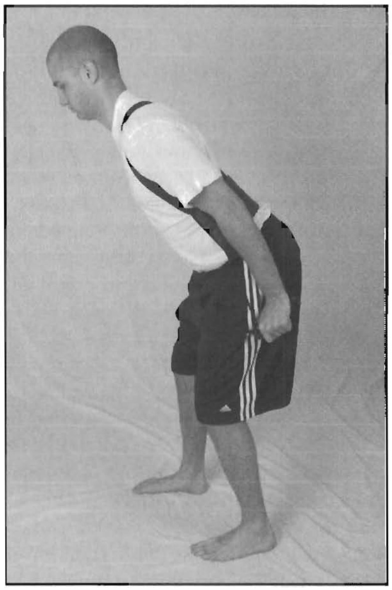

Figure 25-32. The PostureJac Bow.

with all motion occurring at the hips while the spine remains stable in its neutral posture (Figure 25-30).

4. Three sit-to-stand maneuvers are performed at least 3 times per day.

Stage 2: The PostureJac Lunge

1. The standing patient places his or her lumbopelvic region in a neutral position with one foot slightly in front of the other.
2. The PostureJac handles are then engaged in a downward direction.
3. A standing lunge is performed, first landing on the right foot (Figure 25-31) then on the left.

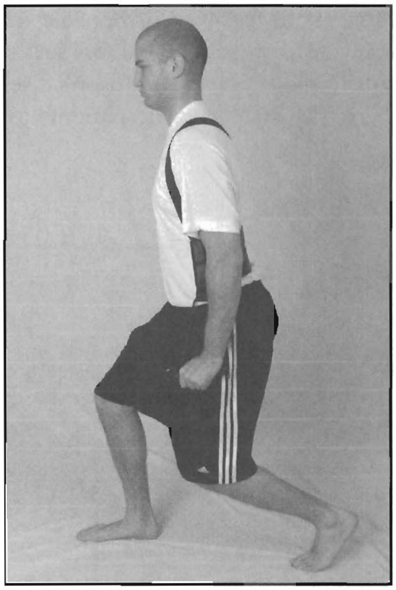

Figure 25-31. The PostureJac Lunge.

4. Three repetitions are performed on each side.

The PostureJac lunge trains core stability, hip mobility, and lower extremity strength.

Stage 3: The PostureJac Bow

1. The standing patient places his or her lumbopelvic region in a neutral position with one foot slightly in front of the other.
2. The PostureJac handles are then engaged in a downward direction.
3. A standing bow is performed by bending at the hips and not at the waist (maintain slight pressure against the back strap of the PostureJac at all times to ensure proper lumbar position throughout the maneuver).
4. At least 3 bows are performed (Figure 25-32) as described.

In the book, *Anatomy Trains: Myofascial Meridians for Manual and Movement Therapists*, Thomas Myers[62] describes an elegant lattice of tensional bands and bony spacers that accounts for ideal posture on the one hand; for the physical impairments that result from abnormal mechanics on the other. In addition, Myers defines the myofascial-skeletal system in terms of tensegrity geometry and states, "A tensegrity structure, like any other, combines tension and compression members, but here the compression members are islands, floating in a sea of continuous tension. The compression members push outwards against the tension members that pull inwards. As long as the two sets of forces are balanced, the structure is stable." Perhaps in the end, it is tensegrity and a tension-compression cycle established in the body that best explains the biomechanical effects of the PostureJac.

This concludes an overview of the theory and clinical utilization of the PostureJac. The information presented in this chapter relates primarily to orthopedic rehabilitation. However, the inventor of this clinical tool sees the potential application to neurological (eg, Parkinson's disease), cardiopulmonary, geriatric, and pediatric patients as well. After all, who is not helped by the improved postural alignment that results from stretching what is tight, mobilizing what is stiff, and strengthening what is weak?

References

1. Definition of Posture: http://www.MedicineNet.com. Accessed August 21, 2009.
2. Kuchera M, Kuchera W. General postural considerations. In: Ward R, ed. *Foundations for Osteopathic Medicine*. Baltimore, MD: Williams and Wilkins; 1997.
3. Peterson Kendall F, Kendall McCreary E, Geise Provance P, McIntyre Rodgers M, Romani WA. *Muscles Testing and Function*. 5th ed. Baltimore, MD: Lippincott Williams and Wilkins; 2005.
4. Pettibon B. Neuro-physiology of spinal subluxation and correction. *The Chiropractic Journal*. January, 2000.
5. Cacciatore TW, Horak FB, Henry SM. Improvement in automatic postural coordination following Alexander technique lessons in a person with low back pain. *Phys Ther*. 2005;85(6):565-578.
6. Kandel ER, James H, Thomas M. *Principles of Neural Science*. 4th ed. New York, NY: McGraw-Hill; 2000.
7. Carr J, Shepherd R. *Movement Science: Foundations for Physical Therapy in Rehabilitation*. 2nd ed. Gaithersburg, MD: Aspen Publishers, Inc; 2000.
8. Bond M. *The New Rules of Posture: How to Sit, Stand, and Move in the Modern World*. Rochester, VT: Healing Arts Press; 2007.
9. Selinger A. Posture. In: Cameron MH, Monroe LG, eds. *Physical Rehabilitation: Evidence-Based Examination, Evaluation, and Intervention*. St. Louis, MO: Saunders Elsevier; 2007.
10. Raine S, Twomey L. Attributes and qualities of human posture and their relationship to dysfunction or musculoskeletal pain. *Crit Rev Phys Rehabil Med*. 1994;6(4):409-437
11. Ludewig PM, Cook TM. Alterations in shoulder kinematics and associated muscle activity in people with symptoms of shoulder impingement. *Phys Ther*. 2000;80(3):276-289.
12. Kebaetse M, McClure P, Pratt NA. Thoracic position effect on shoulder range of motion, strength, and three-dimensional scapular kinematics. *Arch Phys Med Rehabil*. 1999;80:945-950.
13. Solem-Bertoft E, Thuomas K, Westerberg C. The influence of scapular retraction and protraction on the width of the subacromial space: an MRI study. *Clin Ortho Relat Res*. 1993;296:99-103.
14. Lukasiewicz AC, McClure P, Michener L, Pratt N, Sennett B. Comparison of 3-dimensional scapular position and orientation between subjects with and without shoulder impingement. *J Orthop Sports Phys Ther*. 1999;29(10):574-586.
15. Bullock MP, Foster NE, Wright CC. Shoulder impingement: the effect of sitting posture on shoulder pain and range of motion. *Manual Therapy*. 2005;10:28-37.
16. Evcik D, Aksoy O. Relationship between head posture and temporomandibular dysfunction syndrome. *J Musculoskel Pain*. 2004;12(2):19-24.
17. Gonzalez HE, Manns A. Forward head posture: its structural and functional influence on the stomatognathic system, a conceptual study. *J Craniomandib Pract*. 1996;14(1):71-89.
18. Wright EF, Domenech MA, Fischer JR. Usefulness of posture training for patients with temporomandibular disorders. *JADA*. 2000;131:202-210.
19. Fernandez-de-las-Penas C, Alonso-Blanco C, Cuadrado ML, Gerwin RD, Pareja JA. Trigger points in the suboccipital muscles and forward head posture in tension-type headache. *Headache*. 2006;46:454-460.
20. Fernandez-de-las-Penas C, Alonso-Blanco C, Cuadrado ML, Pareja JA. Forward head posture and neck mobility in chronic tension-type headache: a blinded, controlled study. *Cephalalgia*. 2005;26:314-319.
21. McMaster MJ, Glasby MA, Singh HS, Cunningham S. Lung function in congenital kyphosis and kyphoscoliosis. *J Spinal Disord Tech*. 2007;20(3):203-208.
22. Kapreli E, Vourazanis E, Billis E, Oldham JA, Strimpakos N. Respiratory dysfunction in chronic neck pain patient: a pilot study. *Cephalalgia*. 2009;29(7):701-710.
23. Cacciatore TW, Horak FB, Henry SM. Improvement in automatic postural coordination following Alexander technique lessons in a person with low back pain. *Phys Ther*. 2005;85(6):565-578.
24. Nemmers TM. The influence of forward head posture on balance, fall self-efficacy, and physical activity level in community-dwelling women age 60 and older; and the relationship of these variables to self-reported fall history. Doctoral Dissertation, Oklahoma State University, 2006.
25. Hiemeyer K, Lutz R, Menninger H. Dependence of tender points upon posture: a key to the understanding of fibromyalgia syndrome. *J Manual Medicine*. 1990;5:169-174.
26. Keller TS, Harrison DE, Colloca J, Harrison DD, Janik TJ. Prediction of osteoporotic spinal deformity. *Spine*. 2003;28(5):455-462.

27. Evcik D, Aksoy O. Correlation of temporomandibular joint pathologies, neck pain, and postural differences. *J Phys Ther Sci*. 2000;12(2):97-100.
28. Conable B, Conable W. *How to Learn the Alexander Technique, A Manual for Students*. 2nd ed. Columbus, OH: Andover Road Press; 1992.
29. Tavrazich J. Rolfing, Hellerwork, and Soma. In: Davis CM, ed. *Complementary Therapies in Rehabilitation, Holistic Approaches for Prevention and Wellness*. Thorofare, NJ: SLACK Incorporated; 1997.
30. Prsala, J. *Help Yourself to a Healthy Back*. 2nd ed. Halifax, NS: Dalhousie University; 2001.
31. Brennan R. *The Alexander Technique Workbook: Your Personal System for Health, Poise, and Fitness*. London: Element Books Ltd; 1992.
32. Janda V. Muscles and motor control in cervicogenic disorders. In: Grant R, ed. *Physical Therapy of the Cervical and Thoracic Spine*. 3rd ed. New York, NY: Churchill Livingstone; 2002.
33. Murphy DR. *Conservative Management of Cervical Spine Disorders*. New York, NY: McGraw-Hill; 2000.
34. McKenzie RA. *The Cervical and Thoracic Spine, Mechanical Diagnosis and Therapy*. Waikanae, New Zealand: R.A. McKenzie; 1990.
35. Watson DH, Trott PH. Cervical headache: an investigation of natural head posture and upper cervical flexor muscle performance. *Cephalalgia*. 1993;13:272-284.
36. Placzek JD, Pagett BT, Roubal PJ, et al. The influence of the cervical spine on chronic headache in women: a pilot study. *J Manual Manipulative Ther*. 1999;7(1):33-39.
37. Fernandez-De-Las-Penas C, Perez-De-Heredia M, Molero-Sanchez A, Miangolarra-Page JC. Performance of the craniocervical flexion test, forward head posture, and headache; clinical parameters in patients with chronic tension-type headache: a pilot study. *J Orthoped Sports Phys Ther*. 2007;37(2):33-39.
38. Warmerdam A. *Manual Therapy: Improving Muscle and Joint Functioning*. Wantagh, NY: Pine Publications; 1999.
39. Cleland J, Selleck B, Stowell T, et al. Short-term effects of thoracic manipulation on lower trapezius muscle strength. *J Manual Manipulative Ther*. 2004;12:82-90.
40. Makofsky H, Panicker S, Abbruzzese J, Aridas C, Camp M, Drakes J, Franco C, Sileo R. Immediate effect of grade IV inferior hip joint mobilization on hip abductor torque: a pilot study. *J Manual Manipulative Ther*. 2007;15(2):103-111.
41. McPartland JM. Travell trigger points—molecular and osteopathic perspectives. *JAOA*. 2004;104(6):244-249.
42. Shah JP, Phillips TM, Danoff JV Gerber LH. An in vivo microanalytical technique for measuring the local biochemical milieu of human skeletal muscle. *J Appl Physiol*. 2005;99:1977-1984.
43. Simons DG, Travell JG, Simons LS, Cummings BD. *Myofascial Pain and Dysfunction: The Trigger Point Manual, Vol. 1: The Upper Half of the Body*. Baltimore, MD: Lippincott Williams & Wilkins; 1999.
44. Andrade C-K, Clifford P. *Outcome-Based Massage, From Evidence to Practice*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008.
45. Amodie-Storie C, Nash MS, Roussell PM, Knox AW, Crane LD. Head position and its effect on pulmonary function in tetraplegic patients. *Spinal Cord*. 1996;34:602-607.
46. Greenman PE. *Principles of Manual Medicine*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2003.
47. Greenfield B, Catlin PA, Coats PW, Green E, McDonald JJ, North C. Posture in patients with shoulder overuse injuries and healthy individuals. *J Orthoped Sports Phys Ther*. 1995;21(5):287-295.
48. Panjabi M. The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. *J Spinal Disord*. 1989;5:383-389.
49. Panjabi M. The stabilizing system of the spine. Part II. Neutral zone and stability hypothesis. *J Spinal Disord*. 1992;5:393-397.
50. Richardson C, Hodges P, Hides J. *Therapeutic Exercise for Lumbopelvic Stabilization: a Motor Control Approach for the Treatment and Prevention of Low Back Pain*. 2nd ed. Edinburgh: Churchill Livingstone; 2004.
51. Jemmett R. *Spinal Stabilization: the New Science of Back Pain*. 2nd ed. Canada: Libris Hubris Publishing; 2003.
52. McKenzie R, May S. *The Lumbar Spine, Mechanical Diagnosis & Therapy*. Vol 1. Waikanae, New Zealand: Spinal Publications New Zealand Ltd; 2003.
53. McKenzie R, May S. *The Lumbar Spine, Mechanical Diagnosis & Therapy*. Vol 2. Waikanae, New Zealand: Spinal Publications New Zealand Ltd; 2003.
54. Harrington KI, Haskvitz EM. Managing a patient's constipation with physical therapy. *Phys Ther*. 2006;86(11):1511-1519.
55. Koffler KH, Menkes A, Redmond RA, Whitehead WE, Pratley RE, Hurley BF. Strength training accelerates gastrointestinal transit in middle-aged and older men. *Med Sci Sports Exerc*. 1992;24(4):415-419.
56. Noble E. *Essential Exercises for the Childbearing Year: A Guide to Health and Comfort Before and After Your Baby is Born*. 4th ed. Harwich, MA: New Life Images; 1995.
57. Prsala J, Belzer EG, Simpson C, Stalker RE. The ratio of the length of the thoracic-kyphotic curve to that of the lumbar lordotic curve in relation to low-back pain. *Nova Scotia Med J*. 1993;185-187.
58. Godges JJ, Anger MA, Zimmerman G, Delitto A. Effects of education on return-to-work status for people with fear-avoidance beliefs and acute low back pain. *Phys Ther*. 2008;88(2):231-239.
59. Sahrmann SA. *Diagnosis and Treatment of Movement Impairment Syndromes*. St. Louis, MO: Mosby, Inc.; 2002.
60. Isaacs ER, Bookhout MR. *Bourdillon's Spinal Manipulation*. 6th ed. Woburn, MA: Butterworth-Heinemann; 2002.
61. Chiarello C. Spinal disorders. In: Cameron MH, Monroe LG, eds. *Physical Rehabilitation: Evidence-Based Examination, Evaluation, and Intervention*. St. Louis, MO: Saunders Elsevier; 2007.
62. Myers TM. *Anatomy Trains: Myofascial Meridians for Manual and Movement Therapists*. 2nd ed. Edinburgh: Churchill Livingstone Elsevier; 2009.

Section

VII

From the Classroom to the Clinic

26

The Evidence for Spinal Manual Therapy and Therapeutic Exercise

"Looking at the ubiquitous use of manual therapy and my own personal preferences for many manual techniques, it is with sorrow that I observe how the great edifice of manual therapy has been built upon the shakiest of foundations. I understand how the great American patriot John Adams felt when he was forced by his principles to reluctantly face reality and defend British soldiers accused in the Boston massacre. As Adams observed during that defense: 'Facts are stubborn things; and whatever may be our wishes, our inclinations, or the dictates of our passions, they cannot alter the state of facts and evidence.' So it must be with manual therapy.

We lack facts and evidence. Does this mean that manual therapy techniques do not work? No! It means that, whether we like it or not, our profession's endorsement of manual therapy is based on anecdotal observations and a shared faith, a belief that exists in the absence of evidence. I understand this because I too was a believer, one who accepted with enthusiasm and without critical thinking."

Jules M. Rothstein, PhD, PT
Editor's Note, *Physical Therapy*
1992;72(12)

In 1996, Sackett et al[1] defined evidence-based medicine (EBM) as the "conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients." Four years later, Sackett et al[2] described EBM as the "integration of best research evidence with clinical expertise and patient values." To determine "best evidence," Sackett et al rate[2] the type of study employed on a level 1 through 5 scale, with level 1 being the ideal and levels 4 and 5 falling into the category of "lower-level" research. In numerical order, they are as follows: randomized controlled trials (RCTs) = level 1, cohort studies = level 2, case-controlled studies = level 3, case series without a control group = level 4, and expert opinion = level 5. Bogduk uses the acronym RVE to define evidence-based medicine, where R = reliability, V = validity, and E = efficacy. His philosophy is that EBM aspires to use procedures that are reliable and valid, treatments that are known to be effective, and avoids practices that lack reliability, validity, and efficacy.[3]

In this chapter, the author seeks to demonstrate that manual therapy is not built upon "the shakiest of foundations." Although more high quality RCTs[4] are needed to demonstrate treatment efficacy, there is more than "anecdotal observations" and a "shared faith" to support the use of manual therapy within the larger context of physical therapy. In essence, the late Dr. Jules Rothstein challenged the manual therapy community to either "put up or shut up." Such a challenge should provide the impetus for scholarly activity at a time when it is most needed. Manual physical therapists have had 17 years since this challenge was given by the late Dr. Rothstein to address this "absence of evidence," and progress has definitely been made. However, this is no time to rest. Many more high-quality RCTs need to be done, supporting the utilization of manual therapy and therapeutic exercises, before we can say that the "absence of evidence" issue has been adequately addressed. We must continue to be held accountable for the claims that we make and ultimately for the living that we

Makofsky HW
Spinal Manual Therapy, 2nd ed. (pp 243-266)

earn. Not only has there been a lack of evidence to support the use of manual therapy in the past, but there is a growing body of evidence in support of other approaches such as the biopsychosocial model.[5] Whereas some may suggest that the biopsychosocial model replace manual therapy (eg, in the management of LBP), this author is of the opinion that the integration of several treatment models (ie, manual therapy, therapeutic exercise, biopsychosocial, spinal surgery, etc), all of which must be evidence-based, is in the best interest of the patient. According to Sackett et al,[2,6] practitioners of manual therapy can claim "clinical expertise and the patient's unique values and circumstances," but that is unacceptable in today's healthcare environment. As manual physical therapists who routinely incorporate mobilization/manipulation and therapeutic exercise into patient care, we must be able to demonstrate the clinical effectiveness of these interventions with sound science as well.

To this end, the author has selected representative studies on various aspects of the practice of spinal manual therapy and therapeutic exercise (ie, diagnostic and interventional applications) with emphasis on patient outcomes.

Study #1: Jull G, Bogduk N, Marsland A. The accuracy of manual diagnosis for cervical zygapophyseal joint pain syndromes. *Med J Aust.* 1988;148(5):233-236.

Twenty consecutive patients from the Pain Clinic at the Princess Alexandra Hospital entered the study. There were 7 men and 13 women. Fourteen patients complained of neck pain and headache, 3 patients complained of neck and arm pain, and 3 patients complained of neck pain alone. All patients had chronic neck pain for at least 12 months.

In 11 patients, radiologically-controlled diagnostic nerve blocks were used to determine the presence or absence of a symptomatic zygapophyseal joint in the cervical spine. All 11 patients were then seen by a manipulative physiotherapist who had no knowledge of the results of the diagnostic nerve block. In the remaining 9 patients, the above sequence of events was reversed.

The manipulative physiotherapist, using a combination of PAIVMs and PPIVMs, correctly identified all 15 patients with proven symptomatic zygapophyseal joints. None of the 5 patients with asymptomatic joints were misdiagnosed. Furthermore, the therapist specified the correct segmental level of the symptomatic joint in each instance.

The researchers concluded the following, "Manual diagnosis by a trained manipulative therapist can be as accurate as radiologically-controlled diagnostic blocks in the diagnosis of cervical zygapophyseal syndromes." The authors do suggest, however, that further research into intertherapist reliability be performed before generalized claims about the reliability of manual diagnosis can be made.

Study #2: Donelson R, Aprill C, Medcalf R, Grant W. A prospective study of centralization of lumbar and referred pain: a predictor of symptomatic discs and annular competence. *Spine.* 1997;22(10):1115-1122.

Sixty-three patients (41 men, 22 women) with LBP and varying degrees of lower extremity pain/altered sensation participated in this prospective, blinded study. Patients with a history of prior lumbar surgery, including chemonucleolysis, were excluded. The average age was 39.6 years, and all patient symptoms were present for greater than 3 months.

Upon entering the radiology clinic for the scheduled lumbar discography, each patient underwent a McKenzie assessment using repeated end-range lumbar test movements. Each examiner was a Diplomat in mechanical diagnosis and therapy, as well as a faculty member of the McKenzie Institute. One of 3 effects on pain was identified during each patient's mechanical assessment. They were rapid centralization or abolition of the referred pain ("centralizers"); no centralization, but peripheralization of pain in one or more directions ("peripheralizers"); and no change in the distal-most pain location or intensity ("no change").

Immediately after the mechanical assessment, patients underwent lumbar discography by a single investigator "blinded" to the findings of the McKenzie exam. During disc injection, each patient was assessed for pain response by the discographer and a second observer. Provocation discography provides direct information about nuclear morphology and the status of the nuclear envelope; it is the "gold standard" for determining whether a disc is painful.

Results of the McKenzie assessment indicated that 31 patients (49.2%) were "centralizers," 16 patients (25.4%) were "peripheralizers," and the remaining 16 patients (25.4%) experienced "no change." Furthermore, of the 31 patients who were "centralizers," 23 (74%) had a positive discogram ($P<.0007$). Of those 23, the annular wall of the positive disc was competent in 21 patients or 91% ($P<.001$). Of the 16 patients (25.4%) who were "peripheralizers," 11 (69%) had a positive discogram ($P<.004$). Of those 11, the annular wall of the positive disc was competent in 6 (54%) patients ($P=.093$). Of the 16 patients (25.4%) whose pain showed "no change," only 2 (12.5%) had a positive discogram ($P<.001$); the annular walls of these 2 positive discs were both competent. Considering the high incidence of positive discograms in "centralizers" and "peripheralizers" and the low incidence in the "no change" group, the ability to distinguish between a positive and a negative discogram on the basis of pain responses alone was highly significant ($P<.001$). In patients with positive discograms, the "centralizers" demonstrated a significantly greater incidence of annular competence as compared to the "peripheralizers" ($P<.042$).

Based on these data, the researchers concluded that the McKenzie assessment process reliably distinguished discogenic from nondiscogenic pain ($P<.001$) as well as a competent from an incompetent annulus ($P<.042$) in symptomatic discs. In their discussion of spinal imaging procedures (eg, radiography, CT, MRI, and myelography), the authors point out that, unlike the McKenzie system, these procedures are unable to determine the source of the patient's painful symptoms. Although this is not the case with invasive discography, the authors are quick to suggest that the McKenzie assessment system, unlike discography, can easily and safely be implemented in the acute setting, allowing for the early identification of relevant response groups with minimal risk to the patient.

In summary, these researchers have demonstrated the clinical relationship between the centralization phenomenon and a contained intervertebral disc herniation, the peripheralization phenomenon and the likelihood of a noncontained extruded disc, and the "no change" pattern of a nondiscogenic impairment. Consequently, these findings suggest an important role for the McKenzie method, not only in the mechanical diagnosis of discogenic symptoms, but also as a means of identifying those disc patients who are the most likely to benefit from nonsurgical, mechanical therapy. Although the findings of this study support the validity of the McKenzie internal disc model and the role of the annulus as a pain generator, the researchers still admit to not fully understanding "the precise neural mechanism by which pain centralizes."

Study #3: Schoensee SK, Jensen G, Nicholson G, Gossman M, Katholi C. The effect of mobilization on cervical headaches. *J Orthop Sports Phys Ther.* 1995;21(4):184-196.

Twelve subjects (between the ages of 20 and 50), satisfying the diagnostic criteria for cervical headache, were recruited, but 10 subjects (3 males and 7 females) went on to complete the study (one subject was hospitalized for appendicitis and the second subject was not included because of incomplete data). A single case A-B-A design was used for each of the 10 subjects in the study. The A phase consisted of data collection on headache frequency, duration, and intensity. The B phase, or treatment phase, consisted of 2 to 3 mobilization sessions per week, for 4 to 5 weeks, for a total of 9 to 11 treatment sessions. The subject then entered the second A, or withdrawal phase, duplicating the first phase and lasting approximately 1 month.

Treatment consisted of mobilization techniques to the limited or painful upper cervical segments (O-C1; C1,2; and C2,3) found on passive accessory and physiologic testing. The mobilizations included central and unilateral PA pressures described by Maitland and the following techniques described by Paris:

- Inhibitory distraction
- Physiological rotation of C1,2 in sitting
- Occipital nod on the atlas
- Lateral pressures on the atlas
- Upslides and downslides on the upper cervical facets

A one-way ANOVA for repeated measures on headache frequency, duration, and intensity was found to be statistically significant. Visual analysis of data plots also revealed a decrease in headache frequency, duration, and intensity from the baseline phase to the treatment phase. This improvement continued through the second A phase for frequency, but leveled off for both duration and intensity. Complete headache relief was obtained in 1 of the 10 subjects. The placebo effect was partially countered by the use of an additional baseline after treatment. The authors report finding the greatest impairment of motion at C2,3, which is in agreement with the findings of related studies on the topic.

In summary, this study revealed that mobilization of the upper cervical spine had a therapeutic effect in reducing the frequency, duration, and intensity of headaches in 10 patients suffering from cervical headache, with 1 of the 10 experiencing complete headache relief.

Study #4: Schenk R, MacDiarmid A, Rousselle J. The effects of muscle energy technique on lumbar range of motion. *Journal of Manual & Manipulative Therapy.* 1997;5(4):179-183.

Considering that the goal of manipulation is to "restore maximal pain-free motion within postural balance," the Schenk et al study, with its focus on range of motion, is particularly relevant to our discussion of patient outcomes. When considering the connection between limited lumbar extension and the incidence of disc derangements in young adults, there is added significance.

The researchers included 26 subjects with limited lumbar spine extension. Subjects were randomly assigned to either the treatment group, which consisted of 8 males and 5 females, or to the control, consisting of 5 males and 8 females. The average age of the subjects was 25 years.

The study was a pretest-posttest design, comparing the effects of MET on lumbar extension mobility in the treatment group versus an untreated control. The independent variable was the application of osteopathic MET; the dependent variable was the change in extension range of motion of the lumbar spine. Lumbar extension was measured with the bubble inclinometer (intrarater and interrater reliability for lumbar extension were r = 0.93 and r = 0.89, respectively).

The experimental group underwent 8 sessions of MET (twice per week for 4 weeks), performed by a board certified orthopedic clinical specialist who was also certified in orthopedic manual physical therapy. At the conclusion of the intervention, all subjects were re-examined for changes in lumbar spine extension.

An independent group t-test revealed a statistically significant increase ($P<.05$) in lumbar extension range of motion in those who were treated with manual therapy versus those who were not. The average range of lumbar extension for the treatment group was 13.8 degrees at pretest and 20.7 degrees at posttest. The average range of lumbar extension for the untreated control was 17.1 degrees at pretest and 16.7 degrees at posttest.

This study demonstrates the ability of a well-executed manual therapy intervention to significantly alter impaired lumbar extension for the better. Although the sample size was small and improvements in study design could be made (eg, improved examiner "blinding," enhanced placebo-control), the Schenk et al study does provide evidence in support of manipulative therapy. It is also written for clinicians by clinicians; unlike many outcome studies, the reader does not need a PhD to understand it.

Study #5: Mitchell UH, Wooden MJ, McKeough DM. The short-term effect of lumbar positional distraction. *Journal of Manual & Manipulative Therapy*. 2001;9(4):213-221.

A convenience sample of 30 patients presenting with low back pain and unilateral radiating symptoms, secondary to nerve-root irritation and associated with dermatomal sensory loss or weakness in a specific myotome, were included in this study. Patients with previous back surgeries, spinal stenosis, and unstable spondylolisthesis were excluded from the study. Patients were randomly assigned to the treatment or control group. The treatment group included 15 patients (9 females and 6 males) with an age range of 19 to 65 years. The control group also included 15 patients (8 females and 7 males) with an age range of 37 to 54 years. Each patient underwent a thorough examination, including a neuromuscular assessment to determine their eligibility for the study (13 patients in the treatment group exhibited specific sensory and 2 patients, specific motor impairment, while 15 patients in the control had specific sensory and 3 had weakness in a specific myotome). After signing the informed consent, the patients with even numbers were assigned to the treatment group consisting of 5 minutes of lumbar positional distraction; those with odd numbers were assigned to the control and were asked to "comfortably lie on the pain-free side," also for 5 minutes. A second examiner, blinded to the patient's group assignment, performed the postintervention examination, which was identical to the initial examination and included an assessment of pain using a verbal digital scale (1 to 10), an assessment of the pain site with a body diagram, and a measurement of SLR height.

The Wilcoxon signed-ranks test was employed to assess the difference between the pre- and post-pain-score in both groups. In the treatment group, there was a statistically significant improvement in symptoms (P=.001), whereas in the control there was no significant change noted (P=.506). Regarding pain location, 10 patients in the treatment group reported centralization, 1 reported peripheralization, and 2 reported "no change" in response to 5 minutes of positional distraction. In the control group, 3 patients reported "centralization," 2 reported peripheralization, and 10 reported "no change" in response to 5 minutes of comfortable side lying. Regarding SLR height, the treatment group demonstrated a statistically significant increase in height (P=.005), whereas the control group did not (P=.884). The authors indicate the "very high" reliability of the test and re-test SLR data in this study (ie, correlation coefficients of 0.99 and 0.989, respectively).

This study supports the clinical efficacy of lumbar positional distraction in 3 areas: diminished pain intensity, centralization of painful symptoms, and improvement in neurodynamic testing of the sciatic nerve with the classical SLR. The purpose of lumbar positional distraction is to decompress painful nerve roots by increasing space in the intervertebral foramina. Unlike the McKenzie approach, which claims to reduce the joint derangement and therefore cause centralization, positional distraction is a temporary measure that modulates, but does not correct, the underlying disorder. However, an intervention that readily decompresses "pinched nerves" with the simple use of a towel roll is welcome news to patients who suffer with these afflictions. In addition, where the McKenzie approach has limited effectiveness in achieving nerve root decompression (ie, extrusion of the disc with an incompetent annulus or stenosis of the lateral recess), positional distraction is an excellent alternative.

On the negative side, the researchers acknowledge that the sample size was small and that "only the very immediate effect of positional distraction was investigated and that the statistically significant difference should not be confused with a clinically important change." Be that as it may, manual physical therapists welcome the Mitchell et al study and look forward to seeing other clinical studies that confirm, with evidence, what clinicians have known experientially for years.

Study #6: Donelson R, Grant W, Kamps C, Medcalf R. Pain response to sagittal end-range spinal motion: a prospective, randomized, multicentered trial. *Spine*. 1991;16(6):S206-S212.

Patients with nonspecific LBP, with or without referred leg pain, presenting at 12 physical therapy clinics in 5 different countries (Australia, Canada, New Zealand, the United Kingdom, and the United States), were considered potential participants. Seventeen different examiners, 14 of whom had extensive experience with the McKenzie method, were involved in data collection. A total of 267 patients provided informed consent; however, after exclusions, 145 patients were included in the final study sample. Information on the location of a patient's pain at the time of the study was used to determine their Quebec Task Force (QTF) classification. Patients were then entered into 1 of 2 protocols based upon month of birth.

All patients were asked to record the location of symptoms on a pain drawing and the intensity of their central and most distal symptom on an analog scale. Thereafter, the protocols consisted of a sequence of single and repeated flexion and extension movements performed to the patient's available end-range, first standing and then while recumbent. The 2 protocols differed only in the order in which the flexion and extension movements were performed. Following each single movement and each repeated movement sequence, patients again recorded the intensity of their central pain and the location and intensity of their most distal symptom on a standardized form. Movements were repeated to a maximum of 4 sets of 10 repetitions with brief rest periods between each set of 10. Movements were terminated if the pain intensified or peripheralized.

Mantel-Haenszel chi-square analysis was used to explore group differences for categorical data and the Yates correction for continuity (z) to test distributions of proportions. The student t-test for unpaired samples was used to evaluate initial group differences in continuous variables; an analysis of covariance for repeated measures was employed to standardize patient's initial responses to a common starting point. Data analysis proceeded along 2 main lines of inquiry. The first involved whether changes in central pain intensity (CI), distal pain intensity (DI), and distal to most peripheral pain (DIST) could be attributed to the protocol used. The second was concerned with the relationship between symptom differences and the direction of movement. Based on the preliminary data analysis, an analysis model was constructed to assess differences recorded as patients were moved through the 2 protocols. Because CI, DI, and DIST changed after the performance of each movement sequence and consequently affected the patient's starting point for the subsequent movement sequence, analysis of covariance techniques statistically adjusted the results of one movement sequence to serve as a baseline for the next. Ultimately, a modified regression equation determined whether the groups known to be different from one another could be identified by the data and described by the model. Because the outcome variable (protocol) was categorical, logistic regression techniques were used to allow for the development of models of prediction for noncontinuous outcome variables. Specific design structures used to reduce potential bias included the multicentered, randomized, and prospective research design; standardization of the assessment process consisting of a set of written instructions; and the inclusion of examiners who had little or no experience in spinal care and who, therefore, were believed to have had no expectations of outcome.

Results demonstrated no significant differences between the 2 protocol groups for gender, age, QTF classification, work status, back and leg symptoms, or the number of past painful episodes. However, significant differences in responses to flexion and extension were found. Regardless of whether flexion preceded or followed extension, flexion increased intensity and peripheralized pain (ie, an increase in DIST) for the mean of both study groups, while extension decreased intensity and centralized pain (ie, a decrease in DIST). The statistical results from the testing of this model were all significant at $P<.0001$. Only 1 of the 145 patients noted improvement in both flexion and extension during standing testing; no subject showed improvement in both directions while recumbent. This is an important finding when considering that spinal activity in general is considered beneficial for LBP patients. Forty percent improved with extension and worsened with flexion, whereas 7% improved with flexion and worsened with extension. This preference was highly significant ($P<.001$). Not only did one direction clearly centralize their symptoms, but the opposite direction typically intensified or peripheralized them. It was apparent that the performance of a single test movement frequently resulted in a different pain response than performing the same movement repetitively. The inclusion of single movements confounded the differences noted; when single movement responses were deleted, results of analysis of repeated test movements were definitive.

In summary, regardless of the order of spinal movements, there were highly significant differences between the effects of flexion and extension test movements on pain intensity and distal location. Whereas end-range extension significantly decreased central and distal intensity and centralized referred pain, lumbar flexion significantly increased central and distal pain intensity and peripheralized the patient's symptoms. Furthermore, individual patients clearly had statistically significant directional preferences (ie, 40% of this study group improved with extension and 7% improved with flexion). In a previous study by Donelson et al,[7] a centralizing effect was identified in 87% of the patients compared to 47% in this study. The difference can be partially explained by the fact that, in the former study, the test movements were not limited to the sagittal plane. McKenzie has often stated that many patients whose pain does not centralize with repeated flexion or extension will experience centralization with lateral or rotational test movements. In the Donelson et al[7] study, the McKenzie method of achieving centralization, while concurrently discouraging positions and movements that cause peripheralization, yielded excellent patient outcomes in 92% of the cases and good outcomes in 6% of patients when these patients had symptoms for less than 4 weeks. In patients with symptoms lasting longer than 12 weeks, excellent or good outcomes were achieved in 81% of the cases.

Lastly and perhaps most importantly, this study demonstrates the ability of the McKenzie system to assist the clinician in determining the appropriate mechanical intervention for the patient. The notion that any form of exercise for LBP is therapeutic, regardless of its direction, must be reconsidered based upon these data.

Study #7: Farrell JP, Twomey LT. Acute low back pain: comparison of two conservative treatment approaches. *Med J Aust.* 1982;1:160-164.

Of 56 eligible patients, 48 completed the study. Patients of either gender were accepted into the study if they met the following inclusion criteria:

- Age range, 20 to 65 years
- LBP experienced with lumbar movements or SLR
- Intermittent or constant pain between T12 and the gluteal folds
- LBP of 3 weeks duration or less
- A pain-free period of 6 months prior to the onset of the current episode

Patients were excluded if they had other treatment for the current episode, were pregnant, presented with frank neurological signs, had prior lumbar surgery, had a history of a lower thoracic/lumbar fracture, and showed evidence of systemic disease.

The patients were randomly assigned to 1 of 2 groups. The experimental group received passive mobilization and manipulation of the lumbar spine as described by Stoddard[8] and Maitland.[9] The control group received a regimen of microwave diathermy, isometric abdominal exercises, and ergonomic instructions. There were no significant differences between the treatment and control groups in terms of age, gender, LBP history, and the duration of symptoms before treatment. All personnel involved in the examination/evaluation and intervention were physiotherapists who followed standardized measurement and treatment procedures. The assessment included an evaluation of functional limitations, pain severity, active lumbar movements with a lumbar spondylometer and rotameter, and straight leg raising with a standard goniometer. Patients were examined before the first treatment, immediately after the first treatment, after the third session, after the final session, and 3 weeks from the date of the initial treatment. Intraobserver tests showed no significant differences between measurements ($P<.01$), thus reinforcing the reliability of these devices. The patients as well as the examiner were "blinded."

Each patient was treated 3 times a week for up to 3 weeks. For a subject to be pronounced "symptom free," it was necessary that he or she could perform all functional activities without difficulty, his or her subjective pain was either 0 or 1 on a 0 through 10 scale, and the objective measures of lumbar movements and SLR were pain free with passive overpressure at the extreme of the patient's active range. If a patient met the criteria for discharge before 3 weeks, treatment was discontinued.

A Mann-Whitney U-test indicated a statistically significant difference between the 2 groups in the number of treatments needed to reach the symptom-free status ($P<.001$). The manipulative group required 3.5 ± 1.6 treatments, while the control required 5.8 ± 2.3 to reach the same result. An analysis of covariance indicated that the manipulative group had significantly greater lumbar extension following the last session ($P<.05$). However, this was not the case for the other active lumbar movements. Overall, the researchers were not impressed with significant differences in active lumbar range of motion between the 2 groups. At the end of the 3 weeks, there was no significant difference between the subjective pain ratings of the 2 groups, although the trend favored the manipulative group. Within 4 weeks of developing symptoms, 91% of all subjects recovered from their symptoms. This is consistent with other studies that report that the vast majority of patients with acute LBP are asymptomatic within 4 weeks of developing symptoms, regardless of the treatment received.

In summary, the findings of this study strongly suggest that patients with acute LBP treated by passive mobilization/manipulation had a shorter mean duration of symptoms compared to those treated with microwave diathermy, isometric abdominal exercises, and ergonomic instructions. Though one can argue that the placebo effect was stronger in the manipulative group because of greater patient contact, it can also be argued that the control had the advantage of being instructed in proper body mechanics. The "bottom line" is that, despite an "advantaged" control group in several respects (ie, pain-relieving treatment, strengthening exercises, and ergonomic training), the manipulative group still demonstrated a superior clinical outcome.

Study #8: Hoehler FK, Tobis JS, Buerger AA. Spinal manipulation for low back pain. *JAMA*. 1981;245(18):1835-1838.

This was a RCT conducted on 95 patients with LBP. Patients were selected from a group of 1880 patients referred to the University of California, Irvine, Medical Center Back Clinic between June 1973 and June 1979. Exclusion criteria consisted of prior manipulative treatment, disability income, pending litigation, prior back surgery, obesity, drug/alcohol abuse, and pain not amenable to manipulative therapy of the lumbosacral area.

After being admitted to the trial and signing the appropriate informed consent, patients were randomly assigned to either the experimental group or the control group. Patients in the experimental group (56 subjects) received rotational manipulations of the lumbosacral spine consisting of a high-velocity thrust maneuver with the intention of gapping the facet joints and stretching the paravertebral muscles of the lumbosacral area. Patients assigned to the control group (39 subjects) received soft tissue massage of the same area without the rotational thrust manipulation. The number of treatments received was at the discretion of the treating physicians. On discharge, each patient was re-examined by the same physician who performed the initial examination. The patient and physician performing the examinations were both "blinded" in this study. Subjective data came from questionnaires; the objective examination consisted of the SLR to the point of both pain and pelvic rotation, and the distance of the fingertips to the floor in standing forward flexion. Nonparametric statistics were used (ie, the Mann-Whitney U-test) because the data were only measurable on an ordinal scale and therefore not normally distributed. Correlations were measured by the nonparametric Spearman rank-order correlation coefficient; the criteria for statistical significance was $P<.05$ for a one-tailed test.

Although the pretreatment comparison of the 2 groups revealed a somewhat higher proportion of patients with "severe" or "very severe" pain complaints in the experimental group, there were no statistically significant differences regarding the origin of pain, rapidity of pain onset, the extent of pain on lateral bending, SLR to pain, SLR to pelvic rotation, and forward flexion. The experimental group also had a somewhat lower proportion of patients with "chronic" pain, but this too was not a statistically significant difference. Regarding the duration of treatment and number of treatments, the manipulative group exceeded the massage group in both categories. The authors acknowledge, "This effect is difficult to interpret and presents problems for any analysis of postdischarge data."

Moving on to outcome parameters, the data demonstrated the following immediate benefits of spinal manipulation over soft tissue massage:

- The manipulation group showed more improvement than the control ($P<.01$) in 4 of 6 subjective measures of spinal flexibility, including walking, bending or twisting, sitting down in a chair, sitting up in bed, reaching, and dressing.
- The manipulative group reported more pain relief than the control ($P<.05$).
- The manipulative group demonstrated a statistically significant increase in SLR to pain after the first treatment ($P<.01$).
- At discharge, the manipulative group demonstrated superior SLR to pelvic rotation, but this difference was not statistically significant ($P>.05$). One explanation for this lack of significance was the reduced number of patients represented in this particular comparison.

Regarding long-term improvement, apart from the perceived effectiveness of manipulation over massage at 3 weeks postdischarge ($P<.05$), manipulation did not appear to be significantly better than soft tissue massage. However, it must be noted that the 2 groups are similar in this regard because both showed substantial improvement. Furthermore, the long-term effectiveness of manipulation is difficult to assess because, given sufficient time, many patients with back pain will recover with or without intervention.

In summary, these data clearly show that spinal manipulation provides immediate subjective alleviation of LBP. The amount of relief produced by manipulation was significantly greater than the amount of relief produced by soft tissue massage of the affected areas. However, at discharge and following, there was no significant difference between the 2 groups because both showed substantial improvement. This raises another consideration in this study. The authors are pleased with their choice of soft tissue massage as the placebo-control intervention; however, one cannot help but see that soft tissue massage, in reality, is another form of manual therapy. To report that there was no significant difference between the 2 groups at discharge is another way of saying that both forms of intervention are equally effective. Consequently, the outcomes after the first session, at discharge, and at 3 weeks postdischarge indicate that these 2 manual therapy interventions were effective in the management of LBP.

Study #9: Sunshine W, Field T, Schanberg S, et al. Massage therapy and transcutaneous electrical stimulation effects on fibromyalgia. *J Clin Rheumatol*. 1996;2:18-22.

Thirty female adult fibromyalgia syndrome (FMS) patients were recruited from local rheumatology practices. (Note: FMS was confirmed by a rheumatologist using criteria established by the American College of Rheumatology.) Patients averaged 49.8 years; were of middle income levels (on average); and were 32% Caucasian, 44% Hispanic, and 24% African American. The patients were randomly assigned (using a table of random numbers) to 1 of 3 groups: massage therapy, transcutaneous electrical nerve stimulation (TENS), and sham TENS. The 3 groups of women did not differ on the demographic variables of ethnicity, income, or age. The researchers responsible for pre- and postassessments were "blinded" to the group assignment of the patients. Assessments were made during the first and final sessions, sessions 1 and 10, respectively.

All pre- and posttests were performed by the same rheumatologist. A global rating of pain was recorded by the rheumatologist on the first and last days of treatment. Patients were required to maintain their pharmacological regimen during the course of the study. The immediate effects of these interventions were measured by the State Trait Anxiety Inventory (STAI), the Profile of Mood States (POMS), and by stress hormone (cortisol) levels. The end-of-study effects (ie, the end versus the beginning of the study period) were assessed by the dolorimeter test; an interview on pain, sleep, and daily functioning; and by the CES-D (depression scale).

Massage therapy sessions consisted of moderate pressure stroking of the head, neck, shoulders, back, arms, hands, legs, and feet for 30 minutes. The TENS group received microamperage stimulation through the electroacuscope roller to the same areas as the massage group. The sham TENS group received the same tactile stimulation with the electroacuscope roller, however, with the machine turned off. Because the dials and knobs of the unit were hidden from view, the therapist and the patient were both "blinded" during this aspect of the study. Obviously, there was no way to double "blind" the massage group.

Analyses of the immediate treatment effects revealed the following:

- The massage therapy group had lower state anxiety STAI ($P=.001$), lower depressed mood (POMS) scores ($P=.05$), and lower salivary cortisol levels ($P=.05$).
- There were no statistically significant immediate treatment effects of either TENS or sham TENS.

Analyses of longer-term effects (first-session/last-session measures) suggested the following:

- The massage group had lower anxiety/depression scores and salivary cortisol ($P=.05$).
- The TENS group demonstrated statistically significant improvement in all 3 measures (ie, anxiety, $P=.01$; depression, $P=.01$; cortisol level, $P=.05$).
- No changes were noted in the sham TENS group.
- The massage group improved on the rheumatologist's rating of clinical condition ($P=.05$) and dolorimeter

value (P=.01). There were significantly fewer symptoms at the end of the study, including less pain, less pain over the last week, less stiffness, less fatigue, and fewer nights of difficult sleeping.

- The TENS group only improved on the physician's assessment of clinical condition.
- The sham TENS group also improved on the physician's assessment of clinical condition but to a lesser degree than the other 2 groups.

In summary, this study of 30 adult female FMS patients demonstrated the following outcomes:

- Soft tissue massage therapy was superior to TENS and sham TENS in reducing anxiety and depression.
- Whereas both therapeutic massage and TENS significantly reduced anxiety, depression, and salivary cortisol levels on the last day of treatment, only massage showed these changes on both the first and last day of treatment.
- Although the rheumatologist's assessment of the subject's clinical condition improved for all 3 groups, only the massage group improved on the dolorimeter and the subject's self-report of pain.
- Only the massage group consistently reported significantly fewer symptoms by the end of the study, including less pain, stiffness, fatigue, and difficulty sleeping.

Whereas the emphasis in physical therapy education has historically been on the physiological effects of soft tissue mobilization/massage, this study clearly demonstrates the psychological benefits of this important manual therapy intervention as well.

Study #10: Saal JA, Saal JS. Nonoperative treatment of herniated lumbar intervertebral disc with radiculopathy: an outcome study. *Spine*. 1989;14(4):431-437.

The researchers used a retrospective cohort study design to analyze the results of a group of patients treated nonoperatively for lumbar intervertebral disc herniation. The available records of patients seen in the San Francisco Spine Institute and the SpineCare Medical Group in Daly City, California with a diagnosis of herniated lumbar intervertebral disc between January 1, 1985 and April 1, 1986 were reviewed. The inclusion criteria were as follows:

- Diagnosed "herniated nucleus pulposus" (HNP) as per CT and/or MRI.
- Diagnosed lumbar radiculopathy based on a primary complaint of leg pain and a secondary complaint of back pain, a positive EMG demonstrating the electrophysiologic presence of lumbar radiculopathy, and a positive SLR test reproducing leg pain at less than 60 degrees elevation.
- Willingness to participate in an "aggressive" treatment program, including back school, exercise training to teach spinal stabilization (ie, dynamic maintenance of postural control, trunk, and general upper/lower body strengthening and flexibility exercises).

Epidural injections or selective nerve root blocks were to be used when indicated for pain control. All patients in the study had failed passive conservative management and were comparable clinically to the patients evaluated in surgical studies of herniated lumbar discs.

Exclusion criteria were previous lumbar spine surgery and the presence of significant spinal stenosis or spondylolisthesis (ie, grade 3 on the Glenn scale).

A standardized questionnaire, including questions from the Oswestry Scale, pain self-rating, work status, and self-rating of outcome, were mailed to each patient who met the above criteria. Self-rating criteria were as follows:

- Excellent: Working full time, performing usual athletic activities
- Good: Working full time but limited in performance of athletic activities
- Fair: Working part time only, unable to participate in athletic activities.
- Poor: Unable to work and unimproved following treatment

Out of a total of 347 consecutively identified patient records reviewed, 64 were included in the group to whom questionnaires were mailed. A total of 58 questionnaires were returned (91% response rate).

Data analysis included calculation of rates of return to work, average sick-leave time, subsequent surgery due to failure of "aggressive" conservative care, and a self-rating of outcome.

Of the 58 patients in the study, there were 36 men and 22 women with a median age of 35.5 ± 1.2 years. Thirteen (22%) were worker's compensation cases. Weakness of at least one grade on a 0 to 5 grading scale was noted in 37 patients (64%). Symptom duration averaged 4.6 ± 0.6 months. The mean postcare follow-up time was 31.1 ± 1.7 months. Six patients required surgery.

The "aggressive" treatment program utilized in this study consisted of 2 phases. The first was the pain control phase, and the second was the exercise training phase. Pain control consisted of physical therapy, pain-relieving modalities, back school, McKenzie exercises, non-narcotic analgesics, facet joint injections, corticosteroid epidural injections, acupuncture, etc. Exercise training included the use of techniques to improve soft tissue flexibility, joint mobility, joint stability, and aerobic capacity.

Results indicated a success rate, defined as excellent or good, of 83% in the entire study population; an impressive 96% success rate in the nonoperative cases. Forty-eight patients returned to work (83 ± 5.2% of the entire study population and 92 ± 3.5% of all nonoperative patients), and 85 ± 5% of all patients returned to their previous jobs. The average sick-leave time was 3.8 ± 1 month; 26 patients (50 ± 6.9%) reported less than 1 week sick leave. The self-rated reports for these patients were 15 excellent, 35 good, 2 fair, and 0 poor. The median Oswestry score for the excellent group was 16.6, the good group was 20, and the fair group was 32. Therefore, 20 patients who categorized themselves as good by self-report could fall into the excellent group. This would yield 31 excellent results and 14 good ones.

Eleven worker's compensation patients returned to work, with an average sick-leave time of 9 ± 3 months. Six patients required subsequent surgery after unsatisfactory improvement with the nonoperative program. Four of these patients had significant stenosis at the time of operation. One patient had progressive weakness and one, unable to complete the program, referred herself to surgery.

Eighteen patients (31%) were seen for a second opinion. All had been advised by a surgeon that they needed surgery as soon as possible to avoid long-term complications. Of these 18, 15 were nonoperative treatment successes, 3 scoring excellent on the self-rating reports and 12 scoring good. All 15 returned to work with an average sick-leave time of 13.6 weeks.

As per CT or MRI scans, extruded discs were seen in 15 patients. Of these, 11 had weakness. Eighty-seven percent (13 of 15) of these patients had good and excellent outcomes. The average sick-leave time for this group was 2 months and 92% of these patients returned to work. Three of the patients with extruded discs required subsequent surgery, one because of progressive weakness, and one who had significant lateral recess stenosis at the time of surgery. The third withdrew from the program and referred herself to surgery.

In summary, this study demonstrated that patients with HNP and radiculopathy can be successfully treated, nonsurgically. The sick-leave time and return to work rates were superior to rates reported for similar patients treated surgically. The presence of weakness did not adversely affect outcome in the treatment cohort. Disc extrusion was successfully managed 87% of the time. The premise that operative patients fare better in the first year, as noted by the average sick-leave time, is contrary to these outcome measures. Four of the 6 patients who failed "aggressive," nonsurgical treatment were found to have stenosis at subsequent lumbar spine surgery. Thus, failed aggressive nonoperative measures should probably warrant greater decompression than disc excision alone. From this study it appears that HNP combined with stenosis is associated with a different prognosis than HNP without. The results of this study also suggest that failed passive, nonoperative therapy is not a sufficient criterion for the decision to operate.

Study #11: Nicolakis P, Erdogmus B, Kopf A, Djaber-Ansari A, Piehslinger E, Fialka-Moser V. Exercise therapy for craniomandibular disorders. *Arch Phys Med Rehabil.* 2000;81:1137-114.

The objective of this "before-after" trial was to evaluate the use of exercise therapy for the treatment of craniomandibular disorders. Thirty patients (28 women and 2 men) with a mean age of 33.1 ± 11.0 years and diagnosed with ADD with reduction participated in this study. Patients were selected consecutively from patients consulting the Craniomandibular Disorders (CMD) Service at the Department of Dentistry, University of Vienna. At the 6-month follow-up, 26 patients remained in the study (2 were not available and 2 were allocated to splint therapy because they were not satisfied with the treatment result).

Inclusion criteria included symptoms lasting at least 3 months, pain in the TMJ region, signs consistent with a diagnosis of TMJ ADD with reduction, joint clicking together with a straight or convex pathway finding on computerized axiography, and evidence of postural dysfunction.

Patients were examined by the same physiatrist in a standardized manner. After the examination, all patients were assigned to a waiting list for exercise therapy, serving as a no-treatment control period. The following outcome measures were used in this study:

- Pain at rest was measured with a visual analogue scale (VAS).
- Maximal pain during the "last 2 days" (pain at stress) was also measured with a VAS.
- Patients were asked to rate their overall impairment in daily life activities with a VAS.
- The MIO was measured in millimeters (mm).
- The change in self-perceived joint clicking from the outset to the end of treatment was measured on a 4-point scale (ie, vanished, better, equal, and worse).
- Perceived improvement of jaw pain was measured on a 7-point scale (ie, excellent, distinct improvement, moderate improvement, equal, moderate, distinct deterioration, severe deterioration).
- Perceived improvement of jaw function was also measured on the same 7-point scale. The first 4 measures were recorded at baseline, immediately before, immediately after, and 6 months after exercise therapy, while the remaining measures (5 to 7) were recorded only at the second, third, and final examination.

Each patient was treated a minimum of 5 times, with each session lasting 30 minutes (usually 2 treatments per week were administered with the last 2 treatments given at intervals of 1 to 2 weeks to establish the home program). Exercise therapy included massage, stretching, gentle isometric exercises, guided opening and closing movements, manual TMJ distraction, disc/condyle mobilization, postural correction, and relaxation techniques. Patients were also instructed in a home program including some of the above-mentioned exercises for the TMJs, as well as postural and relaxation exercises. Exercise therapy was intended to improve coordination of the muscles of mastication, reduce muscle spasm, and alter the jaw-closing pattern.

According to a "before-after" trial, the time on the waiting list served as the control period. However, because time on the waiting list and treatment time were not equal, changes of all numerical parameters (ie, pain at rest, maximal pain, impairment of quality of life, and MIO) were normalized for daily changes for these 2 periods. Differences between this normalized data were analyzed with the t-test for paired samples. Descriptive data were analyzed by the chi-square test (ie, perceived jaw clicking, pain, and function). For statistical evaluation of perceived improvement of jaw pain and function, the 7-point scale was reduced to a 3-point scale as follows: improvement (excellent, distinct improvement), no change (moderate or no improvement), and worse (distinct or severe deterioration). The Wilcoxon test was used to identify differences between baseline and pretreatment investigation, between pretreatment and post-treatment, and between pretreatment and the 6-month control.

Patients experienced symptoms of CMD for a mean of 2.6 years. Mean duration on the waiting list was 27 days and the mean duration of treatment was 39 days. Patients received a mean of 9.9 treatments. All patients completed treatment. Results revealed that the overall mean pain intensity was reduced significantly as a result of treatment. At the end of therapy, 87% of patients rated improvement in jaw pain as excellent or distinctly improved and 13% experienced a moderate pain reduction. Six months after treatment, 80% of the patients experienced improvement in jaw pain, with no patient reporting deterioration in contrast to his or her pretreatment condition. The effects of treatment on pain at rest, pain at stress, perceived improvement in jaw function, and MIO were all statistically significant ($P<.001$). TMJ clicking vanished in 13.3% and was reduced in another 13.3% after therapy. Six months later 11.5% reported that their clicking had not returned, while 15.4% indicated a reduction in joint clicking. However, a deterioration in clicking had occurred in one patient. At the 6-month follow-up, 5 of the remaining 26 patients were in need of treatment, 4 because of pain and 1 because of excessive clicking.

The authors point out that the results obtained in this study were superior to recent studies using occlusal appliances to treat patients with arthrogenous or myogenous temporomandibular pain and at least equal to studies using either physical therapy modalities or a multimodal approach utilizing a stabilization appliance, exercise therapy, muscle injections, and various forms of physical therapy.

The authors conclude this study with the following statement, "Exercise therapy seems to be useful in the treatment of anterior disc displacement with reduction and pain. The impairing symptoms, jaw pain, and restricted movement can be alleviated significantly."

Study #12: Bronfort G, Evans R, Nelson B, Aker PD, Goldsmith CH, Vernon H. A randomized clinical trial of exercise and spinal manipulation for patients with chronic neck pain. *Spine*. 2001;26(7):788-799.

The objective of this prospective, parallel-group, RCT was to compare the relative efficacy of rehabilitative neck exercise and spinal manipulation for the management of patients with chronic neck pain.

Patients 20 to 65 years who had a primary problem of mechanical neck pain persisting for 12 or more weeks were eligible for the study. Patients were excluded for referred neck pain, severe osteopenia, progressive neurologic deficits, vascular disease of the neck or upper extremity, previous cervical spine surgery, current or pending litigation, inability to work because of neck pain, spinal manipulative therapy (SMT) or exercise therapy within 3 months prior to study entry, or concurrent treatment for neck pain by other healthcare workers. Recruitment of patients was conducted over a 22-month period from October 1994 to July 1996. There were a total of 191 patients (113 females, 78 males).

Patients were randomized to 1 of 3 groups on the basis of a computer-generated list using a 1:1:1 allocation ratio. The 3 groups were as follows:

- Spinal manipulation and low-technology exercise (38 females, 26 males, age 45 ± 10.5 years). At each visit, patients underwent treatment by 1 of 9 chiropractors (15 minutes), followed by a supervised low-technology rehabilitative exercise session (45 minutes).
- MedX exercise (38 females, 25 males, age 43.6 ± 10.5 years). These patients were seen by a physical therapist who, following stretching, upper body strengthening, and aerobic exercise using a dual-action stationary bike, performed dynamic, progressive resistive exercises on the MedX cervical extension and rotation machine (MedX Corp, Ocala, FL).
- Spinal manipulation (37 females, 27 males, age 44.3 ± 11.0 years). Patients in the SMT group received 15-minute sessions of chiropractic manipulation using short-lever, low-amplitude, high-velocity thrust to the cervical spine. To balance for time and attention, all the patients attended 20 1-hour visits during the 11-week study period.

Outcome measures included patient self-report questionnaires administered twice at baseline; 5 and 11 weeks after the start of treatment; then 3, 6, and 12 months after treatment. Pain, the primary outcome measure, was rated with an 11-box scale (0 = no symptoms, 10 = highest severity of pain). The Neck Disability Index measured disability, while the Short Form (SF-36) was used to measure functional health status. The patients rated their improvement using a 9-point ordinal scale. Use of over-the-counter pain medication was assessed by a 5-point scale, with choices from "none" to "every day." Finally, satisfaction with care was assessed by a 7-point scale with choices ranging from "completely satisfied" to "completely dissatisfied."

Cervical spine muscle strength, endurance, and range of motion were measured twice at baseline, then after 11 weeks of treatment by observers "blinded" to patient group assignment. Cervical isometric strength was measured by a computerized load-cell transducer dynamometer; the highest of 3 trials assessing maximal voluntary contraction for flexion, extension, and rotation were used for analyses. Static cervical endurance was measured by having the recumbent patient (supine for flexion, prone for extension) elevate his or her head, free of support with an attached weight, for up to 240 seconds. Dynamic endurance was recorded as the number of repetitions until failure. The attached weight for the static test corresponded to 60% of the maximal voluntary contraction; for the dynamic test, the attached weight was 25% of the maximal voluntary contraction. Active cervical rotation, flexion, extension, and lateral bending ranges of motion were measured with the CA6000 Spine Motion Analyzer (Orthopedic Systems Inc, Haywood, CA).

The statistical analysis involved the use of a repeated measures analyses of covariance (ANCOVA) for each of the patient-rated outcomes. Repeated measures multivariate analyses of variance (MANCOVA) were used as overall tests of treatment differences incorporating the 6 patient-oriented outcomes for the short- and long-term. Change scores (week 11 minus baseline) in objective neck performance data were tested for group differences with an analysis of variance (ANOVA). Group differences were determined by the multiple comparison Newman-Keuls test. Effect sizes were calculated to standardize measurement units of the 6 outcomes and to help evaluate the importance of the magnitude of group differences under the curve. These summary measures were tested for group differences with ANOVA, and 95% confidence intervals were placed on group differences. To evaluate potential predictors of outcome, a multiple linear regression analysis was performed. A statistician independent of the study site performed the main analyses.

An analysis of short-term therapeutic outcomes revealed substantial improvement in all 3 study groups. However, except for satisfaction with care, which was significantly higher in the SMT with exercise group than SMT alone, there were no clinically important or statistically significant differences between groups.

Regarding neck performance outcomes, the SMT/exercise group demonstrated greater gains in strength, endurance, and range of motion than SMT alone (P<.05) after 11 weeks of treatment. The SMT/exercise group also demonstrated more improvement in flexion endurance and in flexion and rotation strength than the group treated with MedX (P=.03). Finally, the MedX group showed greater gains in extension strength and flexion-extension range of motion than the SMT group (P<.05).

An analysis of long-term therapeutic outcomes revealed that most of the improvement noted in all outcomes for the 3 groups at the end of the treatment phase was maintained during the post-treatment follow-up year. There was a group difference in patient-rated pain (P=.02) in favor of the 2 exercise groups. There was a group difference in satisfaction with care, with the SMT/exercise group superior to both MedX and SMT alone (P=.002). The remaining outcome measures showed no significant group differences for neck disability. There were no important differences for any of the patient-oriented outcomes between patients who regularly performed the recommended home exercises throughout the follow-up year (n = 46), those who did them occasionally (n = 51), or those who did not do them at all (n = 62). Overall, these analyses showed that, except for satisfaction with care, there were no important differences between SMT/exercise and MedX. The data did show that SMT/exercise was superior to SMT alone in terms of pain, satisfaction, and improvement and that MedX was superior to SMT in terms of pain.

Regression analyses showed that expectation was not a predictor for any of the outcomes. Although such side effects as temporary increases in neck pain or headache were reported in as many as 23 patients, the differential number of side effects across treatments was not statistically significant.

The following highlights of this study are worth repeating:

- In the short term (ie, during the 11 weeks of intervention), all 3 treatments were associated with substantial improvement in patient-reported symptoms.
- The SMT/exercise group was significantly more satisfied with care than the SMT alone group and the MedX group.
- In terms of neck performance, at least twice as much improvement was observed in the SMT/exercise group over SMT alone.
- The SMT/exercise group showed greater improvement in flexion endurance and flexion strength than the MedX group.
- The tendency in the short term for the 2 exercise groups to perform better in the patient-oriented outcomes than the group treated with SMT alone continued throughout the follow-up year and cumulatively resulted in statistically significant group differences.

Based on these findings, the authors conclude their paper by stating, "Overall, the use of strengthening exercise, whether in combination with SMT or in the form of a high technology MedX program, appears to be more beneficial to patients with chronic neck pain than the use of spinal manipulative therapy alone."

In his commentary on this study, Rand S. Swenson, DC, MD, PhD, points out that the data give support to 2 important clinical concepts. The first is that exercises should be incorporated as a regular part of the treatment of patients with chronic neck pain and the second is that the significantly higher level of treatment satisfaction among the SMT/exercise group "could relate to the addition of a 'hands-on' component to the treatment protocol."

Some would say that the Bronfort et al study raises questions about the relative efficacy of spinal manual therapy. Though it is true that the manipulative group alone was inferior to the manipulative/exercise group in many respects, it must be pointed out that the merits of this combined approach to patient care (ie, spinal manual therapy plus therapeutic exercise) are underscored by this outcome study.

Study #13: Hoving JL, Koes BW, de Vet H, et al. Manual therapy, physical therapy, or continued care by a general practitioner for patients with neck pain: a randomized controlled trial. *Ann Intern Med.* 2002;136(10):713-722.

This RCT consisted of 183 patients between the ages of 18 to 70 years of age who had nonspecific neck pain for at least 2 weeks. Patients were referred to 1 of 4 research centers by 42 general practitioners. Patients with nonbenign causes of neck pain (ie, prior neck surgery, malignancy, neurologic disease, fracture, herniated disc, systemic rheumatic disease, etc) were excluded from the study (40 in all).

Patients were randomly assigned to 1 of 3 groups: manual therapy (n = 60), physical therapy (n = 59), and continued care from a general practitioner (n = 64). Manual therapy, consisting of specific nonthrust spinal mobilization, was performed once per week for 6 weeks by 6 experienced manual physical therapists acknowledged by the Netherlands Manual Therapy Association. Physical therapy, consisting of a combination of massage, heat application, interferential stimulation, stretching, manual traction, and active exercise therapy, was performed twice per week for 6 weeks. The treatment was performed by 5 experienced physical therapists with emphasis on therapeutic exercises (ie, postural correction, stretching, relaxation training, functional and active strengthening/range of motion exercises). These physical therapists, unlike the 6 manual physical therapists in the study, were not specialists in manual therapy. The third group received standardized care from his or her general practitioner, including advice on prognosis, psychosocial issues, self-care (eg, heat application, home exercises), ergonomics (eg, pillow size, work position), and encouragement to await further recovery. Patients were prescribed medication, including paracetamol or nonsteroidal anti-inflammatory drugs as needed. Ten-minute follow-up visits scheduled every 2 weeks were optional. Referral during the intervention period was discouraged. Two research assistants (experienced physical therapists), who were "blinded" to treatment allocation, performed physical examinations at baseline and follow-up.

Outcome data were collected after 3 and 7 weeks. Primary outcome measures focused on perceived recovery, pain, and functional disability, which were measured according to the Neck Disability Index. Secondary outcome measures included the severity of the most important functional limitation, rated by the patient on a numeric 11-point scale. Cervical range of motion was measured using the Cybex Electronic Digital Inclinometer 320 (Lumex Inc, Ronkonkoma, NY). General health was measured according to the self-rated health index (scale 0 to 100) of the Euro Quality of Life scale. Patients recorded absences from work and analgesic use in a diary.

The differences in success rates for perceived recovery were analyzed by applying chi-square tests (univariate analysis). Likewise, differences in improvement rates for absence from work and use of analgesics were analyzed. For the continuous outcome measures, univariate analyses of variance were applied to the differences between the baseline measurement and each of the follow-up measurements (mean improvement).

Multivariate analyses (multiple logistic regression and analyses of covariance) were performed to examine the influence of the following covariates: baseline value of an outcome measure, therapist, age, severity, research center, sex, duration of the current episode, prior episodes of neck pain, headache of cervical origin, radiating pain below the elbow, and patient preference for treatment. For all comparisons, a 2-tailed P value of 0.05 was considered statistically significant.

In general, the outcome measures showed distinct differences, both within groups (compared with baseline) and among groups. These differences usually favored manual therapy more than physical therapy and physical therapy more than continued care. The success rate at 7 weeks was twice as high for the manual therapy group (68.3%) as for the continued care group (35.9%). Physical dysfunction, pain, and disability were less severe in the manual therapy group than in the physical therapy and continued care groups. Some differences in outcome measures were already statistically significant at 3 weeks. At 7 weeks, the success rate for physical therapy (50.8%) was higher than for continued care (35.9%), but this difference was not statistically significant. The success rates for manual therapy were statistically significantly higher than those for physical therapy. Manual therapy scored better than physical therapy on all outcome measures; however, not all differences were statistically significant. There were no statistically significant differences between groups on the Neck Disability Index; however, the Euro Quality of Life scale showed a statistically significant difference in favor of manual therapy compared with physical therapy and continued care. Regarding range of motion, both the manual therapy and physical therapy groups improved markedly when compared to the continued care group. Patients receiving manual therapy had fewer absences from work due to neck pain than the other groups, but the differences were not statistically significant. Regarding analgesic use, the manual and physical therapy groups demonstrated significantly less analgesic use compared with the continued care group.

In their discussion of the results, the authors make the following comments:

- Manual therapy was more effective than continued care on almost all outcome measures.
- Physical therapy scored slightly better than continued care, but most of the differences were, except for range of motion, not statistically significant.
- Although manual therapy seemed to be more effective than physical therapy, differences were small for all outcome measures except for perceived recovery, which was statistically significant (the authors state that perceived recovery may be the most responsive outcome because it combines other outcomes, such

as pain, disability, and patient satisfaction). Perceived recovery was also significantly greater when comparing manual therapy to continued care.

- As expected, the manual therapy group demonstrated the largest increase in cervical spine range of motion.
- The low disability scores on the Neck Disability Index at baseline may have left only a small margin for improvement. Other studies using the Neck Disability Index have found that function may not be severely limited in patients with nonspecific neck pain; therefore, it may lack sensitivity in this regard.
- Mobilization, the passive component of the manual therapy strategy, formed the main contrast with physical therapy or continued care and was considered to be the most effective component.

Study #14: Sterling M, Jull G, Wright A. Cervical mobilization: concurrent effects on pain, sympathetic nervous system activity, and motor activity. *Man Ther.* 2001;6(2):72-81.

This study utilized a double blind, placebo-controlled, within-subjects design in which each subject experienced all 3 experimental conditions (ie, treatment, placebo, and control) in a randomized order. Thirty subjects (16 female and 14 male) with a mean age of 35.77 ± 14.92 years were recruited. Inclusion criteria consisted of mid to lower cervical spine pain of insidious onset, greater than 3 months duration with symptoms originating from the C5,6 segment, as determined by a manipulative physiotherapist. Exclusion criteria included a history of trauma or surgery to the cervical spine, evidence of radiculopathy, headache, dizziness or other cervical spine symptoms, diabetes, or peripheral vascular disease.

Three experimental conditions were applied: SMT, placebo, and control. The SMT treatment consisted of a Maitland grade III PA technique to the articular pillar of C5,6 on the symptomatic side, while the placebo condition consisted of a manual contact at the C5,6 articular pillar on the symptomatic side but with no movement of the vertebral segment. The control consisted of no physical contact between the subject and the researcher. The treatment and placebo conditions involved three 1-minute applications with a 1-minute interval between each. Two researchers were involved in the experiment. Researcher A recorded all pre- and post-experimental measures and was blind to the experimental condition applied. The experimental conditions were applied by researcher B who was an experienced manipulative physiotherapist. Researcher B was "blind" to data collection on each subject.

Three pain-related measures were taken, including scores of the subject's neck pain with VAS, pressure pain thresholds (PPTs) over the symptomatic segment, and thermal pain thresholds (TPTs) also recorded at the C5,6 segment, bilaterally. In addition, 2 measures of sympathetic nervous activity were taken (skin conductance and skin temperature) as well as a measure of EMG activity in the sternocleidomastoid muscles during the "craniocervical flexion test" performed in supine. This test involved the use of an air-filled sensor to monitor flattening of the cervical lordosis during contraction of the longus colli. EMG recordings were taken at 22, 24, 26, 28, and 30 mmHg as the subject was asked to hold each position for 5 seconds. Prior to the main study, the reliability of the test measures utilized was established.

Based upon the postexperiment questionnaire, only 3 of the 30 subjects correctly identified the treatment session. Removal of their data did not significantly affect the results. A one-way ANOVA revealed decreased VAS scores at rest (P=.049). The Newman-Keuls test demonstrated a significant difference between treatment and control conditions but no significant difference for treatment versus placebo condition. There was no significant main effect of condition for VAS scores at end of range cervical rotation (P=.381). Regarding the condition of PPTs on the symptomatic side, a 2-way ANOVA revealed a significant main effect (P=.0042). The post-hoc analysis (Newman-Keuls test) demonstrated a significant difference between treatment and placebo and between treatment and control. There was no significant main effect of condition for TPTs.

A 2-way ANOVA demonstrated a significant main effect of treatment condition for skin conductance (P<.002) and skin temperature (P<.002). Post-hoc analysis revealed a significant difference between treatment and placebo and between treatment and control for skin conductance and skin temperature.

A 2-way ANOVA demonstrated a significant main effect of condition for EMG activity of the superficial neck muscles at pressure levels of 22, 24, and 26 mmHg (P<.0002). Post-hoc analysis demonstrated significant differences between treatment and placebo and between treatment and control at 22, 24, and 26 mmHg of pressure. The treatment condition induced decreases in EMG activity in the superficial neck flexor muscles by approximately 28% at 22 mmHg, 34% at 24 mmHg, and 21% at 26 mmHg. There was no significant reduction in EMG activity of the sternocleidomastoid muscles at 28 and 30 mmHg. The placebo condition induced increases in EMG activity of the superficial neck flexors by approximately 40% at 22 mmHg and 27% at 26 mmHg.

The findings of this study demonstrated that SMT had a hypoalgesic effect specific to mechanical nociception, but not thermal nociception; an excitatory effect on sympathetic nervous system activity; and an effect on motor activity in the cervical region, whereby there was significantly less activity of the superficial neck flexors (sternocleidomastoids, scalenes, and infrahyoids) in the staged craniocervical flexion test. This could imply facilitation of the deep neck flexor muscles with a decreased need for coactivation of the superficial neck flexors at the lower pressure levels of 22 to 26 mmHg. Although mechanical pain thresholds were increased in the order of 23% on the side of treatment, the authors acknowledge that the effect of SMT on VAS scores was less than expected, especially at the end of active movement. They suggest that the treatment technique utilized was not an adequate stimulus given that initial pain scores were low and of long duration. In these cases, more vigorous manual therapy techniques are probably indicated.

Given the combination of effects mentioned (ie, hypoalgesia, sympathoexcitation, and motor effects), the authors suggest that SMT may exert its initial effects by activating descending inhibitory pathways from the dorsal periaqueductal gray area of the midbrain.

Study #15: Jull G, Trott P, Potter H, et al. A randomized controlled trial of exercise and manipulative therapy for cervicogenic headache. *Spine*. 2002;27(17):1835-1843.

Two hundred patients, who met the diagnostic criteria for cervicogenic headache, participated in this prospective, multicenter, randomized controlled trial. Participants, ages 18 to 60 years, were recruited from general practitioners or through advertising in 5 centers located in capital cities in Australia. Inclusion criteria consisted of the following: unilateral or unilateral dominant side-consistent headache associated with neck pain and aggravated by neck postures or movement, joint tenderness in at least 1 of the upper 3 cervical joints as detected by manual palpation, and headache frequency of at least 1 per week over a period of 2 months to 10 years. Exclusion criteria specified bilateral headaches (typifying tension-type headache), features suggestive of migraine, any condition that might contraindicate manipulative therapy, involvement in litigation or worker's compensation, and physiotherapy or chiropractic treatment for headache in the previous 12 months. Those who fulfilled the symptomatic criteria underwent a physical examination of the cervical spine, including manual palpation of the upper cervical joints relevant to the inclusion criteria. A preparatory intertherapist reliability study indicated excellent agreement between pairs of assessors in manual joint examination for subject eligibility.

The 200 subjects were then randomized into 4 groups: manipulative therapy group, exercise therapy group, combined therapy group, and a control group. Manipulative therapy consisted of both low- as well as high-velocity cervical mobilization techniques as taught by Maitland. The therapeutic exercise intervention consisted of low-load endurance exercises to train muscle control of the cervicoscapular region, especially the deep neck flexors, which have an important supporting function for the cervical region. The Stabilizer, an air-filled pressure sensor that monitors the slight flattening of the cervical curve that occurs with contraction of the longus colli, was used for feedback purposes. In addition, the serratus anterior and lower trapezius were trained using inner-range holding exercises of scapular adduction and retraction; postural correction exercises were performed regularly throughout the day in the sitting position. The third intervention was a combination of manipulative therapy and exercise therapy applied on the same day. The control group received no physical therapy intervention. Usual medication was not withheld from any participant regardless of group allocation. Active treatment extended over a period of 6 weeks, including a minimum of 8 and a maximum of 12 treatments. Treatment was delivered by 25 experienced physiotherapists across trial centers. The nature of the interventions precluded any blinding of physiotherapists or participants to assigned treatments. However, blinded outcome assessment was conducted.

The primary outcome measure was a change in headache frequency from baseline to immediately after treatment and at month 12. Changes in headache intensity and duration and in neck pain were secondary outcome measures. Frequency was recorded as the number of headache days in the past week. Average intensity was rated on a VAS and duration was the average number of hours that headaches lasted in the past week. Neck pain and disability were measured using the Northwick Park Neck Pain Questionnaire. The participant-perceived effect of treatment and relief gained were rated on VASs. For analysis, pain medication was converted to a defined daily dose of analgesics using the Anatomic Therapeutic Chemical Code. The tertiary physical assessments included pain with neck movements (VAS). The 3 movements with the highest pain scores were evaluated at follow-up assessment. The pain provoked by manual palpation of the upper cervical joints (VAS) and the 2 joints exhibiting the highest tenderness scores at baseline were reassessed. Performance on the craniocervical muscle test, as well as a photographic measure of the craniocervical angle representing FHP, were also included in the assessment. In addition, several prognostic and evaluative assessments were made for baseline comparisons, including a full headache history, an MPQ, and a psychometric evaluation, the Headache-Specific Locus of Control Scale. Participants also rated the global perceived effect of treatment and the headache relief obtained.

Results demonstrated no differences in headache-related and demographic characteristics between the groups at baseline. The loss to follow-up evaluation was 3.5%. Wilcoxon analyses showed that manipulative therapy (MT), exercise therapy (ExT), and the combination thereof (MT + ExT) all significantly reduced headache frequency, intensity, and the neck pain index immediately after treatment; these differences were still evident at month 12 ($P < 0.05$ for all). The combined therapies were not significantly superior to either therapy alone, but 10% more patients gained relief with the combination. The exception was headache duration, for which combining MT + ExT was effective, but for which the effect of ExT was no greater than the control at the 7-week and 12-month end points. At the 12-month follow-up assessment, MT was not significantly different from the control group in terms of headache duration and neck pain. The results of the 2-way ANOVA provided some evidence that MT + ExT was more beneficial initially in reducing pain produced on joint palpation than either therapy alone, but there was no indication that the additive effect was maintained at month 12.

The authors of this solid multicenter trial conclude that the conservative interventions of manipulative therapy and a specific exercise program were effective in the management of cervicogenic headache with statistically significant improvement in headache frequency and intensity and that the effects are maintained in the long term. Although there was no statistical evidence of an additive effect from combining interventions, 10% more participants receiving the combined therapy obtained good and excellent outcomes. This would support the use of combined MT and therapeutic and home exercises in the management of cervicogenic headache.

Study # 16: Fritz JM, Cleland JA, Speckman M, Brennan GP, Hunter SJ. Physical therapy for acute low back pain: associations with subsequent healthcare costs. *Spine*. 2008;33(16):1800-1805.

A retrospective review of patients with acute LBP, was conducted based on data from the clinical outcomes and financial databases maintained by the Rehabilitation Agency of Intermountain Healthcare (IHC), a private, nonprofit, integrated healthcare delivery system, and SelectHealth, a nonprofit health insurance company that is an integrated subsidiary of IHC. The authors compared the results of an evidence-based active physical therapy regimen, consisting of exercise and patient education in self-management, versus a passive treatment approach such as heat/cold methods, electrical stimulation, ultrasound, etc. The authors assessed short-term clinical outcomes and subsequent healthcare utilization and charges accumulated over a 1-year period after the completion of physical therapy. Adherence to active care was based upon billing records. Patients included in this study presented with acute LBP (<90 days), were between 18 and 60 years of age, had at least 3 physical therapy visits, scored >10% on the Modified Oswestry Disability Questionnaire (OSW), and had no history of back surgery. Disability (Oswestry) and pain (numeric pain rating scale) were assessed at the outset and completion of physical therapy.

Four hundred and seventy-one patients were included in this retrospective analysis with 28% receiving active care. Results indicated that adherence to an evidence-based, active care physical therapy regimen was associated with better clinical outcomes and decreased subsequent use prescription medication, MRI, and injections when compared to a passive treatment approach. For example, the active care group had fewer physical therapy visits (mean difference of 1.3 visits, $P<0.05$) lower charges (nontransformed mean difference of \$167, $P<0.05$), greater improvement in pain (mean difference 12.3%, 95% confidence interval), and disability (mean difference 17.6%, 95% confidence interval). In the year following discharge, the active care group demonstrated a lower likelihood of receiving prescription medication (46.2% versus 57.2%, $P<.05$), MRI (8.3% versus 15.9%, $P<.05$) or epidural injections (5.3% versus 12.1%, $P<.05$) as compared to the LBP patients receiving passive care.

The authors concluded that adherence to the evidence-based recommendation for an active approach to physical therapy care for patients with acute LBP (ie, exercise and patient education in self-management), "May have important implications for improving outcomes and reducing subsequent healthcare costs for individuals with LBP."

Study # 17: Wright EF, Domenech MA, Fischer JR. Usefulness of posture training for patients with temporomandibular disorders. *JADA*. 2000;131:202-210.

Sixty patients with a TMD, which was primarily of masticatory muscle origin, who had moderate to severe pain for at least 6 months were included in this study. Subjects were recruited from the TMD specialty clinic, Lakeland Air Force Base, Texas. The study was conducted over a period of 14 months. The subjects were then randomized into 2 groups: the experimental group receiving posture training and TMD self-management instructions while the control group received only TMD self-management instructions. The dependent measures used consisted of the modified symptom severity index (SSI) to assess the masticatory and neck symptoms, maximum pain-free interincisal opening (millimeters), pain thresholds measured with a pressure algometer at a pressure rate of approximately 0.5 kilograms per square centimeter per second, and perceived TMD and neck symptoms. The 1.8 centimeter-diameter tip was placed over the right and left mid area of the masseter muscles and midcervical area of the trapezius muscle to measure the point at which the patient first perceived pain. The examiner was blinded to the assigned groups and the patients in the treatment group were referred to a physical therapist, who was also blinded to the previously collected data.

Posture training in the experimental group consisted of a home program, taught by a physical therapist, and included the following exercises: chin tucks, chest stretch, wall stretch, on-your-back chest stretch, and face-down arm lifts. To compare changes between the 2 groups, student t-tests were used for all continuous variables and X^2 analyses for categorical variables.

Student's paired t-tests were used to test for changes in posture in the experimental group (recorded in centimeters for head translation from the vertical line). Finally, Pearson product-moment correlation analysis was used to test for changes in posture in patients in the treatment group.

The mean reduction in TMD and neck symptoms, as measured by the modified SSI, were 22.8 and 14.5, respectively, for patients in the treatment group, compared with 3.2 and -0.1, respectively (both $P<.05$), for patients in the control group (scores range from 1 to 100, with 100 being the worst symptoms.) The mean maximum pain-free opening increased by 5.3 millimeters for patients in the treatment group, which was significantly greater than the 1.2 mm improvement for patients in the control group. Within the treatment group, the authors found significant correlation between improvement in TMD symptoms and neck symptoms (P<.005). They also found significant correlations between improvements in TMD symptoms and the pretreatment difference in head and shoulder posture measurements (the greater the pretreatment difference, the greater the symptom improvement) (P<.05). This suggests that TMD patients, who hold their heads farther forward relative to the shoulders, have a higher probability of achieving TMD symptom improvement from posture training. In this regard, the physical therapist in this study found that most patients need some modification of their exercise technique at their first follow-up appointment (if done incorrectly, these exercises may cause the patient's TMD or neck symptoms to worsen). When asked how they thought posture training improved their TMD, 16 (53%) of the patients said that the exercises relaxed their neck muscles and thereby caused the masticatory muscles to relax as well. Twenty-seven (90%) of the 30 patients in the treatment group thought that posture training improved their posture.

In conclusion, posture training and self-management instructions are significantly more effective than TMD self-management instructions alone for patients with TMD who have a primary masticatory muscle disorder. On average, patients who received posture training in addition to self-management instructions reported a 42% and 38% reduction in their TMD and neck symptoms, respectively, as well as experiencing significant improvement in mouth opening. More specifically, patients with FHP who received posture training had a high probability of experiencing improvement in TMD symptoms.

This study illustrates one of the bedrock principles covered in this book, namely that neuromusculoskeletal pain must be directed to the source of the somatic impairment. Otherwise we are merely managing symptoms and not getting to the root of the problem.

Study #18: Whitman JM, Wainner RS, Garber MB. A comparison between two physical therapy treatment programs for patients with lumbar spinal stenosis: a randomized clinical trial. *Spine*. 2006;31(22):2541-2549.

Fifty-eight patients with lumbar spinal stenosis were randomized to one of two 6-week physical therapy programs (each patient was seen twice weekly for a total of 12 sessions). Inclusion criteria included pain in the lumbopelvic region and lower extremities, patient greater than 49 years of age, MRI consistent with lumbar spinal stenosis (evidence of compression of lumbar spinal nerve roots by degenerative lesions of the facet joint, disc, and/or ligamentous flavum), and patient rating of sitting as a better position for symptom relief than standing or walking. One program was designated the Manual Physical Therapy, Exercise and Walking Group (MPTExWG); the other, the Flexion Exercise and Walking Group (FExWG). Those patients in the MPTExWG category received manual physical therapy to the thoracic and lumbar spine, pelvis, and lower extremities (eg, thrust and nonthrust manipulation, manual stretching, and muscle strengthening exercises) by 8 experienced manual physical therapists, 7 of whom were Fellows of the American Academy of Orthopedic Manual Physical Therapists (AAOMPT). In addition, these patients were asked to take a daily walk at a pace and distance that did not irritate lower extremity symptoms, perform a home exercise program consisting of specific exercises, and to participate in a bodyweight-supported (BWS) treadmill ambulation program. Treatment for patients in the FExWG category included lumbar flexion exercises, performance of a progressive treadmill walking program, and subtherapeutic ultrasound. Data regarding perceived recovery, disability, pain, satisfaction, and function were collected at baseline, at the end of the treatment program (6 weeks), and at 1 year. Long-term follow-up questionnaires were used to collect data regarding healthcare utilization, medication usage, pain, and perceived recovery. The patient Global Rating of Change Scale (GRC) served as the primary outcome measure. Secondary outcomes included the Modified Oswestry Disability Index (OSW), the Satisfaction Subscale of the Spinal Stenosis Scale (SSS), a Numerical Pain Rating Scale (NPRS) for thigh/leg pain, and a walking tolerance test.

The results of this study were as follows:

- A greater proportion of patients in MPTExWG reported recovery at 6 weeks compared to patients in FExWG (P=.0015).
- At 1 year, 62% of MPTExWG patients and 41% of FExWG patients still met the threshold for recovery.
- Improvements in disability, satisfaction, and treadmill walking tests favored the MPTExWG patients at all follow-up points.

In conclusion, the authors state the following, "Our results suggest that patients treated with nonsurgical physical therapy programs may achieve clinically important improvements at 6 weeks and 1 year. However, patients receiving a program of manual physical therapy, exercise, and BWS treadmill walking reported greater rates of perceived recovery than those receiving a program of flexion exercises, walking, and subtherapeutic ultrasound."

Study #19: Laslett M, Oberg B, Aprill CN, McDonald B. Centralization as a predictor of provocation discography results in chronic low back pain, and the influence of disability and distress on diagnostic power. *The Spine Journal*. 2005;5:370-380.

This study is a prospective, blinded, concurrent, reference standard-related validity design carried out in a private radiology clinic specializing in the diagnosis of chronic spinal pain. The purpose of this study was to estimate the diagnostic power of the "centralization phenomenon" and the influence of disability and patient distress on diagnostic performance, using provocation discography as a criterion standard for diagnosis in chronic LBP patients.

Consecutive patients with persistent LBP were referred to the study clinic by orthopedists and other medical specialists for interventional radiological diagnostic procedures. Patients were typically disabled and displayed high levels of psychosocial distress. The sample included 107 patients, some having undergone previous lumbar surgery and most having a history of unsuccessful conservative therapies. Patients were excluded from the study if they had a normal MRI, severe degeneration with spondylolisthesis, and other relative contraindications for discography. Patients too frail to tolerate a full physical examination were also excluded.

Measurements included pain, which was assessed on a 100-mm visual analogue scale for current, best, and worst pain; the 23-point Roland-Morris Disability Questionnaire to evaluate disability; the Zung Depression Index, the Modified Somatic Perception Questionnaire (MSPQ), and the Distress Risk Assessment Method (DRAM), all of which were used to assess psychosocial distress.

After the initial interview, a history was taken, and a structured examination was performed by a physical therapist with 30 years of clinical experience as a manipulative therapist, who was a former senior instructor for the McKenzie Institute International. The physical examination included a McKenzie-style assessment in which McKenzie's centralization phenomenon (CP) was recorded if the pain in the furthermost region (ie, buttock, thigh, calf, or foot) from the midline of the lumbar spine was abolished or significantly reduced by specific lumbar spine repeated movements.

Lumbar provocation discography was carried out by a practitioner with 20 years experience or by a resident under his or her guidance. When at least one disc provoked a concordant pain response and an adjacent disc provoked no pain, a diagnosis of discogenic pain was recorded. Failure of the patient to report pain provocation or the report of atypical/discordant pain during injections resulted in the exclusion of discogenic pain at those levels. Local anesthetic was injected into discs that were painful. After discography, axial computed tomographic sections were obtained through selected discs within 30 minutes to evaluate contrast distribution and fissuring patterns.

The physical therapist conducting the clinical examination was unaware of the results of the previous imaging studies, any previous diagnostic injections, the Roland-Morris Questionnaire, Zung Depression Index Questionnaire, and MSPQ. The discographer was blinded to the results of the physiotherapy examination and diagnostic conclusions, but not to the results of the questionnaires.

A full evaluation was achieved in 69 cases, a partial examination in 21 cases, and no examination in 17. The examining physical therapist offered an opinion regarding the CP for 83 patients even though the repeated movements examination was incomplete in some cases. The effect of potential confounding factors on the diagnostic power of centralization was estimated. Multiple logistic regression analysis revealed that higher Roland-Morris Questionnaire, MSPQ, and "worst" pain intensity scores plus a lower Zung questionnaire were associated with positive discography. In a model that included these variables and the CP, the odds (95% confidence intervals) of positive discography were 14.9 (1.57, 141.1), P=.001.

In the present study, prevalence of positive discography was 75% and the likelihood ratio for centralization was 6.9. Pretest odds of 75:25 changed to posttest odds of 95:5, (ie, a 20% increase and diagnostic confidence was 95%). If the expected prevalence of internal disc disruption is used (39%), improvement from pre- to posttest odds is greater and diagnostic confidence increases from 39% to 82%. However, the wide confidence intervals for the likelihood ratios indicate that caution is appropriate. Current data indicate that the CP has high specificity, especially in patients categorized as not being severely disabled. Consequently, when centralization is reported during the McKenzie evaluation (in the absence of severe disability or psychosocial distress), positive provocation discography is highly likely and a diagnosis of discogenic pain is reasonable. In relation to positive provocation discography, the CP during a McKenzie examination of repeated movements has a specificity of 89%, and among patients without severe disability or distress it is 100%. However, in the presence of severe disability, specificity is reduced to 80%.

In summary, the presence of centralization in nondistressed and not severely disabled chronic LBP patients suggests that discography may be delayed (because the expected result of discography is already known). This is based on the statistical rule that a high specificity (eg, 95% or above) allows the clinician to "rule in" the disorder when the test is positive. Furthermore, the availability of a McKenzie treatment program may improve the patient's symptoms to the point where provocation discography is no longer necessary.

Study #20: Aure OF, Nilsen JH, Vasseljen O. Manual therapy and exercise therapy in patients with chronic low back pain: a randomized controlled trial with 1-year follow-up. *Spine*. 2003;28(6):525-532.

This multicenter, RCT with 1-year follow-up compared the effect of manual therapy to exercise therapy in sick-listed patients, obtained through the local Social Security Office, with chronic LBP (>8 weeks). Inclusion criteria were men and women age 20 to 60 years that had been sick-listed between 8 weeks and 6 months due to LBP with or without leg pain. Exclusion criteria consisted of unemployment or early retirement because of LBP; prolapse with neurologic signs and symptoms requiring surgery; pregnancy; spondylolisthesis; spondylolysis, degenerative olisthesis, fractures; suspicion of malignancy; osteoporosis; previous back surgery; known rheumatic, neurologic, or mental disease; or absence of pain aggravation on active, functional movement tests (ie, indicating nonorganic symptoms). Of the 49 patients in the study, 27 were randomized to manual therapy (MT) and 22 to exercise therapy (ET).

Those in the MT group received spinal and sacroiliac joint manipulation, specific mobilization, and stretching techniques described by Evjenth, Hamberg, and Kaltenborn. These patients also received self-mobilization/stretching based upon clinical findings. Each manual therapy session lasted 45 minutes. Those patients in the ET group were assigned to general exercise therapy involving 45 minutes of training. Following a 10-minute warm-up session on an exercise bicycle, the patients were given general training methods suitable for LBP patients (eg, strengthening, stretching, mobilizing, coordination and stabilizing exercises for the abdominal, back, pelvic, and lower limb muscles based on clinical findings). The training took place with or without training equipment in the physiotherapy clinic. Patients were observed and guided closely by the therapist during each session. All patients in the study were treated on 16 different occasions over a period of 2 months.

Outcome measures included:

- Spinal range of motion as measured by the modified Schober test.
- Pain intensity, due to LBP, as recorded on a 100-mm Visual Analogue Scale. The final outcome measure used was the mean of 3 recordings: pain at the moment, worst pain in the last 14 days, and the mean pain during the last 14 days.
- Functional disability using the Oswestry Low Back Pain Disability Questionnaire.
- General health as measured by the Dartmouth COOP Function Charts.
- Return to work.

All outcome measures, except for spinal range of motion, were scored on questionnaires administered 5 times during the study (ie, within 3 days after the last treatment session, then at 4 weeks, 6 months, and 12 months after the end of treatment). Spinal range of motion was assessed by a blinded examiner and carried out at pre- and posttest only. All pretests were performed after randomization, except for spinal range of motion, which was performed before randomization. Repeated measures ANOVA was used to test differences between groups (MT versus ET) and within groups for pain, Dartmouth, and Oswestry variables. The Tuckey-Kramer test was then used for pair-wise comparisons of means. Variables showing significant differences were retained for further post hoc analyses, and the student t-test was used on the above-mentioned outcome measures to test differences in improvement between the 2 treatment groups at all posttreatment test sessions. A paired t-test was used to investigate changes within groups; the results from posttreatment and follow-up test sessions were compared to the pretreatment results. Wilcoxon signed-ranks test (within groups) and Mann-Whitney U test (between groups) were used for the modified Schober test. The Fisher exact test was used to test group differences in sick-leave status and risk ratio used to estimate the risk of being sick-listed in the MT versus the ET group at all follow-up sessions. The significance level was set to $P < 0.05$.

Results can be summarized as follows:

- Significant improvement in pain, general health, and functional disability were observed in both groups from before to after treatment ($P<.01$) and this improvement was maintained throughout the 1-year follow-up.
- Significantly larger improvements ($P<.05$) were found in the MT group compared to the ET group at all posttreatment test sessions. The mean reduction of pain from pre- to posttest in the MT group was twice that of the ET group, correspondingly for general health, and functional disability. The effects gained from the treatments were stable in the 1-year post-treatment period in both groups.
- Spinal range of motion was measured only at the pre- and posttreatment sessions. Significant improvements were found both within and between groups, with the MT group showing significantly larger improvement. The mean improvement in the MT group was 31 mm (95% CI: 26-36) and in the ET group 9 mm (95% CI: 6-12; $P<.01$).
- At pretest, all patients were fully sick-listed. However, at posttest, 73% in the ET versus 33% in the MT group were partly or fully sick-listed ($P<.01$). The respective numbers at 4 weeks follow-up were 57% versus 30% ($P=.08$), at 6 months 62% versus 11% ($P<.01$), and at 12 months 59% versus 19% ($P<.01$).

The authors conclude by stating, "Improvements were found in both intervention groups, but manual therapy showed significantly greater improvement than exercise therapy in patients with chronic LBP. The effects were reflected on all outcome measures, both on short and long-term follow-up."

Study #21: Harman K, Hubley-Kozey CL, Butler H. Effectiveness of an exercise program to improve FHP in normal adults: a randomized, controlled 10-week trial. *J Manual Manipulative Ther.* 2005;13(3):163-176.

The purpose of this randomized controlled study was to determine if a 10-week, targeted, and progressive home exercise program could improve FHP in asymptomatic adults. The impact of FHP-targeted exercises on cervical range of motion was also assessed.

Potential participants were screened, prior to inclusion, by measuring the horizontal distance between the tragus and posterior angle of the acromion in standing using a customized graduated setsquare. If the tragus was >5 cm anterior, then a participant was referred to the study. Participants also had to be pain-free, healthy, between 20 and 50 years old, and had not sought medical/healthcare for neck, shoulder, or LBP over the past year. The outcome measures included postural evaluation of FHP using the Biotonix Postural Assessment System, pre-and poststudy neck flexion ROM using the CROM instrument, and a physical activity questionnaire.

Twenty-three exercise and 17 control subjects participated in this study. The exercise program consisted of 2 strengthening (deep cervical flexors and shoulder retractors) and 2 stretching (cervical extensors and pectoral muscles) exercises. The exercises involved chin tucks in supine lying with the head in contact with the floor, chin drop in sitting, shoulder retraction first in standing using a Theraband and then progressed to shoulder retraction in prone using weights, and unilateral and bilateral pectoralis stretches. Participants were instructed to complete 3 sets of 12 repetitions of the strengthening exercises and 3 stretching exercises held for 30 seconds each. This program was to be repeated 4 times per week. Each exercise subject returned for a consultation every 2 weeks to be checked for exercise technique and progression, if appropriate. Progress to the next exercise level was indicated if the participant could complete 12 repetitions, 3 times easily with correct form. The same individual performed all instruction and consultation. The attendance scores for the 5 scheduled consultation visits were counted. The compliance rate was calculated from the exercise logs; progression was determined by the level of difficulty achieved for each exercise at the end of 10 weeks. Control subjects did not participate in the exercise program but were asked to carry on with their regular activities and were telephoned at the end of each week to monitor their activity. All participants (exercise and control) in the study were asked to complete an activity log each day. Participants completed a physical activity questionnaire prior to and at the end of the study that included questions about the number of times they had exercised in the past week and the intensity of the exercise. In addition, a 1-page questionnaire was given to all participants upon completion of the study asking questions with respect to whether they felt their posture improved and what they liked and disliked about the study.

T-tests were used to determine if there were any statistically significant differences between the exercise and control groups related to age, body mass, or height. A 3-factor mixed-model ANOVA was used to test between trial, between group, between pretest/posttest and all 2- and 3-way interactions for neck flexion ROM. A 2-factor mixed-model ANOVA was used to test between group and between pretest/posttest, and a group by time interaction for the posture measurements. Scores on the physical activity questionnaires were compared between groups for pre- and poststudy differences, using a 2-factor ANOVA. Appropriate post hoc tests were conducted on significant main effects or interactions using the Bonferroni method. Statistical analyses were performed using Minitab version 13.

Two subjects in the exercise group did not attend the consultation sessions and did not comply with the exercise program; consequently, the final number in the exercise group was 21. The lack of significant differences in age, mass, height, and the percentage of males and females in the control and exercise groups demonstrated an effective randomization procedure and equivalence of groups at the pretest collection phase. Physical activity scores demonstrated no changes between groups on pretest and posttest measurements; therefore, changes in activity patterns were not a covariate in the analyses. Regarding neck flexion ROM, significant differences were found between the pretest control and posttest exercise (P=.005), pretest and posttest for the exercise group (P<.0001), and the exercise and control on the posttest (P=.03). Neck flexion ROM increased by 3.7 degrees for the exercise group. Regarding the quantification of FHP, 6 postural measurements, employing the Biotonix Postural Analysis System (reliability and validity have been demonstrated), were utilized. They consisted of a set of angular and distance measurements to assess the change in FHP and included the following:

- Neck angle (the angle between horizontal and the tragus-to-C7 line, measured in degrees)
- Shoulder to pelvis angle (the angle between vertical and the line joining acromion to mid-point between ASIS and PSIS, measured in degrees)
- Head angle (the angle between horizontal and the glabella-to-tragus line, measured in degrees)
- Head distance (horizontal distance from tragus to vertical plumb line from base of the 5th metatarsal, measured in centimeters)
- Shoulder distance (horizontal distance from acromion to vertical plumb from base of the 5th metatarsal, measured in centimeters)
- HScal (horizontal distance between acromion and tragus, measured in centimeters)

Results showed a statistically significant interaction for shoulder-to-pelvis angle (P<.05) between pre- and posttests and also between the exercise and control groups at posttest. This indicates a change in standing trunk alignment consistent with "straightening up," or pulling the shoulders back. There were statistically significant (P<.05) differences between pretest and posttest measurements for both groups (but no between-group differences) for neck angle, shoulder distance, head distance, and HScal distance.

The authors summarize their findings as follows, "The results demonstrate that a short, home-based targeted exercise program can improve postural alignment related to FHP. These results provide a foundation for further development of postural improvement programs that include an exercise component."

Study #22: Powers CM, Beneck GJ, Kulig K, Landel RF, Fredericson M. Effects of a single session of posterior-to-anterior spinal mobilization and press-up exercise on pain response and lumbar spine extension in people with nonspecific low back pain. *Phys Ther.* 2008;88(4):485-493.

The purpose of this study was to examine the immediate effects of PA mobilization and a press-up exercise in people with nonspecific LBP. The outcome measures were pain and total lumbar spine extension. Thirty patients (19 women and 11 men) between 18 and 45 years with a diagnosis of nonspecific LBP were recruited for this study. Inclusion criteria consisted of recent onset of LBP (<3 months), localized LBP at or above the waist, decreased lumbar extension (assessed qualitatively while standing), and increased local pain with lumbar extension in standing. The primary exclusion criteria were spinal malignancy, cardiovascular disease, evidence of cord compression, aortic aneurysm, hiatal hernia, prior low back surgery, gross spinal deformity, spondylolisthesis, known rheumatic joint disease, and implanted devices that could be a contraindication to MRI. In addition, patients with any indication of lumbar disc herniation (eg, radicular symptoms, muscle weakness, sensory loss, reflex changes, MRI findings) were also excluded from the study.

Prior to the pretreatment MRI assessment, each subject's initial pain level was assessed. Subjects were asked to stand, bend backward with their hands on their hips, and rate their LBP with a visual analogue scale. In the pretreatment MRI assessment, sagittal plane images of the lumbar spine were obtained with subjects at rest and at the end of the press-up exercise (ie, end-range lumbar extension). Subjects were placed on a sliding table in the prone position with a pillow under the abdomen (the sliding table was situated such that the spine and torso were within the opening of the MRI system with the surface coil secured to the lumbar region with adhesive straps). Following subject positioning within the MRI, a series of sagittal-plane "localizers" were obtained to ensure that the image plane captured the vertebral bodies of all lumbar vertebrae. Prior to analysis, all images were transferred from the MRI system console to a Macintosh G3 computer (Apple, Cupertino, CA). Only the images containing the vertebral segments at rest and at end-range of the press-up maneuver were analyzed. Sagittal-plane intervertebral angles of the lumbar spine, as measured by the angle formed by lines defining the end-plates of adjacent vertebrae, were measured with National Institutes of Health Image software. Segmental extension was defined as the difference between the intervertebral angles on the resting and end-range images.

Once the pretreatment pain and MRI evaluations were completed, each subject was randomly assigned to either the passive segmental mobilization (PA mobilization) group or the exercise (press-up) group. Both interventions were administered by a physical therapist with 18 years of manual therapy experience and certification as an Orthopaedic Clinical Specialist by the APTA. The physical therapist was unaware of the findings of the baseline MRI and pain ratings. Subjects in the PA mobilization group (n=15) were treated with methods described by Maitland et al. The premise behind this approach is that treatment should mobilize the most restricted segment(s); if this is properly executed, the entire lumbar spine should improve as demonstrated by increased mobility. Initially, the PA mobilization consisted of graded oscillations applied to the most painful lumbar segment. Three bouts of 40-second oscillations were applied to this segment at a rate of approximately 1 to 2 Hz and at the highest amplitude tolerated without the reproduction of symptoms. Following mobilization of the most painful segment, 2 bouts of 40-second oscillations (up to grade IV but short of symptom reproduction) were administered to each of the remaining lumbar vertebral levels. The total time for the PA mobilization intervention was approximately 10 minutes. Subjects assigned to the press-up group (n=15) were treated with the methods described by McKenzie and May. The subject used the arms to press the top half of the body upward into spinal extension, while the pelvis was allowed to sag with gravity and remain on the treatment table. The subject was instructed to move from the prone position to maximum pain-free lumbar extension over the course of 5 seconds. The end-range position was held for 5 seconds before returning to the starting position. A total of 10 repetitions were performed. During each repetition, the subject was encouraged to move slightly higher, within the limits of discomfort. If, at the end of completion of 10 repetitions the subject's level of pain was the same or less, a second and third series of press-ups were performed. All subjects were able to perform 30 repetitions, which required approximately 10 minutes.

Immediately following the intervention, posttreatment pain and MRI assessments were repeated with the same procedures described above. The investigator coordinating the MRI assessment was unaware of each subject's treatment group assignment. Because it was not possible to replicate the exact resting position of the lumbar spine for the posttreatment MRI assessment, the pretreatment resting position was used to calculate motion during both pre- and posttreatment assessments. Therefore, the change in segmental extension following the intervention was defined as: (posttreatment end-range vertebral angle – pretreatment resting vertebral angle) – (pretreatment end-range vertebral angle – pretreatment resting vertebral angle). Consequently, a positive value indicated an increase in extension, whereas a negative value indicated a decrease in extension for a specific spinal segment. Total lumbar extension was quantified by summing the intervertebral motion at each of the 5 functional units of the lumbar spine. Intratester reliability of the MRI measurements was established on 5 healthy volunteers by performing 2 MRI assessments 1 week apart (intraclass coefficients were found to be excellent, ranging from .95 to .99 for all subjects with a standard error of measurement ranging from 0.40 to 0.66 degrees). The ANOVA results for average pain scores revealed a significant main effect for time ($F = 23.274$; $df = 1.14$; $P<.001$). However, no significant group effect or group x time interaction was observed. On average, subjects in the PA mobilization group reported a posttreatment pain score of 2.4 ± 1.8, which did not differ significantly from the posttreatment pain score of 2.8 ±1.5 reported by subjects in the press-up group. The ANOVA results for average total lumbar extension revealed a significant main effect for time ($F=11.764$; df=1.14; $P=.004$). When averaged across both treatment groups, average lumbar extension was greater after intervention than before (24.3 degrees ± 6.1 versus 21.2 degrees ± 4.7). However, no significant group effect or group x time interaction was observed. On average, subjects in the PA mobilization group demonstrated 23.8 degrees ± 6.5 of lumbar extension, which did not differ significantly from posttreatment lumbar extension of 24.9 degrees ± 6.0 demonstrated by the press-up group. In order to explore the relationship between changes in pain and lumbar spine extension, a post hoc correlation analysis was performed. When subjects in both groups were combined, a statistically significant relationship was found ($r = -.37$, $P=.04$). The negative correlation indicated that greater decreases in pain were associated with greater increases in lumbar extension. Although this finding supports the link between pain and joint motion, the relationship was weak. Furthermore, cause-and-effect relationships cannot be inferred by this analysis.

In conclusion, the immediate effects of PA mobilization and a prone press-up exercise were examined in patients with nonspecific LBP. Following the intervention, subjects in both groups reported significantly less pain with standing extension. In addition, both PA mobilization and press-up exercises resulted in a significant increase in lumbar extension. However, there were no significant differences in pain and lumbar extension between the 2 interventions studied.

Therefore, the findings of this study support the use of both PA mobilization and prone press-ups for improving lumbar extension and relieving symptoms in patients with nonspecific LBP, but there is no basis for selecting one intervention over another based on these data.

This concludes the author's attempt at providing evidence to suggest that the practice of manual therapy is not built upon the "shakiest of foundations." There is, in fact, an evolving science that demonstrates a fair degree of support for the types of interventions discussed in this text. Having said that, the author is well aware of the lack of acceptable science in the world of manipulative therapy. Many studies are not properly randomized; have an insufficient number of subjects; are not prospective, placebo-controlled, properly "blinded," nor statistically analyzed; have often been published in journals that are not peer-reviewed. There is no doubt that we need to do better, and Dr. Rothstein and others, including the Philadelphia Panel Members,[10] were correct in challenging the status quo of manual therapy. In addition, there are well-designed research studies that fail to document treatment efficacy[11-14] and interrater reliability[15-17] or present other challenges to the use of manual physical therapy such as the inability to accurately evaluate SI joint movement impairment.[18]

The point of this literature review, however, is to ensure that the "baby is not thrown out with the bath water." The above studies demonstrate that a body of outcomes data is available to support the use of spinal manual therapy and therapeutic exercise. Are more RCTs needed? Absolutely and sooner rather than later! Because of the clinical scope of this text, evidence from the basic science literature (ie, histology, anatomy, physiology, movement science, motor learning, articular neurology, pain science, etc) was not presented. Many good studies, especially in the area of connective tissue pathophysiology, have demonstrated the beneficial effects of mobilization/manipulation.[19-21] In addition to individual studies on the efficacy of manual therapy and exercise, there are systematic reviews available that have been used to create meta-analyses of related literature. In many cases, these analyses have provided additional support for the use of manual therapy and exercise. For example, the Duke University Evidence-Based Practice Center published *Evidence Report: Behavioral and Physical Treatments for Tension-Type and Cervicogenic Headache*,[22] which concluded, "Manipulation is effective in patients with cervicogenic headache, but its efficacy in patients with tension-type headache is unproven." In his systemic review of the literature, DiFabio[23] concludes, "Overall, there was clear evidence to justify the use of manual therapy, particularly manipulation, in the treatment of patients who have back pain." In their large scale, multisite studies on TMD diagnosis and treatment, Gaudet and Brown found that, "Treated patients (TMD) report statistically and clinically significant levels of improvement... but the weight of the evidence indicates that untreated TMD patients, as a group, do not improve spontaneously over time." Of significance to manual physical therapists is the fact that almost half of the patients (43.7% in the first study and 46.9% in the second study) received "physical modalities" that, upon further inquiry, included the use of manual therapy, and therapeutic and home exercises.[24,25] Regarding the conservative management of mechanical neck disorders, the Cervical Overview Group (COG) has completed 11 systematic reviews to date. Their research indicates that mobilization/manipulation is most effective when used as an adjunct to exercise. The evidence also shows that unsupervised home programs are not beneficial for individuals with chronic mechanical neck disorder (ie, "nonspecific" neck pain) and neck disorder with radicular signs and symptoms.[26]

Conclusion

An attempt has been made to present a defense of the practice of manual therapy and therapeutic exercise based upon the available evidence. As in a courtroom, where an attorney makes his or her best case in support of a given position, this chapter presents the case for the efficacy of manual therapy and therapeutic exercise. To that end, the author has selected 22 studies that are a representative cross-section of clinical approaches within the practice of modern manual physical therapy in hopes of making his case. The objective of this chapter was not only to respond to the critics but also to balance the art of practice with a comparable degree of science so that at the end of the day, physical therapists will rise to the professional level to which we all aspire.

References

1. Sackett DL, Rosenberg WM, Gray JA, et al. Evidence based medicine: what it is and what it isn't. *BMJ*. 1996;312:71-72.
2. Sackett DL, Straus SE, Richardson WS, et al. *Evidence-Based Medicine: How to Practice and Teach EBM*. 2nd ed. New York, NY: Churchill Livingstone; 2000.
3. Bogduk N, McGuirk B. *Management of Acute and Chronic Neck Pain: An Evidence-Based Approach*. Edinburgh: Elsevier; 2006.
4. Craik RL, Maher C. Still "Not Satisfied"...Yet (editorial). *Phys Ther*. 2008;88(4):423-425.
5. Gatchel RJ, Howard KJ, Kishino ND. The biopsychosocial approach to pain management. *Practical Pain Management*. 2008;8(4):28-39.
6. Portney LG, Watkins MP. *Foundations of Clinical Research: Applications to Practice*. 3rd ed. New York, NY: Pearson Prentice Hall; 2009.
7. Donelson RG, Silva G, Murphy K. The centralization phenomenon: its usefulness in evaluating and treating sciatica. *Spine*. 1990;15:211-213.
8. Stoddard A. *Manual of Osteopathic Techniques*. London: The Anchor Press; 1977.
9. Maitland GD. *Vertebral Manipulation*. 5th ed. London: Butterworth-Heinemann; 1986.
10. Albright J, Allman R, Bonfiglio RP, et al. Evidence-based guidelines: application to clinical practice, Philadelphia Panel. *Phys Ther*. 2001;81(10):1629-1730.
11. Assendelft WJJ, Morton SC, Yu EI, Suttorp MJ, Shekelle PG. Spinal manipulative therapy for low back pain: a meta analysis of effectiveness relative to other therapies. *Annal Intern Med*. 2003;138:871-881.
12. Gross AR, Hoving JH, Haines TA, et al. A Cochrane Review for manipulation and mobilization for mechanical neck disorders. *Spine*. 2004;29:1541-1548.
13. Avery S, O'Driscoll M-L. Randomized controlled trials on the efficacy of spinal manipulation therapy in the treatment of low back pain. *Phys Ther Rev*. 2004;9:146-152.
14. Mohseni-Bandpei MA, Stephenson R, Richardson B. Spinal manipulation in the treatment of low back pain: a review of the literature with particular emphasis on randomized controlled trials. *Phys Ther Rev* 1998;3:185-194.
15. Potter NA, Rothstein JM. Intertester reliability for selected clinical tests of the sacroiliac joint. *Phys Ther*. 1985;65:1671-1675.
16. Binkley J, Stratford PW, Gill C. Interrater reliability of lumbar accessory motion mobility testing. *Phys Ther*. 1995;75(9):786-792.
17. Riddle DL, Freburger JK. Evaluation of the presence of sacroiliac joint region dysfunction using a combination of tests: a multicenter intertester reliability study. *Phys Ther*. 2002;82(8):772-781.
18. Goode A, Hegedus EJ, Sizer P, Brismee J-M, Linberg A, Cook CE. Three-dimensional movements of the sacroiliac joint: a systematic review of the literature and assessment of clinical utility. *J Manual Manipulative Ther*. 2008;16(1):25-38.
19. Threlkeld AJ. The effects of manual therapy on connective tissue. *Phys Ther*. 1992;72(12):893-900.
20. Cantu RI, Grodin AJ. *Myofascial Manipulation: Theory and Clinical Application*. 2nd ed. Gaithersburg, MD: Aspen Publishers; 2001.
21. Andrade C-A, Clifford P. *Outcome-Based Massage: From Evidence to Practice*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008.
22. McCrory DC, Penzien DB, Hasselblad V, Gray RN. *Evidence Report: Behavioral and Physical Treatments for Tension-Type and Cervicogenic Headache*. Durham, NC: Duke University Evidence-Based Practice Center; 2001.
23. DiFabio RP. Efficacy of manual therapy. *Phys Ther*. 1992;72(12):
24. Gaudet EL, Brown DT. Temporomandibular disorder treatment outcomes: first report of a large-scale prospective clinical study. *J Craniomandibular Pract*. 2000;18(1):9-20.
25. Brown DT, Gaudet EL. Temporomandibular disorder treatment outcomes: secondary report of a large-scale prospective clinical study. *J Craniomandib Pract*. 2002;20(4):244-253.
26. Gross AR, Haines T, Goldsmith CH, Santaguida L, McLaughlin LM, Peloso P, Burnie S, Hoving J. Knowledge to action: a challenge for neck pain treatment. *J Orthop Sports Phys Ther*. 2009;39(5):351-363.

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Case Studies

In order to apply the concepts and principles discussed in this text, this final chapter consists of a collection of case studies, including questions and answers. These cases are representative of those patients typically seen in an outpatient physical therapy clinic and will hopefully facilitate the transition from the "classroom to the clinic."

Case 1

A 25-year-old female housewife presents with a chief complaint of painful clicking of the right TMJ. The patient reports that she is under stress with 2 small children at home. In addition to her jaw symptoms, she also has recurring headaches, intermittent neck pain and stiffness, and occasional dizziness. The patient has no recollection of head, neck, or jaw trauma. The patient indicates that, according to her husband, she grinds her teeth at night. The patient's family physician is aware of her symptoms and has recommended psychological counseling.

1. Before physical therapy is initiated, to whom should this patient be directed?
   A. A rheumatologist to rule out osteoarthritis
   B. A neurologist to diagnose the cause of her headaches and dizziness
   C. A chronic pain center
   D. A physiatrist to determine which physical therapy modalities are indicated

2. Which of the following dental interventions may prove beneficial for this patient?
   A. Root canal
   B. Orthognathic surgery
   C. The Nociceptive Trigeminal Inhibition Tension Suppression System (NTI-tss) appliance
   D. Dental implantation

3. Facilitation of which cranial nerve is implicated in light of the patient's head, neck, and TMJ/facial complaints?
   A. Trigeminal
   B. Spinal accessory
   C. Facial
   D. Vestibulocochlear

4. The patient's TMJ clicking suggests which diagnosis?
   A. Capsular hypomobility
   B. Capsular hypermobility
   C. Internal derangement
   D. Myofascial pain and dysfunction

5. Which of the following is true of the upper cervical spine?
   A. It can be the source of this patient's headaches
   B. It can be the source of this patient's dizziness
   C. It can contribute to this patient's TMJ impairment through trigeminal nerve facilitation
   D. All of the above

Case 2

A 30-year-old male carpenter presents with a chief complaint of intractable left-sided low back and buttock pain

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secondary to a lifting injury on the job. The pain is acute (ie, less than 2 weeks) and the patient is unable to work. The Oswestry Disability Index, Version 2.0, revealed marked functional limitation in the following categories: lifting, sitting, sleeping, and social life. The patient was referred to your outpatient physical therapy clinic by his internist.

1. Your examination reveals normal neurologic function, but there is a right lateral shift with limited lumbar extension. What does your initial intervention involve?
   A. Correction of the lateral shift
   B. Flexion exercises
   C. Extension exercises
   D. Myofascial release/stretching of the right quadratus lumborum

2. If the patient's symptoms are not worsened by movement nor relieved by rest, what would be the correct course of action?
   A. Use pain-relieving modalities to manage symptoms
   B. Discuss proper body mechanics
   C. Call the patient's physician regarding the possibility of viscerogenic pain referral
   D. Instruct the patient in positional distraction

3. Providing that the patient responded well to the McKenzie approach (ie, lateral shift correction and extension exercises), which of the following is the least likely patient classification?
   A. Derangement syndrome
   B. Dysfunction syndrome
   C. Postural syndrome
   D. All of the above

4. What is the muscle most likely to be tender to palpation?
   A. Right piriformis
   B. Left hamstring
   C. Left TFL
   D. Right iliopsoas

5. Assuming the presence of a lumbar derangement, what is the most likely category?
   A. Derangement 1
   B. Derangement 2
   C. Derangement 3
   D. Derangement 4

Case 3

A 60-year-old female physician presents with a long history of intermittent right-sided headaches. There is no family history of migraine. The patient reports sustaining a neck injury when falling off a horse at age 40. Stress is under control, but the patient spends several hours per week at her computer. The patient's headaches are always right-sided, of moderate to severe intensity, and nonthrobbing in nature. They are not associated with nausea, vomiting, or photophobia/phonophobia. Besides having a right hip replacement at age 55, the patient is otherwise healthy and physically fit.

1. Examination of postural alignment reveals marked forward head position. What is this acquired postural deformity associated with?
   A. Occipital flexion/lower cervical extension
   B. Occipital extension/lower cervical flexion
   C. Occipital flexion/lower cervical flexion
   D. Occipital extension/lower cervical extension

2. Examination also reveals moderate restriction of active cervical rotation to the right. What are the history and physical examination consistent with?
   A. Migraine without aura
   B. Headache secondary to a brain tumor
   C. Cervicogenic headache
   D. Subarachnoid hemorrhage

3. Where would the most symptomatic apophyseal joint in this patient's neck be expected to be found?
   A. C2,3 on the left
   B. C2,3 on the right
   C. C5,6 on the left
   D. C5,6 on the right

4. Entrapment of which of the following nerves gives rise to unilateral headache on the affected side?
   A. The long thoracic nerve
   B. The dorsal scapular nerve
   C. The suprascapular nerve
   D. The greater occipital nerve

5. Which column of gray matter in the CNS mediates headache of cervical origin?
   A. The trigeminocervical nucleus
   B. The locus ceruleus
   C. The nucleus dorsalis
   D. The caudate nucleus

Case 4

A 14-year-old female soccer player presents with chronic pain (ie, over 3 months duration) in the cervical and lumbar spine. She, like her parents, is round shouldered. She also reports an overall sense of restricted mobility, without pain, in the midback area. She was seen by an orthopedic surgeon who diagnosed thoracic spine rotoscoliosis with an accentuated kyphosis. The deformity does not warrant surgery or a spinal orthosis; therefore, physical therapy was recommended. The Present Pain Intensity (PPI) of the standard long-form McGill Pain Questionnaire revealed the pain to be distressing (ie, 3 out of a possible 5).

1. Upon examination, a right thoracic convexity is observed from T5 through T11 with an associated rib hump located where?
   A. Right side
   B. Left side
   C. Right above T8, left below T8
   D. None of the above

2. Which muscle in the scapulothoracic region tends toward inhibition, hypotonicity, and weakness?
   A. The upper trapezius
   B. The levator scapula
   C. The pectoralis minor
   D. The lower trapezius

3. The middle trapezius muscle is inhibited by restricted midthoracic:
   A. Flexion
   B. Extension
   C. Lateral flexion
   D. A and C

4. Tightness of which muscle restricts scapular upward rotation and consequently contributes to impingement of the glenohumeral joint?
   A. The levator scapula
   B. The upper trapezius
   C. The pectoralis minor
   D. A and C

5. This patient demonstrates the common pattern of:
   A. Thoracic hypomobility/cervical hypomobility
   B. Thoracic hypermobility/cervical hypermobility
   C. Thoracic hypomobility/cervical hypermobility
   D. Thoracic hypermobility/cervical hypomobility

Case 5

A 38-year-old male accountant sustained an injury to his right low back area while golfing 2 days prior to arriving in your outpatient clinic. The patient gives a history of "missing the golf ball" during an attempted drive on a par 5 with an immediate onset of pain in the right upper buttock region. The patient has had difficulty weight bearing on the right leg. The patient rated his pain as 7 out of 10 on a numeric pain rating scale (NPRS).

1. Examination reveals a (+) standing flexion test on the right, (+) stork at the upper pole of the right SI joint, and a (+) posterior shear test also on the right. From these findings and the patient's history, what pelvic asymmetries are expected with the patient recumbent?
   A. High ASIS/low PSIS on the right versus the left
   B. High ASIS, PSIS, and iliac crest on the right versus the left
   C. Low ASIS/high PSIS on the right versus the left
   D. Low ASIS, PSIS, and iliac crest on the right versus the left

2. Regarding tissue texture abnormality, which muscle will have expected hypertonicity as a result of this injury?
   A. The left hamstring
   B. The right hamstring
   C. The left iliopsoas
   D. The right iliopsoas

3. Muscle energy technique to correct which iliosacral impairment utilizes the neurophysiologic principle of reciprocal inhibition via an isometric contraction of which of the following?
   A. Right gluteus maximus
   B. Right rectus femoris
   C. Right hamstrings
   D. A and C

4. Which of the following is involved in a grade 3 joint mobilization to the right iliac bone?
   A. Large amplitude oscillations at the end of range
   B. Small amplitude oscillations at the end of range
   C. Large amplitude oscillations at the beginning of range
   D. Small amplitude oscillations at the beginning of range

5. The proper sequence of intervention for this patient's condition includes addressing which of the following in which order?
   A. Reactivity, muscle strength, joint mobility, soft tissue extensibility, and alignment
   B. Reactivity, soft tissue extensibility, joint mobility, alignment, and muscle strength
   C. Alignment, muscle strength, joint mobility, soft tissue extensibility, and reactivity
   D. Soft tissue extensibility, joint mobility, muscle strength, alignment, and reactivity

Case 6

A 45-year-old male English teacher presents with a 6-month history of progressive pain commencing in the right lumbosacral area and spreading into the right lower limb below the knee and into the right foot. The patient's lumbar spine x-rays and MRI were unremarkable. The patient has not responded to prior physical therapy and is interested in a second opinion. The patient has been seen by a family physician, orthopedist, and neurologist and has been given a diagnosis of lumbar radiculopathy. A visual analogue scale revealed pain to be 6 out of 10 during the examination and 9 out of 10 at its worst.

1. Your evaluation includes a detailed history and a physical examination of:
   A. Spinal and pelvic alignment
   B. Active and passive lumbar ROM
   C. Sensation, muscle strength, DTRs, and neurodynamic tests (ie, femoral nerve, proximal sciatic nerve, and its branches)
   D. All of the above

2. The examination reveals the following findings: left lateral lumbar shift, centralization of symptoms with lumbar extension, peripheralization into the right leg with lumbar flexion, (+) straight leg raise on the right at 45 degrees, and enlargement of the right buttock. The above signs are all consistent with a McKenzie derangement 6 except for:
   A. Right buttock enlargement
   B. (+) Straight leg raise on the right
   C. Left lateral lumbar shift
   D. "Centralization" of symptoms with lumbar extension

3. Given the finding of an enlarged right buttock, what should be the next course of action?
   A. Contact the patient's family physician regarding your concern
   B. Proceed with McKenzie management of a lumbar derangement
   C. Apply ice and electrical stimulation to the right buttock
   D. Perform connective tissue techniques and stretching to the right piriformis muscle

4. Therapy is begun on the patient, but at patient rounds your colleagues express concern over the large right buttock. They suggest that you examine the right hip for limitation of hip flexion and to your surprise this motion is considerably limited and causes intense pain. You proceed with a further examination of hip ROM and find that rotations are limited by pain with an "empty end-feel." Now that the Cyriax "sign of the buttock" has emerged, you immediately contact the patient's physician. What will an MRI of the pelvis confirm?
   A. Piriformis spasm on the right
   B. A torn long dorsal SI ligament on the right
   C. A malignant neoplasm of the right iliac bone
   D. Inflammation of the right SI joint

5. In retrospect, you are trying to understand how a neoplasm could present like a McKenzie derangement 6. What was the tumor compressing?
   A. Right sural nerve
   B. Right sciatic nerve
   C. Right lateral femoral cutaneous nerve
   D. Right obturator nerve

Case 7

A 50-year-old male truck driver presents with intermittent left-sided neck pain and muscle spasm. Pain-free periods could last several weeks, but the exacerbations are becoming more frequent and intense and are beginning to interfere with the patient's work. During one of his recent episodes, the patient experienced "tingling" in the middle finger of his left hand for several days. Neurologic examination is normal, but there is considerable impairment of neck mobility and the head-neck region appears laterally shifted to the right. The Neck Disability Index revealed that during an exacerbation, there is moderate disability that interferes with the patient's ability to drive long distances.

1. The patient's complaint of "tingling" in the middle finger of the left hand coupled with his other symptoms points to possible disc derangement at which segment?
   A. C5,6 on the left
   B. C5,6 on the right
   C. C6,7 on the left
   D. C6,7 on the right

2. Restricted segmental extension, rotation, and side-bending left is consistent with an apophyseal joint that is "stuck" in which position?
   A. Closed position on the left
   B. Open position on the left
   C. Closed position on the right
   D. Open position on the right

3. If this patient's symptoms progress to the point of causing neurologic involvement, what would the most likely sign(s) consist of?
   A. Triceps weakness on the left
   B. Biceps weakness on the left
   C. Hypoactive left biceps jerk
   D. A and C

4. A type 2 impairment (FRS right) in the lower cervical spine is treated with PIR of which muscles?
   A. Flexors, left rotators, and left side benders
   B. Flexors, right rotators, and left side benders
   C. Flexors, left rotators, and right side benders
   D. None of the above

5. In the McKenzie system, at the time of the initial visit, this patient presents to you with which derangement?
   A. 1
   B. 2
   C. 3
   D. 4

Case 8

The patient is a 17-year-old female basketball player who presents with recurrent aggravating backache. An MRI exam revealed degenerative disc disease at L4,5 with a mild retrolisthesis of L4. The patient wears a lumbar support during games, which provides temporary relief of painful symptoms. The Dallas Pain Questionnaire revealed that both daily activities interference and work/leisure activities were greater than 50%, indicating significant functional limitation. However, the anxiety/depression and social interest interference were not significantly elevated. The patient's orthopedic surgeon referred the patient for spinal stabilization therapy.

1. What most likely caused the patient's pain?
   A. Capsular hypomobility of the L4,5 facet joints
   B. Clinical "instability" at L4,5
   C. Myofascial pain
   D. Malingering

2. Strengthening of which muscle should prove beneficial to the patient?
   A. Sacrospinalis
   B. Multifidus
   C. Quadratus lumborum
   D. Rectus abdominis

3. Evidence suggests that which muscle plays a key role in providing core stability?
   A. Transversus abdominis
   B. Pubococcygeus
   C. Multifidus
   D. All of the above

4. How is an isolated contraction of the transversus abdominis achieved?
   A. Drawing the navel in toward the spine upon exhalation
   B. Performing abdominal crunches
   C. Performing a PPT
   D. Pulling the abdominal wall in during a deep inhalation

5. The "neutral zone" is characterized by all the following except:
   A. The least symptomatic position
   B. The most stable position
   C. The most efficient position
   D. The close-packed position

Case 9

A 50-year-old female nurse sustained soft tissue injuries to her head and neck in a rear-end motor vehicle accident (MVA) while driving to work. The patient presents to your department 6 weeks post-MVA with complaints of headache, TMJ/facial pain and stiffness, and bilateral neck and shoulder pain. The patient has to work to support her 2 children, but each day is stressful because of the pain, impairment, and functional limitation. On the Pain Rating Index (PRI) of the McGill Pain Questionnaire, the patient had an overall score of 28, involving both sensory and affective aspects of pain.

1. The patient demonstrates 30 mm of mandibular depression and her jaw deflects to the right upon opening. An examination of lateral excursions reveals restriction of motion to the left side. What are the physical findings consistent with?
   A. An ADD without reduction on the right
   B. An ADD with reduction on the right
   C. Capsular hypomobility of the right TMJ
   D. A and C

2. The patient presents with marked FHP. With what is this finding often correlated?
   A. Reduction of the freeway space
   B. Inferior and posterior displacement of the mandible
   C. Decompression of the suboccipital region
   D. All of the above

3. Upon further questioning, the patient reveals that prior to the MVA, her right TMJ would intermittently "click" and "pop" and that on one occasion it momentarily "locked." In light of this information, what do you expect the MRI to be positive for?
   A. Posterior disc displacement on the right
   B. ADD without reduction on the right
   C. An acoustic neuroma
   D. An impacted wisdom tooth

4. The patient also demonstrates an accentuated midthoracic kyphosis with protraction, elevation, and downward rotation of her scapulae. You suspect, but need to test for impairment, of which of the following?
   A. Flexion (T5 to T8)
   B. Extension (T5 to T8)
   C. Lower cervical spine flexion
   D. Glenohumeral internal rotation

5. The Alexander technique would assist this patient with the following:
   A. Restoring the "primary control" mechanism
   B. Releasing tension throughout the head, neck, and shoulder girdle
   C. Shortening the torso
   D. A and B

Case 10

A 70-year-old male college professor presents with a chief complaint of intermittent right calf pain and weakness exacerbated by running. When he runs less than 2 miles he is fine, but after that he is forced to stop and rest because the calf muscles become achy, tight, and weak.

The patient is otherwise healthy and is somewhat discouraged because he has been running competitively most of his adult life. The pain that forces him to stop running is given an intensity of 7 on a 0 through 10 scale (NPRS).

1. Based upon the patient's history, which of the following conditions is a possibility?
   A. Vascular intermittent claudication
   B. Neurogenic intermittent claudication
   C. A and B
   D. None of the above

2. The patient is taken to the track and asked to run until his right calf symptoms appear. Following a brief cool down, the patient is asked to stand still. The patient reports that his symptoms persist in standing, but his symptoms begin to abate following 3 minutes of sitting. What is the most likely diagnosis?
   A. Vascular intermittent claudication
   B. Neurogenic intermittent claudication
   C. A and B
   D. None of the above

3. What pathology is most consistent with this patient's symptomatology?
   A. Type II diabetes
   B. Herniated nucleus pulposus (HNP)
   C. Spinal stenosis
   D. Osteoarthritis of the right hip

4. Given that a positive crossed straight leg raise test (straight leg raising on the patient's well leg elicits pain in the leg with sciatica) has excellent "specificity" (0.90) but low "sensitivity" (0.25), what is this the clinical test of choice for?
   A. Rule-in nerve compression
   B. Rule-out nerve compression
   C. Identify the specific root level involved
   D. All of the above

5. What would placing this patient on a stationary bike and asking him to exercise for 20 minutes most likely result in?
   A. Right calf symptoms
   B. The absence of symptoms
   C. Low back pain
   D. None of the above

Case 11

A 26-year-old female physical therapy student presents with a chief complaint of numbness in her right arm and hand. An MRI of the cervical spine was unremarkable, as was electromyography. All blood work was normal. The patient's neurologist referred her for a physical therapy consult.

1. The patient may have which following condition?
   A. Cervical radiculopathy
   B. Thoracic inlet (outlet) syndrome (TOS)
   C. Carpal tunnel syndrome
   D. All of the above

2. The examination reveals that the Roos or Elevated Arm Stress Test (EAST) is positive on the right,

whereas Spurling's test and Tinel's sign at the wrist are both negative. What is the most likely diagnosis?

A. Cervical radiculopathy
B. TOS
C. Carpal tunnel syndrome
D. None of the above

3. Disc herniation with resultant compression of the C6 nerve root most often occurs at which level?

A. C4,5
B. C5,6
C. C6,7
D. C7, T1

4. Elevation of the first rib may occur in response to hypertonicity of which of the following?

A. Levator scapulae
B. Posterior scalene
C. Anterior scalene
D. B and C

5. Direct fascial technique of which muscle is often beneficial in patients with TOS?

A. The quadratus lumborum
B. The rotator cuff
C. The temporalis
D. The pectoralis minor

Case 12

You were exposed to evidence-based practice in PT school, but your first job is in a department that is far from being evidence based. For the most part, the idea of finding any evidence at all to support a given intervention is rarely discussed, let alone scouring the literature for the "best research evidence."

1. Your plan is to introduce the concept slowly and to start what?

A. Organizing a journal club
B. Speaking with the medical librarian about literature searches
C. Inviting guest speakers who are knowledgeable of the subject
D. All of the above

2. A few therapists in the department are threatened by this concept. They are convinced of the efficacy of their techniques, but are fearful of the changes that may be coming. What is your next step?

A. Tell them to "get with the program"
B. Find a new job
C. Stress the importance of clinical expertise in this paradigm
D. Leave the veterans alone and work with new graduates like yourself

3. The chief therapist realizes the financial liability of not being evidence based. She wants to know what constitutes "best research evidence." What is your answer?

A. Expert opinion
B. RCTs
C. Case studies
D. Retrospective cohort studies

4. One of the recalcitrant "veterans" wants to know whether a patient's input "counts for anything" in this new approach to patient care. What is your answer?

A. Yes
B. No
C. It depends on the patient's knowledge of evidence-based practice
D. They cannot be trusted with such important decisions

5. One year has passed and progress has been made. However, one of the therapists is a proponent of therapy "X" for patients with chronic pain. He has undergone extensive training, but other than patient satisfaction he cannot produce sound scientific validation for the use of this intervention. What is your advice to him?

A. Design an acceptable outcome study given the department's resources
B. Submit a grant application for a randomized controlled trial on therapy "X"
C. A and B
D. "All things must come to an end"

Case 13

A 70-year-old African American male presents with a chief complaint of constant pain confined to the low back region, which has progressively worsened over the past 8 months. There is no history of trauma. The pain is 6 on a 0 to 10 pain scale and is refractory to nonsteroidal anti-inflammatory medication. The pain is worse with movement (8/10) and remains constant with rest (6/10). Coughing or sneezing does not exacerbate the LBP. On further history, the patient notes left-sided chest pain x 4 months that is also aggravated by certain movements. Cardiac, gastrointestinal, and other medical work up for chest pain had previously been unremarkable. Past medical history includes type II diabetes mellitus.

1. Based on the above history, which of the following are you considering as diagnostic possibilities?
   A. Herniated disc
   B. Malignancy
   C. Compression fracture
   D. Osteomyelitis
   E. All the above

2. On a review of systems, the patient notes increasing fatigue and weakness as well as confusion over the past few months. One month ago, the patient was given a course of antibiotics for cellulitis. Approximately 1 year ago, the patient was hospitalized for a strep infection, but denies any other hospitalizations or surgeries. On physical examination, you elicit tenderness over the L3 and L4 SPs. Palpation of the chest wall reveals tenderness over the 5th and 6th ribs on the left side. There are no adverse neural tension signs and the neurological exam is normal. What is the most likely diagnosis? Rank in order of likelihood.
   A. Herniated disc
   B. Malignancy
   C. Compression fracture
   D. Osteomyelitis

3. The patient mentions that he recently had a comprehensive lab work up for life insurance and was told that he has moderate anemia and "something about a lot of calcium." There were other lab findings, but he cannot remember what he was told. Considering the patient's history and symptomatology, select the most likely etiology.
   A. Herniated disc
   B. Malignancy
   C. Compression fracture
   D. Osteomyelitis

4. Of the following malignancies, which do you think is most likely?
   A. Testicular cancer
   B. Prostate cancer
   C. Chronic lymphocytic leukemia (CLL)
   D. Multiple myeloma

5. What do you do next?
   A. Apply moist heat and TENS to the low back without referring to a physician
   B. Apply moist heat and TENS to the low back and refer to a physician
   C. Reassure the patient and see again in 3 months
   D. Refer to a physician for a medical work up

Case 14

A 58-year-old white male presents with severe pain in his right shoulder for approximately 3 months. In the past few weeks he has developed pain in the inner side of his right arm, the elbow, and down into his 4th and 5th fingers. In addition to the throbbing pain, he describes a sensation of tingling on the skin in the same distribution. The pain is constant and only alleviated by narcotic pain medicine he received from his primary care doctor. He cannot recall any trauma or mechanical event at onset of pain.

1. Which of the following are the most likely explanations for his symptoms? Select 3.
   A. He is actively seeking narcotic pain medications and is feigning symptoms for secondary gain.
   B. Shoulder tendonitis
   C. Diabetic neuropathy
   D. Cervical radiculopathy
   E. Thoracic outlet syndrome (TOS)
   F. Malignancy

2. Upon further history, you learn that he works as a mechanic, is a long-time smoker, and has no past medical history other than controlled HTN and mild depression for which he is not medically managed. He does reveal some weight loss in the past few months, but attributes this to his depression, which has been worse since the onset of his terrible pain. Upon examination, you notice that his right eyelid seems to be drooping, which he says he has noticed recently as well but had just attributed it to fatigue. What are you concerned about given this finding?
   A. Not concerned at all. It is likely an incidental finding. He may have had a Bell's palsy in the past.
   B. Cerebrovascular accident, which could explain his facial asymmetry and right upper extremity paresthesias
   C. Malignancy
   D. Cervical radiculopathy
   E. Thoracic outlet syndrome (TOS)

3. You continue your examination and the Spurling's (maximal cervical compression) test, on the right side, is negative for radiculopathy. There is no tenderness to palpation over the cervical vertebrae, and the Roos (EAST) test is negative for TOS. Furthermore, you elicit no proximal muscle weakness, but do find his

grip strength to be diminished on the right. Triceps deep tendon reflex is also diminished. As you are examining him, he tells you that when he sweats while at work, it seems he only sweats on the left side of his face. Immediately you are most concerned about:

A. Diabetes
B. Herniated cervical disc
C. Pancoast tumor
D. Hypothyroidism
E. Myasthenia gravis

4. What other constellation of symptoms will you look for to confirm your suspicion?

A. Swallowing difficulty, slurred speech, double vision, unstable gait
B. Pupil asymmetry, cough, chest wall pain
C. Polyuria, polyphagia, vision changes, dry skin, hair loss
D. Pain worse with coughing or sneezing, muscle spasms in neck

5. What is your next step?

A. Commence pain-relieving modalities (eg, heat, TENS, etc)
B. Refer back to primary care doctor for chest x-ray
C. Put on cervical collar and refer to neurology
D. Commence spinal manual therapy

Answers

Case 1

1. B) Although the patient's headaches appear related to bruxism, stress, and cervical/TMJ impairment, it is always wise to have headache patients worked up by a neurologist in order to rule out secondary headache. The patient may also be suffering from migraine. If that is the case, the patient may respond well to pharmacologic management.

2. C) The NTI-tss appliance has demonstrated efficacy for controlling the effects of bruxism as well as managing migraine and tension-type headache. It is the only appropriate dental intervention listed for this patient.

3. A) The fifth cranial nerve is the primary nociceptive afferent pathway involved with the mediation of head, neck, and TMJ/facial pain.

4. C) The most likely cause of TMJ clicking in a 25-year-old female is internal derangement. Of the possible types, an anterior disc displacement with reduction is the most prevalent.

5. D) The upper cervical spine can be the source of headaches and dizziness, and can contribute to a TMD through both sensory and motor excitation of the trigeminal nerve (ie, masticatory hypertonicity).

Case 2

1. A) Prior to the use of lumbar extension, McKenzie recommends correcting the lateral shift when present. Flexion exercises have not been shown to be effective for posterior derangements. Myofascial intervention of the quadratus lumborum is considered after the derangement is reduced and stable.

2. C) Whenever symptoms are not made worse by movement nor relieved by rest, the therapist should see a "red flag." The proper course of action is to have the physician rule out a nonmechanical disorder as soon as possible. Dr. Stanley Paris believes that physical therapists are experts in dysfunction, whereas physicians are experts in disease.

3. C) Postural syndrome is the least likely option because this patient responded to mechanical interventions consistent with derangement and possibly dysfunction. Postural syndrome by definition may be symptomatic, but there is no limitation of motion or deformity.

4. C) A right lateral shift adducts the left hip and thus places the left TFL under stretch. As a result, the left TFL is the most likely of the 4 choices to become irritated under tension and to be tender.

5. D) This is self-explanatory. See Chapter 17.

Case 3

1. B) Recall that FHP = backward head + forward neck. The patient often appears to have an increased cervical lordosis, but upon radiographic inspection it can be seen that the he or she has a flattened cervical curve. There is evidence in the dental literature that mouth breathing plays a role in the development of FHP in children, which in turn affects the growth and development of the maxillofacial region (ie, retrognathia, malocclusion, TMD, etc). Rocabado has been instrumental in sharing this information with the orthopedic physical therapy community. On the other hand, there are those who hypothesize that poor ergonomics and body mechanics cause the development of FHP as part of the aging process. Whether FHP works its way down from the head or up from the neck, the result is the same and the consequences are significant, as discussed in Chapter 8. It is no wonder that Alexander developed an entire approach to treatment based on the relationship that exists between the head, neck, and upper back.

2. C) Given that the patient's headaches are unilateral, posturally related, and associated with cervical motion loss, the best choice is cervicogenic headache. A history of a neck injury does not necessarily rule out migraine, but it is consistent with headache of cervical origin. Choices B and D are ruled out because of the long headache history.

3. B) CGH is thought to be strongly correlated to C2,3 impairment,[1] although the C1,2 segment is also a likely source.[2] That being said, B is the best choice because the

C5,6 segment refers caudad and not cephalward; the involved segment must be on the same side of the headache, as CGH is side-locked (ie, pain is referred from the neck ipsilateral to its source).

4. D) This is the clear peripheral nerve of choice for headache. As discussed in Chapter 8, greater occipital nerve compression can produce unilateral headache not unlike migraine. Its management, however, is quite different.

5. A) This interconnecting collection of sensory neurons, from the pars caudalis in the lower brainstem to at least the third level in the upper cervical spinal cord, provides the neuroanatomic means whereby the pain related to an upper cervical somatic impairment is able to be felt in the head and TMJ/facial region. It is primarily the ophthalmic division that forms this connection, which explains why CGH is perceived most often in the temporoparietal and frontal region (V1) and not in the maxillary (V2) nor mandibular region (V3).

Case 4

1. A) Based on Fryette's first rule (type 1 spinal mechanics), the thoracic spine, from T5 through T11, will rotate to the right when it is first side bent left. Consequently, the ribs will be displaced posteriorly on the right as a result of the right vertebral rotation that occurs with this patient.

2. D) The other choices are all postural muscles, which according to Janda, tend to become facilitated, hypertonic, and tight.

3. B) As per the arthrokinetic reflex,[3] restricted mid-thoracic extension will inhibit the middle trapezius muscle. Consequently, joint manipulation should always precede muscle strengthening in the presence of weak phasic muscles.

4. D) The levator scapula and pectoralis minor muscles are both downward rotators of the scapula and will restrict upward rotation of the scapula when tight. As a result of limited scapular upward rotation, the suprahumeral tissues (ie, rotator cuff tendons and subdeltoid bursa) are susceptible to compression in the subacromial space during shoulder elevation, resulting in potential impingement.

5. C) Of the choices given, this is the best one. It is thoracic spine hypomobility that often causes compensatory hypermobility in the cervical and lumbar spine, leading to pain in these areas. The astute therapist is always on the "look out" for the AGR. The thoracic spine, like the hips, is a good place to look.

Case 5

1. C) Given the patient's history (ie, missed golf swing) and the physical signs of an iliosacral impairment on the right, it is reasonable to assume that the patient has sustained an anterior rotation subluxation of the right iliac bone. The only set of pelvic landmarks consistent with this diagnosis is choice C.

2. D) This is the muscle that is expected to become hypertonic and short in the presence of an anterior iliac rotation on the right.

3. D) By contracting its antagonistic muscles (ie, the gluteus maximus and hamstrings), the iliopsoas is relaxed through reciprocal inhibition. Once the contractile component of the lesioned complex is minimized, the ilium is free to resume its normal anatomic position on the sacrum. This is the neurophysiologic principle at work with muscle energy technique.

4. A) Large amplitude oscillations at the end of range.

5. B) This is the proper treatment sequence when dealing with tissue dysfunction. When managing a derangement, the sequence is quite different (ie, reduction, maintenance of the reduction, recovery of function, and prevention of recurrence).

Case 6

1. D) All of the above.

2. A) All, except enlargement of the right buttock, are typical of a McKenzie derangement 6 (ie, adverse sciatic tension, lateral lumbar shift, and centralization of symptoms with lumbar extension).

3. A) To ignore this potentially serious sign and proceed with "business as usual" is the wrong course of action. As the saying goes, "When in doubt, don't!"

4. C) The famous British orthopedist, Dr. James Cyriax,[4] described the "sign of the buttock" as an indication of "major lesions in the buttock," including osteomyelitis of the upper femur, chronic septic SI arthritis, ischiorectal abscess, septic arthritis, rheumatic fever with bursitis, neoplasm at the upper femur, iliac neoplasm, and a fractured sacrum. It consists of buttock pain with trunk flexion, hip flexion, and straight leg raising. Passive rotations of the ipsilateral hip are painful, but there is no tissue resistance other than the patient's insistence that the movement be stopped (ie, an "empty end-feel"). Resisted hip movements are often painful, since they alter tensions in the buttock. Inspection of the affected buttock may reveal that it is larger than the other side; palpation may disclose a tumor. With the discovery of these findings, Cyriax recommends that the patient's temperature be taken, a rectal examination be performed, and a radiograph be ordered without delay.

5. B) Given that the tumor was compressing the sciatic nerve in the right buttock area, this patient closely resembled a discogenic patient with sciatic compression. Except for the large buttock and the fact that hip flexion with the knee flexed provoked right buttock pain, this patient would present very much like a patient with a McKenzie derangement 6.

Case 7

1. C) Given symptoms of nerve root irritation in the left C7 dermatome (ie, "tingling" in the middle finger), the most

likely disc derangement would be at the C6,7 level. Because the nerve roots in the cervical spine exit above the pedicle of the corresponding vertebrae, a typical posterolateral cervical disc herniation impinges on the nerve root exiting at the level of the disc. For example, a disc herniation at the C4,5 level compresses the C5 nerve root.

2. B) A combined restriction in extension, rotation, and side bending to the left is referred to as an FRS right. The problem with an FRS right is that the left apophyseal joint is "stuck" in the "open" position and cannot "close."

3. A) Since the triceps muscle is innervated by the efferent fibers of the left C7 nerve root, compression from a herniated disc at C6,7 can potentially lead to triceps weakness, atrophy, and hyporeflexia of the triceps jerk.

4. D) The correct answer would have been the flexors, right rotators, and right side benders. However, because the correct choice is not listed, the answer is D. PIR is an extremely useful intervention in manual therapy. Because there is almost always an element of muscle hypertonicity in somatic impairment of the vertebral and peripheral joints, PIR should routinely be performed prior to mobilization of the noncontractile connective tissue capsule. There are several theories related to the neurophysiology of PIR. These include Golgi tendon organ reflex inhibition, Renshaw cell inhibition of the alpha motoneurons, presynaptic Ia inhibition, reduction in gamma motoneuron activity, and sensorimotor learning. In addition, it is conceivable that PIR achieves increased ROM as a result of the fascial "stretch" produced by the contracting muscle belly. PIR is related to what Hammer[5] refers to as "postfacilitation stretch," which includes an isometric contraction of 7 seconds duration, followed by 12 seconds of stretching.

5. D) This is self-explanatory. See Chapter 8.

Case 8

1. B) As described by Panjabi,[6-8] clinical "instability" is the failure of the "spinal stabilization system" to restrict the neutral zone to the physiologic borders of a segment's ROM. As discussed in the introduction to Chapter 20, the "spinal stabilization system" consists of 3 components, namely the passive, active, and neural control subsystems. In this case, our 17-year-old basketball player already shows signs of degenerative disc disease according to the MRI. According to Macnab,[9] the facet joints in this degenerative state subluxate into hyperextension and are held at the extreme of their limit or what Panjabi calls the elastic zone. Consequently, the extension strains of everyday living tend to push the joints past their physiologically permitted limits and thereby produce pain. In general, segmental "instability" is considered to be anything greater than 3.5 mm of horizontal translation (ie, along the Z axis) and/or 11 degrees of angular motion (ie, rotation around the X axis) on a standing lateral radiograph with the patient moving between flexion and extension.[10-12] It must be kept in mind, however, that the presence of "instability" must always be correlated with the patient's symptoms in the clinical decision-making process.

2. B) Of all the choices, the only muscle that has been shown to control segmental motion of the spine is the multifidus. All the others (sacrospinalis, quadratus lumborum, and rectus abdominis) are postural muscles that tend toward hypertonicity and shortening, which do not provide physiologic stability to the lumbar spine.

3. D) The 3 muscles listed (ie, the transversus abdominis, pubococcygeus, and multifidus) are all core stabilizers. The fourth, which is not listed, is the respiratory diaphragm.

4. A) This is the correct way of eliciting a transversus abdominis contraction, but it is not the easy way. The easy but incorrect way is to "suck the belly in" with a deep inhalation or to perform a PPT. However, neither has been shown to activate the transversus, and the patient must not be permitted to "cheat" by taking these measures.

5. D) It is the close-packed position of segmental hyperextension that causes this young athlete so much grief. The goal of "spinal stabilization training" is to activate the active and neural control subsystems so that the patient regains control of the neutral zone in hopes of recovering function and managing symptoms.

Case 9

1. D) Given the "capsular pattern" of the right TMJ, choices A and C are both possibilities. Based upon physical signs alone, we know that there is limited translation of the right mandibular condyle. However, at this point we don't know whether the capsule is inflamed or the disc is displaced without reduction. Stay tuned!

2. A) The only acceptable answer is A. Whereas head-neck extension increases the interocclusal or freeway space, FHP has been shown to decrease it. It is theorized that the increase in temporalis activity, associated with occipital extension, plays a role in this response by displacing the mandible in a posterior and superior direction. The author submits that the forward translation of the occipital condyles on the atlas vertebra also plays a role by causing a vertical "drop" of the skull onto the mandible (ie, not only does FHP cause the mandible to be "pulled" upward, but it also causes the maxilla to "drop" down on the mandible, which in either case diminishes the freeway space between the upper and lower teeth).

3. B) The difference between question 1 and 3 is the patient's history. The additional information indicates the presence of a right-sided TMJ internal derangement prior to her MVA. Consequently, we would expect the MRI to confirm a nonreducing anterior disc displacement (ie, a closed-lock).

4. B) The most likely restriction in light of an accentuated midthoracic kyphosis with scapular protraction, elevation, and downward rotation is thoracic extension. This can be tested with PAIVMs or PPIVMs as discussed in chapter 4.

5. D) The Alexander technique accomplishes many things, but there are 4 core components. They are restoring "primary control" by allowing the neck to release so that the head can balance forward and up, which in turn facilitates a lengthening and widening of the torso, a lateral release of the shoulders away from the chest wall, and a release of the legs/hips away from the pelvis. Consequently, A and B are both good choices.

Case 10

1. C) Intermittent claudication (ie, limping or "lameness") has generally been attributed to an occlusive vascular disease of the legs. In the mid 1950's, however, Verbiest[13] also linked this clinical picture to narrowing of the spinal canal (ie, spinal stenosis) with resultant compression of the spinal nerve roots. Typically, the patient walks a certain distance until leg symptoms, such as pain, numbness, weakness, etc, force the patient to stop walking. Unlike vascular claudication, which requires only that the patient rests, neurogenic claudication requires that the patient decompress the impinged nerve roots by flexing his or her lumbar spine. Based on the history provided, our professor may have claudication of either a vascular or neurogenic nature. Stay tuned!

2. B) The crucial diagnostic distinction between a vascular and a neurologic disorder, in this instance, is found in the intervention that relieves the patient's symptoms. Because spinal flexion helped whereas rest alone did not, the right calf symptoms are therefore due to neurogenic claudication.

3. C) HNP is the only other choice that comes close. However, HNP is most prevalent in the 25- to 45-year-old range, whereas stenosis is a more likely cause of nerve root compression at age 70 (this is because of the gradual loss of water content in the disc from approximately 90% in early adult life to 70% in the elderly). In addition, the peripheral symptoms associated with an HNP tend to worsen with spinal flexion rather than extension.

4. A) Given the excellent specificity (ie, the ability of a test to correctly identify patients without a disease ["negative in health"]) of the crossed straight leg raising test (0.90), it is more effective as a rule-in test for peripheral nerve compression, whereas ipsilateral SLR is more effective as a rule-out test, given its high degree (0.80) of sensitivity (ie, the proportion of persons with the disease who have a positive test "[positive in disease"]). In other words, because the crossed SLR is rarely positive in health (only 10% of the time), a positive test result almost always indicates impairment. Similarly, the absence of sciatica (or femoral nerve radicular pain) makes a clinically significant disc herniation very unlikely because, being such a sensitive finding (0.95), its absence becomes highly significant.[10]

5. B) Because the patient's lower limb circulatory status is not the problem, the "stationary bike test" should be normal. With intermittent neurogenic claudication secondary to spinal stenosis, the provoking factor is not ischemia, but rather activities that place the lumbar spine into an extended position. In fact, the flexion associated with riding a bicycle could prove to be therapeutic for patients suffering from spinal stenosis.

Case 11

1. D) Based on the history provided, all the choices listed are potential causes of the patient's chief complaint.

2. B) A positive Roos or Elevated Arm Stress Test (EAST) rules-in TOS. This, in conjunction with tests to rule-out the cervical spine (negative Spurling's test) and the carpal tunnel (negative Tinel's sign), makes the diagnosis more likely.

3. B) For the reason given in case 7, question 1, the answer is the C5,6 disc. This formula, however, changes at the cervicothoracic junction and below. Starting at T1 and throughout the remainder of the thoracolumbar spine, the nerve roots exit caudal to the pedicle of the corresponding vertebra. However, in the lower lumbar spine where most of the herniated discs occur, it is still the lower root level that is most often impinged (ie, an L4,5 disc herniation will compress the L5 nerve root). This is because the nerve root is not impinged at the level of the foramina where it exits, but posteriorly as it descends through the spinal canal. However, a large disc herniation and/or lateral recess stenosis may violate this rule and impinge the nerve root above (ie, L4 compression at the L4,5 level). According to Kramer,[14] herniated cervical discs occur most often at C5,6 (41%) followed by C6,7 (33%). Nerve root involvement by level is C5 = 4.1%, C6 = 36.1%, C7 = 34.6%, and C8 = 25.2%.

4. C) This is the only possible choice. The posterior scalene attaches to the second rib, where hypertonicity may cause a superiorly laterally flexed rib.

5. D) Of all the muscles listed, the pectoralis minor is the only one that has direct bearing on TOS. Because it is capable of impinging the neurovascular bundle (subclavian artery, vein, and lower trunk of the brachial plexus), direct fascial technique, for the purpose of releasing muscular tension and restrictions, is often quite effective in the management of TOS.

Case 12

1. D) A through C are all excellent ways of beginning the process. Before your department can be evidence-based, the therapists must learn how to access the literature. In addition, most clinicians require further postgraduate training in critiquing scientific papers. Guest speakers with expertise in research design can get the ball rolling.

2. C) Sackett et al[15] emphasize that EBM is the "integration of best research evidence with clinical expertise and patient values." Consequently, the anxious therapists in this department must realize that their clinical experience and expertise are not completely overlooked in this system. The goal of EBM is not to "throw the baby out with the bath-

water," but rather to apply a systematic review process to all aspects of clinical physical therapy so that clinical practice guidelines can be established. The operative word here is *guideline* and not *mandate*. If presented in this manner, EBM need not be feared but welcomed.

3. B) Of the 5 levels of potential evidence, Sackett et al[15] place the systematic review of high-quality RCTs at the top of the list (ie, level 1). Though not the only acceptable form of evidence, RCTs do qualify as the "best evidence" available.

4. A) Again, as per the definition of EBM mentioned above,[15] "patient values" do enter into the equation. It must always be remembered that above all else, the needs of the patient are paramount. Although EBM is an attempt to utilize interventions that are based upon the best evidence available, we as physical therapists are still treating human beings. Their needs, feelings, expectations, and welfare must always remain our top priority. If, for example, a patient "swears by ultrasound" and is convinced that it has helped in the past, this should then become part of the "evidence" in designing a treatment plan for this patient. It would be wrong, in the opinion of the author, to dismiss this patient's preference, unless there are known contraindications, simply because there was a lack of RCTs on this intervention. In other words, common sense sometimes needs to carry the day!

5. C) In the days of EBM it is no longer acceptable to say, "I know it works and that's all that matters." The retort is, "If it you know it works, then prove it"! In the long run, this therapist will benefit when his therapy "X" is shown to be effective. As someone has wisely said, "It's not science until it's published." The question is wherher to refrain from providing a particular intervention when there are no acceptable studies one way or the other.[16] In this author's opinion, we must exercise extreme caution at this early stage in the development of EBM. The process must be given time and we must be patient!

Case 13

1. E) With the information provided, any of the choices given could explain the patient's chronic LBP.

2. A), B), C), D)

A. Except for the LBP made worse with movement, this pain is not consistent with a herniated disc (eg, constant pain unrelieved with anti-inflammatory medication, pain not worse with coughing or sneezing, absence of adverse neural tension, normal neurological exam).

B. Increasing fatigue and weakness, confusion, and recurrent infections suggest systemic cause such as malignancy.

C. In an elderly patient, point tenderness over a SP, aggravated by movement, could suggest compression fracture. However, compression fractures more commonly occur in white females, especially in those with a history of osteoporosis, chronic steroid use, and vitamin deficiency.

D. Two focal areas of bony pain could suggest osteomyelitis, especially in the setting of recurrent infections. Having type II diabetes places this patient at risk for pyogenic vertebral osteomyelitis. The absence of fever does not necessarily rule out osteomyelitis but does make it less likely. In addition, the absence of erythema, edema, and skin break make this a less likely diagnosis than malignancy.

3. B) Anemia and hypercalcemia in the setting of chronic pain, progressively worsening, plus increasing fatigue and weakness indicate malignancy as a likely cause of the patient's LBP.

4. D) Multiple myeloma is a malignant proliferation of plasma cells in the bone marrow. About 70% of patients have bone pain, usually involving the back and ribs, precipitated by movement. Due to substantial calcium mobilization, there is usually hypercalcemia and associated symptoms such as confusion, constipation, and lethargy. Anemia occurs in 80% of patients because of inhibition of erythropoiesis by tumor products. Recurrent bacterial infections are due to impaired immunoglobulin production by the bone marrow. Multiple myeloma is most common in African Americans and the peak age of incidence is between 50 and 60 years. Prostate cancer would be a reasonable choice as it is more prevalent in African Americans and commonly metastasizes to the vertebral column. However, the concurrent rib pain and lab findings suggest an alternate diagnosis. CLL is the most common hematologic malignancy in the elderly, but does not present with bony pain. Testicular cancer is much more prevalent in males in the second and third decades of life.

5. D) The obvious choice.

Case 14

1. D), E), F)

A. Although this must also be kept in back of the health care provider's mind it should never be the first conclusion reached. The pain must be thoroughly evaluated and the patient's history trusted unless you are given reason to believe that they are exhibiting "drug seeking" behavior.

B. Because of the distal referral pattern (4th and 5th fingers) and complaint of paresthesia, shoulder tendonitis is an unlikely cause of the patient's symptoms.

C. Diabetic peripheral neuropathy, also called distal symmetric neuropathy or sensorimotor neuropathy, usually presents in a "glove and stocking distribution." Your feet and legs are likely to be affected before your hands and arms.

D. Given the distal referral pattern (ie, below the elbow), complaint of paresthesias, and the severe nature of the pain, cervical radiculopathy must be a consideration at this point.

E. TOS must also be considered, especially in light of symptoms being in the C8, T1 nerve distribution (neurologic involvement in TOS is characterized by pain and paresthesias most commonly in the ulnar nerve distribution).

F. Although this would not be the primary consideration at this point, it is important to leave this in your differential, as the symptoms of malignancy can be vast and unusual. In a patient without clear etiology for pain and in this case neuropathy, an occult malignancy should be considered.

2. C)

A. Although it may be an incidental, unrelated finding it should raise the suspicion for underlying neurological compromise. The unilateral nature of his drooping eyelid also raises suspicion of an etiology other than fatigue. Bell's palsy could present with unilateral drooping, but is usually associated with other cranial nerve VII deficits.

B. A central stroke would not present as a peripheral neuropathy. This patient's pain and paresthesias are clearly in an ulnar distribution.

C. Given the history of smoking and weight loss, the concern for malignancy must remain strong. The drooping eyelid must be considered as related to the arm paresthesias because of the new onset of both of these symptoms. With unusual symptoms that appear chronologically, a systemic process should be suspected.

D. The presence of weight loss could be related to loss of appetite from severe pain, but could also be a "red flag" for a serious, pathological condition; the drooping of the eyelid underscores this possibility. Consequently, more information is needed before proceeding with a diagnosis of cervical radiculopathy.

E. The additional information obtained upon further history (ie, weight loss and eyelid drooping) gives us pause. Though TOS remains a possibility, we must investigate further.

3. C) A pancoast tumor is a subset of lung cancer, usually a bronchogenic carcinoma, that resides in the lung apex, adjacent to the eighth cervical nerve roots, the first and second thoracic trunk distribution, and the sympathetic chain. As the tumor invades the brachial plexus (C8), it is typical to see pain, paresthesias, and weakness in an ulnar distribution. If the tumor extends to the sympathetic chain and stellate ganglion, Horner syndrome (ptosis, meiosis and anhidrosis) may develop on the ipsilateral side of the face and upper extremity. Pain is frequently relentless and unremitting, often requiring narcotics for relief. The patient usually supports the elbow of the affected arm in the hand of the opposite upper extremity to ease the tension on the shoulder and upper arm. The hand muscles may become weak and atrophic, and the triceps reflex may be absent. The first or second rib or vertebtae may be involved by tumor extension and intensify the severity of pain. The spinal canal and spinal cord may be invaded or compressed, with subsequent symptoms of spinal cord tumor or cervical disk disease. Confusion with TOS and cervical disc disease is common in the early clinical course. Careful neurologic examination, electromyographic studies, and ulnar nerve studies are performed to verify the precise diagnosis.

4. B) As previously mentioned, with Horner syndrome one would expect to find meiosis (small pupil on the affected side). Cough would be consistent with the lung cancer. Chest wall pain would be consistent with local invasion of the tumor into the pleura and apical chest wall.

5. B) With any suspicion for an etiology that is nonmechanical and pathological in nature, a prompt referral should be made to the primary care physician.[17]

References

1. Schoensee SK, Jensen G, Nicholson G, Gossman M, Katholi C. The effect of mobilization on cervical headaches. *J Orthop Sports Phys Ther.* 1995;21(4):184-196.
2. Hall T, Robinson K. The flexion-rotation test and active cervical mobility: a comparative measurement study in cervicogenic headache. *Manual Therapy.* 2004;9:197-202.
3. Wyke BD. Articular neurology and manipulative therapy. In: Glasgow EF, Twomey LT, Scull ER, Kleynhans AM, Idczak RMC, eds. *Aspects of Manipulative Therapy.* Melbourne: Churchill Livingstone; 1985.
4. Cyriax J. *Textbook of Orthopaedic Medicine, Vol. 1: Diagnosis of Soft Tissue Lesions.* London: Bailliere Tindall; 1978.
5. Hammer WI. *Functional Soft Tissue Examination and Treatment by Manual Methods: New Perspectives.* 2nd ed. Gaithersburg, MD: Aspen Publishers; 1999.
6. Panjabi M. The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. *J Spinal Disorders.* 1989;5:383-389.
7. Panjabi M. The stabilizing system of the spine. Part II. Neutral zone and stability hypothesis. *J Spinal Disord Tech.* 1992;5:393-397.
8. Panjabi M. Clinical spinal instability and low back pain. J Electromyogr Kinesiol. 2003;13:371-379.
9. Macnab I. *Backache.* Baltimore, MD: Williams & Wilkins; 1977.
10. Weinstein JN, Rydevik BL, Sonntag VKH, eds. *Essentials of the Spine.* New York, NY: Raven Press; 1995.
11. Kasai Y, Morishita K, Kawakita E, Kondo T, Uchida A. A new evaluation method for lumbar spinal instability: Passive lumbar extension test. *Phys Ther.* 2006;86(12):1661-1667.
12. Rocabado, M. The importance of soft tissue mechanics in stability and instability of the cervical spine: A functional diagnosis for treatment planning. *J Craniomandib Pract.* 1987;5(2):130-138.
13. Verbiest H. A radicular syndrome from developmental narrowing of the lumbar spine vertebral canal. *J Bone Joint Surg (Br).* 1954;36B:230-237.
14. Kramer J. *Intervertebral Disk Diseases: Causes, Diagnosis, Treatment and Prophylaxis.* 2nd ed. New York, NY: Thieme Medical Publishers; 1990.
15. Sackett DL. Straus SE, Richardson WS, et al. *Evidence-Based Medicine: How to Practice and Teach EBM.* 2nd ed. New York, NY: Churchill Livingstone; 2000.
16. Medeiros J. Knowledge-based practice. *Journal Manual & Manipulative Ther.* 2002;10(2):64-65.
17. Jette DU, Ardleigh K, Chandler K, McShea L. Decision-making ability of physical therapists: physical therapy intervention or medical referral. *Phys Ther.* 2006;86(12):1619-1629.

Appendix A
Southside Institute for Physical Therapy—Physical Therapy Evaluation: Cervical/TMJ Form

Makofsky HW
Spinal Manual Therapy, 2nd ed. (pp 283-290)

Physical Therapy Evaluation

Date: _____/_____/_________

Cervical/TMJ

Referring Clinician: ______________________________

NAME: ______________________________

Diagnosis: ______________________________

____

____

Last physician office visit: _____/_____/_________

Occupation: ______________________________

___Not presently employed, ___Retired, ___Disabled- Since _____/_____/_________

If Employed- Presently at Work: Yes______, No______

Sport/ Exercise Program: ______________________________

Patient's Complaint: ______________________________

____

How Sustained: ______________________________

Areas Examined:

Lower Quarter_____ Hip_____ Knee_____ Foot/Ankle_____

Upper Quarter_____ Shoulder_____ Elbow_____ Wrist_____ Hand_____

Pelvic Girdle_____ Lumbar_____ Thoracic_____ Costal Cage_____ Cervical_____ TMJ_____

Summary of Significant Findings: ______________________________

____

____

____

____

____

____

PDM= Pain During Motion, **ERP**= End Range Pain

Recommended frequency & duration of treatment: ______________________________

Clinical Impression: ______________________________

____

____

____

Page 2

NAME: ______________________________

Short Term Goals: To be acheived in _____ weeks

___Reduce pain intensity from __/10 to__/10 in ________________ & from__/10 to __/10 in ____________
___Reduce pain intensity from__/10 to __/10 in right LE, from __/10 to __/10 in left LE
___Reduce pain intensity from__/10 to __/10 in right UE, from __/10 to __/10 in left UE
___Centralize peripheral symptoms from _______________ to _________________
___Centralize peripheral symptoms from _______________ to _________________
___Increase muscle strength of ____
____

___Increase muscle length of ________________ from_________ to ____________________________
____

___Normalize end feel of ___________________ from _________ to _________
___Normalize end feel of ___________________ from _________ to _________
___Decrease palpable reactivity from _________ to _________ in ____________________________
____

___Increase ROM of Cervical spine region: Flexion to ___, Extension to ___, SB Right to ___, SB Left to ___
Rotation Right to ___, Rotation Left to ___
___Increase ROM of ____
____

___Increase joint mobility from severe/ moderate/ minimal hypomobility to moderate/ minimal/ normal at ____
___Increase joint mobility from severe/ moderate/ minimal hypomobility to moderate/ minimal/ normal at ____
___Reduce postural dysfunction: Reduce sev/mod/min FHP to mod/min/no FHP, ____________________
____

___Patient to demonstrate independence in home exercise program
___Patient to demonstrate independence with proper body mechanics
___Improve endurance from a_________ to a _________ grade
___Improve gait pattern: ____
___Improve balance: standing static from a _________ to a _________ grade & standing dynamic from a
_________ to a _________ grade
___Decrease nerve tension reactivity of ______________ from _________ to _________
___Improve Function: ____
____
____
____
____

Long Term Goals:

____
____
____

Treatment Plan:

___P.T. Modalities: Ultrasound, Phonophoresis, E. Stimulation, Iontophoresis, Ice, Moist Heat, ___Home TENS Unit
___Postural Education, ___Instruction of proper body mechanics, ___Instruction of H.E.P., ___Joint M.E.T.'s,
___Joint Mobilization/Joint Manipulation, ___Soft Tissue Mobilization, ___M.F.R., ___L/S Stabilization Exercises,
___C/T Stabilization, ___Therapeutic Ex.s, ___Cervical McKenzie Ex.s: (___extension protocol ___flexion protocol)
___L/Q Strengthening, ___L/Q Stretching, ___U/Q Strengthening, ___U/Q Stretching, ___Passive ROM, ___TMJ
Translation/ Rotation & Stabilization Ex.s, ___Gait Training, ___Balance Training, ___STJ Neutral Foot/Ankle
Orthotics, ___Heel Lift, ___Nerve Tension Mob., ___Craniobase Release, ___Manual Traction, ___Cardiovascular/
Endurance Ex.s, ___ Plyometrics, ___Work Conditioning Ex.s, ___Surgical protocol according to ____________
____
____

Signature: ______________________________

NAME: ______________________

History/Complaint:
(See History Form for PMH, PSH, Meds, etc....)
(Age, sex, onset, mechanism of injury & course of condition)

____

____

____

____

____

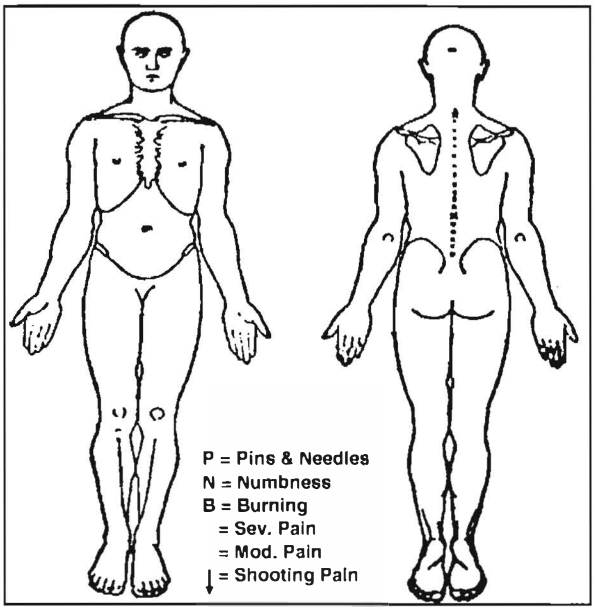
____

Previous episodes of current condition: (circle) 0, 1-5, 6-10, 11+ , Year of first episode: ______
Past Treatment for this condition: ______________________

Equipment\Orthosis: ______________________

Work Activities: ___% sitting, ___% standing/walking, ___lbs. lifting max., other: ______
Work limitations: ______________________
___Performs routine household chores (circle) indoors/ outdoors
ADL Limitations: ______________________
Pyschosocial: Lives with (circle) spouse, friend, sibling, parent(s), ___children.
Lives in (circle) apartment, condo/townhouse, house
___steps going into dwelling, ___steps inside home required to access______
Stress: minimal/ moderate/ severe
Leisure Activities:__________, Sport/ Exercise Program:__________
Any Scheduled Surgery: Y/ N ______________________
Disturbed Sleep: Y/N, If yes, what is preventing patient from sleeping ______
Bed: (circle) Firm, Soft, Sagging, Waterbed. Number of Pillows and used where? ______
Sleep: (circle) Prone/ Supine/ Right side lying/ Left side lying/ No one position
Buttock/Calf pain following walking Y / N, Relieved by Sitting / Standing Still Y / N
Is Pain Better (B), Worse (W), or Unchanged (U):
lying ___, sitting ___, turning ___, standing still ___, walking ___, bending ___, lifting ___, reaching over head ___
coughing/ sneezing ___, valsalva +/ -, in the am __, as the day progresses ___, in the pm ___,
Sitting Tolerance:__________, Walking Tolerance:__________, Standing Tolerance:__________
Pain better with: __________, **Pain worse with:** __________
Key: C=Constant, I=Intermittent, O=Occassional, Y=Yes, N=No

Key Questions:	Frequency: (C,I,O,Y,N)	Location/ Other:
dull/ achy		
sharp/shooting		
pulsing/throbbing		
burning pain		
night pain		
numbness		
pins/needles		
weakness		
B&B changes		
dizzines/fainting	/	
headaches		
stiffness		
buckling		
catching/locking	/	/
joint noise		
edema		
caffeine intake		AMT:
smoker		AMT:
SOB/lethargic	/	
tinnitus/ear stuffiness	/	L/ R ear / L/ R ear
weight loss/ gain		



NAME: ______________________________

Postural Exam:

Standing: FHP- min/ mod/ sev, C/S Lordosis- ↑ / ↓ / N, T/S Kyphosis- Upper- ↑ / ↓ / N. Mid- ↑ / ↓ / N, Lower T/S- ↑ / ↓ /N, L/S Lordosis- ↑ / ↓ / N, ______________________________

Convexities: C/S- L / R, Upper T/S- L / R , Mid T/S- L / R , Lower T/S- L / R , L/S- L / R, No Convexities

Sitting: C/S Lordosis- ↑ / ↓ / N, T/S Kyphosis- Upper- ↑/ ↓ / N. Mid- ↑ / ↓ / N, Lower- ↑/ ↓ /N, L/S Lordosis- ↑/ ↓ / N, ______________________________

<u>Key</u>:
Tender= **T**, Dense= **D**, Tight= **Ti**, Pain= **P**
Restricted= **R**, Holding= **H**, Min= **1**, Mod= **2**, Sev= **3**

Palpation	L	R
Inferior clavicle		
Subcoracoid		
Cervical facets		
Longus colli		
Hyoid mobility		
Infrahyoids		
Suprahyoids		
Digastrics		
Lateral pole		
E.A.M.		
Temporalis		
Medial pterygoid		
Coronoid		
Frontalis		
Orbicularis oculi		
Masseter		
Scaleni		
Dorsal scap. nerve		
Suboccipitals		
Gr. Occipital nerve		
Levator scapulae		
Rhomboids		
Trapezius		
SCM		
Post. cervical mm.		
Post. thoracic mm.		
Thoracic facets		
First rib		

Sensory Screen	Left UE	Right UE
Sharp/ Dull		
Light Touch		
Deep Pressure		
Proprioception		

<u>**Key:**</u>
0=Absent
1+=Decreased
2+=Normal
3+=Increased
4+=Clonus
R =Reinforced

Reflexes	L	R
C5,C6 Biceps		
C5,C6 Brachio		
C7 Triceps		

<u>**Key:**</u> **0**= No Contraction, **1**= Trace, **2**= Poor
3= Fair, **4**= Good, **5**= Normal

Myotome Screen	Left	Right
C1,2 Neck Flexors		
C2,3,4 Upper Trapezius		
C5 Biceps		
C6 Wrist Extensors		
C7 Triceps		
C8 Thumb Extensors		
T1 Hand Intrinsics		

R —————————— **L**

Oral/Facial:

Structure/ Tongue position: ______________________________

____

Muscle Strength: ______________________________
Palpable Click: *R opening. R closing, L opening, L closing*
Audible Click: *R opening, R closing, L opening, L closing*

Depression: _______ mm
Left Lateral Deviation: _______ mm
Right Lateral Deviation: _______ mm
Protrusion: _______ mm
Joint Play (0-6 Mobility Scale): Distraction: L____, R____
Anterior Glide: L____, R____ / Lateral Glide: L____, R____

NAME: ______________________

Key: **ERP**= End Range Pain
PDM= Pain During Motion, **OPP**= Over Pressure Pain

Cervical AROM	ROM	PDM	ERP	OPP	Symptoms
Flexion (60)					
Extension (50)					
SB Right (45)					
SB Left (45)					
Rotate Right (90)					
Rotate Left (90)					

Special Tests	L	R
Alar Odontoid		
Sharp Pursor test		
Hautard's V.A. test		
DeKleyn's V.A. test		
C/S Quadrant test		
Interscalene TOS		
Costoclavicular TOS		
Subcoracoid TOS		
Upper limb tension		
ROOS test		
Hoffman test		

Upper Limb Tension Tests (ULTT)

	Left/ Right
ULTT 1 (Median)	GH Abd__/__, Wrist/Finger Ext__/__, Sup__/__, GH ER__/__, Elb Ext__/__, C/S SB__/__
ULTT 2a (Median)	Shld. Dep__/__, Elb Ext__/__, Arm ER__/__ Wrist/ Finger Ext__/__, Shld. Abduction prn__/__
ULTT 2B (Radial)	Shld. Dep__/__, Elbow Ext__/__, Arm IR__/__ Wrist/ Thumb Flexion__/__, Shld. Abduction prn__/__
ULTT 3 (Ulnar)	Shld. Dep__/__, Arm ER__/__, Shld. Abd__/__ Forearm Pronation__/__, Wrist/Finger Ext__/__

Derangement Screen

Test Movements	Symptoms Before Testing		Pain During Motion		End Range Pain		Symptoms After Testing	
	S	L	S	L	S	L	S	L
Protrusion								
Rep. protrusion								
Flexion								
Rep. Flexion								
Retraction								
Rep. Retraction								
Retr. w/ ext.								
Rep. Retr. w/ ext.								
SB right								
Rep. SB right								
SB left								
Rep. SB left								
Rot. right								
Rep. rot. right								
Rot. left								
Rep. Rot. Left								

Key: ***Pain during motion:*** **P**- no symptom at rest & motion creates pain, **I**- increase existing pain, **D**- decrease existing pain, **A**- abolishes previously existing pain

Pain after testing: **W**- worse as a result of motion, **NW**- not worsened as a result of motion, **NB**- symptoms are decreased with each movement, but do not remain decreased, **NE**- movement has no effect on symptoms at all

NAME: ______________________

Passive Intervertebral Mobility Testing:

sidebending/ sidegliding

Segment	Neutral		Flexion		Extension		P-A Spring
	L	R	L	R	L	R	
O/A							
A/O							
A/A							
A/A Rot							
C2/C3							
C3/C4							
C4/C5							
C5/C6							
C6/C7							

Key:
- **0-** Ankylosed
- **1-** Considerable Restriction
- **2-** Slight Restriction
- **3-** Normal
- **4-** Slight Increase
- **5-** Considerable Increase

Positional Testing:

Segment	Neutral	Flexion	Extension	P-A Spring
C7/T1				
T1/T2				
T2/T3				
T3/T4				
T4/T5				
T5/T6				
T6/T7				
T7/T8				
T8/T9				
T9/T10				
T10/T11				
T11/T12				
T12/L1				

Key:
- **P=** Posterior transverse process on that side
- **T=** Tenderness
- **TC=** Tender Central
- **TR=** Tender Right TP
- **TL=** Tender Left TP
- **TB=** Tender Bilat. TPs
- **1=** Minimal
- **2=** Moderate
- **3=** Severe

Summary of Mobility/ Positional Testing: ______________________

____

Extremity: ______________________

____

Gait: ______________________

Cranial Nerve Screen

Cranial Nerve		Desription of Assessment	Positive	Negative
I	Olfactory	Smelling disturbance- Y or N	Y	N
II	Optic	Vision disturbance- Y or N	Y	N
III	Oculomotor	Can patient move eyes up & to the right & left- Y or N	N	Y
IV	Troclear	Can patient move eyes down- Y or N	N	Y
V	Trigeminal	Can patient close jaw tightly- Y or N	N	Y
VI	Abducens	Can patient look purely to right or left- Y or N	N	Y
VII	Facial	Can patient smile or pout- Y or N	N	Y
VIII	Vestibulococlear	Any hearing disturbances- Y or N	Y	N
IX	Glossopharyngeal	Gag reflex- Y or N	N	Y
X	Vagus	Gag reflex- Y or N	N	Y
XI	Accessory	Weakness of SCM or trapezius- Y or N	Y	N
XII	Hypoglossal	Can patient actively move tongue- Y or N	N	Y

Courtesy of Dr. Kevin Cerrone, Southside Institute for Physical Therapy, Bay Shore, NY.

Appendix B
Southside Institute for Physical Therapy—Physical Therapy Evaluation: Spinal Form

Makofsky HW
Spinal Manual Therapy, 2nd ed. (pp 291-298)

Physical Therapy Evaluation

Date: _____/_____/_________

SPINAL

Referring Clinician: ______________________

NAME: ______________________________

Diagnosis: ______________________

Last physician office visit: ____/_____/__________

Occupation: ____

___Not presently employed, ___Retired, ___Disabled- Since _____/_____/__________

If Employed- Presently at Work: Yes______, No______

Sport/ Exercise Program: ______________________________________

Patient's Complaint: ______________________________________

How Sustained: ______________________________________

Areas Examined:

Lower Quarter_____ Hip_____ Knee_____ Foot/Ankle_____

Upper Quarter_____ Shoulder_____ Elbow_____ Wrist_____ Hand_____

Pelvic Girdle_____ Lumbar_____ Thoracic_____ Costal Cage_____ Cervical_____ TMJ_____

Summary of Significant Findings: ______________________________________

PDM= Pain During Motion, **ERP**= End Range Pain

Recommended frequency & duration of treatment: ______________________

Clinical Impression: ______________________________________

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NAME: ____________________

Short Term Goals: To be acheived in _____ weeks

___Reduce pain intensity from ___/10 to ___/10 in ________________ & from___/10 to ___/10 in ______________
___Reduce pain intensity from___/10 to ___/10 in right LE, from ___/10 to ___/10 in left LE
___Reduce pain intensity from___/10 to ___/10 in right UE, from ___/10 to ___/10 in left UE
___Centralize peripheral symptoms from ______________ to ________________
___Centralize peripheral symptoms from ______________ to ________________
___Increase muscle strength of ____

___Increase muscle length of ________________ from__________ to ____________________

___Normalize end feel of __________________ from __________ to __________
___Normalize end feel of __________________ from __________ to __________
___Decrease palpable reactivity from __________ to __________ in ______________________
___Increase ROM of Lumbo-thoracic region: Flexion to___, Extension to___, SB Right to___, SB Left to___, Rotation Right to___, Rotation Left to ___
___Increase ROM of Cervical spine region: Flexion to ___, Extension to ___, SB Right to ___, SB Left to ___ Rotation Right to ___, Rotation Left to ___
___Increase ROM of ____
___Decrease edema of ____
___Increase joint mobility from severe/ moderate/ minimal hypomobility to moderate/ minimal/ normal at ____
___Increase joint mobility from severe/ moderate/ minimal hypomobility to moderate/ minimal/ normal at ____
___Reduce postural dysfunction: Reduce sev/mod/min FHP to mod/min/no FHP, ____________________

___Patient to demonstrate independence in home exercise program
___Patient to demonstrate independence with proper body mechanics
___Improve endurance from a_________ to a _________ grade
___Improve gait pattern: ____
___Improve balance: standing static from a _________ to a _________ grade & standing dynamic from a _________ to a _________ grade
___Decrease nerve tension reactivity of ______________ from _________ to _________
___Improve Function: ____

Long Term Goals:

Treatment Plan:

___P.T. Modalities: Ultrasound, Phonophoresis, E. Stimulation, Iontophoresis, Ice, Moist Heat, ___Home TENS Unit ___Postural Education, ___Instruction of proper body mechanics, ___Instruction of H.E.P., ___Joint M.E.T.'s, ___Joint Mobilization/Joint Manipulation, ___Soft Tissue Mobilization, ___M.F.R., ___L/S Stabilization Exercises, ___C/T Stabilization, ___Therapeutic Ex.s, ___McKenzie Ex.s: (___extension protocol ___flexion protocol) for___L/S, for___C/S, ___L/Q Strengthening, ___L/Q Stretching, ___U/Q Strengthening, ___U/Q Stretching, ___Passive ROM, ___TMJ Translation/ Rotation & Stabilization Ex.s, ___Gait Training, ___Balance Training, ___STJ Neutral Foot/Ankle Orthotics, ___Heel Lift, ___Nerve Tension Mob., ___Craniobase Release, ___Manual Traction, ___Cardiovascular/ Endurance Ex.s, ___ Plyometrics, ___Work Conditioning Ex.s, ___Surgical protocol according to ____________

Signature: ____________________

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NAME: ______________________

History/Complaint:
(See History Form for PMH, PSH, Meds, etc....)
(Onset, how sustained/ mechanism of injury & course of condition)

____
____
____
____
____
____

Does patient have any of the following *(circle)*: night pain, valsalva, B/B changes, dizziness, fainting, headaches, tinnitus
buckling, catching, locking, joint noise ,other: ______________________

Pyschosocial: Lives with (circle) alone, spouse, friend, sibling, parent(s), ___children.
Lives in (circle) apartment, condo/townhouse, house, other: ______________
___steps going into dwelling, ___steps inside home required to access______________

Prior level of function:______________________

Postural Exam:

Standing: FHP- min/ mod/ sev, C/S Lordosis- ↑ / ↓ / N, T/S Kyphosis- Upper- ↑ / ↓ / N. Mid- ↑ / ↓ / N,
Lower T/S- ↑ / ↓ /N, L/S Lordosis- ↑ / ↓ / N, ______________

Convexities: C/S- L / R, Upper T/S- L / R , Mid T/S- L / R , Lower T/S- L / R , L/S- L / R, No Convexities

Sitting: C/S Lordosis- ↑ / ↓ / N, T/S Kyphosis- Upper- ↑/ ↓ / N. Mid- ↑ / ↓ / N, Lower- ↑/ ↓ /N,
L/S Lordosis- ↑/ ↓ / N, ______________

LUMBAR/ THORACIC ACTIVE ROM

Movement	**ROM**	**PDM**	**ERP**	**Symptoms**
Flexion (90)				
Extension (60)				
SB Right (40)				
SB Left (40)				
Rotate Right (45)				
Rotate Left (45)				

Key: **PDM**= Pain During Motion, **ERP**= **End Range Pain** Other Comments: ______________

Sensory Screen	**Left UE**	**Left LE**	**Right UE**	**Right LE**
Sharp/Dull				
Light Touch				
Deep Pressure				
Proprioception				

Myotome Screen	**L**	**R**
C1,2 Neck Flexors		
C2,3,4 Upper Traps		
C5 Biceps		
C6 Wrist Extensors		
C7 Triceps		
C8 Thumb extensors		
T1 Hand Intrinsics		
L1,2 Iliopsoas		
L3 Quads		
L4 Tib. Anterior		
L5 EHL		
S1 FHL		
S1,2 Gastroc		

Key:
0-No Contraction
1-Trace
2-Poor
3-Fair
4-Good
5-Normal

Reflexes	**L**	**R**
C5,6 Biceps		
C7 Triceps		
C5,6 Brachio.		
L3 Knee Jerk		
S1 Ankle Jerk		

Key:
0-Absent
1+-Decreased
2+-Normal
3+-Increased
4+-Clonus
R-Reinforced

Extremity Clearance:

____
____
____

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NAME: ______________________________

Key: **T**=Tender, **Ti**=Tight, (**+**)=Positive, (**-**)=Negative, ↑=Increase, ↓=Decrease, **P**=Pain, Length resrictions: min/mod/sev

Standing	L	R	Equal
Head Position			
Shoulder Position			
Scapular Position			
Iliac crest			
PSIS			
ASIS			
Greater Trochanter			
Fibular Head			
Calcaneal Position			
Navicular Position			
Stand. Flexion Test			
Marchers Test			
Ant. Ilial Rot. Test			
Hip Drop Test			

Supine	L	R	Equal
TFL Length			
IP Length			
RF Length			
Adductor Length			
ASIS			
FABERE			
HS 90/90			
SLR			
Kernig			
Laseque			
Clonus			
Babinski			
Abdom. MMT			
IP palpation			
LLD ASIS-Lat Mall			
LLD Umb.-Lat Mall			

Hip Screen	L	R	Equal
External Rotation			
Internal Rotation			
Flexion			
Extension			
Abduction			
Distraction			
Hip Scour Test			

Sidelying	L	R	Equal
Ober test			
Glute med. MMT			

Prone	L	R	Equal
PSIS			
Ischial tub.			
Sacraltub. lig			
Coccyx dev			
Med. mall. long			
Piriformis length			
Piriformis palp.			
L/S paraspinals			
Quad. lumborum			
Gastroc length			
RF length			
Trunk ext MMT			
Neck ext MMT			
Glute max. MMT			

Seated	L	R	Equal
Iliac crest			
PSIS			
ASIS			
Pos flexion test			

Neural Tension Tests:

Supine SLR TESTS: ***Left/ Right***

Sciatic: SLR + Hip Add___/___, Hip IR___/___, L/S SB away___/___, C/S Flex___/___

Tibial: SLR= Ank DF/Ever___/___, Toe Ext___/___

Peroneal: SLR + Ank PF/Inv___/___

Sural: SLR + Ank DF/Inv___/___

Sitting Slump Tests: *general*

L/S Flex___/___, C/S Flex___/___, Knee Ext___/___

Ankle Df___/___

Prone/Side Lying Upper Lumbar Tests: Prone Knee Bend=PKB

Femoral: PKB___/___, Hip IR/ER___/___, Hip Abd/Add___/___, Df___/___, PF/Inv___,___

Saphenous: Prone Hip Abd/ Knee Ext___/___, Hip Ext/ER___/___, Ankle DF/Ever___/___

Obturator: Sidelye Hip Abd/Ext___/___

Lat. Fem. Cut.: Sidelye Flex C/S & L/S/ Flex Knee___/___

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NAME: ______________________

Positional Testing:

Segment	Flexion sit	Flexion prone	Neutral sit	Neutral prone	Extension sit	Extension prone
ILA						
SAC BASE						
L5						
L4						
L3						
L2						
L1						
T12						
T11						
T10						
T9						
T8						
T7						
T6						
T5						
T4						
T3						
T2						
T1						

Key: **L**= Rotated Left, **R**= Rotated Right, **E**= Equal
1= Minimal, **2**= Moderate, **3**= Severe

Mobility Testing:

Segment	FB	BB	SBL	SBR	P-A Spring
ILA					
Sac Base					
L5-S1					
L4-L5					
L3-L4					
L2-L3					
L1-L2					
T12-L1					
T11-T12					
T10-T11					
T9-T10					
T8-T9					
T7-T8					
T6-T7					
T5-T6					
T4-T5					

Key: **0**= Anyklosed
1= Moderate restr.
2= Slight restriction
3= Normal
4= Slight Increase
5= Considerable Inc.
6= Unstable

TC= Tender Central
TR= Tender Right TP
TL= Tender Left TP
TB= Tender Bilat. TPs
1= Minimal
2= Moderate
3= Severe

Summary of Positional & Mobility Testing: ______________________

____

____

DERANGEMENT SCREEN

Test Movements	Symptoms Before Testing	Pain During Motion	End Range Pain	Symptoms After Testing
FIS				
Rep FIS				
EIS				
Rep EIS				
SGRIS				
Rep SGRIS				
SGLIS				
Rep SGLIS				
FIL				
Rep FIL				
EIL				
Rep EIL				

Key: ***Pain during motion:*** **P**- no symptoms rest & motion creates pain, ↑ increase existing pain, ↓ dec. existing pain, **A-** abolishes previously existing pain

Symptoms after testing: **W-** worse as a result of motion, **NW-** symptoms are increased with each movement, but do not remained worsened as a result of motion, **B-** better as a result of motion, **NB-** symtoms are decreased with each movement, but do not remain decreased **NE-** movement has no effect on symptoms at all

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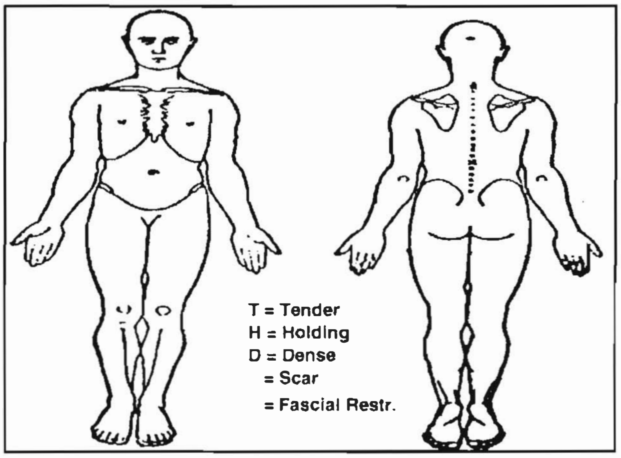
NAME: ______________________

Myofascial Assessment:

C/TJunction______________________
____
T/LJunction______________________
____
L/S Junction ______________________
____
LE______________________
____
UE______________________
____
General______________________
____
____



Courtesy of Dr. Kevin Cerrone, Southside Institute for Physical Therapy, Bay Shore, NY.

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