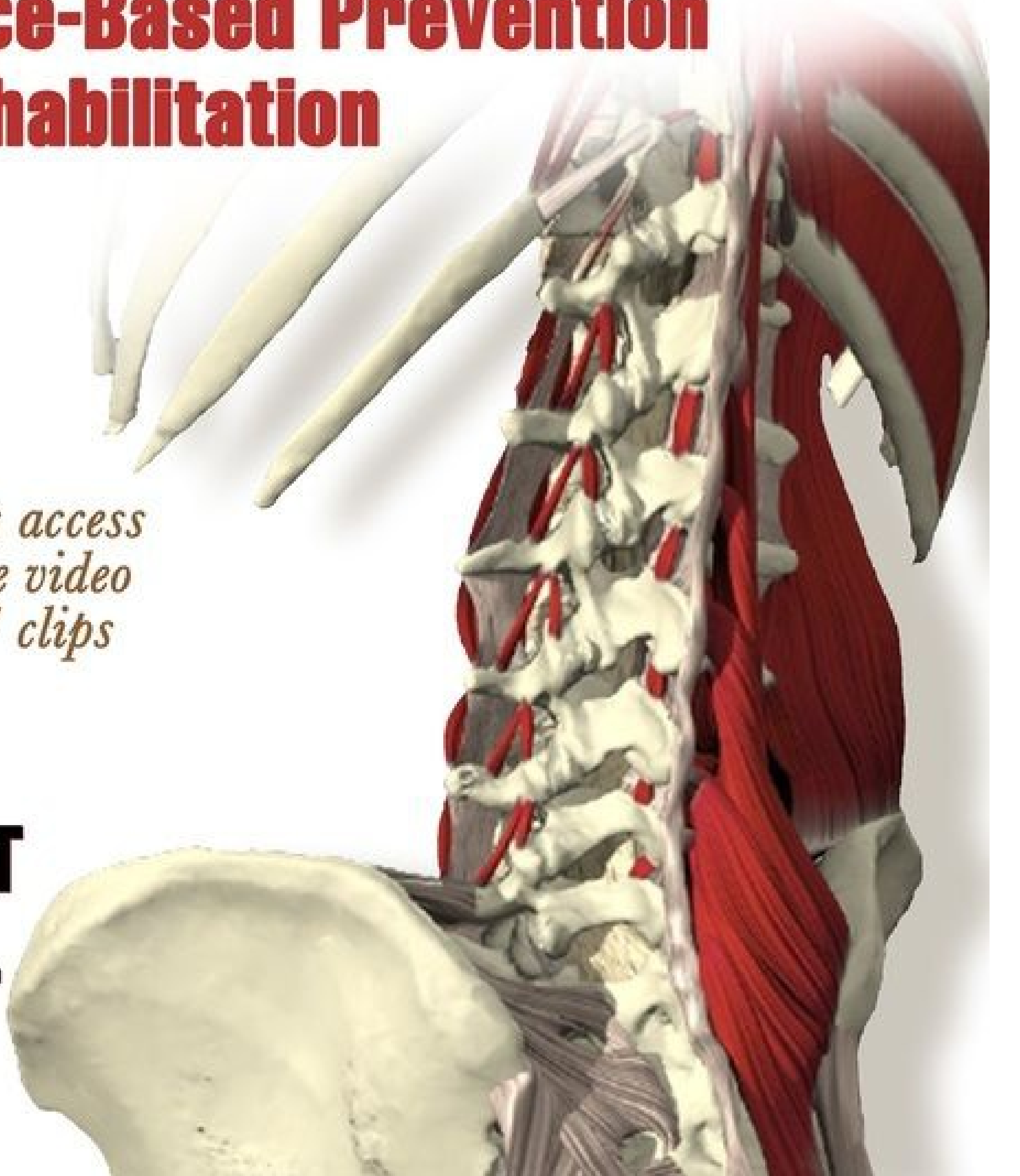
Third Edition

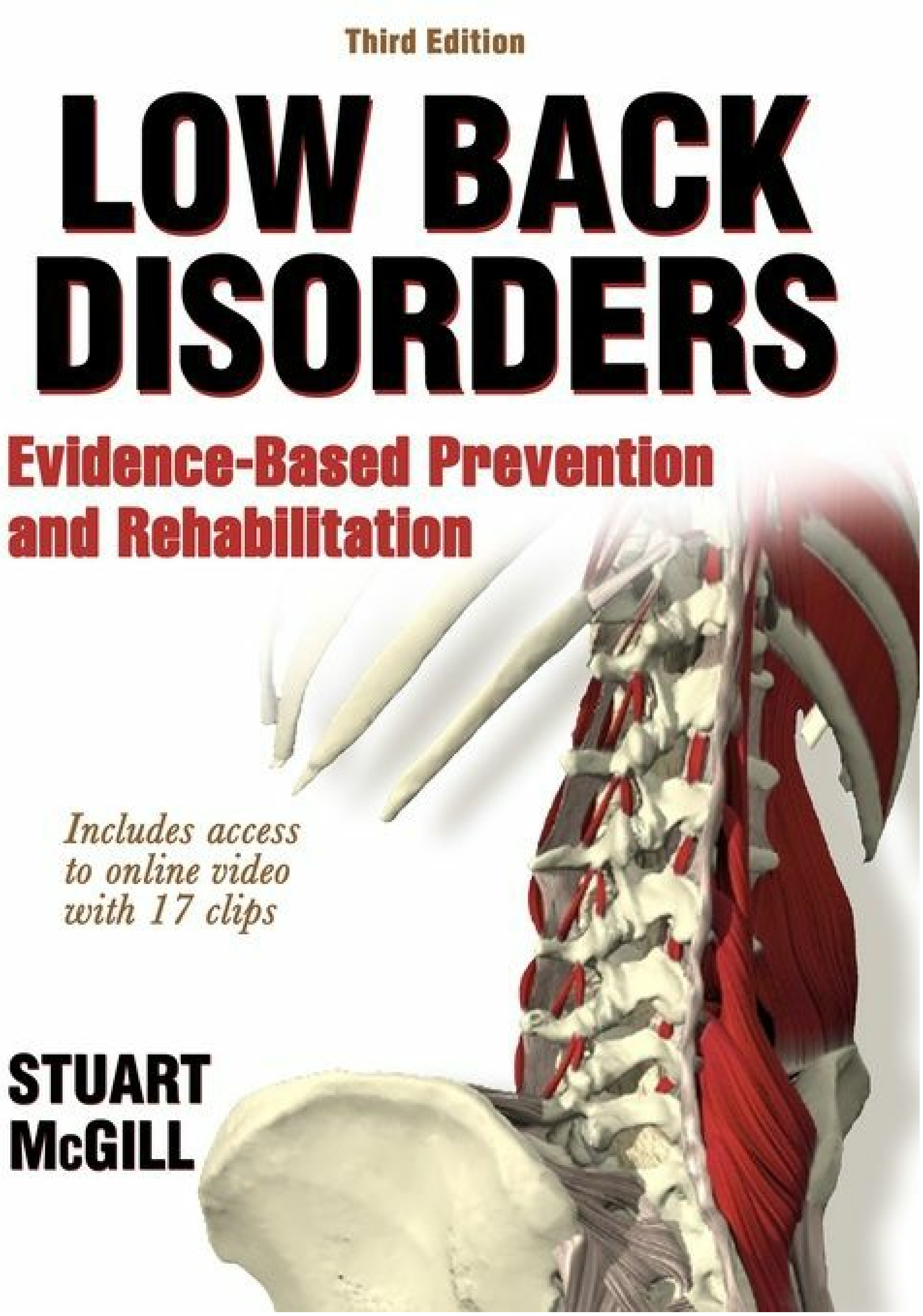
LOW BACK DISORDERS

Evidence-Based Prevention and Rehabilitation



Includes access to online video with 17 clips

STUART McGILL


Third Edition
LOW BACK DISORDERS
Evidence-Based Prevention and Rehabilitation
Includes access to online video with 17 clips
STUART McGILL

Low Back Disorders

Evidence-Based Prevention and Rehabilitation

Third Edition

Stuart McGill, PhD
University of Waterloo
Canada

Human Kinetics

Library of Congress Cataloging-in-Publication Data
McGill, Stuart, 1957- , author.
Low back disorders : evidence-based prevention and rehabilitation / Stuart McGill. -- 3e.
p. ; cm.
Includes bibliographical references and index.
I. Title.
[DNLM: 1. Back Injuries--prevention & control. 2. Low Back Pain--prevention & control. 3. Back Injuries--rehabilitation. 4. Evidence-Based Medicine. 5. Low Back Pain--rehabilitation. WE 755]
RD771.B217
617.5'64052--dc23
2014049488
ISBN: 978-1-4504-7291-3 (print)

Permission notices for material reprinted in this book from other sources can be found on page xvii.
The web addresses cited in this text were current as of April 2015, unless otherwise noted.
Acquisitions Editor: Amy N. Tocco
Developmental Editor: Melissa J. Zavala
Associate Managing Editor: Shevone Myrick
Copyeditor: Patsy Fortney
Indexer: Patsy Fortney
Permissions Manager: Dalene Reeder
Graphic Designer: Joe Buck
Cover Designer: Keith Blomberg
Photograph (cover): © Primal Pictures
Photographer (interior): Neil Bernstein, unless otherwise noted
Photographs (interior): © Human Kinetics, unless otherwise noted
Photo Asset Manager: Laura Fitch
Visual Production Assistant: Joyce Brumfield
Photo Production Manager: Jason Allen
Art Manager: Kelly Hendren
Associate Art Manager: Alan L. Wilborn
Illustrations: © Human Kinetics, unless otherwise noted
Printer: Sheridan Books

Printed in the United States of America
10 9 8 7 6 5 4 3 2 1
The paper in this book is certified under a sustainable forestry program.
Human Kinetics
Website: www.HumanKinetics.com
United States: Human Kinetics
P.O. Box 5076
Champaign, IL 61825-5076
800-747-4457
e-mail: humank@hkusa.com
Canada: Human Kinetics
475 Devonshire Road Unit 100

Windsor, ON N8Y 2L5
800-465-7301 (in Canada only)
e-mail: info@hkcanada.com
Europe: Human Kinetics
107 Bradford Road
Stanningley
Leeds LS28 6AT, United Kingdom
+44 (0) 113 255 5665
e-mail: hk@hkeurope.com
Australia: Human Kinetics
57A Price Avenue
Lower Mitcham, South Australia 5062
08 8372 0999
e-mail: info@hkaustralia.com
New Zealand: Human Kinetics
P.O. Box 80
Mitcham Shopping Centre, South Australia 5062
0800 222 062
e-mail: info@hknewzealand.com
E6220

Check Out the Web Resource!

You will notice a reference throughout this version of Low Back Disorders, Third Edition, to a web resource. This resource is available to supplement your e-book. The web resource includes access to online video with 17 clips. We are certain you will enjoy this unique online learning experience.

Follow these steps to purchase access to the web resource:

1. Visit www.tinyurl.com/LowBackDisordersWR3E.
2. Click the Add to Cart button and complete the purchase process.
3. After you have successfully completed your purchase, visit the book's website at www.HumanKinetics.com/LowBackDisorders**.**
4. Click the third edition link next to the corresponding third edition book cover.
5. Click the Sign In link on the left or top of the page and enter the e-mail address and password that you used during the purchase process. Once you sign in, your online product will appear in the Ancillary Items box. Click on the title of the web resource to access it.
6. Once purchased, a link to your product will permanently appear in the menu on the left. All you need to do to access your web resource on subsequent visits is sign in to www.HumanKinetics.com/LowBackDisorders and follow the link!

Click the Need Help? button on the book's website if you need assistance along the way.

I dedicate this book to my teachers. To my parents, John and Elizabeth, who gave what they could. To the few high school teachers who were able to see past my impatience and occasionally misguided enthusiasm—in particular, Ralph Colucci, who spent countless hours coaching on the track and football fields and teaching life's lessons. He practiced the highest example of daily dedication and the drive to continue after others had given up. My university professors in the tough early years, particularly Dr. Klambauer, who was able to transform mathematics from abstract magic into something very mechanical that I could suddenly feel in my hands. This was a turning point. Professor David Winter, who taught me, among many other things, how to read a scientific paper. Professor Robert Norman, my PhD supervisor and mentor, who is the embodiment of integrity and the master of viewing any issue from the highest of intellectual vantage points. The many great academic personalities in the spine world with whom I have had discussions and debates, and whose writings I have studied, have enhanced my education and perspective—Harry Farfan, Bill Kirkaldy-Willis, Don Chaffin, Bill Marras, Nik Bogduk, Manohar Panjabi, Lance Twomey, and Mike Adams. And the academic clinicians who have stimulated me with their written and spoken thoughts—Vlad Janda, Shirley Sahrmann, Dick Erhart, Paul Hodges, Andry Vleeming, Craig Liebenson, Peter O'Sullivan, Gray Cook, Joachim Wilke, Robin McKenzie, and Clayton Skaggs. My many research colleagues from around the world, and visiting scholars to our lab, too numerous to mention here, who have taught me the many perspectives needed to temper the arrogance that comes so naturally when one is the only person privy to new research results. And to my wonderful graduate students—Jacek Cholewicki, Vanessa Yingling, Jack Callaghan, Sylvain Grenier, Lina Santaguida, Crisanto Sutarno, John Peach, Craig Axler, Lisa Brereton, Greg Lehman, Jennifer Gunning, Richard Preuss, Joan Scannell, David Bereznick, Kim Ross, Kelly Walker, Natasa Kavcic, Simon Wang, John Gray, Steve Brown, Janice Moreside, Sam Howarth, Leigh Marshall, Justin Yates, Stephanie Freeman, Rupesh Patel, Dave Frost, Dianne Ikeda, Christian Balkovec, Natalie Sidorkewicz, Ben Lee, Jordan Cannon, and Drs. Ed Cambridge, Doug Richards, and Claudio Tampier. Our lab technicians—Amy Karpowicz, and Chad Fenwick. The old dog has learned a few of your new tricks. And finally, to the many performance gurus with whom I have had many discussions and training sessions to try experimental approaches—these have assisted me in knowing what important aspects to test. Although it is difficult to pick out a few, I will mention, in particular, the late Mel Siff, Juan Carlos Santana, Al Vermeil, Jerzy Gregorek, Pavel Tsatsouline, Mark McCoy, Art Horne, Dan John, Jon Chaimberg, and Bill Kazmaier—all immensely clever and generous men. For this third edition I also wish to thank many of you who have e-mailed me with your kind and encouraging words. All of you have contributed to my forging. Any remaining personal failings I can only attribute to the unfortunate combination of running Canadian software on Irish hardware.

Finally, to my wife and children, Kathryn, John, and Sarah, who have taught me to see the joy of the moment and enjoy the security of unconditional love. Quoting John Anderson, "I'm just a chunk of coal, but I'm gonna be a diamond some day!"

Contents

Preface

Over the years since the first and second editions hit the bookstores, I have appreciated the testimonials and feedback from patients, clinicians, and educators who have used the book. This has motivated my research team to refocus on expanding our knowledge and best practice in several areas, which we were able to synthesize into an improved edition. Although updates are to be found throughout, major additions were made to several chapters, as noted here.

Ensuring a stable spine was the focus of the first edition. The second edition also addressed dealing with patients who have the opposite difficulty; that is, they have too much stiffness and crush their spines with chronic contraction, and are often locked in problematic postures. Yet others have regional stiffness, and most of the motion occurs at a single segment. We call this the spinal hinge. More often than not, these hinges are the site of pain. New studies have shown that these also predict where future back troubles will occur. Buttressing them with muscular straps or girdles is the key—and this is explained in this third edition.

Patient assessment has been extensively enhanced with more information about provocative testing. This will help you determine the cause of back troubles and, in turn, the best ways to eliminate the causes so that you can make better therapeutic exercise decisions. Our intention was to create a more thorough resource and reference for clinicians and savvy individuals. I have written a book for the lay public with back pain (Back Mechanic: The Step-by-Step McGill Method to Fix Back Pain) that is much more accessible to those unfamiliar with medical jargon. However, because back pain is often not simple to resolve, this resource remains essential.

Another emphasis of the first two editions was finding versions of stabilization exercises that could be conducted pain free. As new findings have emerged in our laboratory, we have been able to incorporate more information about tweaking the exercises to establish pain-free rehabilitation and training. Expansion of the pain-free motion repertoire is another critical component.

Too many patients believe that performing some exercises will take their pain away, and they simply add them to their daily routine. This is a mistake.

The first, and most critical, stage is to remove the cause of pain. This may be a poor choice of movement pattern that overstresses the paining tissue. More emphasis on choosing less stressful ways to move, together with reducing loading in the early stage of rehabilitation is provided in this latest edition. Also, the assessment section has been expanded.

The first edition was printed over a decade ago. Since that time several studies have tested the approaches we described and found them to be successful in reducing and eliminating pain in both individuals and populations such as the military. Some of this supporting evidence has been documented in this edition.

New to this edition is an accompanying web resource available at www.HumanKinetics.com/LowBackDisorders. The web resource includes video clips that showcase 17 exercises and tests explained throughout the book. These videos will help you apply the techniques discussed in this book to your own clients and patients. The web resource also provides blank copies of the handouts appearing at the back of this book. These handouts are presented as fillable PDFs that you may download and customize with instructions unique to patients' needs.

Also new to this edition is an image bank of the art, content photos, and tables used in the book. Instructors may access the image bank at www.HumanKinetics.com/LowBackDisorders and use the files to create customized presentations.

Finally, graduating into performance exercise is outlined in this edition. Although detailed data and guidance are contained in my other book, Ultimate Back Fitness and Performance (www.backfitpro.com), this text explains the critical stages of the performance pyramid:

1. 1. Design appropriate corrective exercise to address the causes of the disorder.
2. 2. Build joint and whole-body stability (and, by default, mobility).
3. 3. Enhance endurance.
4. 4. Train true strength.
5. 5. Transition to ultimate performance with superstiffness techniques, speed, power, and agility.

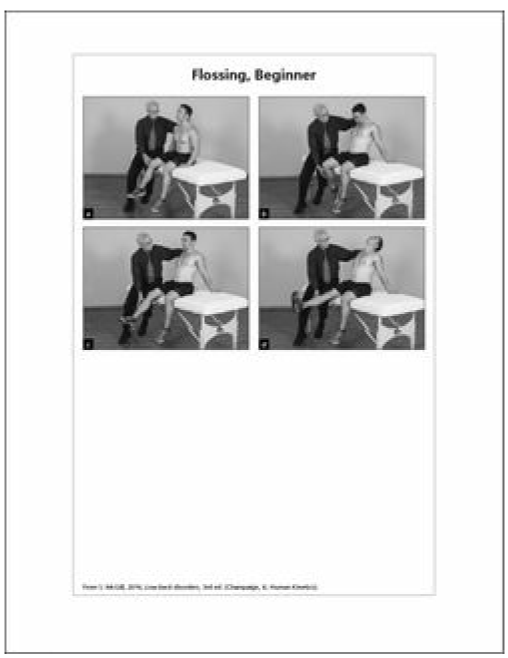
The back is not like any other joint or area of the body. It follows different rules—learn the rules and you will rule the back.

Accessing and Using the Web Resource

The web resource for Low Back Disorders, Third Edition, features 20 handouts in PDF format that clinicians may customize with detailed exercise instructions specific to individual clients. All of these handouts are accompanied by at least one photo showing a key movement of the exercise. The handouts can be printed out and distributed to clients for their use. In addition, the web resource features high-definition video of 17 of the tests and exercises described in the book.

To access the web resource, visit www.HumanKinetics.com/LowBackDisorders. If you purchased a new print book, follow the directions included on the orange-framed page at the front of your book. That page includes access steps and the unique key code that you'll need the first time you visit the Low Back Disorders, Third Edition, website. If you purchased an e-book from HumanKinetics.com, follow the access instructions that were e-mailed to you following your purchase.

To download and print out the handouts, select Web Resource in the ancillary items box in the upper left corner of the screen. You will now be taken to a page with two buttons: One for the handouts and one for the videos. Click the button for the handouts. At the bottom of the screen, select the link to open a handout.



To view the video clips, select Videos button on the Web Resource page. Select the link for the chapter you want. Once you select a chapter link, you'll see a video player. The video numbers along the right side of the player correspond with video number cross-references in the book, and the title under the player corresponds with the exercise title in the book. Scroll through the list of clips until you find the video you want to watch. Select that clip and the full video will play.

Here is a listing of the exercise handouts in the web resource, including an asterisk to denote the exercises that are demonstrated in video:

- Bird dog, remedial*
- Bird dog, beginner*
- Bird dog, intermediate*
- Bird dog, advanced*
- Bird dog, highest level*
- Cat–camel
- Curl-up, beginner*
- Curl-up, intermediate*
- Curl-up, advanced*
- Curl-up, highest level*
- Educating all back extensor muscle motor units
- Flossing, beginner*
- Isometric exercises for the neck
- Potty squat
- Remedial side bridge for deconditioned, chronic patient
- Remedial side bridge for painful shoulders 1
- Remedial side bridge for painful shoulders 2
- Side bridge, beginner
- Side bridge, intermediate

- Side bridge, intermediate variation
- Side bridge, advanced

Acknowledgments

No one will know the effort involved in writing a book until they have done so themselves. Many people have been part of the team. I specifically want to thank and acknowledge my editors at Human Kinetics. Fifteen years ago Loarn Robertson suggested that I write a book, which resulted in the first edition. Elaine Mustain masterfully grasped mechanical and medical concepts and helped guide my writing far beyond what was possible by my English teachers during the rewriting stage of the first and second editions. For this third edition, Melissa Zavala took over the editing duties that improved the extensive additions, while Neil Bernstein made me look good in the photos. We have all become good friends.

Why And How You Should Read This Book

Given the myriad stretch and strengthening programs available for the professional or layperson with a bad back, why write another book offering guidelines for better prevention and rehabilitation of low back troubles? The reason is that very few of the available books and manuals are actually based on scientific studies that investigate the mechanics of back function and the faulty mechanics that lead to pain. This has led to some programs recommending routines that replicate the mechanical causes of the damage! Such routines may actually increase the risk of developing back troubles or exacerbate existing ones. Other, more specialized books promote spine stabilization exercises without quantifying stability, thus basing the recommended exercises on a good guess—albeit a guess based on years of clinical observation. Scientific foundations to justify the recommendations have been thin at best. This book is not about perpetuating clinical myths—it is about challenging them and proposing valid and scientifically justifiable alternatives.

How the Book Is Organized

The intention of this book is to provide the best available scientific evidence to optimize injury prevention and rehabilitation efforts. It is designed to assist those who work to prevent low back troubles and those who are charged with the responsibility of rehabilitating them. Part I begins with a scan of the landscape related to the low back question. Here the main issues are introduced together with a brief introduction to the legislative and institutional constraints—some of which assist efforts to enhance back health, whereas others act as direct barriers. This is followed with evidence as to how the low back structure and associated tissues work during conditions of good health and how they become injured. These chapters describe the following:

- Injury mechanisms involving individual tissues
- Injury mechanisms involving the full lumbar mechanism
- Myths and realities of lumbar stability and mobility

Specific information—such as tissue loads, muscle activation levels, and measures of stability—is provided so that you can meaningfully compare approaches when deciding how to prevent or rehabilitate low back injury. Thus, part I provides the scientific foundation for the rest of the book.

Identifying and implementing the best programs result from considering the evidence. Thus, part II supports evidence-based practice by applying the scientific foundation in part I to the prevention of low back injury. Part III applies this same information to the rehabilitation of low back injury. No rehabilitation exercise program will be successful without removing the cause of the tissue sensitivity and pain.

Focus and Features of the Text

This text will be of value to those involved in designing and delivering back trouble prevention and rehabilitation programs—physical therapists, physiatrists, chiropractors, kinesiologists, ergonomists, exercise therapists, fitness experts, athletic trainers, coaches, and orthopedic specialists.

Low Back Disorders features a number of elements besides the basic text to ensure that all segments of the very diverse readership that deals with low back issues will be able to absorb and use the material. Special elements in some chapters highlight issues of methodological concern or simply provide further elucidation of some concepts or terms that are not common across disciplines. Terms that may be unfamiliar to some readers are defined in a glossary. In addition, a wide variety of occupational and athletic examples throughout the book can be generalized to all activities of daily living.

A major theme of this book is the benefits of blending rehabilitation efforts with ergonomics and prevention. Traditionally, ergonomists and industrial engineers have been responsible for prevention efforts mainly in the workplace, whereas the medical community has been responsible for rehabilitation. This natural division has caused an unfortunate compromise. Rehabilitation efforts will not be effective without preventing the cause of the tissue irritants, and prevention efforts will be retarded without an understanding of the variables obtained from medical investigations into the causes of tissue damage. No clinician will be effective if the cause of the patient's troubles is not removed. The expertise required for optimal prevention and rehabilitation can be found in the fields of ergonomics and rehabilitation medicine, which are naturally symbiotic. Many in the sport and business world have debated whether it is better to develop a stronger offense or defense. The obvious answer is that it is best to have both. I hope that this book will foster a better process resulting in more effective injury prevention and successful rehabilitation.

Some Encouraging Words

Some readers may be tempted to apply the material in parts II and III without digesting the foundation material contained in part I. This is a mistake. People who have attended my courses and were previously familiar with our published scientific papers often state that they were unable to fully synthesize the story and integrate the critical information. Once aware of the full story contained in the course and told in this book, they were much more effective at preventing low back disorders and prescribing exercises that help patients become better rather than worse.

A Note to Lay Readers

Although this book was written for clinicians, many laypeople have read it. As a result, they have written letters expressing their appreciation for their better understanding of the science and their own back troubles, and many have reported substantial improvement. However, we all recognize that having expertise enhances the reading experience of this book (I recently wrote Back Mechanic: The Step-by-Step McGill Method to Fix Back Pain for the lay public). This leads me to add that although successful self-treatment is gratifying, it removes some of the safeguards in the rehabilitation process. If you have a troubled back and are not a clinician, I remind you always to consult a physician at the outset to rule out any condition such as a tumor or a contraindication for exercise.

Part I
Scientific Foundation

The very best professionals with expertise in low back injury prevention and rehabilitation have two things in common: an insight and wisdom developed from experience and a strong scientific foundation to enable evidence-based practice. Each problematic back is part of a whole person, who in turn is the sum of influences that range from those at the cellular level to those at the societal level. Some refer to this as the biopsychosocial approach. No two problematic backs (or patients) are the same, suggesting that optimal intervention would be different for each one. The best approach for optimal rehabilitation and prevention of those factors that exacerbate painful backs results from wise and scientifically based decisions. Part I of this book provides a foundation to enable you to make better decisions in your practice.

Chapter 1
Introduction to the Issues and Scientific Approach Unique to This Book

There is no shortage of manuals and books offering wisdom on low back health. Authors range from those with formal medical or rehabilitation training to laypeople who have found an approach to alleviate their own back troubles and become self-proclaimed low back health prophets. Their intentions are honorable, but their advice is rarely based on a sound scientific foundation. Too many of these authors offer inappropriate recommendations or even harmful suggestions. Years ago, as I began to develop scientific investigations into various aspects of low back problems, I would ask my graduate students to find the scientific foundation for many of the so-called commonsense recommendations I was hearing both in the clinic and in industrial settings. To my surprise they often reported that the literature yielded no, or very thin, evidence (note that I choose my students carefully and that they are very competent and reliable). Examples of such thinly supported "commonsense" recommendations include the following:

- Bend the knees to perform a sit-up.
- Bend the knees and keep the back straight to perform a lift.
- Take yoga and Pilates classes—they are good for the back.
- Reduce the load handled to reduce the risk of back troubles.
- Stretch the hamstrings if you have back pain or leg pain.

In fact, each of these recommendations may be appropriate in some situations but, as will be shown, not in all.

The famous economist John Kenneth Galbraith was well known for demonstrating that actions based on common wisdom, at least in economic terms, were often doomed to fail. He stated that common wisdom is generally neither common nor wise. Galbraith eloquently expressed exactly what I had experienced with "clinical wisdom" pertaining to the low back. Many attempts at preventing low back troubles and rehabilitating symptomatic ones have failed simply because they relied on ill-conceived clinical wisdom. This history of failed attempts is particularly unfortunate because it has lent credence to the assertions of a number of increasingly well-known authorities that low back injury prevention and rehabilitation programs are a waste of resources. These authorities claim that the majority of low back problems are not organic at all—that, for example, most of these difficulties have materialized because workers are paid too much for injury compensation, have been subject to psychosocial influences, or crave sympathy. These dismissals of back injury are not justified. Back injury prevention and rehabilitation programs with strong scientific foundations, executions, and follow-up can be effective.

Having stated this, I must acknowledge that justifying improved practice based on scientific evidence is a dynamic process. With new evidence, the foundation will change. To account for such inevitable shifts, I have developed a balanced approach in these pages, reviewing the assets and liabilities and opposing views of an argument where appropriate. But I offer fair warning! As you read this book, be prepared to challenge current thoughts and rethink currently accepted practices of injury prevention and approaches to rehabilitation.

This chapter addresses some of the debatable issues regarding low back function, together with some opinions that rehabilitation professionals hold about patients, diagnosis, compensation, and disability. It also explores the circumstances that lead to back injury and discusses the need to apply this knowledge to improve low back injury prevention and rehabilitation.

Legislative Landscape: The Unfortunate Adverse Impact on Painful Backs

Although most legislation and legal activity involving back pain is enacted with good intentions, some has been counterproductive. A good example is the issue of spine range of motion (ROM). For years the American Medical Association (AMA) guidelines (1990) for quantifying the degree of back disability were based mostly on loss of spine ROM. Lawyers and compensation boards that needed numbers to define disability and award compensation latched onto spine ROM as an objective and easily measured factor. In the legal arena, therapy was considered successful when the ROM was restored or at least improved. This was changed a few years ago when range of motion was abandoned in favor of diagnosis-based estimates of disability (AMA, 2008). Now, charts link varying inability to perform functions of daily living, medical image findings, and clinically appropriate signs to specify impairment ratings. The contentious issues now include diagnosis of the specific condition, objectively quantifying pain, the existence of a candidate injury mechanism, whether exam findings are clinically relevant, and the ability of a medical image to show pathology. For example, a disc herniation often shrinks and grows depending on the prior loading history. How it appears on the day of the image acquisition may not represent the days in which pain is disabling.

These are scientifically supported and positive advances by the AMA. However, practice is much slower to change, and range of motion remains an emphasis in many clinics. After back injury, many people do not do well with an emphasis on enhancing spine mobility. In some cases, back problems are actually exacerbated by this approach. In fact, evidence shows that many back injuries improve with stabilizing approaches—motor control training, enhancement of muscle endurance, and training with the spine in a neutral position (Saal and Saal, 1989, may be considered the classic work). Our work showed that three-dimensional low back ROM has no correlation to functional test scores or even the ability to perform occupational work (Parks et al., 2003). Most recent work following soldiers in the Finnish military (Suni et al., 2013) showed that conscious training to reduce their lumbar range of motion during the conduct of duties reduced back pain over time. They used the principles in this book to reduce back pain incidence in a large population. In the best practice, spine flexibility may not be emphasized until the very late stages of rehabilitation, if ever.

The idea of flexibility as the best measure of successful rehabilitation became entrenched for reasons of legal convenience rather than as a result of a positive impact on low back pain. It will take time for practice to fully recognize the importance of distinguishing between variables important for rating disability and those important for reducing pain and restoring ability. Another example illustrates the perverse impact of well-intended equity legislation. We all have equal rights under the law, but we are not physical equals. Although individual variance is present in every group, different populations within society demonstrate quite different capabilities. For example, the data of Jager and colleagues (1991), compiled from many studies, clearly showed that young men can tolerate more compressive load down their lumbar spines than can older men, and similarly, that men can tolerate about an additional third more load than can women when matched for age. Yet human rights legislation, which is designed to create fairness and equity by discouraging distinctions among groups, actually puts older females at greater risk than younger men. By not allowing a 64-year-old osteoporotic woman to be treated (and protected) differently from a 20-year-old, fit, 200 lb (90 kg) male in terms of tolerating spine load, the legislation presents a major barrier for intelligently implementing tolerance-based guidelines for protecting workers.

Deficiencies in Current Diagnostic Practices

It is currently popular for many authorities to suggest that back trouble is not a medical condition. They assert that physical loading has little to do with low back injury compensation claims; rather, they believe workers complain of back problems to benefit from overly generous compensation packages or to convince physicians that they are sick. According to this view, any biomechanically based injury prevention or rehabilitation program is useless. Variables within the psychosocial sphere dominate any biological or mechanical variable. If this is true, then this book is of no value—it should be about psychosocial intervention.

Those who contend that psychosocial factors dominate low back issues are well-published scientists and physicians. For example, Professor Richard Deyo (1998) summarized a common view: “Consider the following paradox. The American economy is increasingly postindustrial, with less heavy labor, more automation and more robotics, and medicine has consistently improved diagnostic imaging of the spine and developed new forms of surgical and nonsurgical therapy. But work disability caused by back pain has steadily risen.” This line of reasoning assumes that modern work (i.e., more repetitive, more sedentary) is healthier for the back than the predominantly physical labor of past generations. The evidence suggests, however, that the repetitive motions required by some specialized modern work, or the sedentary nature characterizing others, produces damaging biomechanical stressors. In fact, the variety of work performed by our great-grandparents may have been far healthier than our own. Deyo also seems to assume that nonsurgical therapy has been appropriately chosen for each person, whereas I suggest that inappropriate therapy prescriptions remain quite common.

Furthermore, although I agree that there is more reliance on medical imaging, I suspect that this reliance has resulted in a loss in the mechanically based diagnostic skills that are a crucial factor in accurate diagnosis. Interestingly, magnetic resonance imaging (MRI) has been documented to find—among other features—disc bulges, trophic facet joints, and “degenerative disc disease,” yet these have little relationship to whether the person has pain. The static images are commonly acquired when the person is in an unloaded, recumbent posture. Dynamic images (multiple X-ray, fluoroscopy, dynamic MRI) show much more movement pathology where the dynamics can be directly related to pain. Discogenic pain, for example, has been shown to follow a natural history in which the zenith of pain is associated with the unstable phase, but the condition eventually ends with a very desiccated disc on a medical image and pain that has subsequently burned out. Then, abundant evidence shows, the history continues as changes in joint function influence the mechanical loading of the facet joints. Eventually, the facet joints become arthritic, shifting the pain source from disc to facet. Central sensitization further influences the process and links among pain, mechanical and functional factors, and patient corrections to wind down the heightened neural response. No wonder Savage, Whitehouse, and Roberts (1997) found little correlation between the image and the patient's symptoms.

Too often, the MRI impression or diagnosis is not correctly linked to the pain mechanism, and patients receive the incorrect treatment (Wassenaar et al., 2012), including surgery. Gibson, Martin, and Terry (1980) presented evidence regarding plain film radiographs; and McGill and Yingling (1999) and Zhao and colleagues (2005) discussed why such images are compromised in showing actual damage. Quite simply, bone displacement is needed for a radiograph to reveal damage. Yet we routinely observe quite substantial fractures that are held together by periosteum, which completely masks the damage.

Without question, the images are of great value for the surgeon who must "cut out the pain," but only if the image finding corroborates the specific clinical symptoms of the patient. However, Chou and colleagues rightly pointed out that the images do not improve outcomes except in patients with severe neurological deficits (Chou, Deyo, and Jarvik, 2012). We have found that a thorough assessment reveals the cause of pain better than any image. The assessment is explained in chapter 9.

Professor Alf Nachemson (1992) wrote that "most case control studies of cross-sectional design that have addressed the mechanical and psychosocial factors influencing LBP (low back pain), including job satisfaction, have concluded that the latter play a more important role than the extensively studied mechanical factors." Yet none of the several references cited to support this opinion made reasonable quantification of the physical job demands. Generally, these studies showed that psychosocial variables were related to low back troubles, but in the absence of measuring mechanical loading, they had no chance to evaluate a loading relationship.

Finally, Dr. Nortin Hadler (2001) has been rather outspoken, stating, for example, that “it is unclear whether there is any meaningful association between task content and disabling regional musculoskeletal disorders for a wide range of physical disorders” and that “on the other hand, nearly all multivariate cross sectional and longitudinal studies designed to probe for associations beyond the physical demand of tasks, detect associations with the psychosocial context of working.” Also, "the backache can be disabling nonetheless—not because of what is lifted, but whether or when it is to be lifted. Too often a salutary outcome proves elusive" (Hadler et al., 2007).

Recent evidence clearly shows that, although psychosocial factors can be important in modulating patient behavior, biomechanical components are important in the etiology of low back disorders and in their prevention. The position that biomechanics plays no role in back health and activity tolerance can be held only by those who have never performed physical labor and have not experienced firsthand the work methods that must be employed to avoid disabling injury. Although the scientific evidence is absolutely necessary, it will only confirm the obvious to those who have this experience. I find it perversely satisfying when physicians tell me that they are now, after missing work as a result of a nasty back episode related to physical work, able to relate to their patients. Perhaps experience with a variety of heavy work and with disabling pain should be required for some medics!

The implication of this literature is that the links between pain and biomechanical factors are variables over the natural history of back pain. Ignoring integrated natural history concepts obscures a mature interpretation of what the evidence and a thorough assessment really show: that the links among pain, biomechanics, and images are complex and patient specific, and that rigor is required to understand them.

It is, then, essential to investigate and understand the links among loading, tissue damage or irritation, psychosocial factors, and performance to provide clues for the design and implementation of better prevention and rehabilitation strategies for low back troubles. Founded on this rubric, chapters 8 and 9 strengthen the case for performing provocative testing to discover the cause of a patient’s pain and provide an algorithm to guide that approach. The following sections address several commonly held beliefs about back injury.

Are 85% of Back Troubles of Unknown Etiology?

Low back injury reports often mention the statistic that 85% of low back troubles are of unknown etiology. This has led to the popular belief that disabling back troubles are inevitable and just happen, a statement that defies the plethora of literature linking specific mechanical scenarios to specific tissue damage. Some have argued that this statement is simply the product of poor diagnosis—or of clinicians reaching the limit of their expertise (e.g., Finch, 1999). In fairness I must point out that diagnosis often depends on the profession of the diagnostician. Each group attempts to identify the primary dysfunction according to its particular type of treatment. For example, a physical therapist will diagnose based on manual therapy approaches, whereas a surgeon may find a diagnosis directed toward making surgical decisions more helpful. Some clinicians (surgeons, for example) seek a specific tissue as a pain candidate. From this perspective, nerve block procedures have shown conclusive pain source diagnoses in well over 50% of cases (e.g., Bogduk et al., 1996; Finch, 1999; Lord et al., 1996). This has prompted research into which tissues are innervated and are candidates as pain generators. Biomechanists often argue that this may be irrelevant because a spine with altered biomechanics has altered tissue stresses. Thus, a damaged tissue may cause overload on another tissue, causing pain whether the damaged tissue is innervated or not.

This is why other clinicians employ skilled provocative mechanical loading of specific tissues to reveal those that hurt, or at least to reveal loading patterns or motion patterns that cause pain. These types of functional diagnoses are helpful in designing therapy and in developing less painful motion patterns, but the process of functional diagnoses will be hindered by a poor understanding of spine biomechanics. Furthermore, those with a thorough understanding of the biomechanics of tissue damage can be guided to a general diagnosis by reconstructing the instigating mechanical scenario. An additional benefit of this approach is that, once the cause is understood, it can be removed or reduced. Unfortunately, many patients continue to have troubles simply because they continue to engage in the mechanical cause. Familiarity with spine mechanics will dispel this myth of undiagnosable back trouble and reduce the percentage of those with back troubles of no known cause.

Even with a tissue-based diagnosis, the practice of treating all patients who have a specific diagnosis with a singular therapy has not proven productive (Rose, 1989). For example, success rates with many cancer therapies greatly improved with the combination of chemotherapy and radiotherapy. Optimal back rehabilitation requires the removal of the cause and the addition perhaps of stability, manual soft tissue therapy, or something else depending on the patient. Few patients fall into a complete fit for functional diagnosis in which a singular approach will yield optimal results. Both for interpretation of the literature and for clinical decision making, it would appear prudent to question the diagnostic criteria needed before assigning a diagnosis.

Limitations in tissue-based diagnosis should not be used to suggest that determining the cause of back troubles is irrelevant or that the manual or medical treatment in some cases is fruitless, leaving psychosocial approaches to prevail by default. As presented later in this book, identifying the motions, postures, and loads that cause pain, or relieve pain, results in a precise diagnosis. The levels of pain triggers are determined. Understanding spine function enables links between pain patterns and causation to be established. This diagnostic approach is productive for guiding prevention and rehabilitation approaches. But what is meant by this approach? Read on!

Diagnosis by Hypothesis Testing

The opinion that precise diagnosis for back pain is not possible is now old-fashioned. Geoff Maitland (1987), the Australian physiotherapist, years ago promoted the concept of examining the patient and forming a working hypothesis. The hypothesis was used to guide treatment and project the prognosis. The hypothesis was then tested and refined as rehabilitation progressed. Our approach, which incorporates a strong biomechanical foundation and blends expertise from various biomedical and psychosocial disciplines, is strongly aligned with Maitland's proposition. An initial impression is formed from the first meeting of patients in the waiting room—from observing their sitting posture, how they rise from the chair, their initial gait pattern, and so on. Then a history is taken to look for possible candidate injury mechanisms as well as perceived pain exacerbators and relievers. Observation continues during some basic motion patterns as the evaluation process proceeds, delving further into the mechanics and nature of the symptoms. Then provocative tests are performed to either strengthen or weaken the hypothesis. Motion and motor patterns that are tolerated are identified. All information is used to formulate the plan for corrective exercise and the starting dosage of tolerable therapeutic exercise. The progression concludes with functional screens and tests that are chosen based on information obtained in the preceding process. In this way a functional diagnosis is ensured that is sufficient for considering exercise choice and rates of subsequent progression. In some cases the tissue causing the pain is clearly identified. In others the cause is precisely identified in terms of specific motions, postures, and loads together with symptoms of abnormal motion and motor patterns. The course of prevention and rehabilitation now has some quantitative guiding parameters. The patient and clinician are able to eliminate the variables that cause pain and build progressions that avoid pain.

Are Most Chronic Back Complaints Rooted in Psychological Factors?

Although there is no doubt that many chronic back cases have psychological overlays, the significance of psychology for back problems is often greatly exaggerated. Dr. Ellen Thompson (1997) coined the phrase bankrupt expertise when referring to spine docs who are unable to guide improvement in their patients and default to blaming the patients and their psychoses. These physicians either dismiss mechanical causation or assume that mechanical causation has been adequately addressed. These are the ones who will state that the pain is in the patient's head or that the patient is noncompliant with the ill-prescribed therapy.

At our university clinic I see patients who have been referred by physicians for consult. These are either elite performers or those with very difficult chronic bad backs who have failed with all other approaches. In spite of the fact that these people have received very thorough attention, I am continually heartbroken to hear about the minimal notice paid to ongoing back stressors and about the exercises that these “basket case backs” have been prescribed that have only exacerbated their conditions. The day before I wrote this section, I saw a classic example.

A woman had suffered for 5 years on disability and had seen no fewer than 12 specialists from a variety of disciplines. Although several had acknowledged that she had physical concerns, her troubles were largely attributed to mental depression. She consistently reported being unable to tolerate specific activities while being able to tolerate others. Some provocative testing confirmed her report and uncovered a previously undiagnosed arthritic hip. For years she had been faithfully following the instructions of her health care providers to perform pelvic tilts, knees-to-chest stretches first thing in the morning, and sit-ups; to take her large dog for walks; and so on. All of these ill-chosen suggestions had prevented her posterior disc (with sciatica)–based troubles from improving. As we will see later, these types of troubles typically do not recover with flexion-based approaches—particularly first thing in the morning. Moreover, the lead-imposed torsional loads that she experienced every time she walked her dog exceeded her tolerance. Although she reported vacuuming as a major exacerbator of her troubles, her health care providers had never shown her how to vacuum her home in a way to spare her back. I suggested that removing these daily activities and replacing the flexion stretches with neutral spine position awareness training and isometric torso challenges would likely start a slow, progressive recovery pattern. I believed that her psychological concerns would probably disappear with her back symptoms if she fell into the typical pattern. This patient, with this typical story, has a reasonably good chance to enjoy life once again. (Note: This patient was back to work and off her antidepressant medication at the time of proofing this manuscript.)

None of the experts this woman had seen—including physical therapists, chiropractors, psychologists, physiatrists, neurologists, and orthopedic surgeons—addressed mechanical concerns. This is not to condemn these professions, but rather to suggest that sharing experiences and approaches will help us to be more successful in helping people with bad backs. Perhaps these professionals were unaware of the principles of spine function, the types of loads that are imposed on the spine tissues during certain activities, and how these activities and spine postures can be changed to greatly reduce the loads—in other words, the biomechanical components. The links between the physical and the psychological variables are becoming better understood. Virtually all of these are well addressed with mechanical approaches. For example, reduction in pain-catastrophizing behavior reduces just as effectively with mechanical approaches as it does with the usual cognitive behavioral therapy approaches (Smeets et al., 2006).

This book is an attempt to heighten the awareness and potential of this mechanical approach. Although it sounds very harsh, I have found relatively few experts who appear willing to adequately address the causes of back troubles while working to find the most appropriate therapy. My years of laboratory-based work, combined with collaboration with my clinical colleagues, have provided me with unique insight. As a result, I am not so quick to blame the chronic patient.

Does Pain Cause Activity Intolerance?

Evidence that mechanical tissue overload causes damage is conclusive. But does the damage cause pain, and does the chronic pain cause work intolerance? Although this seems intuitive, the research needed to confirm these links is difficult to perform. Several, but limited numbers of, studies have documented the mechanical or chemical stimulation of tissues to reproduce clinical pain patterns. (The absence of definitive, large-scale studies is due to the ethical issues of performing invasive procedures and probably not to lack of scientific merit in such studies.) For example, the pioneering work of Hirsch and colleagues (1963-64) documented pain from the injection of hypertonic saline into specific spine tissues thought to be candidates for damage. Subsequent to this work, several other studies suggested the link between mechanical stimulation and pain—for example, the work of Hsu and colleagues (1988) documenting pain in damaged discs. Ortiz and Bordia (2011) were able to document the links among loading, damage, and resulting pain in those who develop osteoporotic vertebral compression fractures. A preexisting degenerated disc disrupts the normal axial transfer of load, thus acting as the injury vector resulting in compromised mechanics and adjacent compression fractures, and pain.

There is irrefutable evidence that vertebral disc end-plate fractures are very common and result only from mechanical overload (Brinckmann, Biggemann, and Hilweg, 1989; Gunning, Callaghan, and McGill, 2001). That these fractures are also found in necropsy specimens that were subjected to whiplash (Taylor, Twomey, and Corker, 1990) also strengthens another facet of this relationship. Hsu performed discograms (injections of radio contrast) into 692 discs, of which 14 demonstrated leakage into the vertebral body, confirming an end-plate fracture. Four of these discs (28%) produced severe pain, nine (64%) produced fully concordant pain, and one produced mildly discordant pain. In contrast, only 11% of the remaining 678 discs with no end-plate disruption produced severe pain, 31% produced concordant pain, 17% produced mild pain, and 41% produced no pain. This evidence provides strong support for the notion that loading causes damage and damage causes pain.

Even though pain can limit function and activity in other areas of the body, some still suggest that these are not linked when a bad back is at issue. Teasell (1997) provided an interesting perspective when he argued that in sports medicine, as opposed to occupational medicine, it is well accepted that some injuries require months of therapy or can even cause retirement from the activity. He noted that athletes receiving specialized sports medicine care are an interesting group to consider because many are highly motivated, are in top physical condition, are well paid, have access to good medical care, and are fully compensated even while injured. Their injuries and pain can cause absence from play for substantial amounts of time and can even end their lucrative careers. Teasell reminded us that not all long-term chronic pain is an entirely psychosocial concern, as implied by some clinicians. These clinicians' dismissal of the usefulness of physical approaches simply because they have not been successful in reducing long-term troubles is a disservice to the patient.

Inadequacies in Current Care and Prevention

Many back patients can testify that the care they have received for their troubles is not satisfactory. Conversations with family doctors have left me with the general impression that they do not know what to do with the back pain case except to provide analgesics and perhaps a referral to a surgeon or therapist. They have neither the time nor the expertise to provide specific help matched to the patient. Suggesting that they engage in a nonspecific activity such as yoga may be just as helpful as harmful—the net gain is zero. Carey and colleagues (2009) documented practice patterns in 5,357 households in North Carolina. Of the 732 adults with chronic LBP, 60% had used narcotics in the previous month and over one-third had advanced imaging in the previous year, yet only 3% had engaged in formal spine rehabilitation. The discrepancy between the overemphasis on expensive treatments and the underutilization of pain-reducing movement and exercise is clear.

What are some of the factors that contribute to the inadequacy of patient experience? Certainly one is the fact that the epidemiological evidence on which many professionals base their treatment recommendations can be quite confusing. Following are some examples of the issues that cause confusion.

- Plethora of studies on “backache.” Nonspecific backache is nearly impossible to quantify and, even if it could be quantified, offers no guidance for intervention. As such, any study of treatment interventions on nonspecific backache is of little use. Some people suffer from discogenic problems, for example, and will respond quite differently from those with ligamentous damage or facet-based problems. Efficacy studies that do not subclassify bad backs end up with nonspecific average responses. This has led to the belief that nothing works—or that everything does, but to a limited degree. More studies on nonspecific backache treatment will not be helpful, nor will the large epidemiological reviews of these studies offer real insight. In contrast, patients with treatments matched to their conditions experience greater short- and long-term reductions in disability than those receiving unmatched treatments (Brennan et al., 2006; Fritz, Cleland, and Childs, 2007).

- U-shaped function of loading and resulting injury risk. Like many health-related phenomena, the relationship of low back tissue loading to injury risk appears to form a U-shaped function—not a monotonically rising line. For example, virtually every nutrient will cause poisoning with excessive dosage levels, but health suffers in their absence; thus, there is a moderate optimum. In the case of low back loading, evidence suggests that two regions in the U-shaped relationship are problematic—too much and too little. Porter (1987) suggested that heavy work is good for the back—but how does one define heavy? Porter was probably referring to work of sufficient challenge and variability to reach the bottom of the U and hence lower symptoms. From a biological perspective, sufficient loading is necessary to cause strengthening and toughening of tissues, but excessive levels will result in weakening. In other words, it is not a matter of doing exercise or not, or engaging in activity or not. Heneweer, Vanhees, and Picavet (2009) expressed the need to match the appropriate activity and its level for seeking the optimal point on the U-shaped relation for back pain.
- Relationship of intensity, duration of loading, and rest periods. As Ferguson and Marras (1997) pointed out, some studies suggest that a certain type of loading is not related to pain, injury, or disability, whereas others suggest it is, depending on how the exposure was measured and where the moderate optimum for tissue health resides for the experimental population. The subjectivity of such studies is further underscored when we consider the question of whether there is a clinical difference between tissue irritation and tissue damage. Loading experiments on human and animal tissues to produce damage reveal the ultimate tolerable load beyond which injuries cause biomechanical changes, pain, and gross failure to structures. In real life, any of us could irritate tissues to produce tremendous pain at loading levels well below the cadaver determined tolerance by repeated and prolonged loading. In fact, evidence presented by Videman and colleagues (1995) and Niemelainen and colleagues (2008) suggests that the progressive development of conditions such as spinal stenosis results from years of specific subfailure activity. The fundamental question is Could such conditions be avoided by evidence-based prevention strategies that include optimal loading, rest periods, and controlling the duration of exposure?

Ill-Advised Rehabilitation Recommendations

These failures to appropriately frame research and its results have resulted in many oversimplifications about low back treatment, which have in turn led to some inadequate treatment practices and recommendations. A few of the most common recommendations for back health are discussed here.

- Strengthen muscles in the torso to protect the back. Despite the clinical emphasis on increasing back muscle strength, several studies have shown that muscle strength cannot predict who will have back troubles (Biering-Sorenson, 1984). On the other hand, Luoto and colleagues (1995) and Stroyer and Jensen (2008) have shown that muscular endurance (as opposed to strength) is protective. Why, then, do many therapeutic programs continue to emphasize strength and neglect endurance? Perhaps it is a holdover influence from the athletic world, in which the goal of training is to enhance performance. Perhaps it is an influence from the pervasive use of bodybuilding approaches in rehabilitation. As will be shown, optimal exercise therapy occurs when the emphasis shifts away from the enhancement of performance and toward the establishment of improved health. In many cases the two are mutually exclusive!
- Bend the knees when performing sit-ups. Clinicians widely recommend bending the knees during a sit-up, but on what evidence? A frustrating literature search suggests that this perception may be the result of so-called clinical wisdom. Interestingly, Axler and McGill (1997) demonstrated that there is little advantage to one knee position over the other, and in fact the issue is probably moot because there are far better ways to challenge the abdominal musculature and impose lower lumbar spine loads (traditional sit-ups cause spine loading conditions that greatly elevate the risk of injury). This issue is one of many that will be challenged in this book.
- Performing sit-ups will increase back health. Is this a true statement or an artifact of experimental methodology? Despite what many would like to believe, there is only mild literature support for the belief that people who are fit have less back trouble (although positive evidence is increasing—for example, Stevenson et al., 2001). Interestingly, many of the studies attempting to evaluate the role of increased fitness in back

health actually included exercises that have been known to cause back troubles in many people. For example, many have attempted to enhance abdominal strength with sit-ups. After examining the lumbar compression that results from performing sit-ups with full flexion of the lumbar spine, together with excessive disc annulus stresses, it is clear that enough sit-ups will cause damage in most people. Each sit-up produces low back compression levels close to the U.S. National Institute for Occupational Safety and Health (NIOSH) action limit, and repeatedly compressing the spine to levels higher than the NIOSH action limit has been shown to increase the risk of back disorders (Axler and McGill, 1997). Thus, reaching a conclusion about the role of fitness from the published literature has been obscured by ill-chosen exercises. Increased fitness does have support, but the way fitness is increased appears to be critical.

- To avoid back injury when lifting, bend the knees, not the back. Probably the most common advice given by the clinician to the patient who must lift is to bend the knees and keep the back straight. In addition, this forms the foundation for virtually every set of ergonomic guidelines provided to reduce the risk of work-related injury. Very few jobs can be performed this way. Further, despite the research that has compared stooping and squatting styles of lifting, no conclusion as to which is better has been reached. The issue of whether to stoop or squat during a lift depends on the dimensions and properties of the load, the characteristics of the lifter, the number of times the lift is to be repeated, and so forth, and there may in fact be safer techniques altogether. Much more justifiable guidelines are provided later in this text.
- Tight hamstrings and unequal leg length lead to back troubles. It would seem intuitive that shortened, or tight, hamstrings would apply deleterious torque to the pelvis and lead to back troubles. A similar argument could be mounted for unequal leg length, which would tilt the pelvis and impose bending stresses on the lumbar spine. This line of reasoning appears to have driven popular clinical practice. Interestingly, there is little support for these notions. A longitudinal study of young men throughout their military service did not reveal any link between current back pain and hip flexion restrictions (interpreted as hamstring tightness) (Hellsing, 1988). Neither could pain be predicted in this study or in the well-conducted studies of Biering-Sorenson (1984) and Van

Nieuwenhuyse and colleagues (2009) on the Belgian Low Back Cohort. Developing pain while standing was not linked to hamstring extensibility (Raftry and Marshall, 2012). An interesting study by Ashmen, Swanik, and Lephart (1996) suggested that although reduced hip flexion may not be associated with back pain, asymmetry between sides may be. Interestingly, many high-performance athletes who run and jump have tight hamstrings that they use as springs. Note that hip joint restriction is a different issue; evidence from several perspectives links hip pathology with back symptoms. For example, Cibulka and colleagues 1998 noted the association between internal/external rotation and sacroiliac dysfunction and back pain. Our work has surveyed restricted hip rotation in workers with demanding jobs and chronic recurrent back pain (McGill et al., 2003). Unequal leg length has been shown to have a link with back pain in only the most extreme of length discrepancies; even those with a 2 in. (5 cm) difference rarely develop chronic pain (Grundy and Roberts, 1984). Further, a significant link between leg length inequality and lumbar scoliosis does not seem to exist, at least for inequalities of 0.4 in. (1 cm) or less (Hoikka, Ylikoski, and Tallroth, 1989). All of this suggests caution when assessing patients and attributing symptoms. Perform the provocative tests and discover whether these postural variables are true exacerbators in the individual and thus are justifiable targets for therapy.

- A single exercise or back stability program is adequate for all cases. It is currently popular to promote the training of single muscles to enhance spine stability. Although the original research was motivated by the intention to reeducate perturbed motor patterns that were documented to be the result of injury, others have misinterpreted the data and are promoting exercises to train muscles they believe are the most important stabilizers of the spine. Unfortunately, they did not quantify stability. The process of quantifying the contribution of the anatomical components to stability reveals that virtually all muscles can be important, but their importance continually changes with the demands of the activity and task. It is true that damage to any of the spinal tissues from mechanical overload results in unstable joint behavior. Because of biomechanical changes to the joint, however, the perturbed tissue is rarely linked to the symptomatology in a simple way. More likely, other tissues become involved, and which ones are involved will result in different accompanying motor disturbances. This variety in possible etiologies means that a single, simple rehabilitation approach often will not work. Is mobility to be restored at the expense of normal joint stability? Or is stability to be established first, with enhanced mobility as a secondary delayed rehabilitation goal? Or is the clinical picture complex—for example, when spine stability is needed but tonic psoas activity causing chronic hip flexure necessitates hip mobilization? This example, one of many that could have been chosen, illustrates the challenge of ensuring sufficient stability for the spinal tissues. No simple, or single, approach will produce the best results in all cases. The description and data presented in this book will help guide the formulation of exercises that ensure spine stability.

Can Back Rehabilitation Be Completed in 6 to 12 Weeks?

Some have suggested that damaged tissues should heal within 6 to 12 weeks. In fact, many have used this argument to support the notion that work intolerance exceeding this period has no pathoanatomical basis (e.g., Fordyce, 1995) but stems from psychosocial issues. Further, some have suggested that patient recovery would be better served by redirecting rehabilitative efforts away from physical approaches. This position can be refuted by data and indicates a misunderstanding of the complexities of spine pathomechanics. The concept that tissues heal within 6 to 12 weeks appears to be originally based on animal studies (reviewed in Spitzer, 1993), the majority of which were of rodent muscle. However, not all human patients get better so quickly (Mendelson, 1982), and the follow-up studies from some defined disorders such as whiplash are compelling in the support of lingering tissue disruption (e.g., Radanov et al., 1994; Curatolo et al., 2011). A systematic review by Itz and colleagues (2012) challenged the claim that most first-time back pain episodes resolve in the first 6 weeks. They showed that spontaneous recovery is not true because 65% of these people reported back pain after 1 year.

Evidence is presented in later chapters of both mechanical and neurological changes that linger for years subsequent to injury. This includes loss of various motor control parameters together with documented asymmetrical muscle atrophy and other disorders. This suggests that postinjury changes are not a simple matter of gross damage healing. Following are only a few of the types of damage that can be long-term indeed:

- Specific tissues such as ligaments, for example, have been shown to take years to recover from relatively minor insult (Woo, Gomez, and Akeson, 1985).
- The intervertebral motion units form a complex mechanism involving an intricate interplay among the parts such that damage to one part changes the biomechanics and loading on another part. From the perspective of pathomechanics, many reports have documented the cascade of biomechanical change associated with initial disc damage (Galbusera et al., 2011) and subsequent joint instability and secondary arthritis, which may take years to progress (e.g., Brinckmann, 1985; Kirkaldy-Willis, 1998).
- Videman and colleagues (1995) and Niemelainen and colleagues (2008) documented that vertebral osteophytes were most highly associated with end-plate irregularities and disc bulging. Osteophytes are generally accepted to be secondary to disc and end-plate trauma but take years to develop.

Thus, to suggest that back troubles are not mechanically based if they linger longer than a few months only demonstrates limited expertise.

Another question is Can these back troubles linger for a lifetime? In this connection, it is interesting that elderly people appear to complain about bad backs less than younger people do. Valkenburg and Haanen (1982) showed that back troubles are more frequent during the younger years. Weber (1983) provided further insight by reporting on patients 10 years after disc herniations (some of them had had surgery, whereas others had not) who were engaged in strenuous daily activity—yet all were still receiving total disability benefits! It would appear that the cascade of changes resulting from some forms of tissue damage can take years, but generally not longer than 10 years. Although the bad news is that the affected joints stiffen during the cascade of change, the good news is that eventually the pain is gone.

To summarize, the expectation that damaged low back tissues should heal within a matter of weeks has no foundation. In fact, longer-term troubles do have a substantial biomechanical or pathoanatomical basis. On the other hand, troublesome backs are generally not a life sentence.

Should the Primary Goal of Rehabilitation Be Restoring Range of Motion?

Research has shown that an increased ROM in the spine can increase the risk of back troubles (e.g., Battie et al., 1990; Biering-Sorenson, 1984; Burton, Tillotson, and Troup, 1989). Why, then, does increasing ROM remain a rehabilitation objective? The first reason, discussed earlier, is the need to quantify reduced disability as defined by the AMA. Second, there is a holdover philosophy from the athletic world that increased ROM enhances performance. This may be true for some activities, but it is untrue for others. As will be shown, this philosophy may work for other joints, but it generally does not work for the back (note that loss of hip mobility contributes to eventual low back disorders; the specific loss has a specific effect on the spine [Van Dillen et al., 2008]). In fact, successful rehabilitation for the back is generally retarded when athletic principles are followed.

When Is Surgery Justifiable?

In practice I see the people who are worse off because of surgery; surgeons refer to them as failed backs (note: I do not see the surgical successes). I strongly suspect that many had the surgery too soon. They thought they had exhausted conservative approaches, but a review of their files revealed that they had received inappropriate treatment. The therapeutic exercises prescribed to them were not matched to their specific pain mechanisms and caused more pain. They were not shown the specific causes of their pain, so they failed in preventing pain. Or their surgeon's declaration that they "failed conservative care” simply meant that a specific amount of time had passed.

There are assets and liabilities to surgery and the particular approach. Whereas fusion has been shown to increase adjacent segment level facet arthritis, disc replacement has been shown to spare them, on average (Siepe et al., 2010). However, it is noted that facet degeneration at the level of the replacement is increased (Hellum et al., 2012), suspected to be due to the natural center of joint rotation not replicated by the prosthesis, which results in stress concentrations on the facet. Fusion is intended to stop motion and pain, whereas replacement is intended to restore motion and stop pain. Justifiable guidance for patient selection remains lacking. Note that there were still failures resulting from both approaches.

Vertebroplasty is a technique of injecting cement into a fractured osteoporotic vertebra with the intention of relieving pain and restoring load-bearing ability. Buchbinder and colleagues (2009) studied over 70 patients and questioned the efficacy of this approach. Microdiscectomy techniques are intended to reduce tissue disruption for disc herniation and nerve root decompression. Arts and colleagues (2009) documented a well-conducted trial comparing tubular discectomy (operating through a scope tube) with conventional discectomy. Once again, the small changes favored conventional microdiscectomy techniques for pain reduction, although the outcome would be heavily influenced by surgeon familiarity and skill, and patient selection. Obviously, given that the tubular approach is less invasive, the risk of inadvertently disrupting the dura and other critical tissues is greater because of the reduced vision of the area. Conversely, the greater the surgical exposure with the conventional approach, the greater the gross tissue damage; however, sensitive tissues are better seen and thus protected. Abdu and colleagues (2009) studied pain from spondylolisthesis by following three methods of hardware-based fusion. They concluded that the patients were better off after the surgery, but there was no difference among approaches.

Follow-up of postsurgical people compared with nonsurgical people can be sobering. Nguyen and colleagues (2011) followed up a historical group of compensated back-injured workers over the 3-year period from 1999 to 2001. A total of 725 had had lumbar fusion, compared with 725 nonsurgical control back-injured workers. After 2 years, 67% of nonsurgical people had returned to work compared with 26% of those who had had surgery. Reoperations were conducted on 27% of the surgical cases, and 36% reported complications and higher long-term opioid use. Permanent disability occurred in 11% of postsurgical patients and 2% of nonsurgical patients. The notion that surgery will eliminate back pain does not have statistical support. People have to manage their postsurgical backs just as much as they would their nonsurgical backs.

When interpreting any of these studies, the key is understanding the variability of response, not only the average effect of the surgical technique.

With every technique, some patients got better; and others, worse. Be aware of the selection of patients, which is usually quite defined for each study. Understanding the variables that explain this divergent response will lead to better outcomes in the future. Once again, patient selection appears to be of paramount importance.

Following is a set of rules that I compiled that I give to patients considering surgery:

- First, always try the virtual surgery game and consider surgery only when it fails. Some patients believe that daily workouts will cure their back pain and that failing to do so will cause weight gain and more pain. Usually, it is the exercises and movement that are keeping them patients! In reality, they need to stop irritating and sensitizing the pained tissues. The virtual surgery game is this: Pretend you had surgery today. Tomorrow is the first day of recovery and is characterized by gentle movements and activities, but generally it is a day of forced rest. Subsequent days follow a typical postsurgical progression in restricted activity. The positive response in symptoms, on average, is impressive.
- Consider surgery when neurological issues are substantial, such as loss of bowel and bladder control. This does not include radiating symptoms such as sciatica. Radiating pain symptoms, peripheral numbness, muscle atrophy, and so on, are all signs of trapped or compromised spine nerve roots. We have been successful in each of these conditions using nerve mobilization approaches. Always try these first. Caution: These are special techniques that require expertise. Performed improperly or too aggressively, they will increase symptoms. Approaches to reduce the cause of the nerve compression or irritation are also required in stubborn cases (see the discussion of nerve flossing techniques in chapter 10).
- Consider surgery in cases of trauma. In such cases, the structure is unstable and needs stabilizing.
- Consider surgery only when pain has been unrelenting and severe for a substantial period of time. Patients who have severe pain for 3 weeks and then have surgery have been some of the most disabled postsurgical cases I have seen.
- Select the surgeon. Everyone likes to state that they had the best surgeon. I have observed sloppy and uncaring work, yet these surgeons continue to practice. I have found that asking the nurses and physical

therapists at the hospital which surgeon has the best results is a wise approach. Simply because a surgeon is the head of the department or may be speaking at a medical conference does not indicate the skill in his hands.

- Discuss the pain with the surgeon. What is the pain generator, and can she cut it out? If several tissues are involved, the chances for success go down. If there is damage at several spine levels, the chance for success drops substantially.
- Clarify what the success rate is. The word success is loaded. In some medical reports this means the patient did not die. In others it means that the patient did well for a brief period following the operation. You are most interested in the long-term success rate compared to any other option, as well as the risks and benefits.
- Beware of new treatments. I have observed so many new devices that have been put into people who end up with poor long-term results. Discs have been stiffened with papain injections, heated with a catheter to stiffen them, and cored and screwed with titanium cages, to name a few. None of these lived up to initial claims (read an insightful review of surgical interventions by Carragee and colleagues, 2009).
- Beware of disc replacement, an approach that I am very skeptical of. I have not seen a successful case yet—that is, full resumption of former activities without pain and the ability to bear substantial load. Most spine joint pain results from motion and loading. Traditionally, the surgeons fuse the joint in a variety of ways. But an artificial disc does the opposite. Its goal is to restore motion to the joint. But herein lies the flaw in logic. The disc is just one of three joints at each spine level. The artificial disc creates an axis of rotation that does not mimic the natural axes of the natural disc. This places more stress on the other two facet joints, so that, over time, they become arthritic and intolerant to motion. Long-term spine health is nearly impossible to achieve this way. The process of patient selection for disc replacement also puzzles me. I have reviewed images of spines with replacements at one level; however, there was clear evidence of substantial instability at multiple levels with large traction spurs and the like. What sort of selection criteria led to a surgical opinion that replacement at a single level would take the pain away? My advice now: Ask the surgeon to set up a conversation with a couple of former patients so that you can be assured of their satisfaction.

- Always exhaust the conservative options. You may believe that because you tried physical therapy, or any other approach, and it failed, only a surgical option remains. It may be that the therapy was not appropriate for you.
- Beware of institutes that offer to view medical images and, with no other information, advise patients on surgery. Pictures are not linked to pain; a thorough clinical assessment is absolutely essential.

I must conclude by noting that there are excellent surgeons. I am familiar with one who insists that any potential patient sign a contract with him. The first condition is that the patient agrees to engage in a structured preoperative program of a specified duration per day and per week, incorporating principles similar to those outlined in this book. The second condition is that if the outcome from this approach is not satisfactory and surgery occurs, the patient also agrees to postsurgical rehabilitation of a certain duration each day to enhance spine-sparing biomechanical principles and pain-free function. That is outstanding leadership!

Several essays in the medical literature assert that back pain is not a medical problem but a social problem. These authors claim that patients are catastrophizing their pain. I disagree. I see the problem as being with the system and the practitioners. Medical practitioners will never be motivated to enhance their understanding of mechanical causes, appropriate diagnoses, and treatments when surgeons who operate are paid 100 times what they would be paid to consult with the patient about what causes pain. Patients often get 3 or 4 minutes for a consult and similar attention for preoperation consults. The practice of using medical images as indicators of pain has replaced a thorough examination that would reveal the pain-causing mechanisms. Only a substantial understanding of biomechanical function and the variables that influence a patient's presentation will results in the best treatment approach.

A Better Alternative to Dealing With Painful Backs

Painful backs are the result of a variety of causes; this book proposes approaches to identify the cause in the individual. Understanding the specific cause, or exacerbator, in each person directs efforts to remove the cause, and ensures that the cause is not replicated in the therapy. This approach works

(Ikeda and McGill, 2012). Tissues in the back become irritated with repeated loading. Consider accidentally stubbing a toe or biting the lip repeatedly—eventually the slightest touch causes pain. This is symptom magnification because the tissues are hypersensitized—not because of psychosocial modulators. Reduction of hypersensitivity in the toe or lip only occurs following a substantial amount of time after the accidental stub or bite has stopped. Tissues in the back are continually “hit” because of aberrant motion or motor patterns. For example, people with flexion bending intolerance of the spine may replicate this every time they rise from a chair. Correcting this movement fault, metaphorically taking the hits away, results in less sensitized tissues, an increased repertoire of pain-free tasks, and a return of motion. Motion returns once the pain goes away. Resist attempting to restore function with a mobilizing approach too soon. This often retards progress.

Mechanical Loading and the Process of Injury: A Low Back Tissue Injury Primer

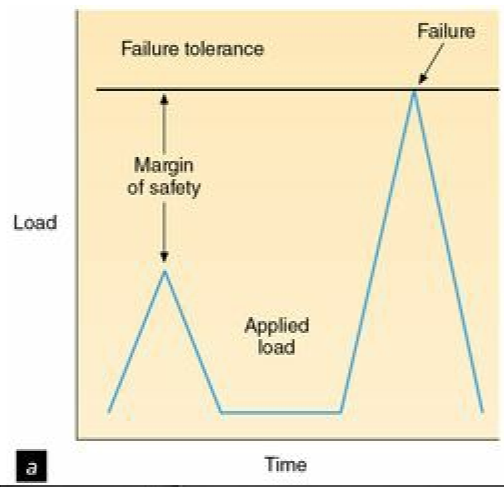
Any clinician completing a worker or patient compensation form is required to identify the event that caused the injury. Very few back injuries, however, result from a single event. This section documents the more common cumulative trauma pathways leading to the culminating event of a back injury. Because the culminating event is falsely presumed to be the cause, prevention efforts are focused on that event. This misdirection of efforts fails to deal with the real cause of the cumulative trauma.

Although a generic scenario for injury is presented here, chapter 4 offers a more in-depth discussion of injury from repeated and prolonged loading to specific tissue. The purpose of this section is to promote the consideration of the many factors that modulate the risk of tissue failure and to encourage probing to generate appropriate hypotheses about injury etiology.

Injury, or failure of a tissue, occurs when the applied load exceeds the failure tolerance (or strength of the tissue). For the purposes of this discussion, injury is defined as the full continuum from the most minor of tissue irritation (but microtrauma nonetheless) to the grossest of tissue failure, such as vertebral fracture or ligament avulsion. We will proceed on the premise that such damage generates pain.

Obviously, a load that exceeds the failure tolerance of the tissue, applied once, produces injury (see figure 1.1b, in which a Canadian snowmobiler airborne and about to experience an axial impact with the spine fully flexed is at risk of posterior disc herniation upon landing). This injury process is depicted in figure 1.1a, in which a margin of safety is observed in the first cycle of subfailure load. In the second loading cycle, the applied load increases in magnitude, simultaneously decreasing the margin of safety to zero, at which point an injury occurs. Although this description of low back injury is common, particularly among medical practitioners who are required to identify an injury-causing event when completing forms for workers' compensation reports, my experience suggests that relatively few low back injuries occur in this manner.

Figure 1.1 (a) A margin of safety is observed in the first cycle of subfailure load. In the second loading cycle, the applied load increases in magnitude, simultaneously decreasing the margin of safety to zero, at which point an injury occurs. (b) The Canadian snowmobile driver (the author in this case from many years ago, who should have known better) is about to experience an axial compressive impact load to a fully flexed spine. A one-time application of load can reduce the margin of safety to zero because the applied load exceeds the strength or failure tolerance of the supporting tissues.

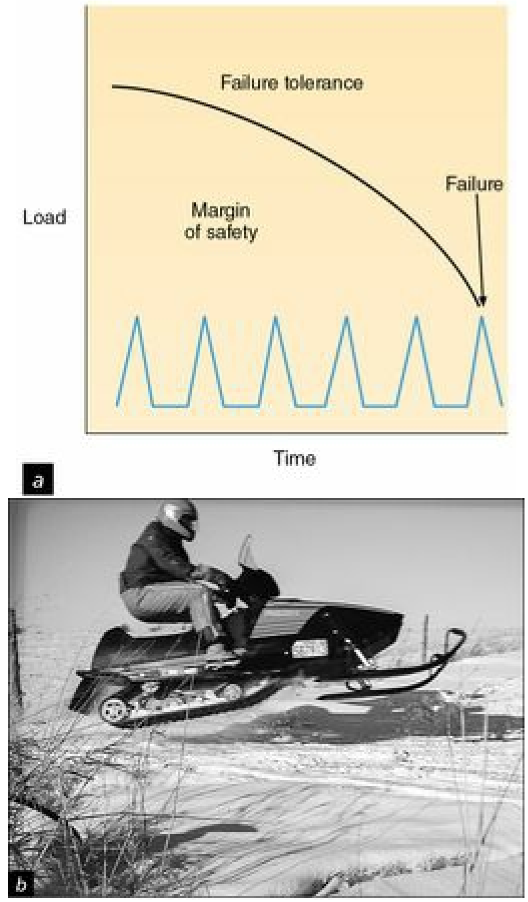


(a) Reprinted from Journal of Biomechanics, 30 (5), S.M. McGill, “Invited paper: Biomechanics of low back injury: Implications on current practice and the clinic,” 456-475, 1997, with permission from Elsevier Science.

(b) Stuart McGill

More commonly, injury during occupational and athletic endeavors involves cumulative trauma from repetitive subfailure magnitude loads. In such cases, injury is the result of accumulated trauma produced by either the repeated application of relatively low load or the application of a sustained load for a long duration (as in a sitting task). A person lifting boxes onto a pallet who is repeatedly loading the tissues of the low back (several tissues could be at risk) to a subfailure level (see figure 1.2, a and b) experiences a slow degradation of failure tolerance (e.g., vertebrae; Adams and Hutton, 1985; Brinckmann, Biggemann, and Hilweg, 1989; Balkovec and McGill, 2012). As tissues fatigue with each cycle of load and correspondingly the failure tolerance lowers, the margin of safety eventually approaches zero, at which point this person will experience low back injury. Obviously, the accumulation of trauma is more rapid with higher loads (Callaghan, et al., 2012).

Figure 1.2 (a) Repeated subfailure loads lead to tissue fatigue, reducing the failure tolerance, leading to (b) failure on the Nth repetition of load, or box lift in this example.



(a) Reprinted from Journal of Biomechanics, 30 (5), S.M. McGill, “Invited paper: Biomechanics of low back injury: Implications on current practice and the clinic,” 456-475, 1997, with permission from Elsevier Science.

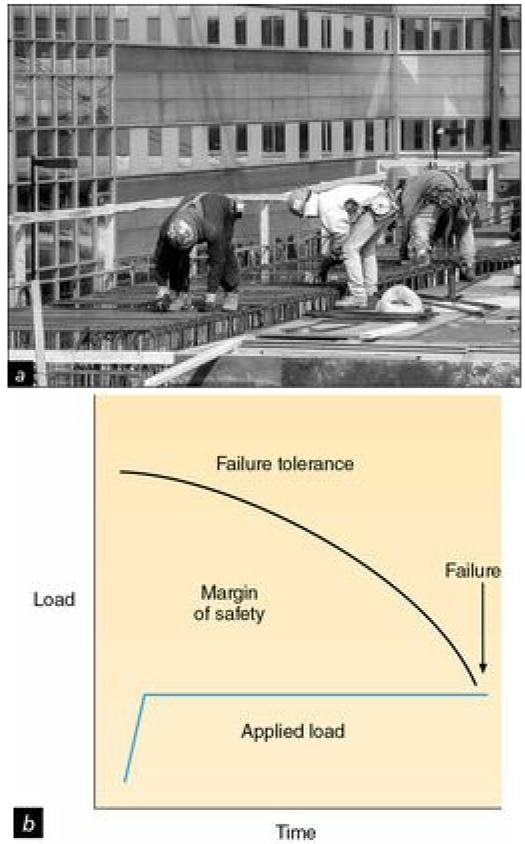
(b) Stuart McGill

Yet another way to produce injury with a subfailure load is to sustain stresses constantly over a period of time. The rodmen shown in figure 1.3a, with their spines fully flexed for a prolonged period of time, are loading the posterior passive tissues and initiating time-dependent changes in disc mechanics (figure 1.3b). Under sustained loads these viscoelastic tissues slowly deform and creep. The sustained load and resultant creep cause a progressive reduction in the tissue strength. Correspondingly, the margin of safety also declines until injury occurs at a specific percentage of tissue strain (i.e., at the breaking strain of that particular tissue). Note that these workers are not lifting a heavy load; simply staying in this posture long enough will eventually result in injurious damage. The injury may involve a single tissue, or a complex picture may emerge in which several tissues become involved. For example, the prolonged stooped posture imposes loads on the posterior ligaments of the spine and posterior fibers of the intervertebral disc. The associated creep deformation that ultimately produces microfailure (e.g., Adams, Hutton, and Stott, 1980; McGill and Brown, 1992) may initiate another chain of events. Stretched ligaments increase joint laxity, which can lead to hyperflexion injury (to the disc) and to the following sequence of events:

1. Local instability
2. Injury of unisegmental structures
3. Ever-increasing shearing and bending loads on the neural arch

This laxity remains for a substantial period after the prolonged stoop (Solomonow, 2012).

Figure 1.3 (a) These rodmen with fully flexed lumbar spines are loading posterior passive tissues for a long duration, (b) reducing the failure tolerance leading to failure at the Nth% of tissue strain.

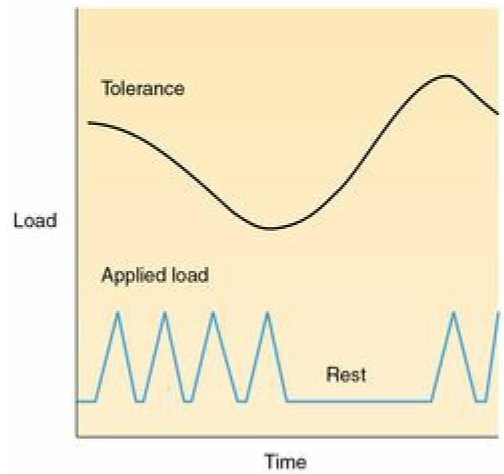


(a) Stuart McGill

Understanding the process of tissue damage in this way clarifies why simple injury prevention approaches often fail. More effective injury intervention strategies recognize and address the complexities of tissue overload.

The avoidance of loading altogether is undesirable. The objective of injury prevention strategies is to ensure that tissue adaptation stimulated from exposure to load keeps pace with, and ideally exceeds, the accumulated tissue damage. Thus, exposure to load is necessary, but in the process of accumulation of microtrauma, the applied loads must be removed (with rest) to allow the healing and adaptation process to gradually increase the failure tolerance to a higher level. We have already seen how tissue loading and injury risk form an optimal U-shaped relationship of not too much and not too little load. Determining the optimal load for health encompasses both the art and science of medicine and tissue biomechanics. Figure 1.4 presents a final load–time history to demonstrate the links among loading, rest, and adaptive tissue tolerance.

Figure 1.4 Loading is necessary for optimal tissue health. When loading and the subsequent degradation of tolerance are followed by a period of rest, an adaptive tissue response increases tolerance. Tissue "training" results from the optimal blend of art and science in medicine and tissue biomechanics.



In summary, the injury process may be associated with either very high loads or relatively low loads that are repeated or sustained. This either/or causation necessitates a rigorous examination of the injury and tissue loading history for substantial periods of time prior to the culminating injury event. It is important to recognize that simply focusing on a single variable such as one-time load magnitude may not result in a successful index of risk of injury, particularly across a wide variety of activities.

Excellent Clinicians and Excellent Practice

When I am asked to refer a patient to a clinician in a distant country, I try to think of a great one. They are rare indeed. Great clinicians perform assessments to uncover the cause of pain. Then they work to eliminate the cause first, followed by an intervention to reduce the risk of recurrence and enhance the robustness of the patient. This must include a component of moving well. Every system in the body requires movement for optimal function and performance. Few clinicians know how to select movement exercises, tune the dosage, and guide the progression.

Clinicians often dismiss the need for a thorough investigation of a patient's pain by stating that finding the pathological source is not possible. I believe that such clinicians are simply unknowledgeable and unskilled. Understanding injury pathways is nonnegotiable because this is the cornerstone of excellent practice. We know what causes disc herniations, Schmorl’s nodes, spondylolisthesis, osteoporotic fractures, torn spine ligaments, facet arthritis, and stenosis, to name a few. Finding these causes is not always simple. Just as a homicide detective must amass enough circumstantial evidence to become convincing, the clinician tests diagnostic hypotheses with more follow-up tests until the evidence is overwhelming. The great clinicians work at the acceptable level of uncertainty. There is no such thing as nonspecific pain to the excellent clinician.

Assessment involves both art and science. Both are needed to interpret the interplay among neural, mechanical, psychological, and physiological variables. Each patient displays signs that must be interpreted. Some are false signs and need to be assessed with sham tests. Great clinicians have eclectic training experience and many tools in their toolboxes. Is specific exercise needed or manual tissue work, or perhaps both? Are the pain and symptoms from a single source or multiple sources? Are the radiating symptoms neural in origin (and if so, what is causing the nerve tension or compression), or are they ischemic? Is the pain organic to a tissue or a neural representation of pain similar to phantom limb pain (which I am convinced explains some of the myalgic syndromes)? These are just a few situations that are not dismissed by the excellent clinician. Worse yet are clinicians who fail to address the cause of the pain and then default to blame the patient for having a psychosocial disorder.

I close with one further generalization. A clinician who provides only passive treatments to a patient has little evidence documenting that the patient will have fewer symptoms in the future. For example, the clinician may perform an injection into the back (no supporting evidence for reducing back pain; Staal et al., 2009), manipulate some joints in the spine, or stretch the patient on a machine. Passive treatments may be helpful at the beginning of a larger treatment plan for certain people. In this case, the patient is progressed quickly into an active approach. The evidence does support the importance of the patients' being fully invested in understanding the cause of pain and practicing strategies throughout the day to avoid these pain triggers. Then they must commit to exercise approaches that suit their conditions.

Unique Scientific Foundation of This Book

This book contains many nontraditional viewpoints on how the spine functions and becomes damaged. Most of these perspectives have emerged from a unique biomechanically based methodological approach that we have been developing for over 30 years at the laboratory and clinic at the University of Waterloo in Canada. This chapter will familiarize you with the unique general approach to obtaining much of the data in this text and will help you understand both the limitations of and the unique insights provided by this approach.

As spine biomechanists, our methods of inquiry are similar to those used by mechanical or civil engineers. For example, a civil engineer charged with the task of building a bridge needs three types of information:

- Traffic to be accommodated, or the design load
- Structure to be used (e.g., space truss or Roman arch), because each architecture possesses specific mechanical traits and features
- Characteristics of the proposed materials that will affect strength, endurance, stability, resistance to structural fatigue, and so on

Our approach to investigating spine function is similar to that of our engineering colleagues. We begin with the following relationship to predict the risk of tissue damage:

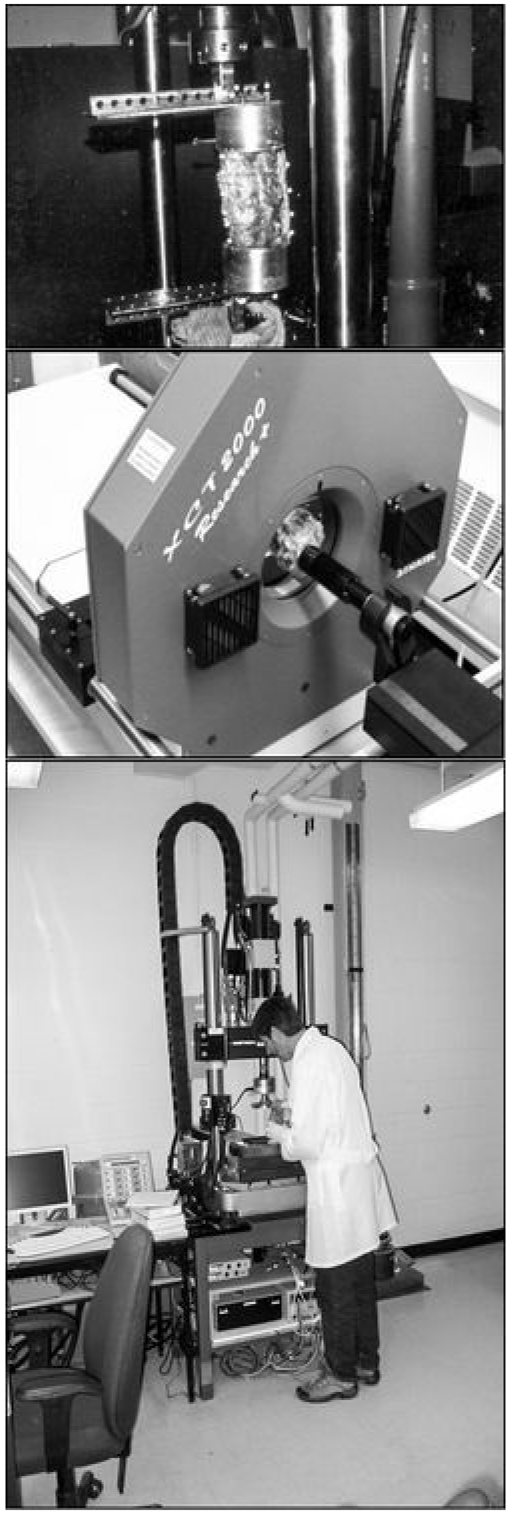
Applied load > tissue strength = tissue failure (injury)

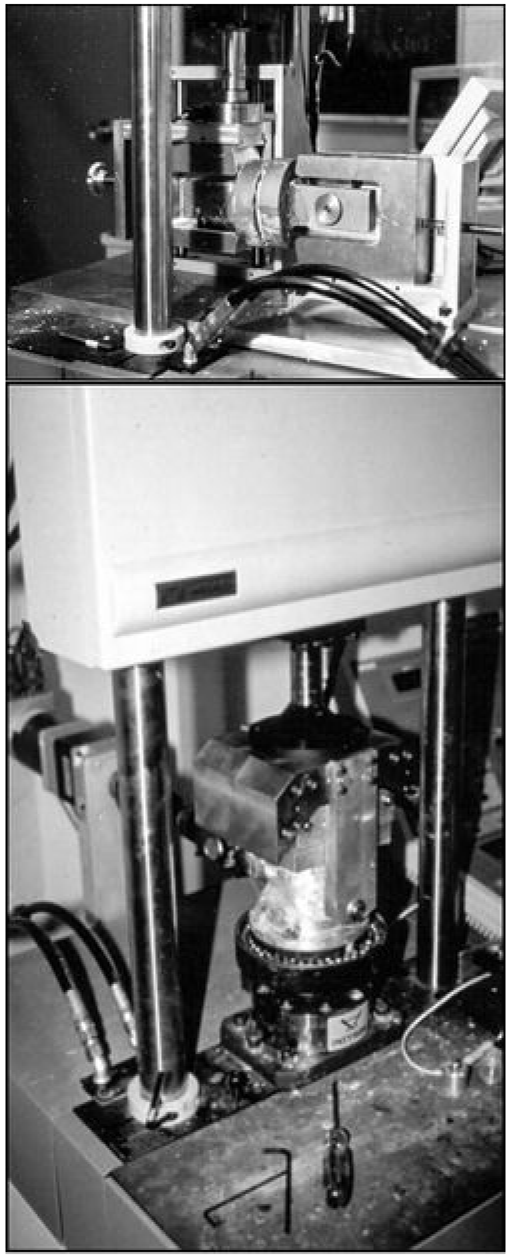
Recall from the tissue injury primer earlier in this chapter that tissue strength is reduced by repeated and prolonged loading but is increased with subsequent rest and adaptation. Analyzing tissue failure in this way requires two distinct methodological approaches. This is why we developed two quite distinct laboratories, which led to much of the progress documented in this book. (The “we” used in this chapter includes my research team of graduate students, visiting scholars, and technicians.) Our first lab is equipped for in vitro testing of spines, in which we purposefully try to create herniated discs, damaged end plates, and other tissue-specific injuries. The second lab is the in vivo lab, where living people (both those with and those without low back issues) are tested for their responses to stress and loading. Individual tissue loads are obtained from sophisticated modeling procedures. The final stage is to test the new findings in the clinic. Following is a brief explanation of the process.

In Vitro Lab

The in vitro lab is equipped with loading machines, an acceleration rack, tissue sectioning equipment, and an X-ray suite to document progressive tissue damage (see figure 1.5). For example, by performing discograms with radio-opaque contrast liquids, we can document the mechanics of progressive disc herniation. We investigate any other injury mechanisms in the same way —that is, by applying physiological loads and motion patterns and then documenting the damage with appropriate technology.

Figure 1.5 We employ many custom-made fixation jigs to mount and load spines. We then document the tissue damage with radiologic imaging and micro dissection.





Stuart McGill

Because many technique issues can affect the experimental results, the decision to use one over another is governed by the research question. For example, because a matched set of human spines to run a controlled failure test cannot be obtained, animal models must be used. Here, control is exercised over genetic homogeneity, diet, physical activity, and so forth, to contrast an experimental cohort with a matched set of control spines.

Of course, the results must be validated and interpreted to be relevant to humans. In addition, identifying the limitations for relevance in interpretation is critical. Some hypotheses that demand the use of human material are compromised by the lack of available young, healthy, undegenerated specimens. Having healthy specimens is critical because biomechanics and injury mechanisms radically change with age. Other major methodological issues include the way biological tissues are loaded, perhaps at specific load rates or at specific rates of displacement. The researcher must decide which has the most relevance to the issue at hand. Devising these experiments is not a trivial task.

In Vivo Lab

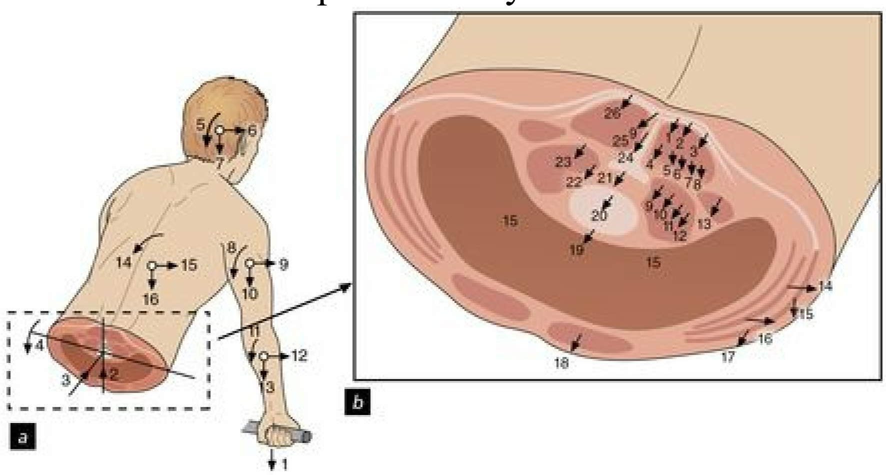
The in vivo lab is unique in its approach to documenting the loads on the many lumbar tissues in vivo. This knowledge lends powerful insight into spine mechanics, of both healthy and injured spines. Because transducers cannot be routinely implanted in the tissues to measure force, noninvasive methods are necessary. The intention of the basic approach is to create a virtual spine. This virtual model must accurately represent the anatomy that responds dynamically to the three-dimensional motion patterns of each test subject or patient and must mimic the muscle activation patterns chosen by the person. In so doing, it enables us to evaluate subjects' unique motor patterns and the consequences of their choices and skill.

How the Virtual Spine Works

Although two groups (the Marras group [e.g., Granata and Marras, 1993] and the McGill group) have devoted much effort to the development of biologically driven models, the McGill model is described here given its familiarity to the author. The model—a dynamic, three-dimensional, anatomically complex, biologically driven approach to predicting individual lumbar tissue loads—is composed of two parts: a linked-segment model and a highly detailed spine model that determines tissue loads and spine stability.

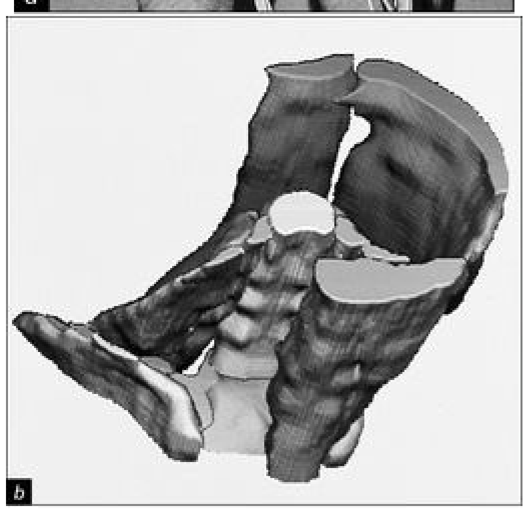
- The first part of the McGill model is a three-dimensional linked-segment representation of the body using a dynamic load in the hands as input. Two or more video cameras at 30 Hz record joint displacements to reconstruct the joints and body segments in three dimensions. Working through the arm and trunk linkage using linked-segment mechanics, reaction forces and moments are computed about a joint in the low back (usually L4-L5) (described in McGill and Norman, 1985) (see figure 1.6a). Using pelvic and spine markers, the three reaction moments are converted into moments about the three orthopedic axes of the low back (flexion–extension, lateral bend, and axial twist).

Figure 1.6 The tissue load prediction approach requires two models. (a) The first is a dynamic three-dimensional linked-segment model to obtain the three reaction moments about the low back. (b) The second model partitions the moments into tissue forces (muscle forces 1-18; ligaments 19-26; and moment contributions from deformed disc, gut, and skin in bending). These forces are applied to the various components, and the stresses are calculated and compared to the structure strengths to assess the risk of pain and injury. Finally, muscle stiffness is calculated, which allows the measurement of spine stability.



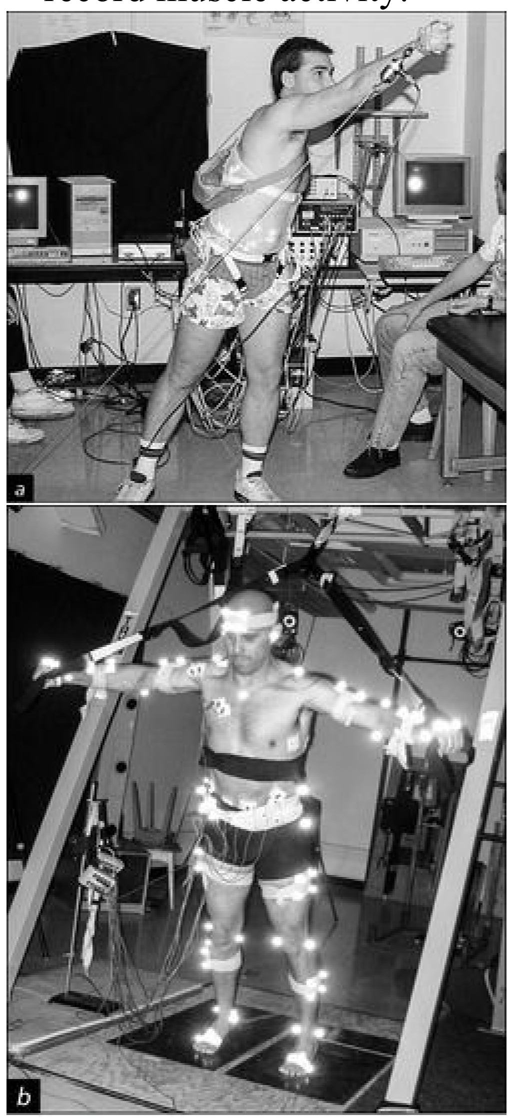
- The second part of the McGill model enables the partitioning of the reaction moments obtained from the linked-segment model into the substantial restorative moment components (supporting tissues) using an anatomically detailed three-dimensional representation of the skeleton, muscles, ligaments, nonlinear elastic intervertebral discs, and so on (see figure 1.6b). This part of the model was first described by McGill and Norman (1986); full three-dimensional methods were described by McGill (1992). Several anatomical additions and updates were provided by Cholewicki and McGill (1996); Vera Garcia, Moreside, and McGill (2009); Brown and McGill (2009); Grenier and McGill (2008); and Brown and McGill (2008a, 2008b). In total, over 120 back and torso muscles are represented. Very briefly, first the passive tissue forces are predicted by assuming stress–strain, or load deformation, relationships for the individual passive tissues. Deformations are modeled from the three-dimensional lumbar kinematics measured from the subject, which drive the vertebral kinematics of the model. Passive tissue stresses are calibrated for the differences in flexibility of each subject by normalizing the stress–strain curves to the passive range of motion of the subject. Electromagnetic instrumentation, which monitors the relative lumbar angles in three dimensions, detects the isolated lumbar motion. The remaining moment is then partitioned among the many fascicles of muscle based on their activation profiles (measured from electromyography [EMG]) and their physiological cross-sectional area. The moment is then modulated with known relationships for instantaneous muscle length of either shortening or lengthening velocity. Sutarno and McGill (1995) described some improvements of the force–velocity relationship. In this way, the modeled spine moves according to the movements of the subject's spine, and the virtual muscles are activated according to the activation measured directly from the subject (see figures 1.7, a and b, and 1.8).

Figure 1.7 (a) A subject monitored with EMG electrodes and electromagnetic instrumentation to directly measure three-dimensional lumbar kinematics and muscle activity. (b) The modeled spine (partially reconstructed for illustration purposes, although for the purposes of analysis it remains in mathematical form) moves in accordance with the subject's spine. The virtual muscles are activated by the EMG signals recorded from the subject's muscles.



Photos from Stuart McGill

Figure 1.8 (a) In this historical photo, an instrumented subject simulates the complex three-dimensional task of tossing an object. The instrumentation includes three-dimensional video to capture body segment kinematics, a recording of the three-dimensional force vector applied to the hand, a 3-Space electromagnetic device to record isolated three-dimensional lumbar motion and assist in partitioning the passive tissue forces, and 16 channels of EMG electrodes to capture muscle activation patterns. (b) A more modern data collection is shown on the right, where force plates register the forces under the feet to begin mapping the forces up to the spine. Infrared cameras (12 of them around the lab room) reconstruct the body in 3-D, and EMG electrodes record muscle activity.



Photos from Stuart McGill

Using biological signals in this fashion to solve the indeterminacy of multiple load-bearing tissues facilitates the assessment of the many ways we choose to support loads. Such an assessment is necessary for evaluating injury mechanisms and formulating injury-avoidance initiatives. From a clinical perspective, this ability to mimic individual spine motions and muscle activation patterns enables us to evaluate the consequences of a chosen motor control strategy. For example, we can see that some people can stabilize their backs and spare their lumbar tissues from overload when performing specific tasks. Conversely, we can evaluate the consequences of poorly chosen motor strategies. In this way we can identify people with perturbed motor patterns and devise specific therapies to regroove healthy motor patterns that ensure sufficient spine stability and spare their tissues from damaging load.

Our challenge has been to ensure sufficient biological fidelity so that estimations of tissue forces are valid and robust over a wide variety of activities. The three-dimensional anatomy is represented in computer memory (muscle areas are provided in appendix A.1). On occasion, if the expense is warranted, we create a virtual spine from a three-dimensional reconstruction of serial magnetic resonance imaging (MRI) slices from the hip trochanter to T4 (see figure 1.7b). This component of the modeling process is well documented for the interested reader in McGill and Norman (1986) and McGill (1992). A list of the large number of associated research papers pertaining to the many detailed aspects of the process is provided in the references and additional readings section at the end of this book. See figures 1.9 and 1.10 for a flowchart and example of the modeling process.

Figure 1.9 The model input and output are illustrated in this flowchart up to the point of the calculation of moments and tissue loads. Spine stability is calculated with an additional module.

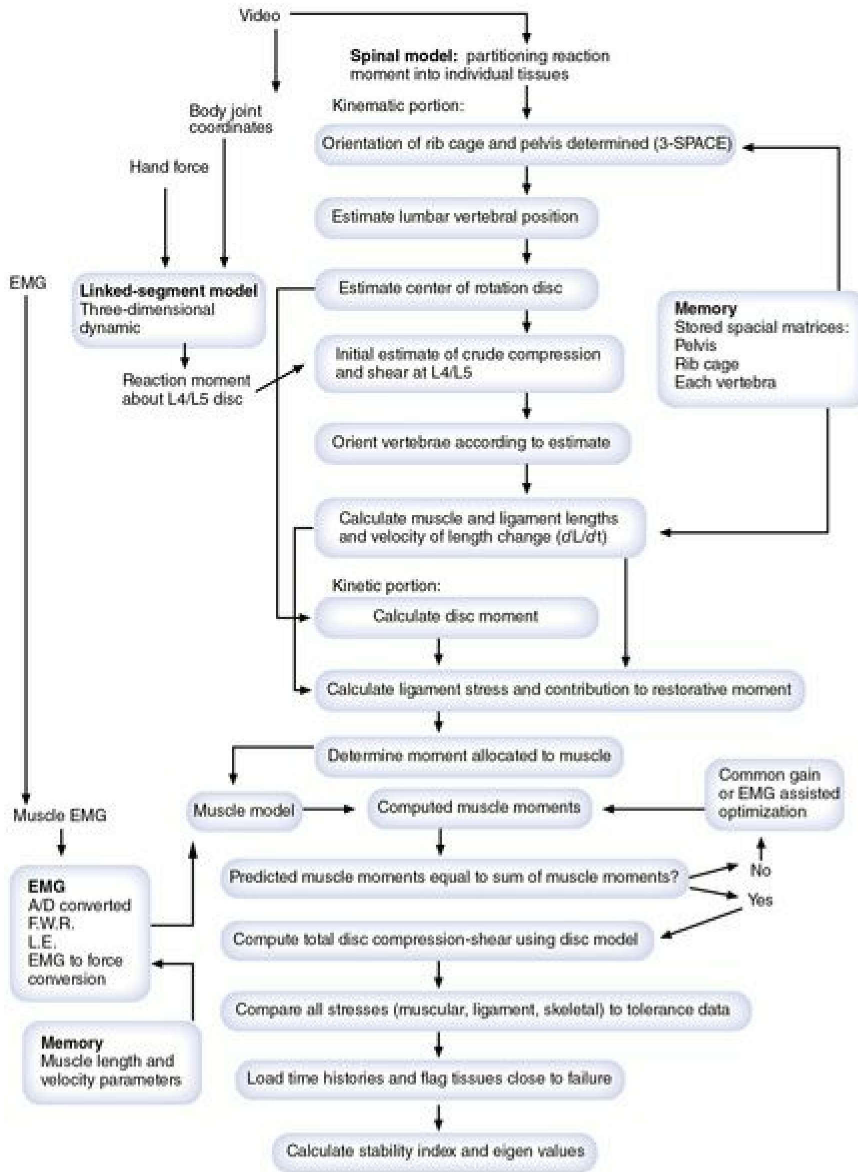
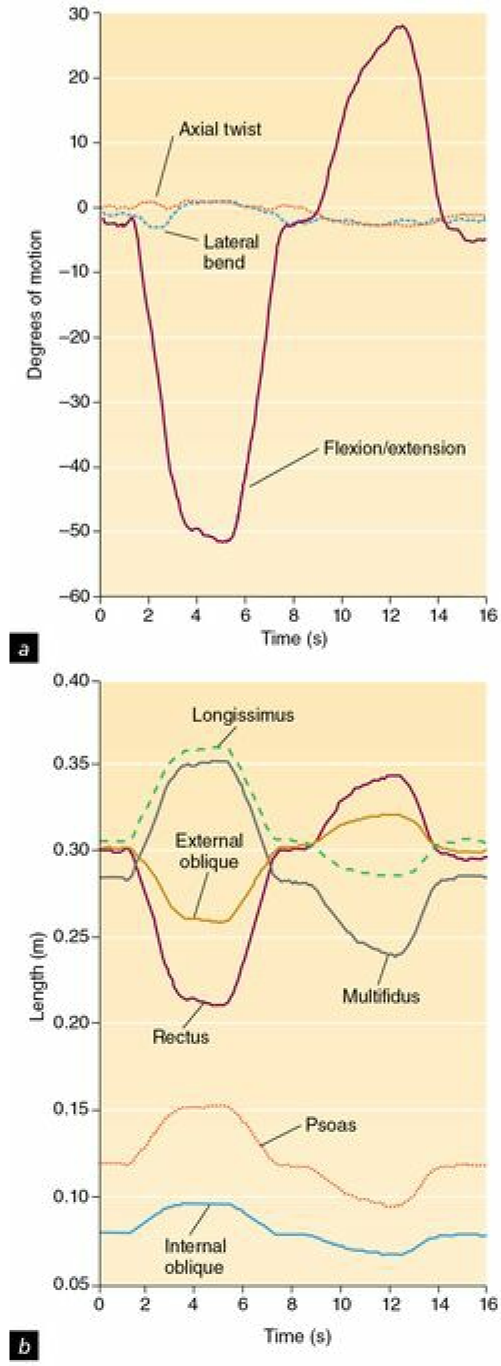
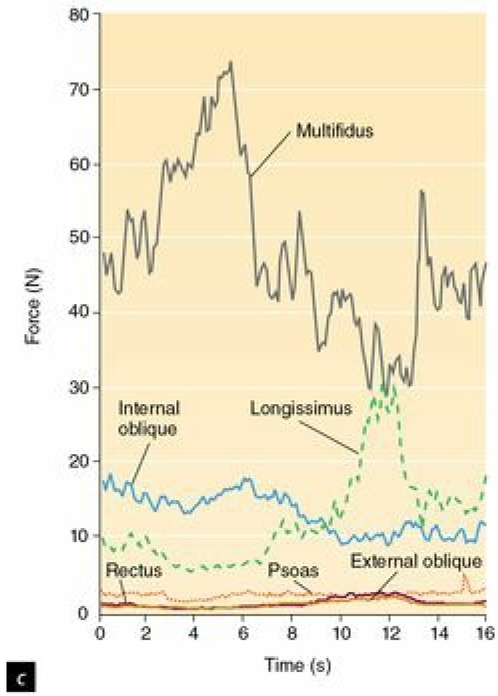
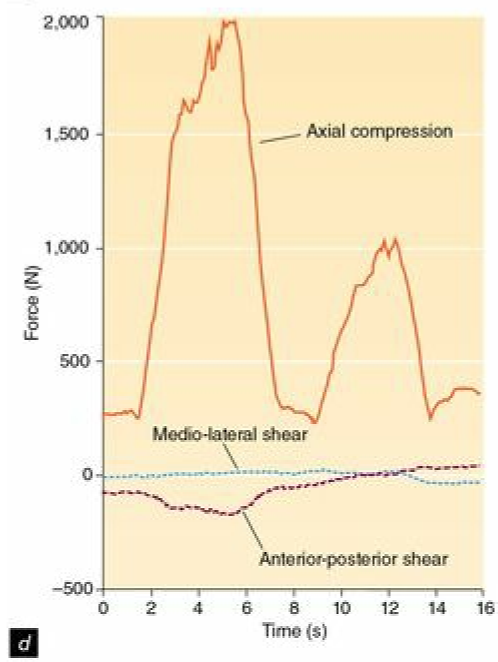


Figure 1.10 Stages of model output in this example of a subject flexing, picking up a weight, and extending: (a) lumbar motion about the three axes—flexion–extension, lateral bend, and axial twist; (b) lengths of a few selected muscles throughout the motion; (c) some muscle forces; (d) L4-L5 joint forces of compression and shear; (e) a stability index, in which larger positive numbers indicate higher stability, and a zero or negative number suggests that unstable behavior is possible.




Multifidus
Internal oblique
Longissimus
Rectus
Psoas
External oblique
Force (N)
Time (s)
c


Axial compression
Medio-lateral shear
Anterior-posterior shear
Force (N)
Time (s)
d

Development of the Virtual Spine

The development of the virtual spine approach has been an evolutionary process spanning 20 years. In that time we have had to confront issues of validity and how to handle deep muscles that are inaccessible with surface EMG electrodes. Briefly, although we have tried intramuscular electrodes in highly selected conditions, this is a limiting invasive procedure. Generally, we estimate the deep muscle activation amplitudes from movement synergists (McGill, Juker, and Kropf, 1996). This method is limited, however, because it requires prior knowledge of muscle patterns for a given moment combination in a specific task. We try to incorporate the highest level of content validity by using detailed representations of the anatomy and physiological cross-sectional areas, recording stress–strain relationships, and incorporating known modulators of muscle force such as length and velocity. One of our validation exercises is to compare the three measured reaction moments with the sum of individual tissue moments predicted by the virtual spine. A close match suggests that we have succeeded in accurately representing force.

Over the past 15 years, this approach for predicting individual lumbar tissue loads has evolved to enable us to document spine stability. In this way, we can evaluate a person's motor patterns and identify strategies that ensure safety and those that could result in injury. This is not a trivial task. It requires converting tissue forces to stiffness and using convergence algorithms to separate the forces needed to create the torques that sustain postures and movements from the additional forces needed to ensure stability. Potential energy-based analyses are then employed to identify the stability index in each degree of freedom of the joint, which in turn reveals the joint's ability to survive a given loading scenario. This level of modeling represents the highest level of sophistication currently available. It has enabled us to challenge concepts pertaining to spine stability—more on this in chapters 5 and 10. The latest initiative, which is still in progress, is to complete the model to include legs via the hip joints and a cervical spine. This will extend our capability to assess the interplay with hip and neck function and provide a better understanding of disorders.

A Final Note

The controversies introduced in this chapter illustrate the need for the evidence presented in the rest of the text and the relevance of the discussions that follow. The scientific approaches used to build the basic scientific foundation are unique, which assist for interpreting the patient trials that guide how the clinical approaches are designed and prescribed. Resist the urge to assume that conventional wisdom is correct: First consider the evidence, and then form your own opinions.

Chapter 2
Epidemiological Studies and What They Really Mean

Definitive experiments are rare in the fields of science and medicine. Instead, conclusions generally emerge from the integration and synthesis of evidence from a variety of sources. Using a similar approach, lawyers argue cases in which each piece of evidence is considered circumstantial in the hope that eventually the circumstantial evidence will become overwhelming. Like lawyers, scientific researchers gather and regather circumstantial evidence from several perspectives with the goal of understanding cause and effect. By studying the relationships among variables and investigating mechanisms, they are able to form perspectives that are robust and plausible. This type of research work, together with longitudinal studies, tests the causative factors identified in the mechanistic studies.

Other chapters in this book address the mechanisms of the low back and their links to good health and disability. This chapter focuses on the study of associations of variables through various epidemiological approaches. Some readers will find this a boring chapter—so my students inform me. You may choose to skip this chapter. But before you do, consider that I will show you that many of these studies have been misinterpreted, or misrepresented, to serve the agenda of a group. Those wishing a fuller understanding of the challenges of building a strong foundation for the very best injury prevention and rehabilitation programs are encouraged to read on. Doing so will enable you to appreciate the epidemiological perspective, to have a more complete comprehension of the positions taken in the text as a whole to reduce the economic impact of low back disorders, and to understand certain subsequent approaches for prevention and rehabilitation.

Several sections in this chapter will help you understand the risk factors for low back trouble—specifically, the changes in personal factors and whether they cause back troubles or are a consequence of having them. For the purpose of this review, purported disabling low back troubles and possibly related work intolerance will be referred to collectively as low back disorders (LBDs). Furthermore, the term personal factors can include anthropometric and fitness variables, as well as motor control ability, injury history, and so forth.

Influence of Randomized Controlled Trials and Other Epidemiological Approaches

What is the truth regarding back pain cause and treatment? Generally, the randomized controlled trial (RCT) is regarded as the gold standard in medicine. However, this has led to several questionable popular opinions regarding back pain. This section addresses the role of RCTs for back pain.

Back pain is not a homogeneous condition. Any therapeutic approach that relieves one person's pain will exacerbate another's. Epidemiological studies of back pain will never reveal cause and effect, or efficacy, because each person will respond to a different approach and different dosage. Thus, a controlled study on back pain will result in the conclusion of no effect. However, when patients are categorized into subgroups based on pathomechanics, or pain patterns, or history, or even psychosocial variables, results in the evolving literature are generally positive. Fersum, Dankaerts, and O'Sullivan (2009) opined that "non-specific exposures, with non-specific treatments, with non-specific back pain guarantees a null finding." Each patient is unique. Patients sit differently and use different strategies that either keep them in pain or out of pain (Dankaerts et al., 2006), and they react differently to different movement corrections (Ikeda and McGill, 2012). By their very nature, RCTs are not suitable for unclassified low back pain.

The evolution of the spine literature lags behind that on the knee and related leg pain. As an exercise in logic, let's replace the discussion of back pain with that of leg pain and observe how the back literature has been misinterpreted. No study of nonspecific leg pain would be expected to reveal cause and effect (the genesis of pain could be due to mechanical, vascular, hereditary, or many other factors). Similarly, no credible person would use such logic to state that there is no evidence of links between mechanical factors and nonspecific leg pain. Nor would such an assertion be published. However, a very specific leg disorder such as ACL disruption in female basketball players has been substantially reduced by a specific intervention to alter biomechanics (e.g., Hewett et al., 1999). One could argue that the treatment effect is underestimated. Even in these controlled clinical trials, everyone in the group received the same treatment. However, in excellent clinical practice, each intervention is tuned and adjusted to each patient, suggesting that efficacy in real life on a patient-by-patient basis is probably better than any controlled trial would suggest. Well-presented examples of the interactions among individual anatomical, morphological, mechanical, and neuromechanical factors have been compiled by McLean and Beaulieu (2010), implying the need for individual assessment and treatment.

Some have opined that the relationships studied for the leg do not exist for the spine. Yet evidence exists on disc shape, to choose just one example from our own laboratory (Yates, Giangregorio, and McGill 2010), influencing the patterns of annulus disruption from specific modes of loading resulting from specific movement patterns. This supports individual assessment of anatomical features to justify appropriate clinical treatment to alter offending movement. The complex interactions of structure and biomechanics suggest that evidence from controlled trials of back pain must be coupled and interpreted with treatment studies of appropriately subclassified patients, even to the point of case studies, to delineate the complex interactions. I hope this convinces you that the back pain literature needs a higher level of perspective to converge on the truth.

The mechanism of injury must be considered before a systematic review on back pain is valid. For example, discogenic back injury and pain are associated with repeated and prolonged disc flexion. This occurs in occupational lifting from lower heights, but it also occurs in office workers who sit for prolonged periods. Both scenarios are potent causative factors (see a review by Adams et al., 2002). Reviewing studies that compared workers engaged in lifting activities to those who do not lift but have sedentary jobs (i.e., sitting) does not test lifting and pain, but instead tests the mechanism (i.e., disc flexion). Many reviews ignore known mechanisms that obscure any association or cause and effect. A textbook such as Marras' The Working Back (2008) integrates the mechanisms and defines links between back disorders and occupational exposure—for example, the hazards of lifting from the floor versus from table height, particularly for aging workers with stiffer hips. These concerns have motivated others to call for a more extensive study of the mechanisms of back pain to provide a better base for what is tested in an RCT (Gordon, 2010). The dose is important, but what is the effective dose?

Another widely held clinical belief is that a diagnostic test must be reliable to be of value (for a full essay on the topic, see McGill, 2013). This means that five clinicians would reach the same conclusion testing the same patient. Simple tests are reliable. Measuring core temperature in a case of infection, or blood chemistry in a diabetic, are reliable tests that lead to a homogeneous treatment. But musculoskeletal disorders are different: the subjects are highly variable in terms of pain, there is often more than one source of pain, the dosage of intervention is critical (because too much exacerbates and too little has no effect), and the outcome is highly variable in terms of duration and effectiveness. Why should two clinicians obtain the same impression when examining a biological system that is continually in a state of flux? If skill was equal, and the patient remained static, then prediction may be possible and even justifiable. But this is not the case with most musculoskeletal disorders. Thus, the typical rules for reliability associated with evidence-based medicine need a liberal amount of reflection for logical application in back pain situations.

Do clinicians have similar levels of clinical skill? Clinical skill involves perceiving via touch; interpreting what the patient verbalizes and displays with body language; knowing how much force to apply; and knowing how to explore the end range and arc of motion with subtle trajectory variations to interpret joint capsule, bony interaction, ligament spring, and associated muscle tone, to name just a few variables. A simple test, such as one to objectify the range of motion, becomes much more revealing in the hands of a skilled clinician who probes tissues through a range. A less skilled clinician will overlook modulators of pain and function and should not be expected to replicate the clinical impression.

So what is the better test? The simple test to obtain a range of motion score that is reliable, or the test that facilitates a branching decision tree using variables and relationships that are nonlinear, change over time, and are not repeatable among clinicians of differing skill levels? Should reliability be used to identify a good test, and by logical extension, should reliability be a metric for inclusion into clinical guidelines? This argument illustrates how skilled diagnosticians using less reliable tests but more complex decision trees will obtain more insight into the patient than unskilled ones.

Tragically, the more complex cases, many of which fail to be sorted out by clinical guidelines advocating reliable diagnostic tests, are dismissed by the health care system. I see many of these sad cases in my consulting practice. Skilled clinicians must be encouraged to maintain and develop their skills in the more complex, but more insightful (but unreliable) assessment tests. Over my 30 years of observation, my impression is that this has declined. Unfortunately, political forces via medical journal publication policies are influencing the development of clinical skill. I am against the growing emphasis on developing only tests that are reliable, and I am even more against the practice of journals now requiring a high test reliability score to qualify for publication. This practice is retarding expert assessment skill acquisition and development, and optimal patient outcomes for all branches of manual medicine.

Misunderstandings of Epidemiology

Epidemiological studies have been used almost exclusively to drive policy change that affects back pain issues. The compensation board in New Zealand (Accident Compensation Corporation) now denies any association between the type of work exposure and the development of disabling back pain. How could this be, given the multitude of studies confirming the link, and opposing policies of other countries? They appear to have based their position and policy on a study of twins (Battie et al., 2009), claiming that genetics explains the majority of variance. This study assessed MRI images of 147 monozygotic twins and 153 dizygotic twins that had exposure discordance. It was estimated that 61% of variance in disc degeneration could be explained by their common genes, whereas 16% was explained by occupation. McLean and colleagues (2011) stated that this was based on a misunderstanding. They noted that variation is not the same as causation, and that what is inherited is not the same as genetic determination. They give examples of specific, well-documented, inherited diseases that occurs only in the presence of the specific exposure trigger. Thus, the cause is both 100% genetic and 100% environmental. They also used smoking to illustrate the complexities of ferreting out exposure. If every person smoked one pack of cigarettes per day, smoking would not emerge as a causative factor. Only genetic predisposition to smoke would appear significant. Thus, the finding that 61% of variation in disc degeneration is explained by genetics does not mean that 61% of back pain is caused by genetics; in fact, it does not show anything in terms of causation. The same principle holds true for excessive noise exposure and hearing loss, and exposure to radiation and cancer.

As my good colleague Professor Marras has stated, "Genetics loads the gun, but exposure pulls the trigger." However, there is a difference with back pain. Exposure to physical loads is not a linear relationship—no load and too much load are both harmful. Some load is healthful. This will become clear in the following pages.

Multidimensional Links Among Biomechanical, Psychosocial, and Personal Variables

As noted in chapter 1, several prominent people have declared that psychosocial variables are the most significant factors in LBD. This is an important issue because, to be effective, intervention must address the real causes and consequences of back troubles. This section shows that virtually all studies that properly measured or calculated the physical demands of tasks revealed that people subjected to specific mechanical stressors are at higher risk of LBD than others, but that there also appear to be some mitigating issues.

Four Important Studies

The following four studies are reviewed here because of their lasting historical influence.

- Bigos and colleagues. In 1986 Bigos and colleagues performed a highly quoted study—one that has been very influential in shaping opinion regarding injury prevention and rehabilitation—at the Boeing plant in Washington state in the United States. This retrospective investigation analyzed 4,645 injuries (of which 900 were to the low back) over a 15-month period in 1979-1980. The authors reported a correlation between the incidence of back injury and poor appraisal ratings of employees performed by their supervisors within 6 months prior to the reported injury. The authors considered the poor ratings to represent a psychosocial factor. In 1991 Bigos and colleagues conducted a longitudinal prospective study of 3,020 employees at Boeing, during which there were 279 reported low back injuries. The researchers collected personality inventories as well as questionnaires regarding family and coworker support and job satisfaction. They also analyzed personal factors such as isometric strength, flexibility, aerobic capacity, height, and weight. The authors concluded that psychosocial measures—particularly those related to job enjoyment—had the strongest influence of all the variables analyzed. In fact, those workers who stated that they did not enjoy their job were 1.85 times more likely to report a back injury (odds ratio = 1.85). Job satisfaction counted for less than 15% of the variance as an injury risk factor, meaning that more than 85% of the variance was unaccounted for. In other words, psychosocial factors failed to account for 85% of the causation of LBDs. The authors concluded "that the statistically significant, though clinically modest, predictive power of work perceptions and psychosocial factors for reports of acute back pain among industrial workers argues against the exclusive use of an injury model to explain such problems." This is a fair summary of the implications of their work. It does not mean that mechanical loading is unimportant. Yet, this study is often quoted to support the viewpoint that psychosocial factors are the most important causes of back disorders. Interestingly, Marras and colleagues (1993) also found similar odds ratios for job satisfaction (1.56) in a massive study of 400 repetitive industrial lifting jobs across 48 industries.

Very few epidemiologically based studies have employed reasonably robust quantifications of biomechanical, psychosocial, and personal factors. Two important studies meet this requirement.

Odds Ratios

Perhaps the most lucid definition of an odds ratio can be achieved through an example. If smokers have three times the risk of developing lung cancer that nonsmokers do (perhaps 6 out of 10 smokers as opposed to 2 out of 10 in nonsmokers), they have an odds ratio of 3. Thus, an odds ratio greater than 1 suggests an increased risk from a specific factor.

- Marras and colleagues. The first important study **that considered multiple factors** was reported by Marras and colleagues (1995), who surveyed over 400 industrial lifting jobs across 48 industries. They examined medical records in these industries to classify each type of job as being low, medium, or high risk for causing LBD. They documented a variety of mechanical variables as well as reporting job satisfaction. The most powerful variable for predicting those jobs with LBD was maximal low back moment. This resulted in a predictive odds ratio of

4.04 between low- and medium-risk groups and a ratio of 3.32 between the low- and high-risk groups. Other single variables produced impressive odds ratios; for example, sagittal trunk velocity (odds ratio = 2.48) for the low- and high-risk comparison and 2.42 for maximal weight handled between the low- and high-risk groups. Job satisfaction produced an odds ratio of 1.48 between the low- and high-risk groups and of 1.32 between the low- and medium-risk groups. The researchers entered the single variables into a multiple logistic regression model. The group of measures selected by the model described the risk index well between the low- and high-risk groups and also between the low- and medium-risk groups. Suitably varying the five measures chosen by the regression process (maximal load moment, maximal lateral trunk angular velocity, average trunk twisting velocity, lifting frequency, and maximal sagittal trunk angle) decreased the odds of being a member of the high-risk LBD group over 10 times (odds ratio = 10.6). This was an important study for specifying certain physical characteristics of job design that reduce the risk of LBD and linking epidemiological findings with quantitative biomechanical analysis and psychosocial factors across a large working population. Marras' group has continued with similar studies to strengthen the understanding of biomechanical and psychosocial risk factors in predicting meaningful low back functional losses (e.g., Ferguson et al., 2012).

- Norman and colleagues. The second important study to successfully integrate biomechanical, psychosocial, and personal factors was conducted by Norman and colleagues (1998), who examined injuries that occurred in an auto assembly plant that employed more than 10,000 hourly paid workers. During a 2-year period of observation in the plant, the authors reported analyses on 104 cases and 130 randomly selected controls. Cases were people who reported low back pain (LBP) to a nursing station; controls were people randomly selected from company rosters who did not report pain. This is a notable study because the authors attempted to obtain good-quality psychosocial, personal, and psychophysical data on all participants from an interviewer-assisted questionnaire, as well as good-quality, directly measured biomechanical data on the physical demands of the jobs of all participants. (Note that the psychophysical approach is based on worker self-perceived stresses.) The study revealed that several independent and highly significant biomechanical, psychosocial, and psychophysical factors (identified as risk factors) existed in those who reported LBP. The personal risk factors that were included were much less important. After adjusting for personal risk factors, the statistically independent biomechanical risk factors that emerged were peak lumbar shear force (conservatively estimated odds ratio = 1.7), lumbar disc compression integrated over the work shift (odds ratio = 2.0), and peak force on the hands (odds ratio = 1.9). The odds ratios for the independent psychosocial risk factors, from among many studied, were worker perceptions of poorer workplace social environment (2.6), higher job satisfaction (not lower, as shown in the Boeing study) (1.7), higher coworker support (1.6), and perception of being more highly educated (2.2). Perceptions of higher physical exertion, a psychophysical factor, resulted in an odds ratio of 3.0, which is possibly related to the capacity of the worker relative to the job demands. Nearly 45% of the total variance was accounted for by these risk factors, with approximately 12% accounted for by the psychosocial factors and 31% by the biomechanical factors. These results are very consistent with those reported by Marras and colleagues (1995) and by Punnett and colleagues (1991). Only a few of the personal factors were associated with reporting LBP: body mass index (odds ratio = 2.0) and prior compensation claim (odds ratio = 2.2). This case–control study is of high quality because it used a battery of many of the best measurement methods available for field use to assess many psychosocial, biomechanical, and personal factors of all participants in the data pool.

- Coenen and colleagues. This Dutch group (2013) studied the spine loads of workers and documented personal and psychosocial factors. Three years later, a follow-up documented the occurrence of low back pain. Cumulative low back load had a greater association with the onset of pain than any other factor including those considered as personal and psychosocial. Interestingly, a more detailed analysis revealed that this relationship was strengthened when workers spent more time with their spines flexed and lifts of loads weighing more than 25 kg (55 lb). The authors' position on the importance of motions, postures, and loads causing low back pain strengthens throughout this monograph.

The evidence from the preceding comprehensive studies suggests that both psychosocial and biomechanical variables are important risk factors for LBD. In particular, cumulative loading, joint moments, and spine shear forces are important. Those claiming that only psychosocial factors are important or that only physical loading factors are important cannot mount a creditable, data-based defense, because it appears that the data they quote fail to measure properly either physical or psychosocial variables, or both.

Do Workers Experience LBDs Because They Are Paid to Act Disabled?

Some papers in the literature appear to dismiss the link between pain and disability. Most of these papers clearly state that this notion is restricted to nonspecific back pain, noting that specific diagnoses do impair the ability of a worker to perform a demanding job. However, some authors base their arguments on the concept that low back tissue injury heals in 6 to 12 weeks, whereas others base their arguments on a behavioral model of chronic pain that is not totally consistent with the findings of other scientific approaches. A short discussion of the issues and evidence related to physician diagnosis, compensation, tissue damage, and pain is necessary.

The position that chronic pain and disability are a function of compensation (and not mechanical factors) is contradicted by evidence that low back troubles continue after legal settlement of injury compensation (Mendelson, 1982). Hadler, Tait, and Chibnall (2007) believe that the contest between the patient and the medical officer charged with determining the compensatory award causes the patient to act disabled and thwarts any incentive to act well. Teasell and Shapiro (1998) shared this opinion in a review of several chronic pain disorders, as did Rainville and colleagues (1997) in another high quality study that specifically addressed chronic LBP. Hadler has taken the position that mechanical factors are, for the most part, of little importance in either causing or rehabilitating bad backs when compared with the psychosocial modulators (e.g., Hadler, 2001). His selective citation of the literature excludes evidence linking mechanical overload to tissue damage and ignores several important intervention studies. For example, Werneke and Hart (2001) showed that pain patterns on patient presentation, specifically whether the pain "centralizes" or not, are much more powerful predictors of chronicity than the psychosocial variables they studied.

This rather common perception that injury compensation negatively affects the health recovery of injured workers has been challenged by Spearing and Connelly (2011). They reviewed the evidence often cited to support this perception for both verifiable and nonverifiable injury. They refuted the notion that strong evidence exists of an association between compensation and poorer health outcome, even for one of the most documented disorders—whiplash. They showed that the laws and legal processes are heterogeneous and that any link of a disorder to compensation is extremely difficult to control. Of the patients I have seen for whom compensation has been suggested to modulate their behavior and recovery, I have found an alternate explanation in nearly every case. In some, the rehab program assigned to them was ill-conceived and hurt them more. Some were never shown the mechanical causes of their pain. The jobs of others were so physically demanding that few healthy people would survive the daily rigors. These jobs demanded close to 100% of their healthy capabilities, meaning that 90% capability was not sufficient. Their doctors did not have the tools to measure the jobs' physical demands nor their capabilities. Yet some jurisdictions have started to move to limiting access to compensation, believing the association between health and compensation exists, is common, and is simple. It is not.

Of the several parties involved in the compensation system, all wish for a healthy patient. However, several factors conspire to militate against an optimal process and experience for all. There is much heartache and unfairness. Some unfortunate patients are rejected from the comp system because they fail to get better, or actually get worse, and are labeled noncompliant. This is largely because the comp system usually employs a rehab approach that brings finality to the case. This approach is often known as work hardening and is characterized by physical tasks that have a systematic schedule for increased challenge. Some claim that the type of challenge or exercise or work task is not important—only that it be performed. Patients are encouraged to work through the pain. There is no question that many patients thrive under this approach and are successfully discharged as employed workers. However, this approach is not for every patient. Typically, these sorts of programs have significant dropout rates. The comp system usually labels these people as noncompliant, and they are dismissed from payouts. Psychosocial issues are usually given as reason for their inability to cope. My opinion is different. Many of these people have backs that may be unstable, and the work-hardening program incorporates the injury mechanism as part of the treatment and makes them worse. Butler (e.g., 1991, 2000) has documented for years that central sensitization mechanisms and secondary hyperalgesia are based on measurable changes in nervous structures and that more pain during movement only heightens the syndrome. The mechanisms of sensitization are nicely described by Woolf (2010). Because corrective exercise has not been prescribed, the tissues are further damaged, or at least prevented from healing. For these patients we work to eliminate the cause of their pain and ensure pain-free therapeutic exercises specifically designed to address their deficits and the actual cause of tissue overload. Even with people who have the most disabled of backs, those who have been classified as failures and labeled with no hope for recovery (i.e., 0% chance of returning to work), this approach gets about 35% back to work.

Thus, although the topic of compensation is important, it is irrelevant in discussions of the links between loading and LBD. Compensation issues should not be used to argue against the existence of a mechanical link between injury and work tolerance or, worse yet, to suggest that the removal of compensation will eliminate the cause of tissue damage.

Does Pain Have an Organic Basis—Or Is It All in the Head?

Much has been written about the apparent absence of an organic basis for chronic low back disability (and other chronic pain syndromes). As noted in chapter 1, as high as 85% of disabling LBD cases are claimed to have no definitive pathoanatomical diagnosis (White and Gordon, 1982). Two conclusions have been proposed:

- Many LBD patients present with “nonorganic signs,” suggesting psychological disturbance as the cause of their condition.
- Poor diagnostic techniques, either from inadequately trained doctors or from the limitations of widely available diagnostic technology, have precluded the making of many solid diagnoses.

The first conclusion suggests that the correct course of action in these undiagnosed cases is to ignore physiological issues and address only psychological or psychosocial factors. The second conclusion suggests that one should not ignore the possibilities that more thorough diagnostic techniques could unearth physical causes and that rehabilitation based on that assumption could be more effective than rehabilitation for psychological disturbance alone. Let’s examine each of these arguments.

Does Absence of Diagnosis Imply Psychological Cause?

Waddell and colleagues wrote many manuscripts and guidelines (e.g., 1980, 1984, 1987) on nonorganic signs in patients to support the notion of psychological disturbance overriding any pathoanatomical tissue damage (because none was diagnosed). Yet many of these so-called nonorganic signs are precisely what we will use clinically to detect hypersensitivity to loads that cause pain. For example, a compressive injury often creates pain under very mild compression when the person is in a flexed or slouched posture. Light touch and reported pain in a painful region may indicate a highly centrally sensitized patient. Although the nonorganic signs may be very helpful in defining risk for surgical candidates, we take the position that they do not indicate psychological overlay—in fact, the behavior may be physiologically based. Only a rigorous and complete exam that includes provocative testing could provide evidence to support the diagnostic hypothesis.

Teasell and Shapiro (1998) wrote a nice summary of an extensive experimental literature suggesting that these pain symptoms may indeed have a physiological basis. They reviewed the recent science on the spread of neuron excitability and sensitization of adjacent neurons to explain the sensation of radiating pain in chronic conditions. Changes in neuroanatomy are coupled with biochemical changes with chronic pain. For example, in fibromyalgia patients, numerous studies have shown levels of substance P in the cerebrospinal fluid elevated two to three times over that in controls (reported in a review by Teasell, 1997). Although the nonorganic signs described in Waddell’s papers are important considerations in many cases and are a contribution to clinical practice, strong evidence suggests that many nonorganic signs may not be exclusive of a pathoanatomical mechanism that has eluded diagnosis.

Some have argued that no link exists between pain and tissue damage and activity intolerance. In the absence of no direct evidence, some groups have simply assumed that nociceptive pain that is not surgically correctable or that has not improved within six weeks should not be regarded as disabling (e.g., Fordyce, 1995). In fact, the Fordyce monograph initiated much discussion, including several letters (e.g., Thompson/Merskey/Teasell/Fordyce, 1996). Fordyce's 1996 influential statements that "the course we are presently on threatens disaster" and that "we change or go broke" were particularly revealing. The high cost of treating chronic pain and disability appears to have motivated the elevation of the importance of the psychosocial factors so emphasized in this monograph. It is also interesting to observe the absence of any eminent biomechanical expert among the author list of the Fordyce report. The Canadian Pain Society stated that the Fordyce report literature review "is incomplete and does not reflect the contemporary understanding of chronic low back pain" (Thompson/Merskey/Teasell/Fordyce, 1996). Moreover, the Fordyce report largely ignored the evidence linking mechanical overload to measurable changes in spine biomechanics and spinal pain neuromechanical mechanisms. Another assumption of the report was that most spine LBDs are nonspecific, meaning simply that a diagnosis was not made.

Could Inadequate Diagnosis Be a Factor in Nonorganic LBP?

Bogduk and colleagues (1996) argued that pain arising from many spinal tissues can be attributed to a detectable painful lesion. For example, the facet joints produce pain upon stimulation (McCall, Park, and O'Brien, 1979). Bogduk's point is certainly correct. However, because not all lesions are easily detectable, one cannot argue that if a lesion is not detected, there is no organic basis for pain. For example, fractures and meniscal tears that have been detected in postmortem studies have not shown radiologically on planar X-ray (Jonsson et al., 1991; Taylor, Twomey, and Corker, 1990) or on computed tomography (CT) (Schwarzer et al., 1995). Nor have freshly produced fractures and articular damage been outwardly detectable radiographically in animal models (Yingling and McGill, 2000). Yet these are the typical diagnostic procedures used. Roudsari and Jarvik (2010) questioned the use of the MRI imaging procedure after reviewing the high prevalence of abnormal MRI findings in people with low back pain. However, the problem is more with the common practice of a clinician, most often a surgeon, putting the images on the viewer before a thorough examination of the patient has been conducted. Of course, all sorts of features can be stated as the cause of the patient's symptoms, but this is poor practice. Better practice is to assess the patient and then hypothesize the pain source from the observed patterns. This diagnostic hypothesis is then refuted or confirmed with the MRI image.

Developments in technology such as fMRI have provided a deeper understanding of tissue irritation and pain sensitivity. Kobayashi and colleagues (2009) mechanically stimulated pain with controlled cutaneous pressure lateral to L4/L5 in both patients with chronic back pain and pain-free controls. The patients reported higher levels of pain, and also demonstrated higher levels of activation, in the prefrontal, insular, and posterior cingulate cortices but not in the S1 and S2 surface somatosensory areas, suggesting stimulation of painful deeper structures. These areas of brain activity are associated with the commonly reported pain matrix from a variety of truly painful nociceptive agents (Peyron, Laurent, and Garcia-Larrea, 2000). Back pain was a real organic phenomenon, but the elevated sensitization appears to be a trait of those in chronic pain. Later in this text, cleverly designed exercises are shown to be a potent approach for winding down the sensitivity.

Interestingly, Bogduk and colleagues (1996) and Lord and colleagues (1996) showed that the injection of an anesthetic (placebo-controlled diagnostic blocks) convincingly demonstrates that facet joints are often the site of pain origin. Further, disc studies examining the pain response of well over 1,000 discs in over 400 people undergoing discography by Vanharanta and colleagues (1987) (subsequently reappraised by Moneta and colleagues in 1994 and reported by Bogduk and colleagues in 1996) showed a clear and statistically significant correlation between disc pain and grade 3 fissures of the annulus fibrosus. Most physicians would probably not detect these deep fissures of the annulus. In a rigorous and systematic study of diagnosis based on anesthetic blocks, Schwarzer and colleagues (1995) were able to diagnose over 60% of the LBD cases cited in the study by Bogduk and colleagues (1996) as being internal disc disruption (39%), facet joint pain (15%), or sacroiliac pain (12%). Bogduk and colleagues (1996) stated, "If inappropriate tests such as EMG and imaging are used, nothing will be found in the majority of cases, falsely justifying the impression that nothing can be found." Clearly, we must question the statement that 85% of LBD cases are idiopathic or have no definitive pathoanatomical cause.

At the World Congress for Lumbopelvic Pain in 1995, Professor Bogduk proposed many thought-provoking reasons to caution against defaulting to a psychosocial cause of back troubles, as follows:

- First consult: The usual practice of a 10-minute consult is simply poor practice because it provides no chance for a full assessment of causal mechanisms.
- Evidence-based management: Faith in passive interventions continues, leading to frustration in all parties given poor efficacy.
- Issue of certificate: Rather than engaging patients, uncovering causes of their troubles, and so on, a clinician makes the decree with the issuance of a certificate for time off, light duty, or so forth.
- Workplace intervention: Was the occupationally related cause of the back problem addressed?
- Correct treatment: Was the treatment appropriate or more a consequence of convenience or what may be socially correct?
- Investigations: Prolonged pain requires a thorough investigation. False positives from tests are an iatrogenic nightmare, and their possibility must be considered in these cases.
- Treatment of chronic LBP: Simply the wrong treatment or therapeutic exercise was pursued.
- Lodging a claim: The compensation board has made an incorrect decision.
- Lawyers: Encourage retainment of the disability for maximal claim.
- Expert witness: False witness claiming the cause to be psychosocial.

I must admit that I have had to deal with every one of these points while working with patients, medical management groups, and the legal process. They are important considerations.

Helpful Strategies for Undiagnosed LBDs

Some physicians are clearly frustrated with the delayed improvement of undiagnosed chronic LBD patients. This frustration, together with concern for the financial health of the compensation system, may have motivated the authors of the Fordyce report to emphasize psychosocial modulators rather than organically based variables to explain intolerance to certain types of activity. But dealing with frustration on the basis of false assumptions will not help the situation. What, then, are more useful approaches to treating undiagnosed chronic cases of LBD?

As noted earlier, many clinicians do not have the expertise or tools to diagnose back troubles at a tissue-based level. Provocative testing will enable many physicians to identify painful motions and loading. By integrating biomechanics with such testing, physicians may be aided in making functional diagnoses.

In a pioneering study, Delitto, Erhard, and Bowling (1995) suggested that appropriately classified back pain sufferers (those with functional classifications rather than tissue-specific diagnoses) do better with specific treatments. Further, as patients progress through the rehabilitation process, they seem to require different treatment approaches. For example, several studies suggest that manipulation can be beneficial for acute short-term troubles, whereas physical therapy and exercise approaches appear better for chronic conditions (Skargren, Carlsson, and Oberg, 1998). Brennan and colleagues (2007) strengthened this impression with a trial showing superior results from matching the therapy and exercise approach with the clinical test results from the patient. For example, those with newer pain, confined to the back, and with stiffer spine segments did better with manipulation (those with longer pain histories did not). Those with repeated episodes, aberrant motion, and painful movement catches did better with stabilization exercise, and those with pain centralization on either flexion or extension did better with exercises matched to the direction of relief. Fritz, Cleland, and Childs (2007) wrote a commentary discussing the evolution of patient classification, which included several refining notes for specific tests. A major remaining limitation of these studies is that none has assessed progressive treatments that change as the patient progresses through the rehabilitation process; rather, they all have assessed only single treatment approaches. Future studies must assess the efficacy of staged programs in which categorized patients follow progressive treatment involving several sequenced approaches.

In summary, the position suggesting that there is no detectable pathoanatomical basis for pain and activity intolerance in some patients and thus that these are functions of only psychosocial variables does not appear to be defendable. Improved tissue-based diagnosis, improved provocative testing and improved functional diagnosis, and a better understanding of the interactions of psychological variables with pathoanatomical variables support this notion and will continually evolve to help improve treatment outcomes.

Are Biomechanical Variables and Psychosocial Variables Distinct?

Are biomechanical variables and psychosocial variables distinct, or is there an interplay between them that, if understood, would underpin a more evidenced-based intervention program? Most reports have made a clear separation between psychosocial and biomechanical factors. But is there any evidence that psychosocial factors could modulate musculoskeletal loading—or vice versa?

Consider that highly respected pain scientists present volumes of empirical data demonstrating that pain perception is modulated by sensory, neurophysiological, and psychological mechanisms, suggesting that separating the two for analysis is folly (e.g., Melzack and Wall, 1983). Teasell (1997) argued quite convincingly that, although psychological factors have been cited as being causative of pain and disability, in fact, psychological difficulties arise as the consequence of chronic pain (see also Gatchel, Polatin, and Mayer, 1995; Radanov et al., 1994) and disappear upon its resolution (see also Hicks et al., 2005; Mannion et al., 2001; Wallis, Lord, and Bogduk, 1997). Interestingly, when depression is tracked as a variable, some evidence suggests that back pain is a predictor of depression (e.g., Currie and Wang, 2004), yet depression can predispose people to pain (Lepine and Briley, 2004). The evidence is compelling—pain and psychological variables appear to be linked in a bidirectional relationship.

Is there more direct evidence that psychosocial factors are inextricably linked with biomechanical factors? Marras and colleagues (2000) noted that certain personality factors, together with some psychosocial variables, appear to increase spinal loads by up to 27% in some personality types via muscular cocontraction. This appears to occur at moderate levels of loading, whereas biomechanical loading overrides any psychosocial effects under larger task demands. This conclusion was strengthened by a field study linking these general mechanistic observations with reported LBD in workers. In summary, LBDs appear to be associated with both loading and psychosocial factors, and these factors seem to be related and multifactorial. The same conclusion appears to be valid for many types of chronic pain conditions (Gamsa, 1990).

Does an expectation of impending pain and catastrophizing the expectation influence the eventual pain experience? It appears so. Edwards and colleagues (2008) identified a pathway whereby catastrophizing pain elevated levels of the proinflammatory cytokine interleukin-6. This cytokine increases pain sensitivity and is associated with persisting pain syndromes (Thacker et al., 2007). The next question is how to reduce catastrophizing in patients with back pain? Smeets and colleagues (2006) conducted a randomized controlled trial of 211 patients with back pain with three treatment groups: active physical treatments, cognitive-behavioral treatment, and a combination of the two. All three groups reduced pain and pain catastrophizing, whereas the control group did not. Treating the pain, even with approaches that are physical and do not include specific cognitive-behavioral components, is effective. This series of works provides some insight into the mechanism. This begs one final question: How effective are the purely psychosocial interventions for back pain? Van der Windt and colleagues (2008) reviewed the existing reports of negative efficacy and suggested that a refocus of the research agenda is needed for a better understanding of the potential. It may exist; it may not.

Consider this final tale and logic. My loving dog developed a painful knee. My simply reaching toward her knee caused her to sink her teeth into my arm as she recoiled. Was she displaying psychosocial disturbances causing her pain or a natural response to avoid mechanical loading that she knew would hurt her?

What Is the Significance of First-Time Injury Data for Cause and Prevention?

One of the best indicators of future back troubles is a previous history of back troubles (Bigos et al., 1991; Burton, Tillotson, and Troup, 1989; Troup et al., 1987). This suggests that studies of first-time back trouble episodes may be quite revealing for causative factors. Burton, Tillotson, Symonds, and colleagues (1996) studied police officers in Northern Ireland wearing >8 kg (18 lb) of body armor in a jacket (this additional load was borne by the low back). This group demonstrated a shorter period of time to their first onset of pain when compared to officers in an English force who did not wear the body armor. The authors also found that spending more than 2 hours per day in a vehicle constituted a separate risk for first-time onset of LBP. A recent study of American soldiers in Afghanistan found that wearing body armor was the most influential predictor of back pain incidence other than having a previous history of back pain (Roy, Lopez, and Piva, 2013). Another survey (Troup, Martin, and Lloyd, 1981) noted that falls among employees across a variety of industries were a common cause of first-time onset and were associated with longer periods of sick leave and a greater propensity for recurrence than were injuries caused by other mechanisms. (Ligamentous damage resulting from this type of loading is discussed in chapter 4.) It is also interesting to note that personal factors appear to play some role in first-time occurrence. Biering-Sorensen (1984) tested 449 men and 479 women for a variety of physical characteristics and showed that those with larger amounts of spine mobility and less lumbar extensor muscle endurance (independent factors) had an increased occurrence of first-time back troubles. Luoto and colleagues (1995) reached similar conclusions. Muscular endurance, and not anthropometric variables, appears to be protective.

Some injuries just happen as the result of motor control errors. (This interesting mechanism is introduced in chapter 4, in which we describe witnessing an injury using videofluoroscopy to view the spine.) These may be considered random events and may be more likely in people with poor motor control systems (Brereton and McGill, 1999).

How Do Biomechanical Factors Affect LBD?

Several approaches have provided evidence of links between biomechanical factors and LBD. A few are summarized in this section.

Mechanical Loading and LBD: Field-Based Risk Factors

Of the epidemiological studies that have focused on kinematic and kinetic biomechanical factors, a few investigated loading of low back anatomical structures. These would be considered to be the strongest evidence. The majority of the studies, however, assessed indirect measures that are linked to spinal loading, such as the presence of static work postures, frequent torso bending and twisting, lifting, pushing or pulling exertions, and exertion repetition. Although tissue overload is the cause of tissue damage and related back troubles, these indirect measures of load merely act as surrogates. The attraction of using surrogate measures rather than direct tissue loads per se for epidemiological study is that they are simpler to quantify and survey in the field. However, trade-offs exist among methodological utility, biological reality, and robustness. Risk factors related to specific tissue-based injury mechanisms are addressed in chapters 4, 6, and 7.

Several issues should be kept in mind when interpreting this literature. Virtually all reviews of the epidemiological literature (e.g., Andersson, 1991; Pope, 1989) have noted that specific job titles and types of work are associated with LBD (although LBD is defined differently in different studies). In particular, jobs characterized by the manual handling of materials, sitting in vibrating vehicles, and remaining sedentary are all linked with LBD. However, this type of data does not reveal much about the links between specific characteristics of the work and the risk of suffering LBD; specifically, a dose–response relationship has not been elucidated. Furthermore, a review of 57 papers that surveyed LBDs revealed no consistency between specific risk factors and the development of those disorders (Ferguson and Marras, 1997). This review demonstrated the large differences in the way surveillance was performed and in risk factor measurements. We are reminded once again that epidemiological approaches alone will not elucidate the biological pathway of the development of LBDs, a process that must be understood to develop optimal prevention and rehabilitation strategies.

As noted, the majority of risk factors that are addressed in the epidemiological literature (which is surprisingly sparse) are really surrogate factors, or indirect measures, of spine load. These surrogate factors are static work postures; seated work postures; frequent bending and twisting; lifting, pulling, and pushing; vibration (especially seated); and generation of spine power.

- Static work postures. Research has suggested that work characterized by static postures is an LBD risk factor. Although many studies have suggested a link with static work, the key paper on this topic was presented by Punnett and coworkers (1991), who reviewed 1,995 back injury cases from an auto assembly plant. Analyzing jobs for postural and lifting requirements, they found that LBDs were associated with postures that required maintaining mild trunk flexion (defined as the trunk flexed forward from 21 to 45°) (odds ratio = 4.9), postures involving maintaining severe trunk flexion (defined as the trunk being flexed forward greater than 45°) (odds ratio = 5.7), and postures involving trunk twisting or lateral bending greater than 20° (odds ratio = 5.9). Their results suggested that the risk of back injury increased with exposure to these deviated postures and increased with duration of exposure. Deviated postures greatly increase low back tissue loading, particularly when they must be held (Marras et al., 1993; McGill, 1997).
- Seated work postures. Kelsey (1975) linked the seated work posture to a greater risk of LBD. Subsequently, Liira and colleagues (1996) suggested that although white collar (sedentary) workers who must sit for long periods have a greater risk of low back troubles (8% increase in odds risk), active blue collar workers gain some prophylactic effect from sitting down (14% reduction in odds risk). This suggests that variable work, and not too much of any single activity, may reduce mechanically induced low back troubles. Further, nonneutral spine postures while sitting exacerbate the incidence of occupational low back pain, as noted in subway train and tractor operators (Lis et al., 2007) and in dentists who were not able to use backrests (Rafeemanesh et al., 2013). Using backrests can reduce spine loading up to three times by reducing the

muscle activity needed to sit upright (Morl and Bradl, 2013).

- Frequent bending and twisting. A U.S. Department of Labor report (1982) and many more studies (summarized by Andersson, 1981; Marras et al., 1995; Punnett et al., 1991; and Snook, 1982) noted the increased risk of LBD from frequent bending and twisting. In fact, Marras documented in several studies the increased risk of LBD with higher torso velocities (e.g., Marras et al., 1993, 1995). (Note that this is isolated spine motion and not nonspecific torso motion.) Although these studies did not examine a mechanism to explain a link with LBD, the associated motion within the spine will be shown in chapter 4 to form a pathomechanism for very specific disabling LBDs.
- Lifting, pulling, and pushing. In the United States, the National Institute for Occupational Safety and Health report (NIOSH, 1981) provides a good review linking activities requiring lifting, pushing, and pulling with increased risk of LBD.
- Vibration. Vibration, particularly seated vibration, is linked to elevated rates of LBD (e.g., Kelsey, 1975; Lis et al., 2007; Pope, 1989). Our work on cadaveric spines showed that exposure to vibration similar to occupational exposures increased the progression of disc herniation and any resulting herniations (Yates and McGill, 2011).
- Generation of spine power. The concept of power (force × velocity, or in the context of the spine, spine bending velocity with simultaneous muscle force) has not been well formulated because it is a variable that is calculated rather than measured directly. However, it appears that when the spine muscles are required to generate high forces, the motion needs to be very slow or static to reduce the risk. On the other hand, if the bending velocity is high, then the muscle forces need to be low. Either way, low power is required to minimize risk. This is substantiated by professor Marras' work linking velocity and acceleration to higher risk (Marras, 2008). In addition, in our own laboratory work we have begun experiments loading people's spines isometrically and then with various combinations of load and motion. We had to abandon the experiments as a result of pain generation when higher power levels were reached.

Although all of the risk factors noted here have been epidemiologically linked with an increased incidence of LBD, a subsequent section on tissue damage provides insight into the mechanisms linking mechanical overload, the onset of pain and disability, and the natural history of these injuries as they pertain to job performance and exposure.

Injury and Lasting Physiological, Biomechanical, and Motor Changes

This section addresses the substantial literature documenting performance deficits and subsequent anatomical changes in LBP populations. The following researchers documented a change in muscular function after injury:

- Hodges and Richardson, 1996, 1999. Delayed onset of specific torso muscles during sudden events may impair the spine's ability to achieve protective stability during situations such as slips and falls. These delays have been further refined to specific subcatagories of back pain patients by Silfies and colleagues (2009). Delays in muscular response have been observed to worsen when people focus on their pain, supporting the notion of a neurophysiological pathway (Butler et al., 2010).
- Arendt-Nielson et al., 1995. Changes in torso agonist–antagonist activity during gait.
- Zedka et al., 1999. Inhibition of back extensors in the presence of pain.
- Freeman, Mascia , and McGill, 2013. Inhibition of the gluteal muscles of the hip in the presence of pain.
- Grabiner , Koh , and Ghazawi , 1992. Asymmetrical muscle output during isokinetic torso extensor efforts that alters spine tissue loading.

Anatomical changes following low back injury include asymmetrical atrophy in the multifidus (Beneck and Kulig, 2012; Hides et al., 1994), particularly at the L4 and L5 segmental levels (Hides et al., 2008), although this is controversial. Fiber changes in the multifidus occur even 5 years after surgery (Rantanen et al., 1993). Further, in a very nice study of 108 patients with histories of chronic LBD ranging from 4 months to 20 years, Sihvonen and colleagues (1997) noted that 50% had disturbed joint motion, and 75% of those with radiating pain had abnormal electromyograms of the medial spine extensor muscles.

Finally, a study of those with a history of low back troubles has shown a wide variety of lingering deficits (McGill et al., 2003). The back troubles were sufficient to cause work loss, but the subjects had been back to work for an average of 270 weeks. Generally, those with a history of troubles were heavier and had disturbances in the flexor–extensor strength ratio and the flexor–extensor endurance ratio together with the lateral bend endurance ratio, had diminished gross flexion range of lumbar motion, and had lingering motor difficulties compromising their ability to balance and bend down to pick up a light object. None of these changes could be considered good.

The broad implication of this work is that a history of low back trouble, even when a substantial amount of time has elapsed since the trouble, is associated with a variety of lingering deficits such that a multidisciplinary intervention approach would be required to diminish their presence. This collection of evidence is quite powerful in documenting pathoneuromechanical changes associated with chronic LBD. These changes are lasting years—not 6 to 12 weeks!

Optimal Amount of Loading for a Healthy Spine

A lucid interpretation of the data in the epidemiological literature is limited by the fact that the levels at which tissue damage occurs remain obscure. Many are concerned with the known tissue damage that occurs with high magnitudes of load, repetition, and so on. For example, Herrin and colleagues (1986) found that musculoskeletal injuries were twice as likely when workers' lumbar spines were exposed to compressive forces that exceeded 6,800 N (predicted with a biomechanical model). On the other hand, several of the epidemiological studies have not been able to support a link between heavy work, when crudely measured, and the risk of LBD (e.g., Bigos et al., 1986; Porter, 1987). Mitigating factors appear to include the repetition of similar movements and variety in work.

In the discussion of the U-shaped relationship between activity levels and LBD in chapter 1, we saw that stress at optimal levels strengthens the system, whereas too little or too much is detrimental to health. Kelsey (1975) demonstrated a greater-than-expected increase in disc protrusions among sedentary workers. Although many consider sitting a low-load task, in fact, it creates damaging conditions for the disc—the mechanism of which is explained in chapter 4. This has obscured the emergence of much clearer relationships among biomechanical loading, disc herniation, clinical impressions, and the ability to perform demanding work. Further, Videman, Nurminen, and Troup (1990) studied a cross section of retired workers by comparing their LBD history with their magnetic resonance imaging (MRI) scans. A history of back pain and the visible parameters of spinal pathology were least prevalent in workers whose jobs had included moderate activity and most prevalent in workers with either sedentary or heavy work.

Many studies compare just two levels of work—for example, light versus heavy, light versus moderate, or moderate versus heavy. Because any relationship other than a straight line requires more than two points (or levels of activity or levels of loading), such studies cannot illustrate the U-shaped relationship. Consider the data compiled in a thorough epidemiological study reported by Liira and colleagues (1996) of LBD prevalence and physical work exposures. Even though the authors considered only the upper levels of excessive loading, they concluded that one-quarter of excess back pain morbidity could be explained by physical work exposures. That this is an underestimate is a very real possibility.

Furthermore, the nature of LBD appears to be affected by the type of work. Videman and colleagues (1990) noted a tendency among those who had had sedentary careers to have marked disc degeneration in later years, whereas those who had performed heavy work (defined as not only lifting but also requiring large trunk motions) tended to have classic arthritic changes in the spine (stenosis, osteophytosis, etc.). Recent work by the same group (Wang et al., 2012) noted a strong link between vertebral end-plate damage and pain frequency, secondary disc degeneration, and Schmorl's nodes in those with occupations classified as heavier (meaning more demanding). In a similar study, Battie and colleagues (1995) reported an apparent contribution of genetic factors to various age-related changes in the spine using monozygotic twins, given significantly greater similarities in spinal changes than would be expected by chance.

Porter (1987, 1992) performed two studies that furthered the notion of an optimal loading level for health. The first study tracked miners and nonminers treated at hospitals for back pain. Although significantly more miners reported back trouble compared with nonminers, significantly fewer were diagnosed with disc protrusions, whereas significantly more were reported to have stenosis and nerve root entrapment (conditions associated more with the arthritic spine according to the data of Videman, Nurminen, and Troup, 1990). The second study evaluated questionnaire results from 196 patients with symptomatic disc protrusion and 53 with root entrapment syndrome. They were asked about their history of heavy work between the ages of 15 and 20. Significantly more subjects with disc protrusion had done no heavy physical work in those early years. By contrast, more of those with nerve entrapment syndrome had done 5 full years of heavy work between 15 and 20 years of age. These collective data suggest that different work demands cause different spine conditions and perhaps that the optimal loading is different for different tissues. Nonetheless, the optimal activity appears to be varied work at a moderate level between sedentary and heavy work.

What constitutes an optimal load—a load that is not too much, not too little, not too repetitive, and not too prolonged? Currently, we are in need of assessment tools for determining optimal load to guide the intervention toward optimal health.

Links Between Personal Factors and LBD

Some personal factors such as muscle endurance (not strength) and less spine range of motion (not more) are prophylactic for future back troubles. These are addressed in part III, Low Back Rehabilitation. A few other personal factors are noted here.

A few personal factors appear to affect spine tissue tolerance according to the existing literature; age and gender are two examples. Jager, Luttmann, and Laurig (1991) compiled the available literature on the tolerance of lumbar motion units to bear compressive load that passed their inclusion criteria. Their results revealed that when males and females were matched for age, females were able to sustain only approximately two-thirds of the compressive loads of males. Furthermore, Jager and colleagues' data showed that within a given gender, the 60-year-old spine was able to tolerate only about two-thirds of the load tolerated by the 20-year-old spine. Keep in mind that age and gender are very simple factors.

It appears that other personal factors, such as poor motor control system fitness, can lead to a back injury during ordinarily benign tasks such as picking up a pencil from the floor. The modeling data of Cholewicki and McGill (1996) suggest that the spine can easily buckle during such a task. When the muscle forces are inherently low, a small motor error can cause rotation of a single spinal joint, placing all bending moment support responsibility on the passive tissues. Such scenarios do not constitute excessive tasks, but patients often report them to clinicians as the event that caused their injury. This phenomenon will not be found in the scientific literature, however. Many medical personnel would not record this event as the cause of injury because in many jurisdictions it would not be deemed a compensable injury. These types of injuries seem to be more influenced by the fitness of a person's motor control system than by factors such as strength. McGill, Sharratt, and Seguin (1995) noted that people differ in their ability to hold a load in their hands and breathe heavily. This is very significant because the muscles required to be continuously active to support the spine (and prevent buckling) are also used to breathe by rhythmically contracting. Those who must use their muscles to breathe in this way sacrifice spine stability. Interestingly, we have measured changing strategies in those with compromised lung elasticity (e.g., smokers, those with emphysema) who use their back extensors to assist with lung inflation, resulting in the perverse effect of enhancing stability at the expense of wearing down the endurance capacity of their back muscles (Wang and McGill, 2008). All of these deficient motor control mechanisms heighten biomechanical susceptibility to injury or reinjury (Cholewicki and McGill, 1996) and are highly variable personal characteristics.

As noted previously, muscle endurance is predictive of future episodes of back pain, however those studies measured back extensor endurance. A collection of works measuring gluteal and lateral torso muscles suggests that endurance deficits predict those who will develop pain (Gallagher, Nelson-Wong, and Callaghan, 2011; Marshall, Patel, and Callaghan, 2011; Nelson-Wong and Callaghan, 2010).

Additional factors other than simple load magnitude appear to modulate the risk of tissue damage. The mechanism of disc herniation provides an example. Although disc herniations have been produced under controlled conditions (e.g., Gordon et al., 1991), they have not been produced consistently. Our lab has been able to consistently produce disc herniations by mimicking spine motion and load patterns seen in workers (Callaghan and McGill, 2001). Specifically, it appears that only a very modest amount of spine compression force is required (only 800-1,000 N), but the spine specimen must be repeatedly flexed—mimicking repeated torso–spine flexion from continual bending to a fully flexed posture. The main relevance for this issue is that the way workers elect to move and bend influences the risk of disc herniation. This highlights the need to examine how workers move in standardized tests at the beginning of their careers while they still have virgin backs, making it possible to determine cause and effect. Recent evidence suggests that those with a history of back troubles are more likely to lift flexing the spine and not the hips, increasing the risk of back damage (McGill et al., 2003).

Studies of Exercise Intervention

So far we have introduced the notions that exercise alone is not optimal, and that effort must be directed to eliminate whatever is causing the symptoms. Also, a form of exercise that helps one person will hurt another. Each person is unique in terms of what causes pain, and the dosage that will cause less pain. We have reviewed how exercise can wind down pain sensitivity, strengthen injured tissue, enhance function, and improve movement to reduce the risk of tissue irritation—but only when the type and dose of exercise is tuned to the individual. Therefore, studies of exercise trials in which each person is given the same exercise and dose should not be expected to yield positive results, yet they do. In fact, the exercise studies have the strongest support for positive patient outcome (Bigos et al., 2009), although the effectiveness is for chronic, not acute, pain (Van Middlekoop et al., 2010). Some exercise trials used machines to isolate spine and back muscle movement and claimed good results in reducing pain. However, when these exercises were compared to nonmachine exercises, they were not shown to be superior (Helmhout et al., 2008). Concerns and issues with using machines are introduced later. Again, the studies usually do not report adequate descriptions of the actual exercises, dosage, or patient matching. The studies control the training sessions per week and do not fine-tune exercises for individual pain patterns. When exercise programs are individualized for injury and pain history, current fitness levels or deficits, training goals and other personal descriptors, efficacy increases.

Suni and colleagues implemented an exercise program combined with counseling and coaching to remove poor movement (documented in this book); effectiveness was shown for middle-aged men (2006) and military personnel (2013). Performing the big three exercises, documented in this book, reduced injury rates in women collegiate gymnasts (Durall et al., 2009). Note: Big three exercises was a term coined to describe the original stabilization exercises that we showed to be superior in challenging the spine stabilizing musculature in a way that spared the spine of applied load (McGill, 1998).

Practical Application: What the Evidence Supports

In summary, it is interesting to consider why only some workers become patients. There is no question that damage to tissue can be caused by excessive loading, and that damage causes pain. However, pain is a perception that is modulated by psychosocial variables in addition to physiological injury. Clearly, both psychosocial and biomechanical variables are associated with LBD and are important in preventing low back injury and the ensuing chronicity; collectively, the evidence from several scientific perspectives is overwhelming. The relative importance of either is often difficult to compare across studies because the metrics for each are different —biomechanical variables are reported in newtons, newton-meters, numbers of cycles, and so forth, whereas psychosocial variables are reported in ordinal scales linked to perception (independent risk factors can be compared using odds ratios). Some influential reports have ignored biomechanical evidence and promoted psychosocial variables as more important. However, no study of psychosocial variables has been able to conclusively establish causal links —only association. Some biomechanically based studies, together with the chronic pain literature, are strongly convincing in their establishment of both association and causality. Thank goodness! We can now proceed.

Back injury can begin with damage to one tissue, but this changes the biomechanical function of the joint. Tissue stresses change and other tissues become involved, leading to progressive deterioration with time. Tissue damage does not always result from too high a load magnitude. In the case of disc herniation, repetitive motion, even in the absence of large loads, seems to be a significant causative mechanism. The notion that tissues heal within 6 to 12 weeks and that longer-lasting work intolerance has no pathoanatomical basis appears to be false; tissue injury data and the science of chronic pain mechanisms strongly suggest otherwise.

Understanding the role of biomechanical, psychosocial, and personal factors as well as their interrelationships will build the foundation for better prevention in the future. The challenge is to develop variable tolerance guidelines, psychosocial guidelines, and higher-level medical practice codes.

On balance, the evidence supports the following statements:

- Biomechanical factors are linked to both the incidence of first-time low back troubles and absenteeism, and subsequent episodes.

- Psychosocial factors appear to be important as well, but may be more related to episodes after the initial back-related episode.
- Psychosocial and biomechanical factors appear to influence each other, in terms of both causation of work absence and the course of recovery.
- Treatment to reduce back pain often leads to a reduction in psychosocial issues, not the other way around. There is overwhelming evidence that once patients can obtain a sound night's sleep and can eliminate low-level chronic pain, their psychological constitution and mental toughness return.
- The relationship between loading and LBDs appears to be U-shaped, and the optimal load is at a moderate level.
- Low back tissue damage can initiate a cascade of changes that may cause pain and intolerance to certain activities, and these changes may be disruptive for up to 10 years in an unfortunate few.
- Many types of tissue damage can escape detection in vivo. Even gross damage visible during dissection is often not visible on medical images. Thus, nonspecific diagnosis does not rule out the presence of mechanical damage and must not be used to imply that mechanical factors are not related to LBD.

What Works Clinically

The approach developed in this book is what I use daily with patients and in my consulting for others. We follow up with every patient I see, and we know our clinical score. The patients who are referred to me must qualify by having pain and having been seen by several clinicians who either failed to get results or made the situation worse. These patients are considered the most difficult. I enjoy the challenge.

These challenging back patients have only one thing in common: a track record of failing to get better. Based on their intolerance (the motion or load that triggers symptoms), our follow-up revealed excellent outcome (i.e., cured with no remaining issue) as follows:

- 48% of patients with flexion intolerance
- 75% of patients with extension intolerance
- 80% of patients with flexion and extension intolerance
- 33% of patients with flexion and compression intolerance

When categorizing these people based on their diagnosis (the diagnosis resulted from our series of tests), our follow-up revealed excellent outcome (cured with no remaining issue) as follows:

- 47% of discogenic patients
- 78% of disc and facet patients

Responses to the question Did you experience an improvement after seeing McGill? were as follows:

- 19%: No improvement
- 23%: Moderate improvement
- 58%: Substantial improvement

Responses to the question Did you get worse? were as follows:

- 9%: Yes
- 91%: No

Responses to the question Do you continue with the exercise prescription? were as follows:

- 71%: Yes
- 21%: No (8% didn't answer)

What may be surprising is that I saw each patient for 3 hours, only once. The sessions included an assessment of movement quality and pain provocators, followed by education and instruction on moving to avoid their specific pain trigger, and a session with notes outlining a graded and progressive therapeutic exercise program tuned for them.

Others have implemented the approach in this book on a wide scale. You should expect better results with patients who have not yet become the clinical "failures." Suni and colleagues (2013) documented the outcome of a 6-month neuromuscular exercise and counseling program for new conscripts in the Finnish military who entered with no back pain. Days lost due to back pain were significantly reduced in the experimental subjects compared to the controls.

A Final Note

After warning you not to skip this traditionally boring chapter on epidemiological approaches, I hope that you have found value. Understanding the foundation for the opinions you may hear about back pain makes you an educated consumer. Scientists will find what they look for, and groups will ignore specific evidence when it suits their agendas. I have always found that people make better decisions when informed with fair and balanced evidence.

Chapter 3
Functional Anatomy of the Lumbar Spine

You have most likely already studied the basic anatomy of the spine. This chapter begins by revisiting some anatomical features, possibly in a way not previously considered. Then these features are related to normal function and injury mechanics to lay the foundation for the prevention and rehabilitation strategies that follow. I believe that clinicians and scientists alike who specialize in low back disorders do not devote sufficient effort to simply considering the anatomy. The answers to many questions relevant to the clinician can be found within an anatomical framework, wherein lies the mechanical foundation for preventing and rehabilitating back disorders. The understanding of injury mechanisms presented here will help ensure that you do not unknowingly include injury-exacerbating maneuvers in therapeutic exercise prescriptions.

Anatomy Trains

Many students of anatomy study cadavers and learn the names of the parts, but never learn their functional relevance. The cadaver shows the parts in a static and obviously dead state. This trite statement is a segue into the notion that a living body moves dynamically and that muscles influence joints other than the ones they cross. Thomas Myers wrote the groundbreaking book Anatomy Trains (2014), which introduced the notion of linkages between muscles and connective tissues that span many joints. Muscles, fascia, and other connective tissues transmit force along their length in a series arrangement. This is conventional thought. However, forces are also transmitted laterally, in a parallel sense to adjacent structures. On one hand, these anatomy trains create great efficiency in economy of movement and strength augmentation, but on the other, pathology in one component translates along the linkage affecting distant normal tissues.

Another logical extension of this concept of anatomical linkages is the greater functional role of many muscles and ligaments. For example, consider the gluteal muscles. The best anatomy student will state that they extend and externally rotate the femur via the hip joint. This is correct, yet naive. When a person is standing and squatting, using the gluteal muscles to extend the hips also extends the knees; thus, the gluteal muscles become powerful knee extensors. Poor use of the gluteal muscles eventually leads to knee overload and pain. We can now proceed with a discussion as anatomists, biomechanists, clinicians, and coaches.

Basic Neural Structure

The greatest athletes who are elite performers, and who avoid injury, are very wise in understanding the process of activating muscles and groups of muscles. They are masters at using imagery to control the motor unit recruitment process. We study these athletes to learn their processes so that we can employ these techniques with people who have painful backs. The basics of what you need to know about neural integration, some of which are introduced here, can be found in wonderful resources such as Kandel, Schwartz, and Jessell (2000).

Motion may occur from a conscious thought in the brain that instigates muscle activation, or from a more subconscious process involving an encoded pattern thought to reside in the spinal cord. Traumatic events can recode these patterns to perturbed states, as can chronic and acute pain. Rerecoding these perturbed patterns back to normal is an issue addressed in part III of this book.

Better links between training and neuroanatomy, neurophysiology, and rehabilitation can be found in my textbook Ultimate Back Fitness and Performance (McGill, 2014). Several relevant discussions illustrate, for example, why machines cannot create the many variations of force development within a muscle to stimulate all motor units. In the torso, for example, the oblique muscles have many neuromuscular compartments that must be stimulated with demand. Slow and isolationist approaches typical of bodybuilding do not offer a rich proprioceptive environment that provides variable motion, balance, force projection, and direction challenges involving the full linkage.

Another aspect for designing the best manual therapies is based in neuroanatomy. Although David Butler's book The Sensitive Nervous System (2000) is a wonderful neuroanatomical source, an introduction to the concepts is attempted here. Often, pain that is attributed to muscle turns out to be neurogenic. The tests described later in this book are founded on several principles. Anatomically, the spinal cord and all nervous tissues linked in series (lumbar nerve roots, the sciatic nerve, and so on) can be tensioned, released, mobilized, and flossed with specific and coordinated joint motions. Tensioning nerves only causes more pain—neurogenic pain cannot be stretched away. Sadly, too many patients with so-called tight hamstrings or sciatic symptoms pursue stretching programs that produce only temporary relief. This relief results from the activation of the stretch reflex in the back extensor muscles, but it typically lasts only about 20 minutes. The pain and stiffness return. It is often possible to break the cycle by replacing the stretching with neural mobilization. Butler described well the benefits of mobilizing a nerve along its entire tract together with the mechanics that create local tensions. With coordinated cervical, hip, knee, and ankle motion, the lumbar nerve roots, cauda equina, and sciatic tract can be mobilized and flossed without tensioning (shown in chapter 10). We have too many cases of intransigent sciatica cured with this approach to ignore it.

Vascular Anatomy

All spinal tissues have a vascular supply with the exception of the inner disc. The implications of the avascular nucleus are described in the section Intervertebral Disc later in this chapter. The curious case of vertebral veins is introduced here. The veins leaving the vertebral bodies are the only veins in the body known to lack valves. Venous valves prevent the backflow of blood. But as we will see shortly in the discussion of vertebral mechanics, this anatomic feature is critical. It appears that the veins act as a hydraulic outlet from the vertebral body, enabling the expulsion of blood under high compressive loading. In this way both the arteries and the veins may provide a protective mechanism and the ultimate hydraulic shock-dampening system.

Vertebrae

As you undoubtedly know, the spine has 12 thoracic and 5 lumbar vertebrae. The construction of each vertebral body may be likened to a barrel with round walls made of relatively stiff cortical bone (see figure 3.1). The top and bottom of the barrel are made of a more deformable cartilage plate (end plate) that is approximately 0.6 mm (0.02 in.) thick but thinnest in the central region (Roberts, Menage, and Urban, 1989). The end plate is porous to allow for the transport of nutrients such as oxygen and glucose, whereas the inside of the barrel is filled with cancellous bone. The trabecular arrangement within the cancellous bone is aligned with the stress trajectories that develop during activity. Three orientations dominate—one vertical and two oblique (Gallois and Japoit, 1925) (see figure 3.2).

Figure 3.1 The parts of a typical lumbar vertebra. (a) Lateral view, (b) superior view, and (c) posterior view.

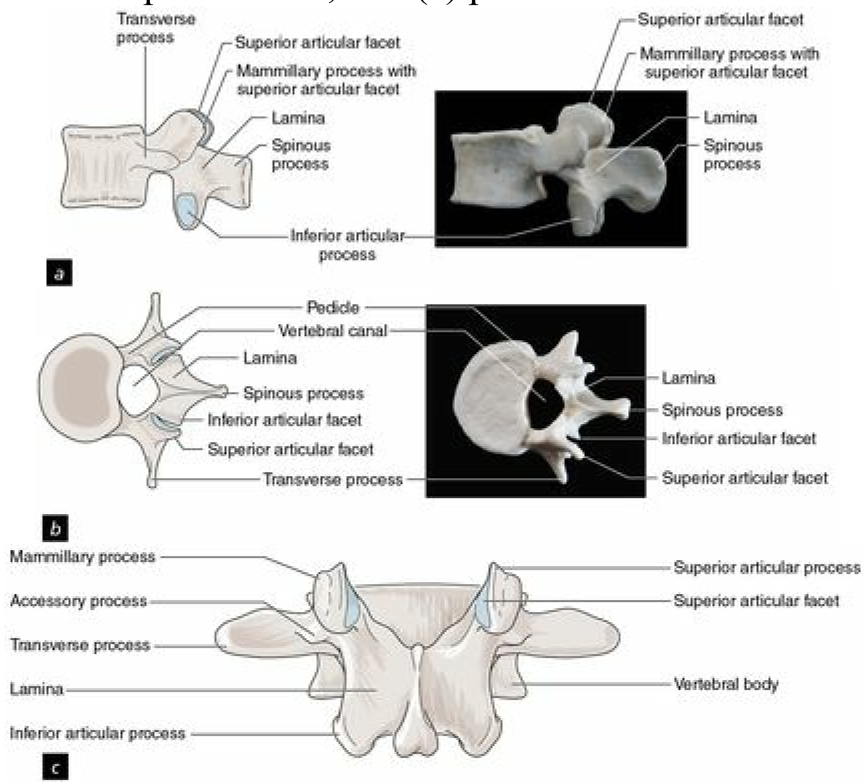
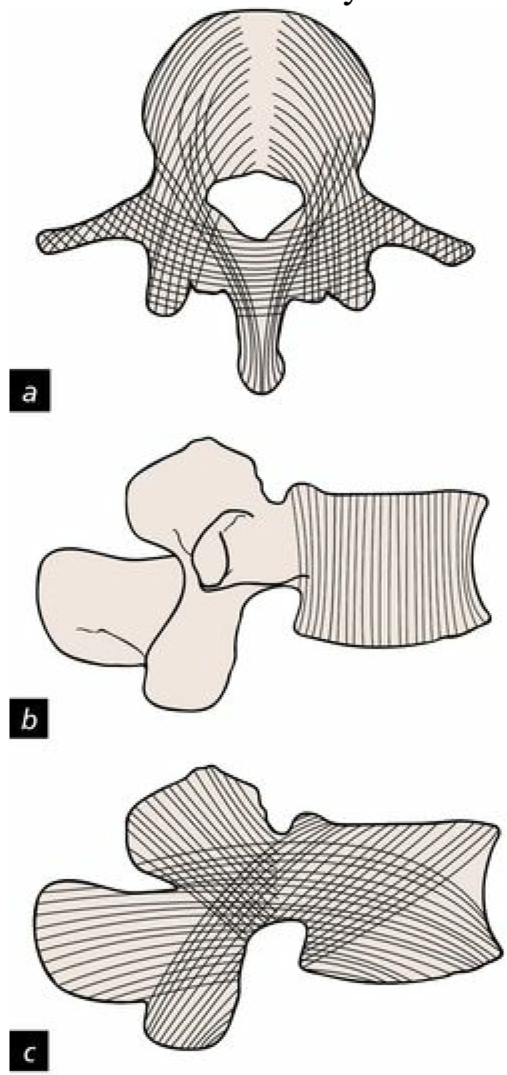


Figure 3.2 The arrangement of the trabeculae (first noted by Gallois and Japoit in 1925) is aligned with the dominant trajectories of stress. (a , b, c) The three trabecular systems.



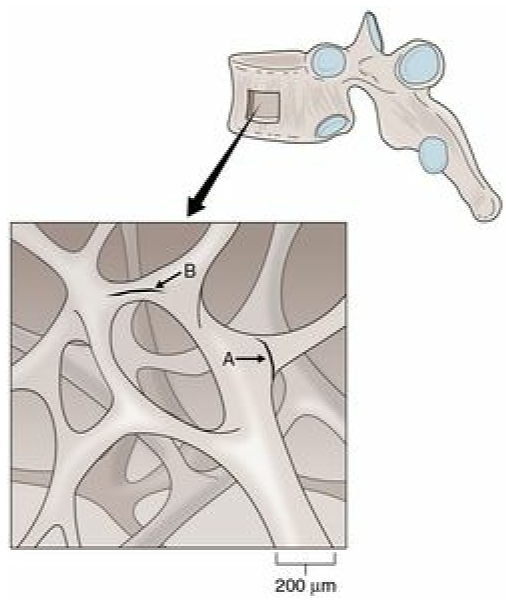
Vertebral Architecture and Load Bearing

The very special architecture of the vertebral bodies determines how they bear compressive load and fail under excessive loading. The walls of the vertebrae (or sides of the barrel) remain rigid upon compression, but the nucleus of the disc pressurizes (the classic work is by Nachemson, 1960, 1966) and causes the cartilaginous end plates of the vertebrae to bulge inward, seemingly to compress the cancellous bone (Brinckmann, Biggemann, and Hilweg, 1989). In fact, under compression the cancellous bone fails first (Gunning, Callaghan, and McGill, 2001), making it the determinant of failure tolerance of the spine (at least when the spine is not positioned at the end range of motion). It is difficult to injure the disc annulus this way (annular failure is discussed later).

Although this notion is contrary to the concept that the vertebral bodies are rigid, the functional interpretation of this anatomy suggests a very clever shock-absorbing and load-bearing system. Farfan (1973) proposed the notion that the vertebral bodies act as shock absorbers of the spine, although he based this more on vertebral body fluid flow than on end-plate bulging. He suggested that the discs were not the major shock absorbers of the spine, contrary to virtually any textbook on the subject. Because the nucleus is an incompressible fluid, bulging end plates suggest fluid expulsion from the vertebral bodies, specifically blood through the perivertebral sinuses (Roaf, 1960). This mechanism suggests a protective dissipation upon quasistatic and dynamic compressive loading of the spine. The case study literature abounds with compression fractures of the vertebral body during dynamic loading in which the disc remained intact (e.g., during tobogganing and sledding; Kelly and Robinson, 2003). More vertebral body–based shock-absorbing mechanisms are documented later. In summary, the common statement found in many textbooks that the discs are the shock absorbers of the spine now appears questionable; rather, the vertebral bodies appear to play a dominant role in performing this function.

The notion of deformable vertebrae is a new one for many. How do the end plates bulge inward into seemingly rigid bone? The answer appears to be in the architecture of the cancellous bone. Vertebral cancellous bone structure is dominated by a system of columns of bone (shown in figure 3.2) that run vertically from end plate to end plate. The vertical columns are tied together with smaller transverse trabeculae. Upon axial compression, as the end plates bulge into the vertebral bodies, these columns experience compression and appear to bend. Under excessive compressive load, the bending columns buckle as the smaller bony transverse trabeculae fracture, as documented by Fyhrie and Schaffler (1994) (see figure 3.3). In this way, the cancellous bone can rebound back to its original shape (at least 95% of the original unloaded shape) when the load is removed, even after suffering fracture and delamination of the transverse trabeculae. This architecture appears to afford excellent elastic deformation, even after marked damage, and then to regain its original structure and function as it heals. Damaged cancellous fractures appear to heal quickly, given the small amount of osteogenic activity needed, at least when compared with the length of time needed to repair collagenous tissues.

Figure 3.3 Under compressive loading, bulging of the end plate causes buckling stresses in the vertical trabeculae, which, when excessive, cause damage in the transverse trabeculae. Note (A) the vertical (from compression) and (B) horizontal (from tension) cracks in the transverse trabeculae.



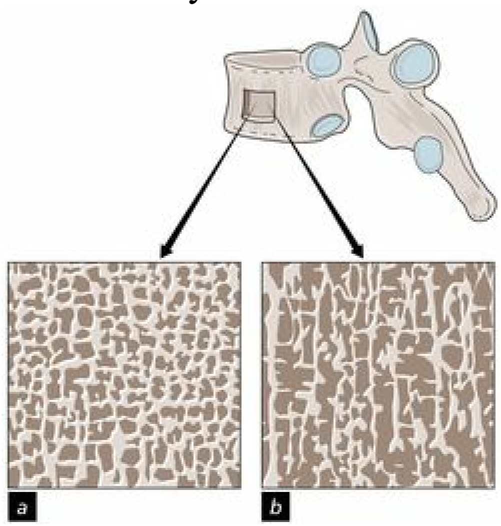
Understanding Vertebral Mechanics

To truly appreciate vertebral behavior, obtain a vertebra from a butcher (bovine or porcine is ideal). Hold the vertebra end plate to end plate between your thumb and finger and squeeze. If you have never done this before, you will be amazed by the deformation and elasticity. The vertebra experiences similar deformation as the incompressible nucleus of the disc presses over the central end plate during spine compression in vivo.

Microfracturing of the trabeculae can occur with repetitive loading at levels well below the failure level from a single cycle of load. Lu and colleagues (2001) demonstrated that cyclic loading at 10% of ultimate failure load caused no damage or change in stiffness; but with 20,000 cycles of load at 20 to 30% of the ultimate failure load, both stiffness and energy absorbed at failure were decreased. Highly repetitive loads, even at quite low magnitudes, appear to cause microdamage.

The osteoporotic vertebra is characterized by mineral loss and declining bone density in the trabeculae. Because transverse trabeculae are far fewer in number than longitudinal trabeculae, and because they are generally of smaller diameter, the transverse trabeculae specifically are the target for mechanical compromise with osteoporotic mineral loss (Silva and Gibson, 1997) (see figure 3.4). Interestingly, the same authors noted a higher tendency for the transverse trabeculae to disappear in females with greater incidence than in males. This loss in mechanical integrity of the transverse trabeculae has a great influence on the compressive strength of the vertebrae via the mechanism described earlier. Thus, the osteoporotic vertebra begins to slowly collapse when exposed to excessive load, with serial buckling of the columns of bone, ultimately developing the classic wedge shape.

Figure 3.4 With aging, the transverse or horizontal trabeculae thin and eventually lose their ability to support the vertical trabeculae, which can then buckle, causing vertebral collapse. (a) A healthy trabecular bone network from a 47-year-old woman. (b) Perforation in a horizontal trabecula in an elderly woman.



It is interesting to contrast the other extreme of the bone density spectrum. The transverse trabeculae harvested from specimens who performed heavy work (in particular, weightlifters) were thick and dense. In addition, where the transverse trabeculae intersected with the vertical columns, the joints were characterized by heavy bony gusseting, similar to what a welder would weld to strengthen a right-angled joint. The transverse trabeculae appear to be crucial in determining compressive strength.

In yet another observed failure mechanism of the vertebral body, termed the slow crush, extensive trabecular damage is observed without concomitant loss of stiffness or abrupt change in the load–deformation relationship (Gunning, Callaghan, and McGill, 2001) (see figure 3.5). Because slope change in the load–deformation relationship is often used to identify the yield point, or the initial tissue damage, this injury can go unnoticed. Interestingly, vertebrae failing under ever-increasing compressive load gradually increase in stiffness, a testament to the wonderful architecture of the transverse and vertical trabeculae. Also interesting is the load-rate dependence of bone strength: the end plate appears to fail first at low load rates, whereas the vertebral bony elements fail at higher load rates (see figure 3.6).

Figure 3.5 (a) Massive trabecular damage found during the dissection and removal of marrow following an excessive slow crush compressive load. (b) Higher magnification of the crush fracture. Even this massive fracture was not clear on X-ray or any other examination method.

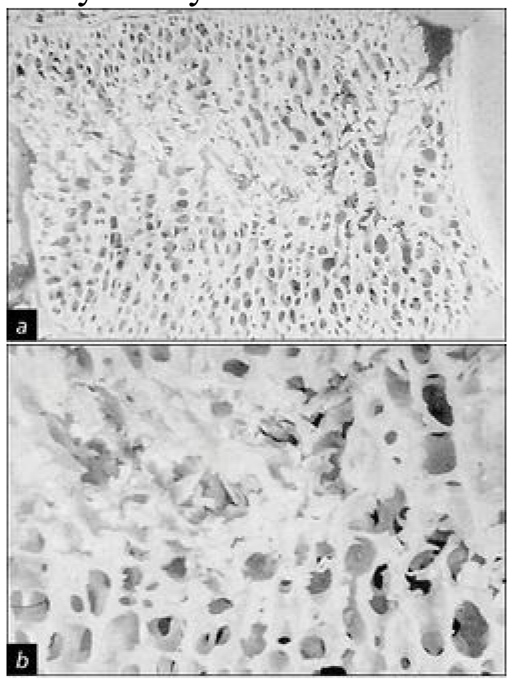
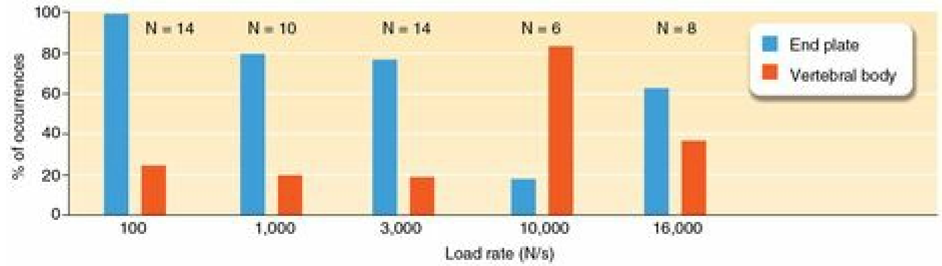


Figure 3.6 Compression injuries at various load rates. At low rates of compressive load, the end plate appears to be the first structure to fail, but bone will fracture first under higher rates of load.



Both the disc and the vertebrae deform while supporting spinal loads. Under excessive compressive loading, the bulging of the end plates into the vertebral bodies also causes radial stresses in the end plate sufficient to cause fracture in a stellate pattern (see figure 3.7). These fractures, or cracks, in the end plate are sometimes sufficiently large to allow the liquid nucleus to squirt through the end plate into the vertebral body (McGill, 1997) (see figures 3.8 and 3.9). Sometimes a local area of bone collapses under the end plate to create a pit or crater that goes on to form the classic Schmorl's node (see figure 3.10). This type of injury is associated with compression of the spine when the spine is within the neutral range of motion (i.e., not flexed, bent, or twisted). In my experience, this type of compressive injury is very common and often misdiagnosed as a herniated disc because of the flattened interdiscal space seen on planar X-rays. However, note that in end-plate fractures, the annulus of the disc remains intact. It is simply a case of the nucleus leaving the disc and progressing through the end plate into the cancellous core of the vertebra (sometimes referred to as a vertical herniation). Over the years we have compressed over 400 spinal units in a neutral posture, and all but 2 resulted in end-plate fractures as the primary tissue damage.

Figure 3.7 A stellate-patterned end-plate fracture (indicated with arrow) occurs as the nucleus is pressurized under compressive load, which causes it to bulge the end plate, imparting tensile stresses.

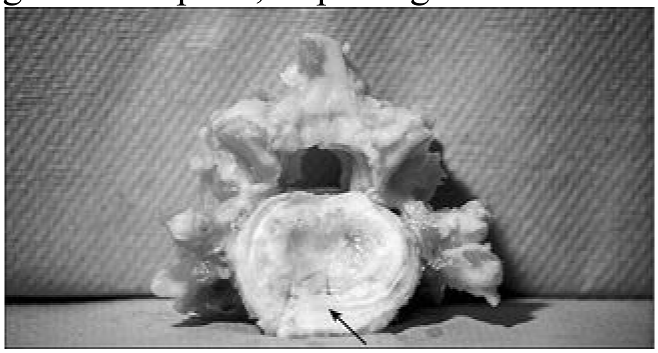


Photo from Stuart McGill

Figure 3.8 Under compressive loading, the nucleus pressurizes, causing the end plate to bulge into the vertebral body. With excessive radial–tensile stress, the end plate will fracture and the viscous nucleus will squirt through the crack into the vertebral body.

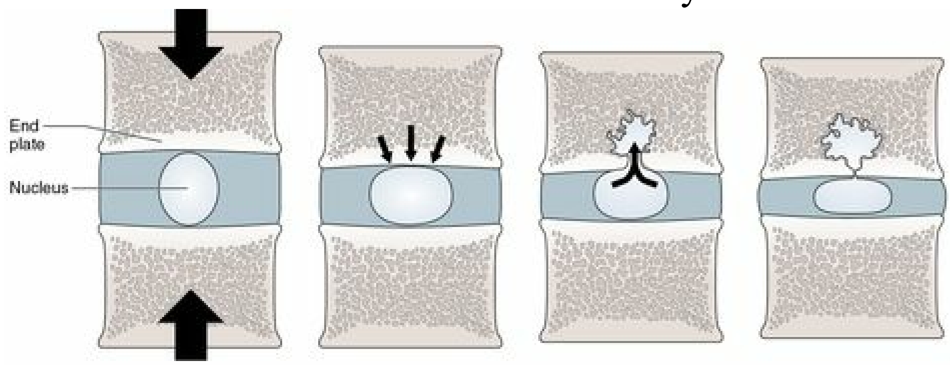


Figure 3.9 Following a more severe end-plate fracture, a portion of the nucleus has squirted through into the vertebral body (shown at the tip of the scalpel and the arrow). This is a porcine specimen.

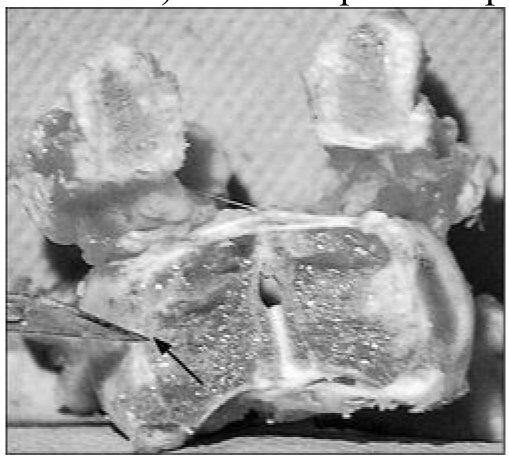


Photo from Stuart McGill

Figure 3.10 In addition to end-plate fractures, other injuries to the end plate under compressive load include pits, which occur as trabecular bone fractures in small areas under the end plate. These go on to form Schmorl's nodes.

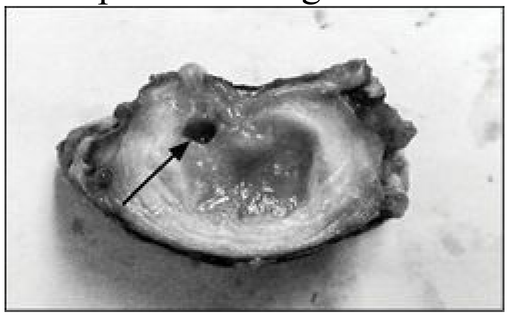
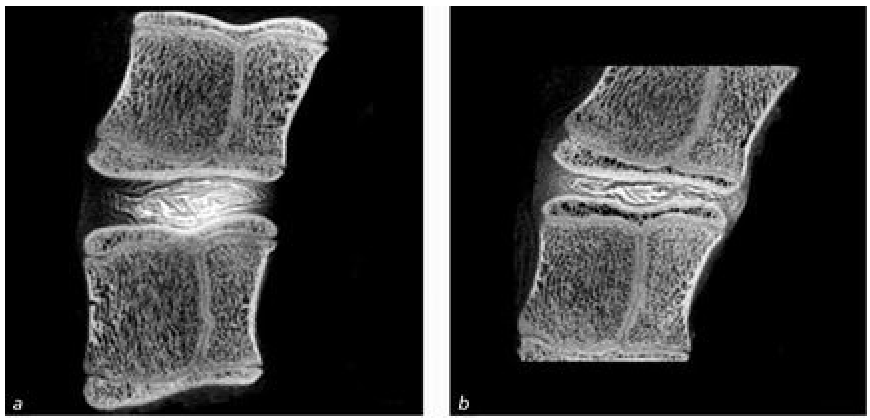


Photo from Stuart McGill.

Our most recent work has mimicked compressive loads similar to those imposed during heavy lifting, such as performing deadlift exercises. Using micro-CT imaging, we documented a crushing of the trabecular framework across the thin growth plate (see figure 3.11) (Balkovec et al., 2014). Although this damage would be clinically important, the resulting weakening of the end plate would make it susceptible to secondary end-plate fracturing and Schmorl's nodes.

Figure 3.11 Micro-CT images reveal the crushed growth plate from compressive loading mimicking a very heavy lift. (a) A virgin spine specimen prior to mechanical loading. (b) The same specimen after undergoing a compressive load with a flexion bending protocol mimicking a heavy deadlift session. Note the distinct regions directly beneath the end plate where the trabecular bone appears to have fractured and the density is reduced.



Reprinted from Journal of Biomechanical Engineering, Vol. 35(6), C. Balkovec, J. Vernengo, and S.M. McGill, "The use of a novel injectable hydrogel nucleus pulposus replacement in restoring the mechanical properties of cyclically fatigued porcine intervertebral discs," pp.: 61004-61005, copyright 2014, with permission of Elsevier.

Osteophytes are a characteristic of older spines classed as degenerated. Michael Adams' group (Al-Rawahi et al., 2011) assessed whether these are degenerative or adaptive. Removing them reduced the ability of the joint to withstand bending loads by up to one-half. Losses in compression resistance were less than 20%. They concluded that osteophytes are an adaptation to joint instability, which stimulates their formation. Further, the presence of osteophytes increased measures of bone mineral density, but such measures may actually underestimate vertebral compressive strength.

End-Plate Fractures

In my experience, end-plate fracture with the loss of nuclear fluids through the crack into the vertebral body (often forming Schmorl's nodes) is a very common compressive injury and perhaps the most misdiagnosed. Loss of the disc nucleus results in a flattened interdiscal space that, when seen on planar X-rays, is usually diagnosed as a herniated disc or degenerated disc.

However, the annulus of the disc remains intact. It's simply a case of the nucleus squirting through the end-plate crack into the cancellous core of the vertebra. True disc herniation requires very special mechanical conditions that are described shortly. When compressing spines in the lab, we hear an audible pop at the instant of end-plate fracture—exactly what patients report when they describe details of the event that resulted in their pain. I also strongly suspect that some fractures are somewhat benign in that no immediate severe pain occurs. Others, however, may be instantaneously acute; it depends on the biomechanical changes that accompany the fracture. If there is substantial loss of the nucleus from the disc (i.e., it is vertically herniated), then immediate loss of disc height and subsequent compromise of nerve root space will result. At this point the end-plate fracture will mimic the symptoms of true herniation—another reason for the common misdiagnosis.

Modic Changes and What They Mean

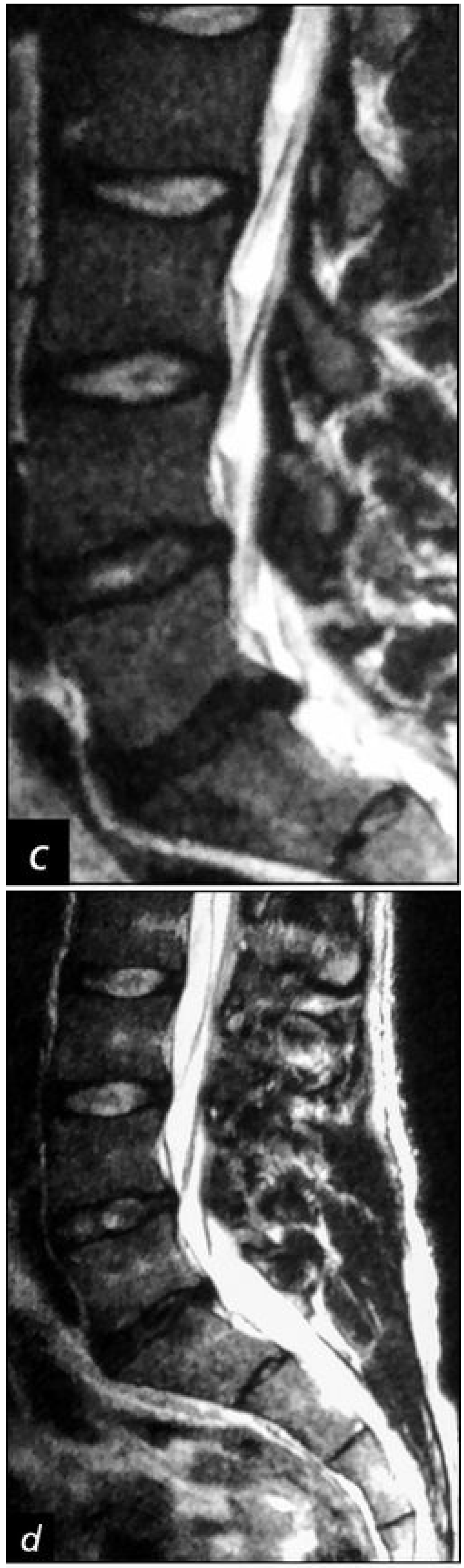
MRI images sometimes show signals in the vertebral body along the end plate. These signals, called Modic changes after Dr. Modic (Modic et al., 1988), have sparked much discussion regarding their clinical significance, if any. Our experience strongly suggests that these findings represent edema in the bone. The edema can have two causative pathways. First, an end-plate fracture sufficient to allow nucleus material to leak into the vertebral body sets off a substantial inflammatory response. The immune system and vascular environment respond to the invading nucleus as foreign material, setting off the reaction. This mechanism suggests damage to the cartilagenous end plate. This is consistent with the report of Zhi and colleagues (2014), who noted more hyaline end-plate cartilage particulate in harvested herniated material during surgery in those with Modic changes than in those without.

The second causative pathway of edema is consistent with what is called a bone bruise. Several athletes we have seen had well-documented training histories as well as annual records of repeated MRI images. These case studies enabled us to analyze the associations among loading, progressive changes in joint function, and MRI images. A typical example would be a person who shows end-plate damage but no other MRI changes. Over the next few years, the joint slowly flattens and loses water signal in the disc.

Then anterior margins of the vertebrae begin to collide during flexion motion (see figure 3.12). Modic changes then appear in the bone at these contact points. I refer to these as bone bruises, because pain is now reported central in the spine, at the damaged level upon flexion movement. Once Modic changes have been documented, longer-lasting pain can be expected; our experience suggests that 12 to 18 months is typical. Avoiding this movement reduces pain sensitivity.

Figure 3.12 The history of a professional athlete showing initial end-plate damage in 2009, disc height loss by 2009, and colliding anterior margins of the vertebrae with bone bruise formation by 2011. (a) Schmorl's node at L5, 2008. (b , c, d) Disc bulge, no bone bruise, 2009. (e , f, g) Bone bruise at L5 and S1, retrolisthesis, October 2011.


b


c
d


e

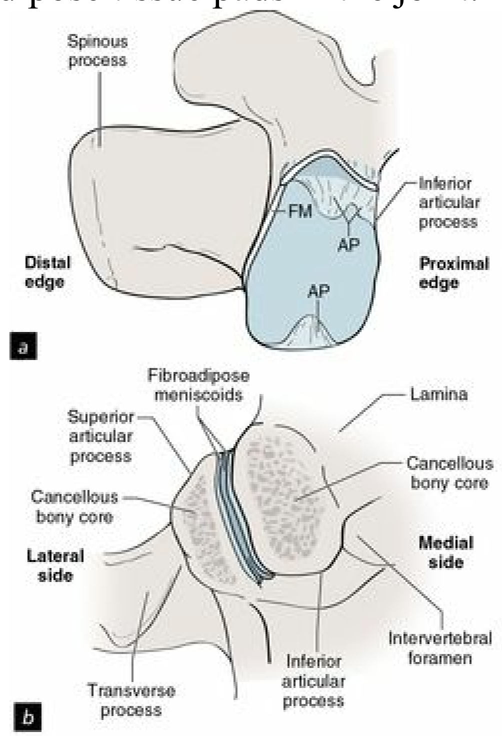

f

Photo from Stuart McGill

Posterior Elements of the Vertebrae

The posterior elements of the vertebrae (pedicles, laminae, spinous processes, and facet joints) have a shell of cortical bone but contain a cancellous bony core in the thick sections. The transverse processes project laterally together with a superior and an inferior pair of facet joints (see figure 4.1). On the lateral surface of the bone that forms the superior facets are the accessory and mamillary processes that, together with the transverse process, are major attachment sites of the longissimus and iliocostalis extensor muscle groups (described later). The facet joints are typical synovial joints in that the articulating surfaces are covered with hyaline cartilage and are contained within a capsule. Fibroadipose enlargements, or meniscoids, are found around the rim of the facet, although mostly at the proximal and distal edges (Bogduk and Engel, 1984), which have been implicated as a possible structure that could bind and lock the facet joint (see figure 3.13, a and b).

Figure 3.13 (a) Lateral view of the inferior articular process, revealing the facet, the fibroadipose meniscoids, and the adipose tissue pad, which have been implicated in joint binding. (b) Cross section of the posterior view of the facet joint, showing the positions of the fibroadipose meniscoids and the adipose tissue pads in the joint.

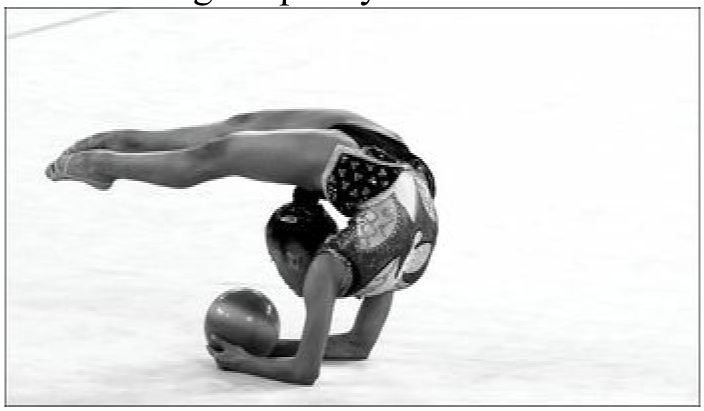


The neural arch in general (pedicles and laminae) appears to be somewhat flexible. In fact, Bedzinski (1992) demonstrated flexibility of the pars interarticularis during flexion–extension of cadaveric spines. Dickey, Pierrynowski, and Bednar (1996) documented up to 3° changes of the right pedicle with respect to the left pedicle during quite mild daily activities using pedicle screws in vivo. Failure of these elements together with facet damage, leading to spondylolisthesis, is sometimes blamed exclusively on anteroposterior shear forces. However, a case could be made from epidemiological evidence in athletes such as gymnasts and Australian cricket bowlers (Hardcastle, Annear, and Foster, 1992) that the damage to these posterior elements may also be associated with full range of motion. In fact, the faster the bowler, the higher the risk of fatigue fracture (Ranawat, Dowell, and Heywood-Waddington, 2003). The cyclic full spine flexion and extension in these sorts of activities fatigue the arch with repeated stress reversals.

Flexibility of the Posterior Elements of the Vertebrae

In the preceding sidebar Understanding Vertebral Mechanics, I recommended obtaining a vertebra from the butcher. Grasp the vertebral body in one hand and the whole neural arch in the other. Bend the arch up and down and note the flexibility. This flexural displacement occurs in each cycle of full spine flexion–extension common in events such as women's gymnastics (figure 3.15). If this cycling continues, the stress reversals eventually cause a fatigue crack in the pars. Further repetition causes the crack to propagate through the full width of the pars, eventually resulting in fracture—and in vivo, the condition of spondylolisthesis.

Figure 3.15 Repetitive gymnastics moves such as this cause stress–strain reversals in the pars. In sufficient numbers they result in fatigue fracture—leading to spondylolisthesis.



Shariffc | Dreamstime.com - Gymnastics Action Photo

On the other hand, there is no doubt that excessive shear forces cause injury to these posterior vertebral elements. Posterior shear of the superior vertebrae can lead to ligamentous damage but also failure in the vertebra itself, because the end plate often avulses from the rest of the vertebral body (Yingling and McGill, 1999b) (see figure 3.14). Both our lab observations and discussions with international colleagues have reinforced our suspicion that this type of failure may be more common in the adolescent and geriatric spine than in the young and middle-aged adult spine. Further work is needed for confirmation.

Figure 3.14 Shear injuries include fracture of the facet base and, on occasion, end-plate avulsion from the vertebrae.

Photo from Stuart McGill

Cripton and colleagues (1995) documented that anterior shear of the superior vertebra causes pars and facet fracture leading to spondylolisthesis with a typical tolerance of an adult lumbar spine of approximately 2,000 N. Although similar injury mechanisms and tolerance values were observed in young porcine spine specimens (Yingling and McGill, 1999a, 1999b), the type of injury appeared to be modulated by loading rate. Specifically, anterior shear forces produced soft tissue injury at low load rates (100 N/s), but fractures of the pars, facet face, and vertebral body were observed at higher load rates (7,000 N/s). Posterior shear forces applied at low load rates produced soft tissue failure and vertebral body fracture, whereas those at higher load rates produced wedge fractures and facet damage.

Although shear tolerance of the vertebral motion unit appears to be in the range of 2,000 to 2,800 N for one-time loading, Norman and colleagues (1998) noticed an increase in reported back pain in vivo in jobs that exposed workers to repetitive shear loads greater than 500 N. We consider these the best guidelines currently available.

Neural Arch Fracture

Spondylolisthesis and neural arch defects are endemic among female gymnasts and cricket bowlers, to name a couple of athlete types. Patients with spondylolisthesis generally do not do well with therapeutic exercise that takes the spine through the range of motion; rather, stability should be the rehabilitation objective. Ranawat, Dowell, and Heywood-Waddington (2003) made a strong case for conservative approaches, which were successful even with cricket bowlers: only a few needed follow-up surgical interventions. I have been involved in litigation cases in which clear spondylolisthesis existed but was alleged to be related to a specific event (e.g., a recent automobile accident). In surgery, however, there was evidence of substantial osteogenic activity, suggesting that the injury was quite old. In fact, these patients (former gymnasts) must have had this damage while competing, but they were so fit and their spines were so stable that they were able to remain in competition. Following retirement and after having children and losing fitness, a rather minor event became the instigator of their symptoms.

A final note is relevant regarding the repeated and prolonged extension postures and motions associated with the McKenzie approach discussed later in this book. Some people have what are called kissing spines, in which adjacent posterior spines collide in full extension at one level. The involved level is usually due to a simple case of anatomical variation. These may become more frequent in people with disc height loss when the posterior spine becomes more approximated. Jim Taylor (Twomey and Taylor, 1987) noted destructive changes in the interspinous ligaments as they are repeatedly crushed at the kissing spine level. He described the changes as the development of a "fibrocartilaginous covering on the colliding bone and a bursa-like cavity surrounded by fat lined with a synovial membrane." There is no question regarding the efficacy of the McKenzie extension routines for some people with acute discogenic back issues. I am cautious about having people continue to engage in these postures following recovery from the acute episode, however—the spine may pay a price.

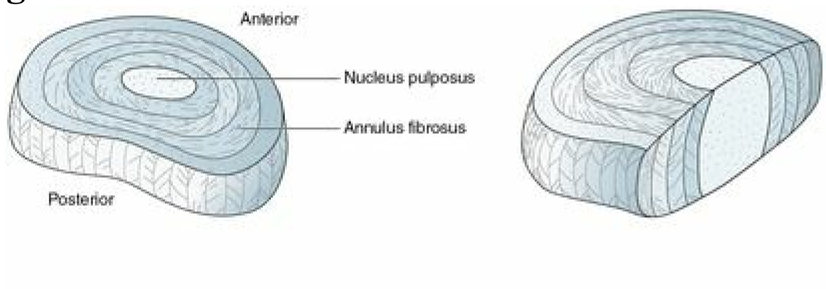
Sacroiliac Joints

The sacroiliac (SI) joints are quite large and have little mobility. This means that large forces are often required during provocative testing. There has been much discussion over the years about the existence of measurable movement, whether there should be movement, whether the movement can be asymmetrical between right and left sides, and how much difference is required to be clinically detectable. The majority of research has been focused on test reliability rather than on mechanisms and clinical technique. As noted later, painful symptoms reported in the sacroiliac joint region are often referred from discogenic disorders irritating sciatic nerve roots. After using diagnostic injections, Young, Aprill, and Laslett (2003) stated that SI joint pain rarely occurred above the L5 level, or around the midline; rather, they stated that it was much more likely to occur unilaterally (exacerbated by rising from a chair) and was correlated with three positive SI joint pain provocation tests. However, like all tests, this depends on clinicians' ability to direct force to the joints while sparing surrounding tissues.

Intervertebral Disc

The intervertebral disc has three major components: the nucleus pulposus, annulus fibrosus, and end plates. The nucleus has a gel-like character with collagen fibrils suspended in a base of water and various mucopolysaccharides, giving it both viscosity and some elastic response when perturbed in vitro. At the risk of sounding crude, the best way to describe a healthy nucleus is that it looks and feels like heavy phlegm. During in vitro testing, we have had it squirt out under pressure and literally stick to the wall. Although there is no distinct border between the nucleus and the annulus, the lamellae of the annulus become more distinct, moving radially outward. The collagen fibers of each lamina are obliquely oriented (the obliquity runs in the opposite direction in each concentric lamella). The ends of the collagen fibers anchor into the vertebral body with Sharpey's fibers in the outermost lamellae, whereas the inner fibers attach to the end plate (the end plate was discussed earlier). The discs in cross section resemble a rounded triangle in the thoracic region and an ellipse in the lumbar region, suggesting anisotropic facilitation of twisting and bending (see figure 3.16).

Figure 3.16 Cross section of the intervertebral disc.



Load-Bearing Abilities

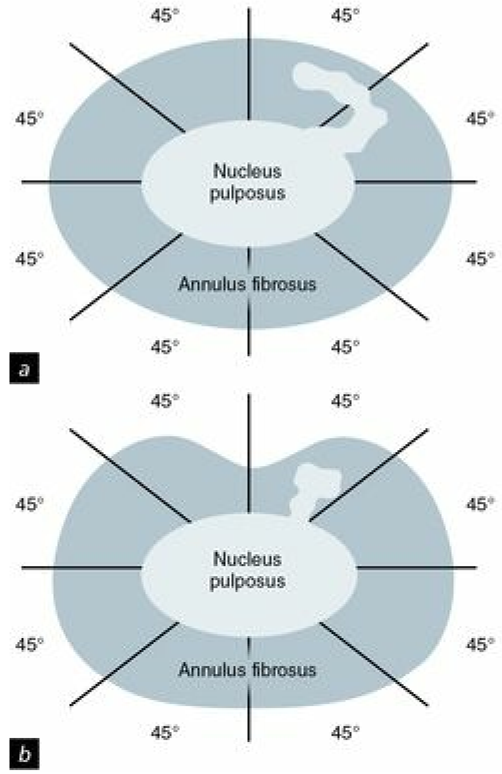
The disc behaves as a hydrostatic structure that allows 6° of motion between vertebrae. Its ability to bear load, however, depends on its shape and geometry. As a result of the orientation of the collagen fibers within the concentric rings of the annulus (one-half of the fibers are oblique to the other half), the annulus is able to resist loads when the disc is twisted. However, only half of the fibers can support this mode of loading; the other half become disabled, resulting in a substantial loss of strength or ability to bear load. The annulus and the nucleus work together to support compressive load when the disc is subjected to bending and compression. Under spine compression, the nucleus pressurizes, applying hydraulic forces to the end plates vertically and to the inner annulus laterally. This causes the annulus collagen fibers to bulge outward and become tensed. Years ago, Markolf and Morris (1974) elegantly demonstrated that a disc with the nucleus removed lost height but preserved much of its properties of axial stiffness, creep, and relaxation rates. The fact that the nucleus appears necessary to preserve disc height has implications for facet loading, shear stiffness, and ligament mechanics.

It is noteworthy that disc damage is most often accompanied by subdiscal bone damage (Gunning, Callaghan, and McGill, 2001). In fact, Keller and colleagues (1993) noted the interdependence of bone status and disc health. Evidence also suggests that excessive compression can lead to altered cell metabolism within the nucleus and increased rates of cell death (apoptosis) (Lotz and Chin, 2000). Thus, the evidence suggests that compressive loading involving lower compressive loads stimulates healthy bone (noted as a correlate of disc health), but that excessive loading leads to tissue breakdown.

The strength of the disc relative to the vertebrae was studied by Skrzpiec and colleagues (2007), who found age and gender effects. They found that the vertebrae lost 69% and 75% of compressive strength in males and females, respectively, from age 48 to 91 years. The annulus strength did not decrease except for the outermost layers in males' spines. The implication is that, given the minimal potential for adaptive change, the annulus is relatively weak in the strengthening spines of young men, but is relatively strong in the weakening spines of elderly females.

Other features of the disc influence load-bearing ability. First, larger discs develop more stress when bending and herniate more easily (Adams and Dolan, 2005). Yates, Giangregorio, and McGill (2010) found that disc shape determined the pattern of collagen delamination and herniation type (see figure 3.17 a and b). Transverse sections reveal whether the disc is predominantly oval or limaçon shaped (shaped like a lima bean with the concave section around the posterior part adjacent to the spinal cord). The magnitude of stress concentrations in the annulus is a function of the radial distance from the neutral axis. This means that thicker discs experience more stress during bending and twisting. Because oval discs do not focus stress concentrations when bending or twisting, the patterns of the nucleus tracking through the annulus are diffuse. In contrast, the limaçon-shaped discs focus stress at the apex of the curve on either side of the concave section. This produces focal bulges posterolaterally (Yates, Giangregorio, and McGill, 2010). This type of bulge appears to respond better to direction-based therapy. There is a reason big athletes (with large-diameter limaçon-shaped discs) who generate very high stress can bear the stress without injury, but do poorly with activities such as yoga. Also, these athletes cannot hit a golf ball far because they bend and twist poorly. The archetypical golfer's spine is slender with ovoid discs that bend and twist with less stress, but it cannot survive high compressive loads. The higher-level implication is that the skilled clinician matches training and rehabilitation approaches with the anatomical features that govern function.

Figure 3.17 Pathway of partial herniation in (a) an oval-shaped disc with volume exceeding an arc of 45°; (b) a limaçon-shaped disc directionally concentrated partial herniation with volume contained within an arc of 45°.

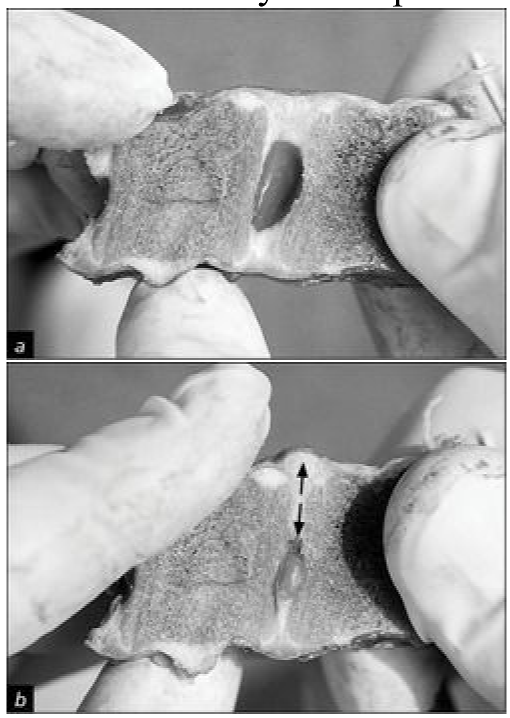


Adapted from Yates, Giangregorio, and McGill 2010.

Progressive Disc Injury

Chapters 1 and 2 addressed the risk of disc injury as a function of occupation and other variables that are proxy measures of load on the disc. Here, a more detailed consideration of progressive disc injury is in order, focusing on primary variables that damage tissue rather than surrogate descriptors. A normal disc under compression deforms mainly the end plates vertically and displays outward bulging of the annulus (Brinckmann, Biggemann, and Hilweg, 1988). However, if little hydrostatic pressure is present, as in the case in which the nucleus has been lost through end-plate fracture or herniation, the outer annulus bulges outward and the inner annulus bulges inward during disc compression (see figure 3.18, a and b). This double-convex bulging causes the laminae of the annulus to separate, or delaminate, and has been hypothesized as a pathway for nuclear material to leak through the lamellae layers and finally extrude, creating a frank herniated disc (Adams and Dolan, 1995).

Figure 3.18 (a) A healthy joint with a contained nucleus has minimal annulus deformation under compressive load. (b) If the nucleus loses pressure (as a result of an end-plate fracture, for example), the annulus compresses, causing radial bulging both outward and inward (arrows) and producing delaminating stresses as the annulus layers are pulled apart.



Photos from Stuart McGill

Professor Michael Adams put forth an interesting proposal (personal communication, June, 2015). He suggested that a healthy disc builds interval pressures under compressive loads that are so high that no nerve or vascular vessel could survive. Following initial end-plate damage, the disc can no longer build substantial pressures so that nerves and blood vessels are able to invade the disc. These are more possibilities to explain the increased vascularization of so-called degenerated discs and their ability to generate pain.

The following two sections review factors that influence disc damage and those that influence recovery.

Influences on Disc Damage

Several features of disc herniation tie together injury mechanisms and therapy. Most herniations have no tearing of the collagen fibers. Rather, the fibers delaminate. They do this in two ways. First, with repeated bending, adjacent fibers within the same layer eventually separate allowing the nucleus gel to flow into this cleft (Tampier et al., 2007). Bending also creates a hydrostatic pressure concentration on these fibers on the outside of the bend (i.e., on the posterior fibers when bending forward). Repeated bending causes this delamination process to continue with the next concentric annulus layer (see figure 3.19). In this way, this type of damage works from the inside out (Veres, Robertson, and Broom, 2009). Time series radiographs (see figure 3.20) demonstrated the progression of the nuclear material tracking through the annulus with successive motion cycles. The herniated disc appears to result from cumulative trauma: even though we have crushed several thousand vertebral motion segments, we have only once or twice observed a herniation without concomitant flexion cycles. Note that we are including both frank herniation and visible disc bulges (see figure 3.21) under this category of injury mechanism.

Figure 3.19 (a) Peeling off some collagen layers reveals the nucleus tracking through the layers of the annulus from the inside out. (b) Removing another layer of the annulus shows how the nucleus tracks through the annulus.

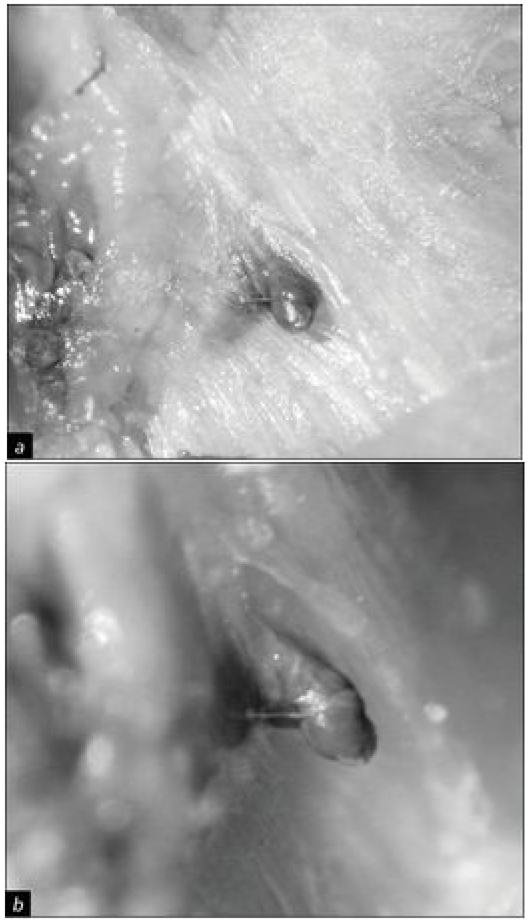
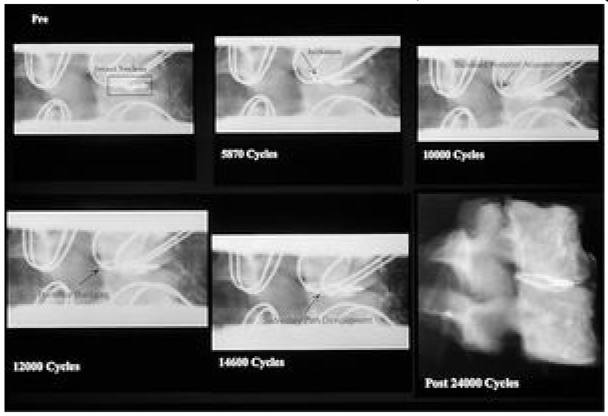


Figure 3.20 Serial radiographs showing the initial containment of the radiocontrast in the nucleus and, with repeated full-flexion motion (with about 1,400 N of compression), the progressive posterior tracking of the contrast until frank herniation after 24,000 bending cycles.



Reprinted from Clinical Biomechanics, 16(1), J.P. Callaghan and S.M. McGill, “Intervertebral disc herniation: Studies on a porcine model exposed to highly repetitive flexion/extension motion with force,” 28-37, 2001, with permission from Elsevier Science.

Figure 3.21 Repeated flexion can result in a frank herniation or a bulge, shown here in a specimen with the lamina removed to expose the posterior disc, which can impinge on nerves, causing symptoms. Note that the nucleus was injected with a dye so that the bulge is more visible (arrow).

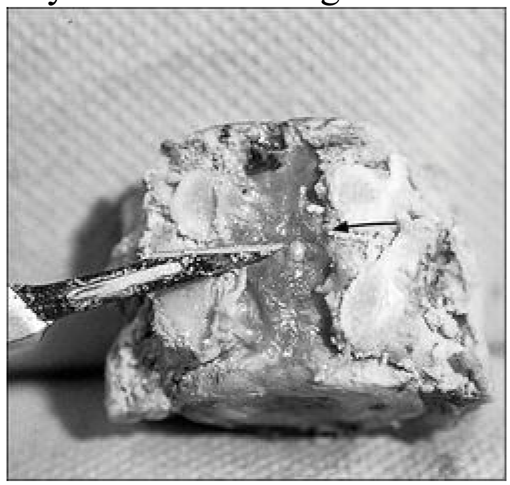


Photo from Stuart McGill

As noted earlier, disc size and shape influence the rate of this progress (Yates, Giangregorio, and McGill, 2010). I also strongly suspect from our animal work that repeated bending softens the collagen matrix so that repeated stretching and bending allows this delamination process to occur even faster. Although several reports have documented broken end-plate pieces extruded with the nucleus material, our work has reported microfracturing of the end plate where the annulus collagen fibers attach (Balkovec et al., 2014). Thus, although the fibers do not appear to rupture along their length, they can break away from their connection to the end plate. This is further evidence that herniation may often begin with an overload of compressive force and finish with a bending cycle. The excessive compression pressurizes the nucleus to the point that stress on the end plate causes cracks within the plate (Brown, Gregory, and McGill 2008) and breaking of the trabecular bone underneath (Balkovec et al., 2014).

This process is initiated and progressed with repeated bending. Additional compressive load accelerates the process. Interestingly we have not been able to initiate the delamination process with prolonged flexion, as would occur with sitting. Rather, once the delamination has begun with bending (probably full flexion; Adams and Hutton, 1982), prolonged flexion exacerbates the flow of nucleus into the delaminated clefts. This means that sitting probably does not initiate disc injury, but once bending initiates the damage, sitting can become painful. This helps to make sense of the epidemiological data that link herniation with sedentary occupations and the sitting posture (Videman, Nurminen, and Troup, 1990).

Several years ago we sought the most potent mechanism leading to disc herniation. We found that repeated flexion motion under simultaneous compressive loading was the easiest way to ensure herniation. In fact, it turned out that the number of cycles of flexion motion was more important than the actual magnitude of compressive load. Although no herniations were produced with 260 N of compressive load and up to 85,000 flexion cycles, herniations were produced with 867 N of load and 22,000 to 28,000 cycles, and with 1,472 N and only 5,000 to 9,500 cycles (Callaghan and McGill, 2001). Although people vary greatly, these numbers illustrate the relationship: herniations are a function of repeated full-flexion motion cycles with only a modest level of accompanying compressive load. In fact, we mimicked the lumbar motion and loading of a typical spine rehabilitation machine in which the seated patient belts down the pelvis to isolate the lumbar spine and then extends the torso repetitively against a resistance over the midback. (Amazingly, some people are trained on this type of machine, even those with known disc herniations!) (See figure 3.22.) Logically, one could follow a McKenzie-type exercise philosophy, believing that the extension motion can reduce a posteriorly displaced nucleus (we observed this in some specimens; Scannell and McGill, 2009). Yet, more recent work (Balkovec and McGill, 2012) in which we measured the accelerated rate of disc flattening with repeated flexion–extension cycles motivated us to seek better alternatives.

Figure 3.22 Rehabilitation devices such as this attempt to isolate lumbar motion by extending against a resistance pad but create simultaneous lumbar compression. We found that replicating the full range of motion from full flexion to neutral and using the compressive loads of these devices was a powerful combination that produced disc herniation.

Other aspects of our work have revealed the dependency of the location of the herniating bulge on the axis of motion (Aultman, Scannell, and McGill, 2005). For example, in 20 motion segments, we flexed them repeatedly about an axis that was 30° rotated from the pure flexion axis (mostly flexion with some lateral bend). One specimen simply failed abruptly and was removed. In the remaining 19, the herniation track was away from the axis of rotation (see figure 3.23).

Figure 3.23 Tilting the flexion axis 30° away from pure flexion caused the nucleus to track in a direction away from the axis (Aultman, Scannell, and McGill, 2005). This motion dependence has powerful potential for the design of exercise for those with known posterolateral disc bulges.

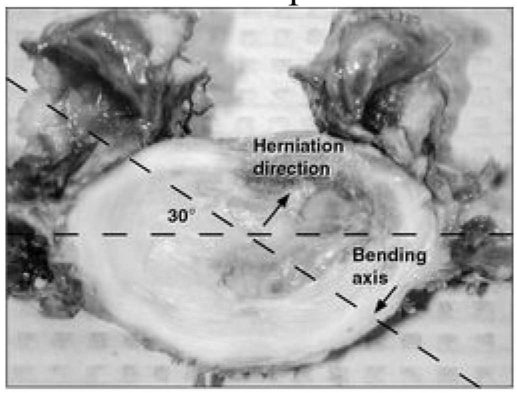


Photo from Stuart McGill

Herniations tend to occur in younger spines (Adams and Hutton, 1985), meaning those with higher water content (Adams and Muir, 1976) and more hydraulic behavior. Older spines do not appear to exhibit classic extrusion of nuclear material, but rather, are characterized by radial cracks that appear to progress with repeated loading (a nice review is provided by Goel, Monroe, et al., 1995).

Twisting initiates a different type of collagen delamination than with pure bending. Here twisting causes stress between the adjacent rings of collagen. More twisting cycles causes stress–strain reversals and clefts between the layers (figure 3.24). Some radiologists report these as disc tears, but we recognize them as subsequent to twisting damage (see figure 3.25 a and b). Thus, both forms of delamination, between fibers and between layers, can be classified as cumulative trauma damage. Although we do not yet know the relationship between the number of cycles and loads, we do know that added torsion reduces the compressive strength of the joint (Aultman et al., 2004).

Figure 3.24 Twisting causes concentric layers of the annulus to delaminate. Here a layer has been dissected away to reveal the tracking of the nucleus between annulus layers.

Photo from Stuart McGill

Figure 3.25 Delamination patterns from repeated twisting. The nucleus was dyed to show separation of concentric rings of collagen. (a) Complete circumferential delamination; (b) partial circumferential delamination.

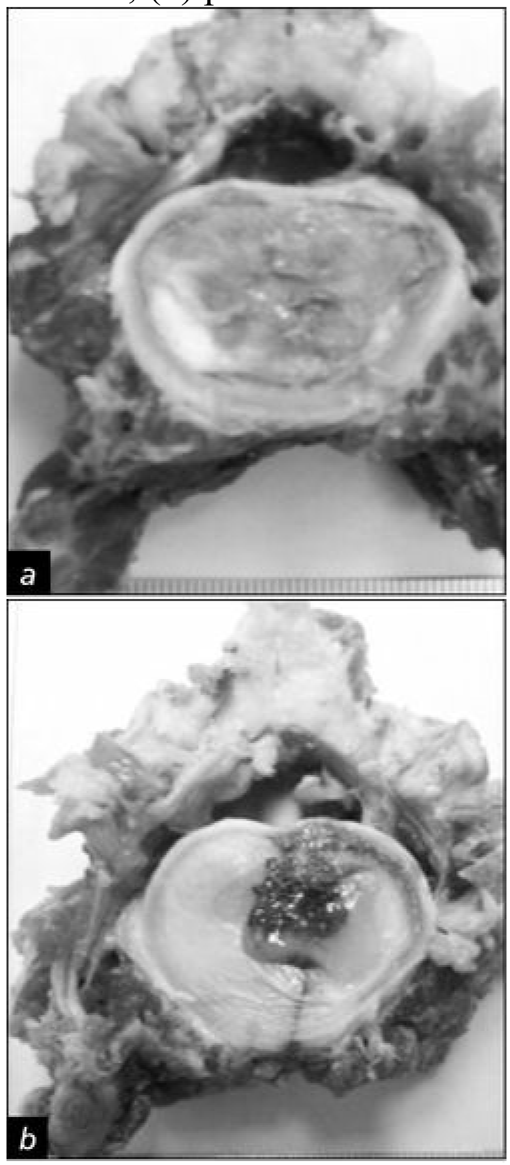


Photo from Stuart McGill

Mechanisms of Annulus Failure (Herniation)

Clinical Relevance

Damage to the annulus of the disc (herniation) appears to be associated with fully flexing the spine for a repeated or prolonged period of time. In fact, herniation of the disc seems almost impossible without full flexion, and without some compressive load. This has implications for exercise prescription particularly for flexion stretching and sit-ups or for activities such as prolonged sitting, all of which are characterized by a flexed spine. Some resistance exercise machines that take the spine to full flexion repeatedly must be reconsidered for those interested in sparing the posterior annulus portions of their discs. Furthermore, the mechanism by which the process can be interrupted appears to be postural dependent, providing some more insight into the mechanism of the McKenzie approach. These summary thoughts provide a basis for the prescription of the cat–camel exercise, for example, as a preferred position for moving the spine. Being on the hands and knees removes the compressive load.

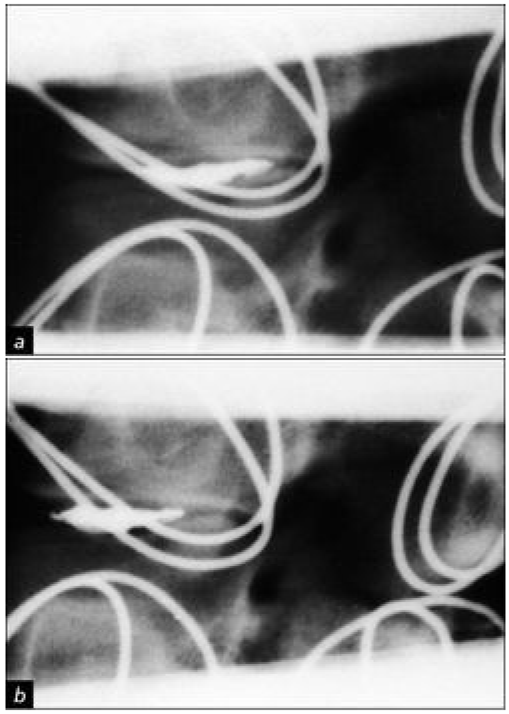
Also worth noting here is the intriguing hypothesis of Bogduk and Twomey, who suggested the possibility of an annular sprain similar to a sprain of the ankle ligaments (Bogduk and Twomey, 1991). They hypothesized that the outer layers of the annulus experience excessive strain under torsion. Given that these authors have presented evidence for the presence of nerve fibers particularly in this region, the annulus appears to be a good candidate for a source of pain.

Influences on Disc Recovery

That disc bulges shrink over time is quite well documented. Benson and colleagues (2010) showed that the overwhelming majority of massive disc bulges shrink over a period of 2 years, as do the symptoms (83% had a complete and sustained resolution of symptoms, and volumetric analysis showed that bulges had reduced 64% in size on average). Several possibilities for a shrinking mechanism exist. The extruded material may be resorbed by the disc or may dehydrate. The inflammatory response may cause digestion of the extruded nucleus. Miyazaki and colleagues (2009) documented the ingrowth of vascular tissues into the extruded material and believed that this mechanism brings macrophages that digest the foreign body. However, subsequent work showed that vascularization was accompanied by granulation tissues on the dura and nerve roots. They linked this clinically to radiating symptoms that disrupted nerve conduction and reduced the pain-free range during the straight-leg raise (Kobayashi et al., 2010). This understanding is the underpinning of some of the nerve flossing techniques introduced later.

We have also been able to add more insight into the McKenzie therapy approach, which is based on extended postures. The theory is that an extended posture drives the nucleus forward within the disc, and quite possibly vacuums in the partially extruded nucleus. As the layers of the annulus delaminate and fill with nucleus material, we now have proof that extended postures can drive the nucleus material that is in the delaminated pockets of the posterior nucleus back toward the central part of the disc (Scannell and McGill, 2005) (see figure 3.26). Physical therapists have claimed that movements such as the floppy push-up milk the nucleus back to the center of the disc. Scannell showed this to be true, but only in discs with 70% of their original disc height remaining. But I am concerned over the trauma to the facets in this dynamic therapy, which results in an extension-intolerant facet patient a couple of years later. Subsequently, we tested spines in a static extended posture (prone with a fist under the chin). It appears that while in this posture, the nucleus material in the posterior annulus slowly returns to the center of the disc, but avoids the trauma to the facets. Once again, it appears to be possible only in discs with 70% of their height remaining. We need to further refine this work.

Figure 3.26 Proof that disc bulges can be reduced with extended postures that appear to vacuum the nucleus that has invaded the posterior annulus. This worked only in discs with 70% of their original height, or more, remaining. (a) The nucleus is working posteriorly with repeated flexion motion; (b) extension reverses the direction of the leading edge of the nucleus.



Photos from Stuart McGill

The technique of repeated dynamic extension movements to reduce displaced nucleus in the annulus causes additional concern. Balkovec and McGill (2012) compared repeated flexion of the spine with repeated flexion and extension. The hypothesis tested was that the extension component of each cycle could prevent the posterior migration of the nucleus. We observed that the added extension simply mashed down the disc faster. In other words, adding repeated extension greatly increased the rate of disc deterioration. We retain our position in favor of static extension postures for acute posterior disc bulges with 70% of the disc height remaining, and discourage dynamic extension movements such as the floppy push-up and the prone press-up.

Finally, some therapists advocate flexing discs postsurgery and postinjury because they believe that this reduces the risk of scar tissue formation and adhesions. One could argue that scarring could be desirable to form a plug of the nucleus leaking in the outer layers of the annulus. Obviously, scars tethering neural tissues are problematic (more on this in the nerve flossing discussion). Adams, Stefanakis, and Dolan (2010) presented an excellent discussion of the many factors that influence the potential for healing. For these reasons we often advocate the cat–camel exercise performed while on the hands and knees. We have shown that minimal load is placed on the disc during the cat–camel given the horizontal orientation, and that bending without compressive load does not appear to exacerbate a disc bulge.

Muscles

Traditional anatomical descriptions of the spine musculature have taken a posterior vantage point. This has hindered insight into the roles of these muscles because many of the functionally relevant aspects are better viewed in the sagittal plane. (For a nice synopsis of the sagittal plane lines of action, see Bogduk, 1980, and Macintosh and Bogduk, 1987.) Furthermore, many have developed their understanding of muscle function by simply interpreting the lines of action and region of attachment, assuming that the muscles act as straight-line cables. This may be misleading.

Muscles of the torso are fundamentally different from those of the limbs from a motor control perspective. Limb muscles create motion; torso muscles more often stop or control motion. There really are no agonists or antagonists in the torso because all of the muscles are required to stabilize and control motion generated elsewhere. Studies of athletic performance show that function is optimized when power is generated at the hips and transmitted through a stiffened core, or torso, with no energy leaks. This has a large impact on the approach to training torso muscle for both rehabilitation and performance enhancement. An examination of the architecture and mechanics of the abdominal wall, for example, shows that the wall uses the three layers as a structural composite to enhance stiffness and produce hoop stresses. The rectus abdominis assists the production and transmission of hoop stresses around the torso via the lateral tendons. Thus, training the muscle structures through the range of motion as in a sit-up, for example, would not address one of the primary functions.

Location of Disc Bulge and Designing Exercise

Clinical Relevance

A posterolateral disc bulge is usually caused by repeated flexion of the disc about an axis cutting across the disc perpendicular to the herniating track. This is powerful knowledge for exercise intervention, because further motion about this axis would exacerbate the herniation. This knowledge gives clues for better prevention. Look for a dominant motion pattern in a patient's daily routine that is consistent with the bulge location, and eliminate it. If the causative motion pattern is an element of an athletic event in which the patient competes, major decisions will need to be made. More motion will only ensure the inevitable. Can the technique be changed to eliminate the causative motion?

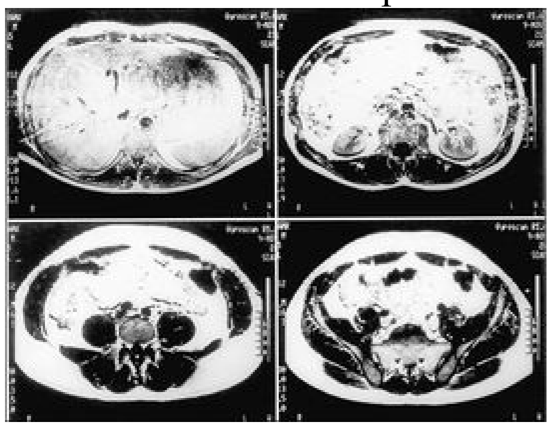
A different approach is required for a full understanding of the function and purpose of each muscle. First, knowledge of static muscle morphology is essential, although it may change over a range of motion. Second, knowledge of activation-time histories of the musculature must be obtained over a wide variety of movement and loading tasks. Muscles create force (and stiffness), but these forces play roles in moment production for movement and in stabilizing joints for safety and performance. A further understanding of the motor control system strategies chosen to support external loads and maintain stability requires the interpretation of anatomy, mechanics, and activation profiles. This section enhances the discussion of anatomically based issues of the spine musculature and blends the results of various electromyographic (EMG) studies to help interpret function and the functional aspects of motor control.

Muscle Size and Force Production

The physiological cross-sectional area (PCSA) of muscle determines the force-producing potential, whereas the line of action and moment arm determine the effect of the force in moment production, stabilization, and so forth. It is erroneous to estimate muscle force based on muscle volume without accounting for fiber architecture or by taking transverse scans to measure anatomical cross-sectional areas (McGill, Patt, and Norman, 1988). Using areas directly obtained from computed tomography (CT) or magnetic resonance imaging (MRI) slices has led to erroneous force estimates and interpretations of spine function. In such cases, because a large number of muscle fibers are not seen in a single transverse scan of a pennated muscle, muscle forces are underestimated. Thus, areas obtained from MRI or CT scans must be corrected for fiber architecture and scan plane obliquity (McGill, Santaguida, and Stevens, 1993).

In figure 3.27, transverse scans of one subject show the changing shape of the torso muscles over the thoracolumbar region, highlighting the need to combine transverse scan data with data documenting fiber architecture obtained from dissection. In this example, the thoracic extensors (longissimus thoracis and iliocostalis lumborum) seen at T9 provide an extensor moment at L4, even though they are not seen in the L4 scan. Only their tendons overlie the L4 extensors. A sagittal plane schematic shows the errors in measuring muscle cross-sectional area from a single transverse slice—which has caused some to underestimate the potential of the muscle.

Figure 3.27 Transverse scans of one subject (supine) at the levels of T9, L1, L4, and S1, showing the musculature in cross section. Note that many muscles seen at more superior levels pass tendons over the lower levels, transmitting force. This illustrates the error in using a single scan to estimate muscle force and moment potential.



Reprinted from Clinical Biomechanics, Vol 8(4), S.M. McGill, L. Santaguida, and J. Stevens., "Measure of the trunk musculature from T6 to L5 using MRI scans of 15 young males corrected for muscle fiber orientation," 171, 1993, with permission from Elsevier Science.

Raw muscle PCSAs and moment arms (McGill, Santaguida, and Stevens, 1993) are provided in appendix A.1. Areas corrected for oblique lines of action are shown in table 3.1 for selected muscles at several levels of the thoracolumbar spine. Guidelines for estimating true physiological areas are provided in McGill, Patt, and Norman (1988). Marras and colleages (2001) is another recent source of raw muscle geometry obtained from MRI for both males and females.

Table 3.1 Corrected Muscle Cross-Sectional Areas

Muscle*		Cross-sectional area (mm)^2	Moment arms (anteroposterior) (mm)	Moment arms (lateral)(mm)
Longissimus pars lumborum	L3-L4	644	51	17
Quadratus lumborum	L1-L2	358	31	43
	L2-L3	507	32	55
	L3-L4	582	29	59
	L4-L5	328	16	39
External oblique	L3-L4	1,121	17	110
Internal oblique	L3-L4	1,154	20	89

*The laminae of longissimus pars lumborum at the L4-L5 level would have been listed here by virtue of their cosines, but were not, because they could not be distinguished on all scan slices.

A few examples of corrected cross-sectional areas, anteroposterior moment arms, and lateral moment arms perpendicular to the muscle fiber line of action using the cosines are listed in McGill, Patt, and Norman (1988). These are the values that should be used in biomechanical models rather than the uncorrected values obtained directly from scan slices.

Moment arms of the abdominal musculature are generally obtained from CT- or MRI-based studies. Generally, the subject lies supine or prone within the MRI or CT scanner, and the distance from the spine to the muscle centroid is measured. Lying on the back in this posture causes the abdominal contents to collapse posteriorly under gravity (McGill, Juker, and Axler, 1996). In real life and during standing, the abdominal muscles are pushed away from the spine by the visceral contents. Recently, research has shown that CT or MRI studies of the abdominal muscle moment arms obtained from subjects in the supine posture underestimated the true values by 30%.

Other variables influence muscle force and stiffness production. Stress describes the amount of force per unit of muscle cross-sectional area. Generally, we have found in our modeling work that this value resides within the range of 35 to 60 N/cm2. Highly trained people would be in the higher end of the range. What we don't know is the difference between individual muscles. Again, given our modeling work, we strongly suspect that the oblique muscles produce more force per cross-sectional area than more bulky, traditionally shaped muscles. Another variable recently investigated is the ultrastructural design in terms of sarcomere length. Ward and colleagues (2009a) noted that the multifidus, for example, has shorter sarcomeres than any other muscle they studied in the body. This means that the multifidus is stiffer and creates more force at lengths longer than rest length. Fiber type and muscle spindle concentration are yet other variables that modulate force production. Although the erector spinae muscles are generally considered to have a higher proportion of less-fatigable Type I muscle fibers, some have suggested that people with back pain are more fatigable and have fewer Type I fibers. Although on average, people with back pain are more easily fatigued, the evidence suggests that their fiber type distribution is no different from that of people who are less easily fatigued (Crossman et al., 2004).

Muscle spindles sense length and modulate force output accordingly. For example, if a muscle is elongated, the spindles instigate a pulse of force via the spindle-driven stretch reflex. Professor Pickar and colleagues studied spindle behavior and noted that prior length history (such as stretching) changes force production. For example, short, static stretches reduced subsequent spindle responsiveness (Ge and Pickar, 2012). This explains some of the performance losses following sitting, for example, which is revisited later in part II on injury prevention.

In summary, understanding the force and mechanical potential of muscles requires an appreciation for the curving line of action, which is best obtained in the anatomy lab. But, unfortunately, these specimens are usually atrophied, eliminating them as a source for muscle size estimates. Muscle areas obtained from medical imaging techniques need to be corrected to account for fiber architecture and contractile components that do not appear in the particular scan level (e.g., only the tendon passes the level). Further, moment arms for muscle lines of action from subjects who are lying down need to be adjusted for the application to upright postures maintained in real life. Thus, several variables influence force and stiffness production, which in turn influences spine stability, performance, and pained conditions. Finally, there is also the tendency to classify muscles as flexors, extensors, and the like. Every muscle is a stabilizer, a proximal stiffener that enables distal motion, and a stiffener of the flexible spine to enable load bearing without buckling. For this reason the muscles are listed in sections as anterior, posterior, and so on.

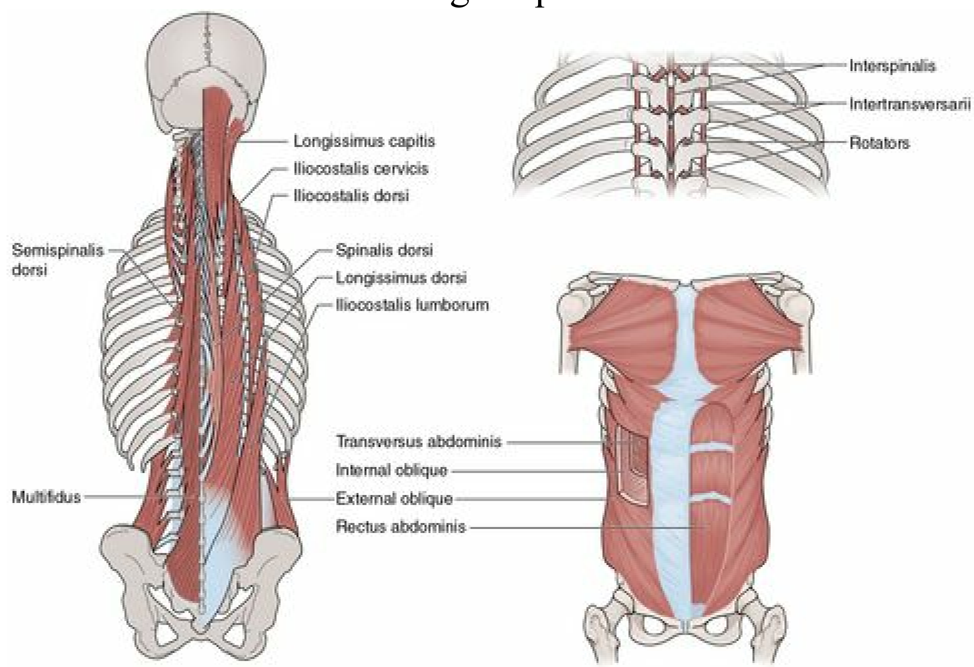
Posterior Muscle Groups

This section describes muscle groups from a functional perspective and introduces some issues fundamental for understanding injury avoidance and the choice of appropriate rehabilitation approach.

Rotatores and Intertransversarii

Many anatomical textbooks describe the function of the small rotator muscles of the spine, which attach to adjacent vertebrae, as creating axial twisting torque. This is consistent with their nomenclature (rotatores). Similarly, the intertransversarii are often assigned the role of lateral flexion. These proposals have several problems. First, these small muscles (see figure 3.28) have such small PCSAs that they can generate only a few newtons of force, and second, they work through such a small moment arm that their total contribution to rotational axial twisting and bending torque is minimal. For these reasons, I believe they serve another function.

Figure 3.28 Short muscles of the spine: levator costarum longi, levator costarum brevi, intertransverse medialis, intertransverse lateralis, rotatores, and interspinalis. They have been described erroneously as creating axial twisting torque.



The rotatores and intertransversarii muscles are highly rich in spindles, approximately 4.5 to 7.3 times richer than the multifidus (Nitz and Peck, 1986). This evidence suggests that they may function as length transducers or vertebral position sensors at every thoracic and lumbar joint. In some EMG experiments we performed a number of years ago, we placed indwelling electrodes very close to the vertebrae. In one case we had a strong suspicion that the electrode was in a rotator muscle. The subject attempted to perform isometric twisting efforts with the spine untwisted (or constrained in a neutral posture) in both directions but produced no EMG activity from the rotator—only the usual activity in the abdominal obliques and so on. However, when the subject tried to twist in one direction (with minimal muscular effort), there was no response, whereas in the other direction there was major activity. This particular rotator seemed not to be activated to create axial twisting torque but rather in response to twisted position change. Thus, its activity was elicited as a function of the twisted position—which was not consistent with the assumed role of creating torque to twist the spine. This is stronger evidence that these muscles are not rotators at all but function as position transducers in the spine proprioception system.

Manual Therapy and the Function of the Rotatores and Intertransversarii

Clinical Relevance

We now suspect that the rotatores and intertransversarii are actually length transducers and thereby position sensors, sensing the positioning of each spinal motion unit. These structures are likely affected during various types of manual therapy with the joint at its end range of motion (a posture used in chiropractic technique, for example).

Longissimus, Iliocostalis, and Multifidus Groups

The major extensors of the thoracolumbar spine are the longissimus, iliocostalis, and multifidus groups. Although the longissimus and iliocostalis groups are often separated in anatomy books, it may be more enlightening in a functional context to recognize the thoracic portions of both of these muscles as one group and the lumbar portions as another group. The lumbar and thoracic portions are architecturally (Bogduk, 1980) and functionally different (McGill and Norman, 1987). Bogduk (1980) partitioned the lumbar and thoracic portions of these muscles into longissimus thoracis pars lumborum and pars thoracis, and iliocostalis lumborum pars lumborum and pars thoracis.

These two functional groups (pars lumborum, which attach to lumbar vertebrae, and pars thoracis, which attach to thoracic vertebrae) form quite a marvelous architecture for several reasons and are discussed in a functional context with this distinction (i.e., pars lumborum versus pars thoracis). Fiber typing studies note differences between the lumbar and thoracic sections: the thoracic sections contain approximately 75% slow-twitch fibers, whereas the lumbar sections are generally evenly mixed (Sirca and Kostevc, 1985). The pars thoracis components of these two muscles attach to the ribs and vertebral components and have relatively short contractile fibers with long tendons that run parallel to the spine to their origins on the posterior surface of the sacrum and medial border of the iliac crests (see figure 3.29). Furthermore, their line of action over the lower thoracic and lumbar region is just underneath the fascia, such that forces in these muscles have the greatest possible moment arm and therefore produce the greatest amount of extensor moment with a minimum of compressive penalty to the spine (see figure 3.30). When seen on a transverse MRI or CT scan at a lumbar level, pars thoracis tendons have the greatest extensor moment arm, overlying the lumbar bulk—often over 10 cm (4 in.) (McGill, Patt, and Norman, 1988; McGill, Santaguida, and Stevens, 1993) (see figure 3.31).

Figure 3.29 An isolated bundle of longissimus thoracis pars thoracis (inserting on the ribs at T6), with tendons lifted by probes, course over the full lumbar spine to their sacral origin. They have a very large extensor moment arm (just underneath the skin).

Photo from Stuart McGill

Figure 3.30 This world-class powerlifter exemplifies the hypertrophied bulk of the iliocostalis and longissimus muscles seen in trained lifters. This muscle bulk is in the thoracic region, but the tendons span the entire lumbar spine.

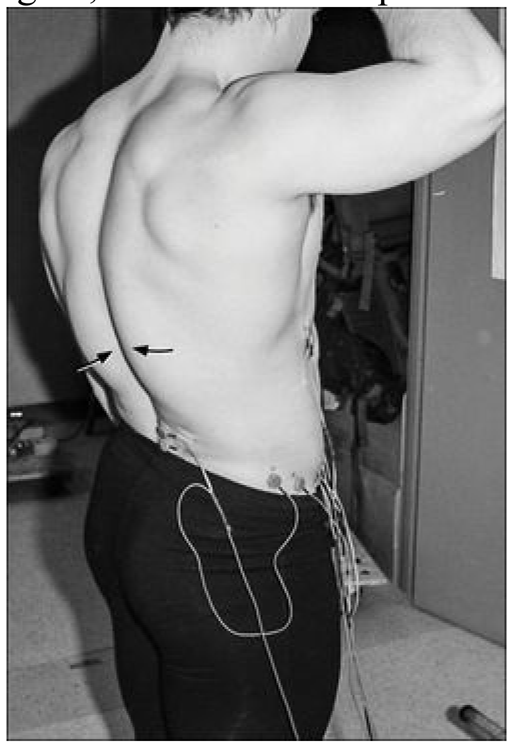
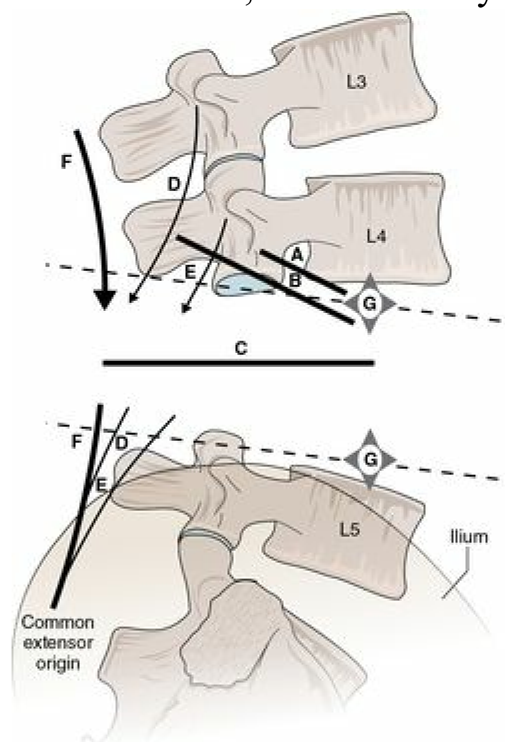


Photo from Stuart McGill

Figure 3.31 (A, B) The moment arms of the pars lumborum portions of iliocostalis and longissimus. (C) The large moment arm of the pars thoracis iliocostalis and longissimus muscles gives them their ability to create major lumbar extensor moments. The lines of action of (D) the pars lumborum portion of longissimus, (E) the pars lumborum portion of iliocostalis, and (F) the pars thoracis portion of longissimus and iliocostalis join at their common point of origin at the sacral spine. (G) The mechanical fulcrum, or the axis about which muscles create moments, is indicated by the diamond shape.



The lumbar components of these muscles (iliocostalis lumborum pars lumborum and longissimus thoracis pars lumborum) are very different anatomically and functionally from their thoracic namesakes. They connect to the mamillary, accessory, and transverse processes of the lumbar vertebrae and originate, once again, over the posterior sacrum and medial aspect of the iliac crest. Each vertebra is connected bilaterally with separate laminae of these muscles (see figure 3.32). Their line of action is not parallel to the compressive axis of the spine but rather has a posterior and caudal direction that causes them to generate posterior shear forces together with extensor moment on the superior vertebrae (see figure 3.33). These posterior shear forces support any anterior reaction shear forces of the upper vertebrae that are produced as the upper body is flexed forward in a typical lifting posture. It is important to clarify that this flexion of the torso is accomplished through hip rotation, not lumbar flexion. These muscles lose their oblique line of action and reorient to the compressive axis of the spine with lumbar flexion (McGill, Hughson, and Parks, 2000), so that a flexed spine is unable to resist damaging shear forces (see figure 3.34, a-d). This possible injury mechanism, together with activation profiles during clinically relevant activities, is addressed in a later section.

Figure 3.32 Iliocostalis lumborum pars lumborum and longissimus thoracis pars lumborum originate over the posterior surface of the sacrum, follow a very superficial pathway, and then dive obliquely to their vertebral attachments. This oblique orientation creates posterior shear (S) forces and extensor moments on each successive superior vertebra. The compressive axis (C) is indicated.

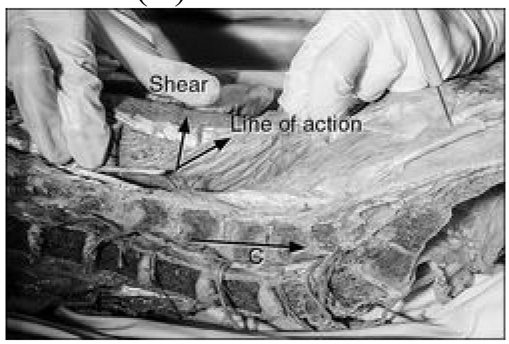


Photo from Stuart McGill

Figure 3.33 The oblique angle of the lumbar portions of longissimus and iliocostalis is seen in vivo in this MRI picture. Their line of force (F) is shown relative to the compressive axis (C).

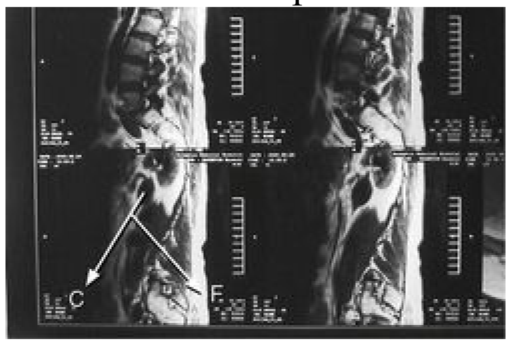


Photo from Stuart McGill

Figure 3.34 The oblique angle of the lumbar portions of the iliocostalis lumborum and longissimus thoracis protects the spine against large anterior shear forces. However, this ability is a function of spine curvature. (a) A neutral spine and (b) the oblique angle of these muscles as viewed with an ultrasound imager. (c) The loss of this angle with spine flexion (d) so that anterior shear forces cannot be counteracted. This ensures shear stability and is another reason to consider adopting a neutral spine during flexed weight-holding tasks.

Photo from Stuart McGill

The multifidus muscles perform quite a different function from those of the longissimus and iliocostalis groups, particularly in the lumbar region where they attach posterior spines of adjacent vertebrae or span two or three segments (see figure 3.35). Their line of action tends to be parallel to the compressive axis, or in some cases runs anteriorly and caudal in an oblique direction. The major mechanically relevant feature of the multifidii, however, is that because they span only a few joints, their forces affect only local areas of the spine. Therefore, the multifidus muscles are involved in producing extensor torque (together with very small amounts of twisting and side-bending torque) but provide only the ability for corrections, or moment support, at specific joints that may be the foci of stresses. Interestingly, the multifidus muscles appear to have quite low muscle spindle density—certainly less than the iliocostalis or longissimus muscles (Amonoo-Kuofi, 1983). This may be due to their more medial location and subsequent smaller length excursions (see table 3.2 for muscle length changes, which were assessed using a number of extreme postures depicted in figure 4.1 in chapter 4). An injury mechanism involving inappropriate neural activation signals to the multifidus is proposed in chapter 4, using an example of injury observed in the laboratory. It is also worth noting here, given the recent emphasis on the multifidus, that some people have considered more lateral portions of the extensors to be multifidus. This has presented some problems in both functional interpretation and rehabilitation.

Figure 3.35 The multifidus is actually a series of laminae that can span one to three vertebral segments. Their lines of action do not support anterior shear of the superior vertebrae but actually contribute to it. Examining their cross-sectional area reveals that the multifidus is a relatively small lumbar extensor.

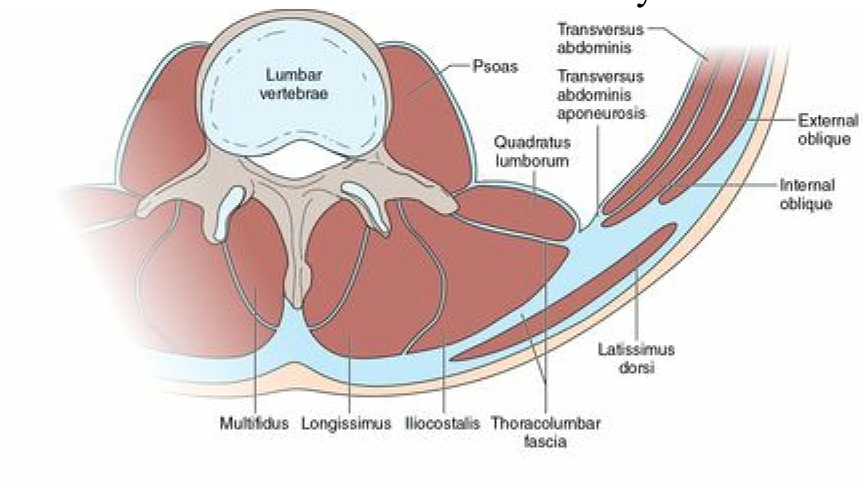


Table 3.2 Muscle Lengths in Centimeters (Including Tendon Length) Obtained From the Upright Standing Position and From Various Extreme Postures

	Upright standing	60° flexion	25° lateral bending	10° twist	Combined[a]
R rectus abdominis	30.1	19.8[b]	27.4	30.0	17.7
L rectus abdominis	30.1	19.8[b]	32.6	30.5	22.9[b]
R external oblique 1	16.7	14.4	12.6[b]	15.5	13.3
L external oblique 1	16.7	14.4	21.3[b]	18.0	19.2
R external oblique 2	15.8	12.9	10.7[b]	15.6	9.5[b]
L external oblique 2	15.8	12.9	21.0[b]	16.2	18.2
R internal oblique 1	11.1	9.9	9.1	12.2	7.6[b]
L internal oblique 1	11.1	9.9	14.1[b]	10.0	12.2
R internal oblique 2	10.3	8.8	7.6[b]	11.2	6.9[b]
L internal oblique 2	10.3	8.8	13.2[b]	9.5	10.7
R psoas lumborum (L1)	15.1	20.0[b]	14.2	15.4	18.7
L psoas lumborum (L1)	15.1	20.0	16.8	15.7	20.7[b]
R psoas lumborum (L2)	12.5	16.4[b]	11.8	12.9	15.6[b]
L psoas lumborum (L2)	12.5	16.4[b]	13.8	13.0	17.0[b]
R psoas lumborum (L3)	9.9	12.9[b]	9.5	10.0	12.5[b]
L psoas lumborum (L3)	9.9	12.9[b]	10.9	9.8	13.3[b]
R psoas lumborum (L4)	7.5	9.4[b]	7.4	7.6	9.3[b]
L psoas lumborum (L4)	7.5	9.4[b]	8.0	7.4	9.5[b]
R iliocostalis lumborum	23.0	29.7[b]	18.9	23.5	26.4
L iliocostalis lumborum	23.0	29.7[b]	25.5	22.8	31.8[b]
R longissimus thoracis	27.5	33.7[b]	25.4	27.6	31.1
L longissimus thoracis	27.5	33.7[b]	28.8	27.4	34.8[b]
R quadratus lumborum	14.6	18.2[b]	11.9	14.4	14.9
L quadratus lumborum	14.6	18.2	17.4	15.1	20.9[b]
R latissimus dorsi (L5)	29.4	32.1	26.8	29.8	29.0
L latissimus dorsi (L5)	29.4	32.1	31.5	29.1	34.6
R multifidus 1	5.3	7.3[b]	5.2	5.1	7.1[b]
L multifidus 1	5.3	7.3[b]	5.4	5.5	7.5[b]
R multifidus 2	5.1	7.2[b]	5.0	5.1	7.01[b]
L multifidus 2	5.1	7.2[b]	5.2	5.1	7.2
R psoas (L1)	29.2	28.6	28.1	29.0	27.2
L psoas (L1)	29.2	28.6	30.2	29.5	29.6
R psoas (L2)	25.8	25.3	25.1	25.7	24.4
L psoas (L2)	25.8	25.3	25.1	25.7	24.4
R psoas (L3)	22.1	21.8	21.7	22.0	21.2
L psoas (L3)	22.1	21.8	22.5	22.2	22.3
R psoas (L4)	18.7	18.6	18.6	18.7	18.4
L psoas (L4)	18.7	18.6	18.9	18.8	18.9

The range of extreme postures used to assess muscle length changes is illustrated in figure 4.1 in chapter 4.

[a]Combinations of 60° flexion, 25° right lateral bend, and 10° counterclockwise twist.

[b]Muscle lengths that differ by more than 20% from those obtained during upright standing.

Reprinted, by permission, from S.M. McGill, 1991, "Kinetic potential of the lumbar trunk musculature about three orthogonal orthopaedic axes in extreme positions," *Spine* 19(7): 809-815.

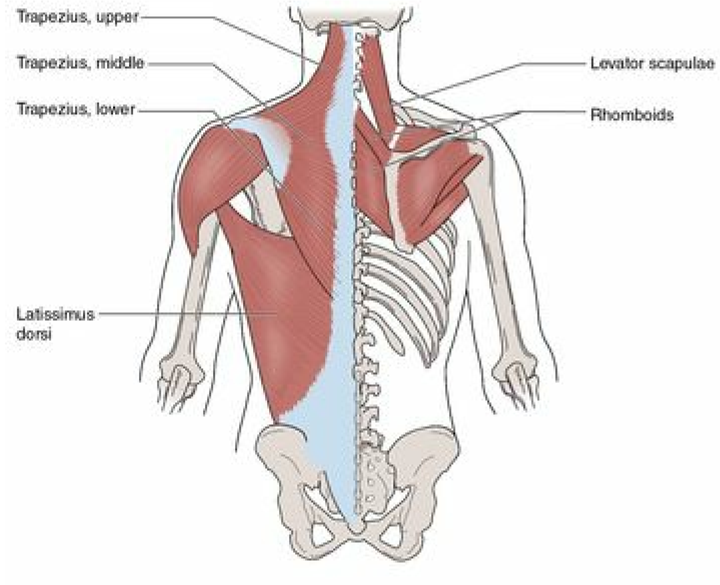
Some clinical groups have recommended focused training of the multifidus based primarily on a report suggesting unilateral atrophy of these muscles in people with pathology at that level (e.g., Hides et al., 1994). This is controversial because more recent work by Zielinski and colleagues (2013) and Le Cara and colleagues (2014) suggested that multifidus geometry was not predictive of people who improve clinically. Intramuscular fat in the multifidus has been shown to be greater in those with chronic low back pain (Kader, Wardlaw, and Smith, 2000; Paalanne et al., 2011), although neither study included the observation of selective muscle atrophy (they may have not been at a pathological level). Is the multifidus very different from other muscles?

Comprehensive studies show that all muscles atrophy. My opinion after studying this issue is that the multifidus is a collection of small slips of muscle. Fat grows between the muscle septa, not within a muscle. Thus, the architecture of the multifidus supports the growth of fat when compared with other extensor muscles. Further, from a clinical standpoint this argument may be moot because people are not able to target specific levels of the multifidus to activate and train the muscle. In fact, it is possible to train the entire erector group, but we have measured only a couple of skilled subjects over 30 years old who had the ability to activate specific components. The techniques they used to do this involved substantial risk of exacerbating pain in a sensitized patient. In summary, we support training all of the erectors for their role in spine stiffness, enhancing load bearing, controlling motion, and supporting torques. If segmental muscle pathology exists, the therapeutic exercise would not change.

A Note on Latissimus Dorsi

The latissimus dorsi is involved in lumbar extensor moment generation and often acts as a major stabilizer. Its origin at each lumbar spinous process via the lumbodorsal fascia and insertion on the humerus gives it a very large extensor moment arm (see figure 3.36). During a pulling and lifting motion, the latissimus is active (see chapter 4), which has implications for its role and how it is trained for functional motion patterns. It is often the muscle of choice for stopping thoracic and high lumbar hinges in painful backs and is also very important in enhancing torso extension in high-performance situations. It is also a critical muscle for power transference between the core and the upper limb (the role in higher-performance activities is outlined in detail in McGill, 2014).

Figure 3.36 The latissimus dorsi originates from each lumbar spinous process via the lumbodorsal fascia and inserts on the humerus to perform both lumbar extension and stabilization roles.



Exercise for the Extensor Muscles of the Low Back

Clinical Relevance

Research has shown that the thoracic extensors (longissimus thoracis pars thoracis and iliocostalis lumborum pars thoracis) that attach in the thoracic region are actually the most efficient lumbar extensors because they have the largest moment arms as they course over the lumbar region. For this reason, it is time to revisit the clinical practice of isolating muscle groups—in this case, the lumbar extensors for the lumbar spine. Specifically, what are referred to as the lumbar extensors (located in the lumbar region) contribute only a portion of the total lumbar extensor moment. Training the lumbar extensor mechanism must involve the extensors that attach to the thoracic vertebrae, whose bulk of contractile fibers lies in the thoracic region but whose tendons pass over the lumbar region and have the greatest mechanical advantage of all the lumbar muscles. Thus, exercises to isolate the lumbar muscles cannot be justified anatomically or from a motor control perspective because all "players in the orchestra" must be challenged during training.

Another important clinical issue involves the anatomical features of the extensors. Although the lumbar sections of the longissimus and iliocostalis muscles that attach to the lumbar vertebrae create extensor torque, they also produce large posterior shear forces to support the shearing loads that develop during torso flexion postures. Some therapists unknowingly disable these shear force protectors by having patients fully flex their spines during exercises, creating myoelectric quiescence in these muscles, or by recommending the pelvic tilt during flexing activities such as lifting. A discussion of this functional anatomy is critical for developing the strategies for injury prevention and rehabilitation described later in this book.

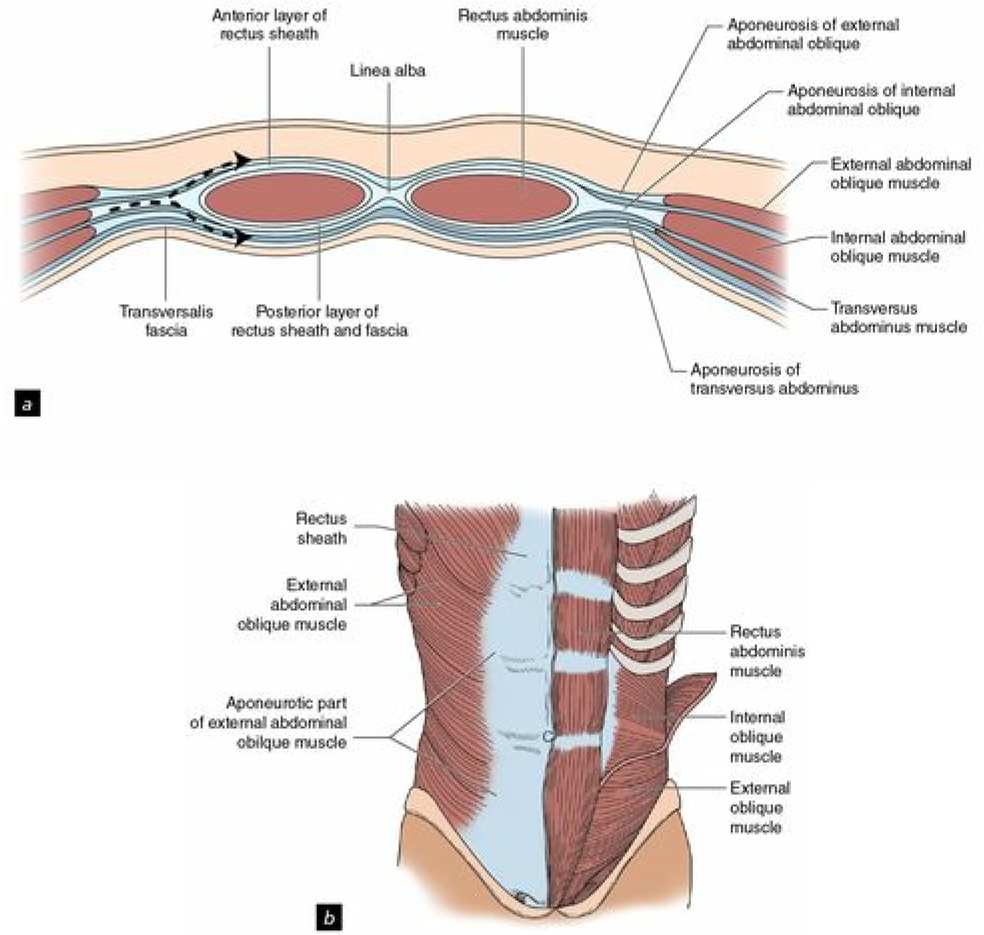
Anterior Muscles

This section addresses several important aspects of lumbar mechanics in which the abdominal muscles are involved.

Abdominal Fascia

The abdominal fascia contains the rectus abdominis and connects laterally to the aponeurosis of the three layers of the abdominal wall. Its functional significance is made more important by connections of the aponeurosis with pectoralis major, together with fascial elements that cross the midline to transmit force to the fascia (and abdominal muscles) on the opposite side of the abdomen (Porterfield and DeRosa, 1998) (see figure 3.37, a and b). Such anatomical features underpin and justify exercises (detailed later) that integrate movement patterns that simultaneously challenge the abdominal muscles, the spine, and the shoulder musculature.

Figure 3.37 The abdominal fascia connects the obliques of the abdominal wall with (a) rectus abdominis and, to a lesser extent, (b) pectoralis major and helps to transmit hoop stresses around the abdomen.

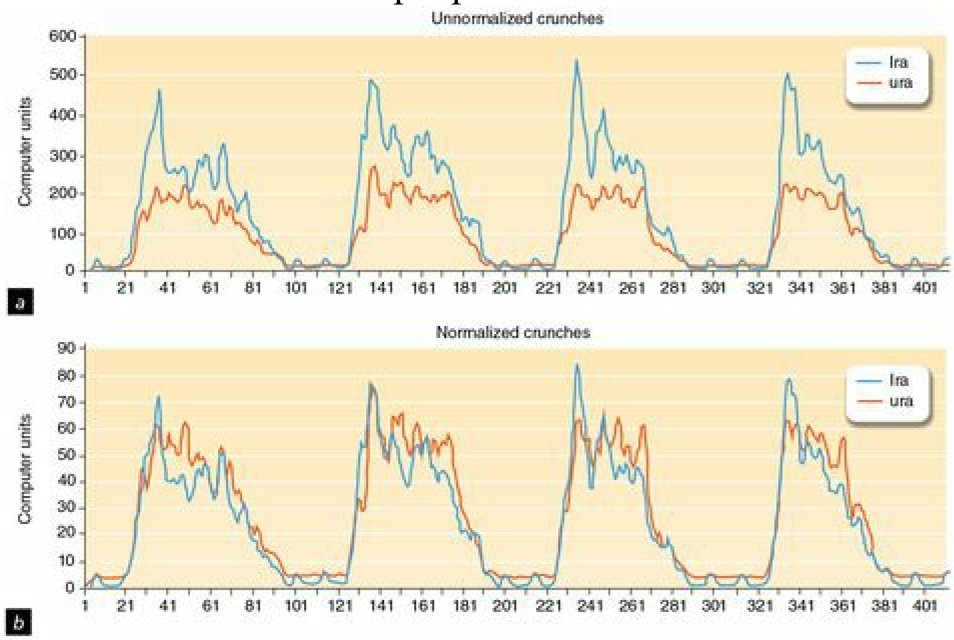


Although many classic anatomy texts consider the abdominal wall to be an important flexor of the trunk, the rectus abdominis appears to be the major trunk flexor—and the most active during sit-ups and curl-ups (Juker, McGill, and Kropf, 1998). Muscle activation amplitudes obtained from both intramuscular and surface electrodes over a variety of tasks are shown in table 4.4 in chapter 4. It is interesting to consider why the rectus abdominis is partitioned into sections rather than being a single long muscle, given that the sections share a common nerve supply and that a single long muscle would have the advantage of broadening the force–length relationship over a greater range of length change. Perhaps a single muscle would bulk upon shortening, compressing the viscera, or be stiff and resistant to bending. Not only does the sectioned rectus abdominis limit bulking upon shortening, but the sections also have a bead effect, which allows bending at each tendon to facilitate torso flexion–extension or abdominal distension or contraction as the visceral contents change volume.

The beaded rectus also performs another role—the lateral transmission of forces from the oblique muscles that form a continuous hoop around the abdomen (Porterfield and DeRosa, 1998). The intermuscular tendons and fascia prevent the fibers of rectus from being ripped apart laterally from these hoop stresses. This aspect of abdominal mechanics is elucidated further in the next section (Abdominal Wall), which addresses the forces developed in the oblique muscles.

Another clinical issue is the controversy regarding upper and lower abdominal muscles. Although the obliques are regionally activated (and have functional separation between upper and lower regions), all sections of the rectus are activated together at similar levels during flexor torque generation. A significant functional separation does not appear to exist between the upper and lower rectus (Lehman and McGill, 2001) in most people. Research reporting differences in upper and lower rectus activation sometimes suffers from the absence of normalization of the EMG signal during processing. Briefly, researchers have used raw amplitudes of myoelectric activity (in millivolts) to conclude that there is more, or less, activity relative to other sections of the muscle, but the magnitudes are affected by local conductivity characteristics. Thus, amplitudes must be normalized to a standardized contraction and expressed as a percentage of this activity (rather than in millivolts) (see figure 3.38). Researchers may also have inadvertently monitored pyramidalis (an optional muscle at the base of the rectus), which would cloud interpretation.

Figure 3.38 (a) Studies that have reported an upper and lower rectus abdominis generally evaluated unnormalized EMG signals—for example, in the study of men performing crunches. (b) However, when the signals are normalized properly, the apparent difference disappears. Although there are regional activation zones in the abdominal obliques, there is little evidence to suggest that a functional upper and lower rectus abdominis exists in most people.



Reprinted from Journal of Manipulative Physiological Therapeutics, 22(9), G. Lehman and S.M. McGill, “The influence of a chiropractic manipulation on lumbar kinematics and electromyography during simple and complex tasks: a case study,” 576-581, Copyright 1999, with permission from National University of Health Sciences.

Upper and Lower Rectus

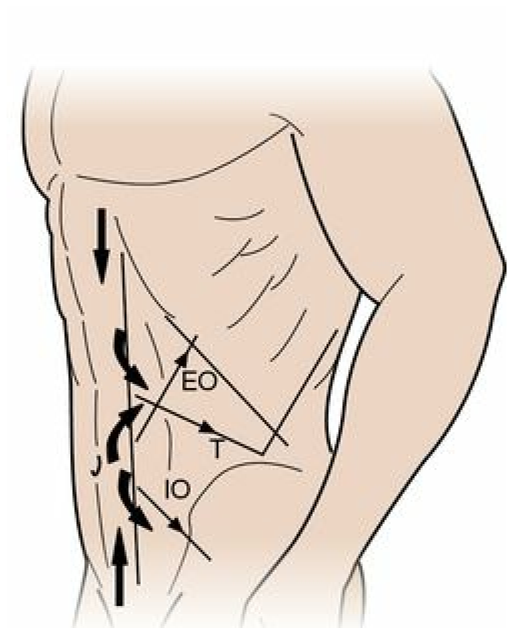
Clinical Relevance

Although we have seen separation in neural drive between upper and lower sections of rectus abdominis in a few Middle Eastern–style belly dancers (out of a larger cohort), these abilities and moves are rare in everyday life. (We made these observations only during very low-level contractions in which the muscles were moved in the absence of substantial flexion torque or muscle load.) In fact, a distinct upper and lower rectus does not exist in most people. Once force is required, the rectus appears to act as a cable with active tension along its entire length. Thus, training the rectus for nearly everyone can be accomplished with a single exercise. This is not true for the obliques, because they have several neural compartments—lateral, medial, upper, and lower.

Abdominal Wall

The three layers of the abdominal wall (external oblique, internal oblique, and transverse abdominis) perform several functions. All three are involved in flexion and appear to have their flexor potential enhanced because of their attachment to the linea semilunaris (see figure 3.39) (McGill, 1996), which redirects the oblique muscle forces down the rectus sheath to effectively increase the flexor moment arm. The obliques are involved in torso twisting (McGill, 1991a, 1991b) and lateral bend (McGill, 1992) and appear to play a role in lumbar stabilization because they increase their activity, to a small degree, when the spine is placed under pure axial compression (Juker, McGill, and Kropf, 1998). (This functional notion is developed later in this chapter.) The obliques are also involved in challenged lung ventilation, assisting with what is called active expiration (Henke et al., 1988). The important functional implication of active expiration is explained in McGill (2014). For example, when athletes grunt at the beginning of the expiration phase, it enhances spring stability.

Figure 3.39 The oblique muscles (EO: anterior portion of external oblique; IO: anterior portion of internal oblique; T: transverse abdominis) transmit force along their fiber lengths and then redirect force along rectus abdominis via their attachment to the linea semilunaris to enhance their effective flexor moment arm.



Researchers have focused a lot of attention on the transverse abdominis for two reasons. First, some believe it is involved in spine stability through its beltlike containment of the abdomen and also in the generation of intra-abdominal pressure (IAP). Richardson and colleagues (1999) nicely summarized the second reason for the focused attention on this muscle by suggesting that transverse has a delayed onset of activation time in some with back troubles, prior to a rapid arm movement. Their hypothesis is that the trunk must first be made stiff and stable. It appears that this is evidence that an aberrant motor pattern exists in rapid activity, but according to Silfies and colleagues (2009), all muscles exhibit these patterns in different subgroups of back pain. Further we cannot find evidence that this motor pattern happens bilaterally, nor could others (e.g., Allison, Morris, and Lay, 2008), implying that it has little to do with stability. The question remains as to whether this is important in more normally paced activities. Delayed onset confirms motor deficits, but a 10 to 30 ms onset delay would be irrelevant during a normal movement in which these muscles are continually cocontracted to ensure stability. Contrary to what is commonly heard in exercise discussions, the evidence does not suggest that transverse abdominis is not recruited. It is also interesting that, again, contrary to popular thought, the transverse and internal oblique are very similar in their fiber orientation. In our very limited intramuscular EMG electrode work (Juker, McGill, and Kropf, 1998), we saw a high degree of coupling between these two muscles in a wide variety of nonballistic exertions (no doubt some myoelectric cross-talk existed). We have been unable to find people who can or cannot activate transverse beyond a level of 2% of maximum. Nonetheless, several groups (Cresswell, Oddsson, and Thorstensson, 1994, and Hodges and Richardson, 1996, are the most experienced) have noted the extra activity of transverse when IAP is elevated, but also that all of the abdominal muscles show this activity. The combination of transverse activation (and almost always concomitant oblique activity) with elevated IAP enhances stability, without question. In fact, Cholewicki, Juluru, and McGill (1999) concluded that building IAP on its own adds spine stability.

In the broader functional perspective, the components of the abdominal muscles (rectus, obliques, and transverse) work together but also independently. Although a variety of sources have provided myoelectric evidence from a variety of tasks, an anatomical and functional interpretation is needed. As noted earlier, the obliques differentially activate to create twisting torque and can enhance flexion torque. Rectus is primarily a flexor —in fact, those people who have a great deal of motor control in the abdominal muscles can preferentially activate each muscle (see figure 3.40, a-d). Finally, some have suggested an upper and lower partitioning of the abdominal muscles. As previously mentioned, this impression is probably an artifact resulting from poor EMG technique; there does not appear to be a functional upper and lower rectus (Lehman and McGill, 2001; Vera-Garcia, Grenier, and McGill, 2000). On the other hand, regional differences do exist in the obliques; some sections can be preferentially recruited both medially and laterally together with upper and lower portions. Finally, the obliques, together with transverse abdominis, form a containing hoop around the entire abdomen; the anterior of the hoop is composed of the abdominal fascia, and the posterior is composed of the lumbodorsal fascia. The resulting hoop stresses and stiffness assist with spine stability.

Figure 3.40 Some trained people have the ability to differentially activate specific portions of the abdominal musculature. This sequence shows (a) an inactive abdominal wall, (b) the abdominal wall ballooned, and (c) the contraction of transverse abdominis, which draws and hollows the wall. (Note that other muscles must relax to do this, which compromises both stability and strength.) (d) Placing the hands on the thighs and pushing allows a flexor moment to develop in which good muscular control is able to activate just the rectus abdominis with the previously activated transverse and little oblique activity.

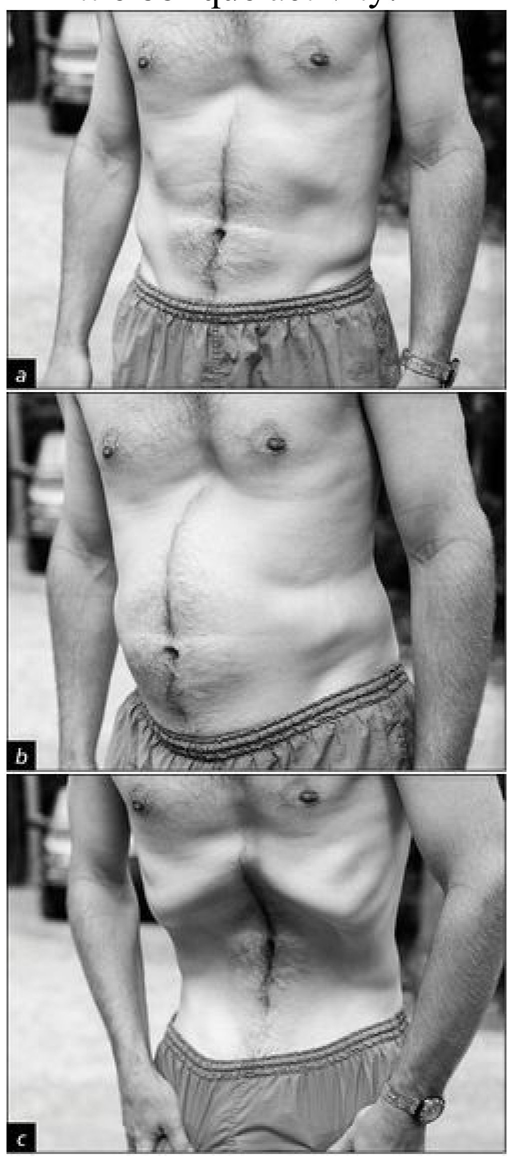


Photo from Stuart McGill

Abdominal Muscle Exercises

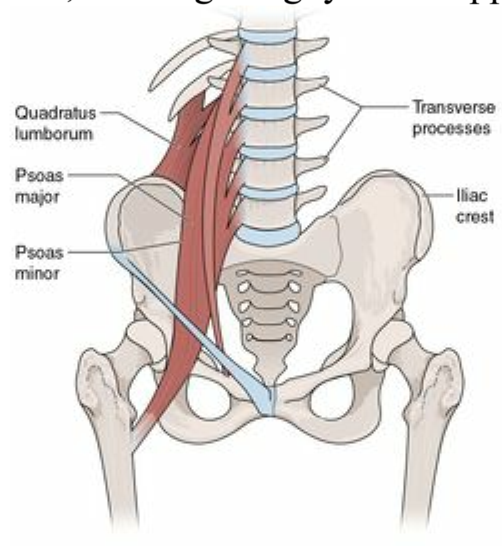
Clinical Relevance

The functional divisions of the abdominal muscles justify the need for several exercise techniques to challenge them in all of their roles: moment generation, spine stability, and heavy breathing. Although the obliques are regionally activated, with several neural compartments, there appears to be no functional separation of upper and lower rectus abdominis. Thus, a curl-up exercise activates all portions of the rectus abdominis. However, upper and lower portions of the oblique abdominal muscles are activated separately depending on the demands placed on the torso. Finally, detailed examination of the fascial connections reveals force transmission among the shoulder musculature, the spine, and the abdominal muscles, justifying exercises incorporating larger movement patterns for the advanced patients who can bear the higher loads. Techniques to obtain high performance from the abdominal muscles enhanced with power breathing are beyond the scope of this book, but are documented in Ultimate Back Fitness and Performance (McGill, 2014).

Psoas

The psoas, a muscle that crosses the spine and hip, is unique for many reasons. Although it has been claimed to be a major stabilizer of the lumbar spine, I believe that this claim needs interpretation. Although the psoas complex attaches to T12 and to every lumbar vertebra on its course over the pelvic ring (see figure 3.41), its activation profile (see Andersson et al., 1995; Juker, McGill, and Kropf, 1998; and Juker, McGill, Kropf, and Steffen, 1998, for indwelling EMG data) is not consistent with that of a spine stabilizer (in the purest sense); rather, it indicates that the role of the psoas is purely as a hip flexor. During our work to implant intramuscular electrodes in this muscle, we performed the insertion technique several times on ourselves. The first time the electrodes were in my own psoas, I tried several low back exertions in an attempt to activate it (flexion, extension, side-bending, and so on.). None of these really caused the psoas to fire. Simply raising the leg while standing, with hip flexion, caused massive activation, clearly indicating that the psoas is a hip flexor.

Figure 3.41 The psoas attaches to each lumbar vertebra (lateral vertebral body and transverse process) where each of these laminae of psoas fuse and form a common tendon that courses through the iliopectineal notch to the femur. The quadratus lumborum attaches each transverse process with the ribs and iliac crest, forming the guy wire support system.



After this pilot work, in a larger study we found that any task requiring hip flexion involved the psoas. Although we obtained this impression from studying the activation profile of the psoas, other considerations stem from its architecture. Why does the psoas traverse the entire lumbar spine and, in fact, course all the way to the lower thoracic spine? Why not let just the iliacus perform hip flexion? If only the iliacus were to flex the hip, the pelvis would be torqued into an anterior pelvic tilt, forcing the lumbar spine into extension. These forces are buttressed by the psoas, which adds stiffness between the pelvis and the lumbar spine. In effect, it can be thought of as a spine stabilizer but only in the presence of significant hip flexor torque. Also, an activated and stiffened psoas will contribute some shear stiffness to the lumbar motion segment—but once again, only when hip flexor torque is required. The fact is that the psoas and iliacus are two separate muscles (see Santaguida and McGill, 1995), functionally, architecturally, and neurally. There is no such thing as an iliopsoas muscle!

In addition, some clinical discussion has been centered on the issue of whether the psoas is an internal or external rotator of the femur and hip. Although it provides some anatomical advantage during external hip rotation, Juker, McGill, Kropf, and Steffen (1998) observed only small activation bias during hip rotation tasks. However, this may have been due to the need for significant hip stabilization, resulting in substantial hip cocontraction. In a recent study, we examined a matched cohort of workers—half of whom had a history of disabling low back troubles, and half of whom had never missed work. Those who had a history, but were asymptomatic at the time of the test, had a significant loss of hip extension and hip internal rotation (but more external rotation). This is an interesting observation, given the extensive clinical discussions regarding the so-called tight psoas. Even though we do not fully understand the neuromechanics, this muscle as a clinical concern is worth studying further.

Psoas Function

Myoelectric evidence and anatomical analysis suggest that the psoas major acts primarily to create hip flexion torque, that its activation is minimally linked to spine demands, and that it imposes substantial lumbar spine compression when activated. Caution is advised when training this muscle because of the substantial compression penalty imposed on the spine when the psoas is activated.

Quadratus Lumborum

The quadratus lumborum (QL) is another special muscle for several reasons. First, the architecture of this muscle provides a stabilizing role by attaching to each lumbar vertebra, effectively buttressing adjacent vertebrae bilaterally, and by attaching to the pelvis and rib cage (see figure 3.41). Specifically, the fibers of the QL cross-link the vertebrae and have a large lateral moment arm via the transverse process attachments. Thus, by its design the QL could buttress shear instability and be effective in stabilizing all loading modes. Typically, under compressive load, the first mode of buckling instability is lateral (Lucas and Bresler, 1961); the QL can play a significant role in local lateral buttressing. Also, the QL hardly changes length during any spine motion (see table 3.2 and McGill, 1991b), suggesting that it contracts virtually isometrically. Further insight into its special function comes from an earlier observation that the motor control system involves this muscle together with the abdominal wall when stability is required in the absence of major moment demands.

Quadratus Lumborum and Spine Stabilization

Clinical Relevance

The QL appears to be highly involved with stabilization of the lumbar spine, together with other muscles, suggesting that a clinical focus on this muscle is warranted. Exercises emphasizing activation of the QL while sparing the spine are described in part III, Low Back Rehabilitation.

The QL muscle appears to be active during a variety of flexion-dominant, extensor-dominant, and lateral bending tasks. (Note that myoelectric access to the QL is quite tricky, and it is difficult to confirm where the intramuscular electrodes are within the muscle. Certainly, our techniques on this muscle were not very precise. In addition, they tend to migrate upon contraction, further clouding interpretation of the signal.) Andersson and colleagues (1996) found that the QL did not relax with the lumbar extensors during the flexion–relaxation phenomenon. The flexion–relaxation phenomenon is an interesting task because there are no substantial lateral or twisting torques and the extensor torque appears to be supported passively, further suggesting some stabilizing role for the QL.

In another experiment (note again that our laboratory techniques to obtain QL activation were rather imprecise at the time), subjects stood upright with a bucket in each hand. We incrementally increased the load in each bucket (resulting in progressively more spine compression). Our data suggest that the QL increased its activation level (together with the obliques) as more stability was required (McGill, Juker, and Kropf, 1996b). This task creates a special situation because only compressive loading is applied to the spine in the absence of any bending moments. In summary, the strength of the evidence from several perspectives leaves one to conclude that the QL performs a very special stabilizing role for the lumbar spine in a wide variety of tasks. Most recently, the Hodges group (Park et al., 2013) strengthened the concept that there can be a redistribution of muscle activity in pain. In particular, there is a tendency for activity to migrate extensor activity to the quadratus lumborum. Given the number of patients with back pain who have perceived spasm and pain in this muscle, experimental follow-up is recommended.

Muscle Summary

This section provides an overview of the roles of the muscles of the trunk in supporting postures and moving and stabilizing the lumbar spine. The posterior muscles were presented in four large functional groups. The muscles in the deepest group (the small rotators) appear to act as position sensors rather than as torque generators. The more superficial extensors (multifidus and iliocostalis lumborum and longissimus thoracis) fall into three categories to do the following:

- Generate large extension moments over the entire lumbar region
- Generate posterior shear
- Affect and control only one or two lumbar segments

The roles of the abdominal muscles in trunk flexion and trunk stabilization were highlighted together with the roles of the psoas and QL. Clearly, many muscles play a large role in ensuring sufficient stiffness to bear load and create movement, while protecting the low back from injury. Chapter 10 applies these findings to exercise regimens for those with low back pain.

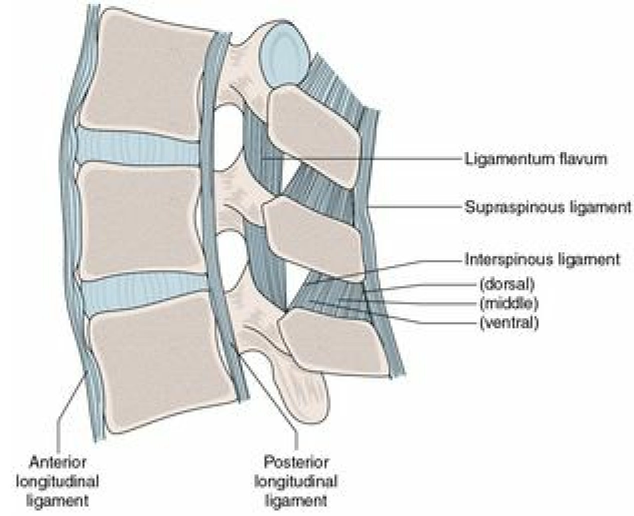
Ligaments

When the lumbar spine is neither flexed nor extended (neutral lordosis), only muscle contributions need be considered in the mechanics to support the spine. However, as the spine flexes, bends, and twists, passive tissues are stressed; the resultant forces of those tissues change the interpretation of injury exacerbation, the discussion of clinical issues, or both. For this reason, I introduce the mechanics of passive tissues in this section, followed by some examples illustrating their effects on clinical mechanics. Also fascinating is the distribution of mechanoreceptors documented in every lumbar ligament and fascia. Solomonow and colleagues' (2000) evidence suggests a significant proprioceptive role for spinal ligaments.

Longitudinal Ligaments

The vertebrae are joined to form the spinal column by two ribbon-like ligaments, the anterior longitudinal and the posterior longitudinal ligaments, which assist in restricting excessive flexion and extension (see figure 3.42). Both ligaments have bony attachments to the vertebral bodies and collagenous attachments to the annulus. Very little evidence exists for the presence of mechanoreceptors in these ligaments. Posterior to the spinal cord is the ligamentum flavum, which is characterized by a composition of approximately 80% elastin and 20% collagen, signifying a very special function for this ligament. It has been proposed that this highly elastic structure, which is under pretension throughout all levels of flexion, acts as a barrier to material that could buckle and encroach on the cord in some regions of the range of motion.

Figure 3.42 Major lumbar ligaments. Note the controversy surrounding the interspinous ligament in figures 3.43 and 3.44.



Interspinous and Supraspinous Ligaments

The interspinous and supraspinous ligaments are often classed as a single structure in anatomy texts, although functionally they appear to have quite different roles. The interspinous ligaments connect adjacent posterior spines but are not oriented parallel to the compressive axis of the spine. Rather, they have a large angle of obliquity, which has been a point of contention. For years most anatomy books have shown these ligaments with an oblique angle that would cause posterior shear of the superior vertebrae (see figure 3.43). This error is believed to have originated around the turn of the century. The hypothesis is that an artist held the vertebral section upside down when drawing; other artists simply copied the previous art rather than look at a spine. This was corrected by Heylings (see figure 3.44), noting that indeed these ligaments have the obliquity to resist posterior shear of the superior vertebrae—but also impose anterior shear forces during full flexion (Heylings, 1978). Although many anatomy textbooks suggest that this ligament serves to protect against excessive flexion (based on erroneous anatomy), I do not believe that this notion is correct. Heylings (1978) suggested that the ligament acts like a collateral ligament similar to those in the knee, whereby the ligament controls the vertebral rotation as it follows an arc throughout the flexion range. This in turn helps the facet joints remain in contact, gliding with rotation. Furthermore, with its oblique line of action, the interspinous ligament protects against posterior shearing of the superior vertebrae and is implicated in an injury scenario discussed later in this chapter.

Figure 3.43 For most of the past 100 years, many anatomical artists have drawn the interspinous ligament upside down, as shown in this example. Such drawings have caused the ligament function to be misinterpreted as that of a supporter of anterior shear, which is incorrect.

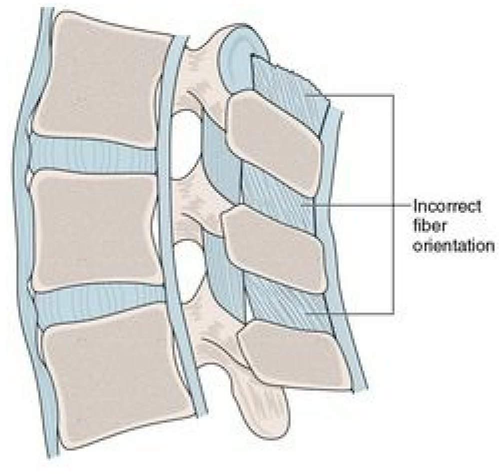
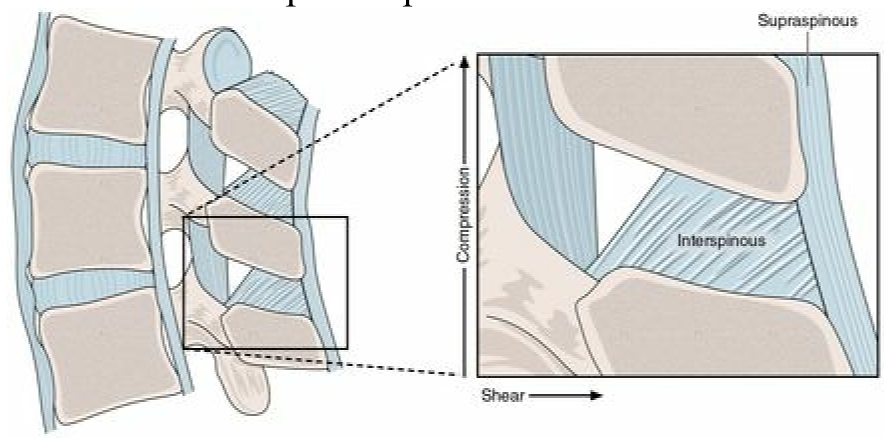


Figure 3.44 The interspinous ligament runs obliquely to the compressive axis and thus has limited capacity to check flexion rotation of the superior vertebrae. Rather, the interspinous ligament may act as a collateral ligament, controlling vertebral rotation and imposing anterior shear forces on the superior vertebrae. (ZG) Zygapophyseal joint or facet joint, (L2), (L3) lumbar spinous processes.



In contrast to the interspinous ligament, the supraspinous ligament is aligned more or less parallel to the compressive axis of the spine, connecting the tips of the posterior spines. It appears to provide resistance against excessive forward flexion. Finally, both supraspinous and interspinous ligaments have an extensive network of free nerve endings (type IV receptors) (Yahia, Newman, and Rivard, 1988) together with Ruffini corpuscles and Pacinian corpuscles (Jiang et al., 1995; Yahia, Newman, and Rivard, 1988). Yahia and colleagues (1988) and Solomonow and colleagues (2000) suggested a proprioceptive role for the ligaments to prevent excessive strain in fully flexed postures and—given their architecture—quite possibly when under excessive shear load.

Interspinous Ligament Architecture

Clinical Relevance

The interspinous ligament appears to provide a collateral action as it guides the sliding motion of the facet joints and checks posterior shear of the superior vertebra. The flip side of this functional role is that during full flexion the superior vertebra is sheared anteriorly, often adding to the reaction shear forces produced in a forward bending posture. Therapeutic exercise recommending full spine flexion stretches must consider the resultant shearing forces imposed on the joint by interspinous ligament strain. Too often, even patients with shear pathology—for example, those with spondylolisthesis—are prescribed flexion stretches. This appears to be ill-advised.

Other Ligaments in the Thoracolumbar Spine

Other ligaments in the thoracolumbar spine include the intertransverse ligaments and the facet capsule.

- Intertransverse ligaments. These ligaments span the transverse processes and have been argued to be sheets of connective tissue rather than true ligaments (Bogduk and Twomey, 1991). In fact, Bogduk and Twomey suggested that the intertransverse ligament membrane forms a septum between the anterior and posterior musculature that is an embryological holdover from the development of these two sections of muscle.
- Facet capsule. The facet capsule consists of connective tissue with bands that restrict both the joint flexion and the distraction of the facet surfaces that result from axial twisting. The ligaments that form the capsule have been documented to be rich in proprioceptive organs—Pacinian and Ruffini corpuscles (Cavanaugh et al., 1996; McLain and Pickar, 1998)—and have been observed to respond to multidirectional stress (Jiang et al., 1995), at least in cats.

Normal Ligament Mechanics and Injury Mechanics

Determining the roles of ligaments has involved qualitative interpretation using their attachments and lines of action together with functional tests in which successive ligaments were cut and the joint motion was reassessed. Early studies attempting to determine the amount of relative contribution of each ligament to restricting flexion were performed on cadaveric preparations that were not preconditioned prior to testing. This usually entails a loading regimen so that the cadaveric specimens better reflect in vivo behavior.

Specifically, the investigators did not take into account the fact that upon death, discs, being hydrophilic, increase their water content and consequently their height. The swollen discs in cadaveric specimens produced an artificial preload on the ligaments closest to the disc, inappropriately suggesting that the capsular and longitudinal ligaments are more important in resisting flexion than they actually are in vivo. For this reason, these early data describing the functional roles of various ligaments were incorrect. The work of Sharma, Langrama, and Rodriguez (1995) showed that the major ligaments for resisting flexion are the supraspinous ligaments. Those early studies showing that the posterior longitudinal ligament and the capsular ligaments are important for resisting flexion did not employ the necessary preconditioning just discussed.

Mechanical failure of the ligaments is worth considering. King (1993) noted that soft tissue injuries are common during high-energy, traumatic events such as automobile collisions. Our own observations on pig and human specimens loaded at slow load rates in bending and shear modes suggest that excessive tension in the longitudinal ligaments causes avulsion or bony failure near the ligament attachment site. Noyes, De Lucas, and Torvik (1994) noted that slow strain rates (0.66%/s) produced more ligament avulsion injuries, whereas fast strain rates (66%/s) resulted in more midligamentous failure, at least in monkey knee ligaments. The clinical results of Rissanen (1960), however, showed that approximately 20% of randomly selected cadaveric spines possessed visibly ruptured lumbar interspinous ligaments, whereas the dorsal and ventral portions of the interspinous ligaments and the supraspinous ligaments were intact.

Patterns of Ligament Injury

Clinical Relevance

Torn spinal ligaments appear to be the result of ballistic loading—particularly slips and falls or traumatic sporting activity with the spine at its end range of motion. Those with recently developing spine symptoms and accompanying spine instability can often recount prior incidents in which the ligaments could have been damaged. If the traumatic event occurs at work but the delayed sequellae develop later during an event at home, is this compensable? Arguing these types of questions requires a solid understanding of injury mechanisms.

These results could be considered to represent the living population. Given the oblique fiber direction of the interspinous complex (see figure 3.44), a very likely scenario of interspinous ligament damage is falling and landing on one’s behind, driving the pelvis forward on impact and creating a posterior shearing of the lumbar joints when the spine is fully flexed. The interspinous ligament is a major load-bearing tissue in this example of high-energy loading characterized by anterior shear displacement combined with full flexion. Considering the available data, I believe that disruption of the ligaments of the spine, particularly the interspinous complex, is not likely during lifting or other normal occupational activities. Rather, ligament damage seems to occur primarily during traumatic events, as described earlier. The subsequent joint laxity is well known to accelerate arthritic changes (Kirkaldy-Willis and Burton, 1992). What has been said in reference to the knee joint, that ligament damage marks the beginning of the end, is also applicable to the spine in terms of being the initiator of a cascade of degenerative change.

Chronic loading of the posterior ligaments, at subfailure loads, occurs during repeated bending and flexing of the spine. Solomonow's group has documented in many studies the changes in proprioceptive function as the ligaments are repeatedly stretched. Their recent work has shown elevated proinflammatory cytokines 7 hours after a repeated loading session and suggested that acute inflammation conditions can occur that can become chronic inflammation with further exposure to ligament loading (D'Ambrosia et al., 2010).

Lumbodorsal Fascia (LDF)

Although a functional interpretation of the lumbodorsal fascia (LDF) (also called the thoracolumbar fascia by some) is provided later, a short anatomical description is given here. First, the fascia has bony attachments on the tips of the spinous process (except the shorter L5 in many people) and to the posterior superior iliac spines (PSIS). Some fascial connections cross the midline, suggesting some force transmission, thus completing the hoop around the abdomen with the previously described abdominal fascia anteriorly. The transverse abdominis and internal oblique muscles obtain their posterior attachment to the fascia, as does the latissimus dorsi over the upper regions of the fascia. The fascia, in wrapping around the back, forms a compartment around the lumbar extensors (the multifidus and pars lumborum groups of the iliocostalis and longissimus) and has been implicated in compartment syndrome (Carr et al., 1985; Styf, 1987) (see figure 3.45). Recent studies attribute various mechanical roles to the LDF. In fact, some have recommended lifting techniques based on these hypotheses. However, are they consistent with experimental evidence? Gracovetsky, Farfan, and Lamy (1981) originally suggested that lateral forces generated by the internal oblique and transverse abdominis muscles are transmitted to the LDF via their attachments to the lateral border, and that the fascia could support substantial extensor moments. They hypothesized that this lateral tension on the LDF increased longitudinal tension by virtue of the collagen fiber obliquity in the LDF, causing the posterior spinous processes to move together, resulting in lumbar extension. This proposed sequence of events formed an attractive proposition because the LDF has the largest moment arm of all the extensor tissues. As a result, any extensor forces within the LDF would impose the smallest compressive penalty to vertebral components of the spine.

Figure 3.45 Collagen fiber arrangement in the LDF binds the lumbar extensor muscles and tendons from the thoracic muscles together as they course to the sacral attachments. Thus, one of the functions of the LDF appears to be acting as an extensor muscle retinaculum—and a natural abdominal–back belt.

Photo from Stuart McGill

However, three studies, all published at about the same time, collectively challenged the viability of this hypothesis: Tesh and colleagues (1987) performed mechanical tests on cadaveric material; Macintosh, Bogduk, and Gracovetsky (1987) recognized the anatomical inconsistencies with the abdominal activation; and McGill and Norman (1988) tested the viability of LDF involvement with the latissimus dorsi as well as with the abdominal muscles (see figure 3.46). These collective works show that the LDF is not a significant active extensor of the spine. Nonetheless, the LDF is a strong tissue with a well-developed lattice of collagen fibers, suggesting that its function may be that of an extensor muscle retinaculum (Bogduk and Macintosh, 1984), or nature's back belt. In addition, the fascia does contain both Ruffini and Pacinian corpuscles together with diffuse innervation (Yahia, Newman, and Rivard, 1998). The tendons of longissimus thoracis and iliocostalis lumborum pass under the LDF to their sacral and iliac attachments. It appears that the LDF may provide a form of retinacular strapping for the low back musculature. Finally, the abdominal wall and the latissimus dorsi forces add tension to the fascia and stiffness to the spine to prevent specific types of unstable behavior and tissue damage (explained in chapter 5 on spine stability).

Figure 3.46 Stress lines in the LDF indicate that the latissimus dorsi is the dominant force activator—at least in this example.

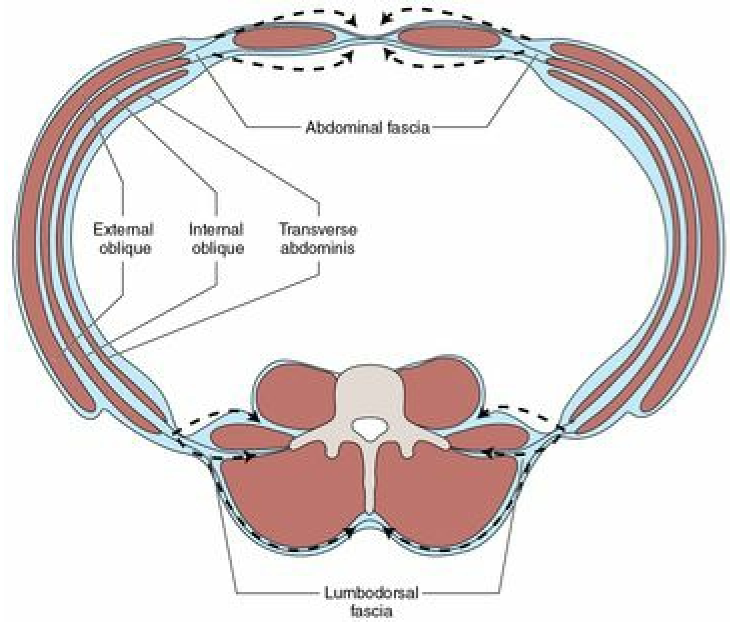
Photo from Stuart McGill

Lumbodorsal Fascia Anatomy: Nature's Back Belt

Clinical Relevance

No evidence justifies specific lifting techniques to involve the LDF for extension of the spine. However, activation of the latissimus dorsi and the deep abdominal obliques contributes stiffening (and stabilizing) forces to the lumbar spine via the fascia (guidelines for activating these muscles are provided in chapter 10). Furthermore, the LDF appears to act as a retinaculum and probably fulfills a proprioceptive function. It is part of a hoop around the abdomen, which consists of the LDF posteriorly, the abdominal fascia anteriorly, and the active abdominal muscles laterally; the three together complete the stabilizing corset (see figure 3.47). As noted earlier, this also appears to be an important elastic energy storage-recovery device for ballistic athletes, tuned by the obliques.

Figure 3.47 The abdominal fascia, anteriorly, and the LDF, posteriorly, are passive parts of the abdominal hoop. The lateral active musculature (primarily the larger internal oblique and external oblique together with the smaller transverse abdominis) serves to tension the hoop (dashed arrows).

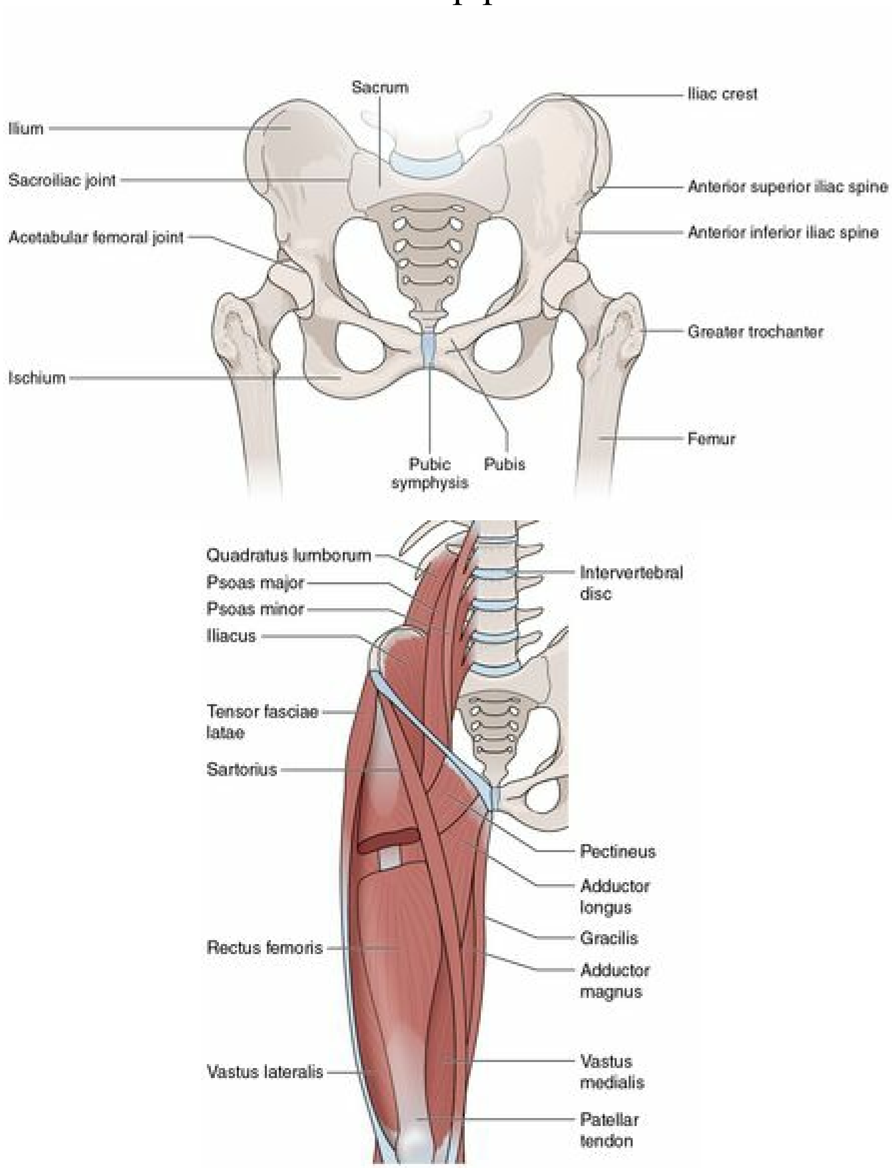


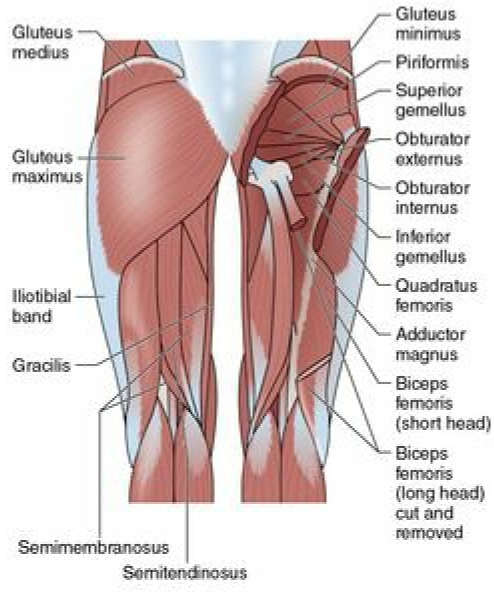
Quick Review of the Pelvis, Hips, and Related Musculature

A healthy back depends on proper function in the pelvis and hips for several reasons. Power is usually generated at the hips, for both performance and safety reasons. Further, the pelvis acts as the platform for the spine. Given the exercises that ensure optimal hip function with spine integration, this brief overview of the relevant anatomy will give context to the exercises later in the book.

The pelvis is usually sectioned into three regions: the ilium, ischium, and pubis. Articulations and small motions occur at the pubis anteriorly and at the sacroiliac joints (see figure 3.48). Several nerve block studies have shown these to be potential causes of pain. Probably the most relevant factor for exercise-related issues is the architecture of the acetabulum and of the muscles. The hip socket depth and shape influence the ability to squat deeply, which in turn influences the bending moment on the lumbar discs, in particular L5/S1. Interestingly, the shape of the acetabulum tends to be associated with different regions of the world (e.g., Aly and Fuji, 2013). For example, Poland has the highest rate of hip dysplasia (indicative of a shallow acetabulum) (Loder and Skopelja, 2011). But this type of hip shape is best suited for deep squatting—it is no wonder the majority of Olympic weightlifters come from this region of the world. They do not come from Scotland! The reason is that the western European Celtic hip is characterized by a very deep acetabulum (Inoue et al., 2000), which accounts for the very high incidence of anterior femoral hip impingement in that region (Gliedt and Scali, 2012). From a performance perspective the power production in hip extension is very poor out of the bottom of the squat. But the standing stability of the hip is supreme. Tests to define the hip architecture and subsequent impact on therapeutic decisions are discussed later. Training implications are well discussed in the book Ultimate Back Fitness and Performance (McGill, 2014).

Figure 3.48 View of the pelvis and hips. Together they function to stabilize and create hip power.





The psoas and iliacus have already been described for their role in hip flexion and stabilization. Gluteus maximus is primarily a hip extensor and external rotator, whereas gluteus medius and gluteus minimus are primarily abductors and external rotators, thus tremendously important for any activity that requires a single-leg stance or a gait with directional change. They assist the spine musculature (such as QL and the obliques) to hold the pelvis up during single-leg support and thus are key players in spine stability during gait. They also externally rotate the femur, which is a functional feature we use to full advantage in the design of squat exercises later in this book.

The gluteus maximus also has fascia connections across the sacroiliac joints that transfer load (Barker et al., 2014). In this way they add stiffness and stability to the pelvic ring and transfer force across the spine–pelvis junction. This architecture also is consistent with the EMG patterns for walking and carrying in which the gluteus on one side is contracted with the quadratus, extensors, and obliques on the other. This is one of the foundations for beginning training progressions with the bird-dog exercise—it challenges the muscles groups with this same synergy. It also surprises many people to learn that the gluteal muscles are efficient extensors of the knee. Even though they do not cross the knee, they work through the skeletal linkage. Here, during a squat, the gluteals extend the hip as it rotates the femur, in turn extending the knee. This unloads the patellar tendon and assists in knee pain in complex hip/knee/back pain syndromes, a technique introduced later. Other "gluteal" muscles known as the deep six (piriformis, obturator internus and externus, gemellus superior and inferior, and quadratus femoris) together assist in controlling internal and external rotation.

The muscles in the hamstring group (biceps femoris, semitendinosus, semimembranosus) extend the thigh, flex the knee, and perform stabilizing roles over each of these joints. In many upright situations, particularly walking and running, their most important role is in braking with eccentric contraction. Yet in stooped postures, particularly lifting, the hamstring group is very important for their contributions to hip extension. The medial thigh muscles adduct the thigh. The quadriceps extend the knee and provide a patellar tendon tracking function. One member of the "quads," rectus femoris, crosses the hip joint anteriorly to create hip flexion. Tight hamstrings are often blamed for back troubles, but as is noted several times in this book, this blame is often misdirected. The bulk of evidence supports neither a link between shorter hamstrings and back troubles nor the idea that stretching enhances strength output (e.g., Avela et al., 2003; Fowles, Sale, and MacDougall, 2000) and offers no protective value against injury risk (e.g., Black and Stevens, 2001). Furthermore, what is often attributed to hamstring tightness is actually neural tension so that stretching only worsens the back and radiating leg symptoms. Evidence suggests that hamstring trauma can lead to neural tension (Butler, 1989), although more often the source of the neural tension is associated with a lumbar nerve root or central stenosis.

Training the Hip Musculature

No single exercise can train all hip muscles, although there are some particularly good ones. For example, the one-legged squat is particularly functional, because the quadriceps are active for knee extension and patella control; the hamstrings, for knee stability and hip extension; the gluteals, for hip extension and hip abduction; and the adductors and gluteal deep six, for hip stabilization. Another clinical issue is the peril in training hip flexion power given the associated loading of the lumbar spine. This is usually reserved for those who no longer have pain, because training hip flexion often retards progress toward the elimination of pain. It is usually wise to build extensive spine stability prior to progressing to building hip power.

Hip mobility is essential for sparing the back from bending movement that causes stress, together with assisting with power transfer to the legs. The question is how best to restore and maintain hip mobility. Moreside and McGill (2011, 2012) performed a suite of studies showing that stretching targeting muscle, nerve, joint capsule, and fascia can indeed enhance the range of motion. However, they found that enhancing core stiffness enhanced hip mobility. Further, simply adding to the hip joint range of motion did not translate into more hip motion during a variety of functional activities. This is because the movement engrams, or encoded patterns of motion, need to be changed. The implication is that if more movement in the hips is desired, a hip mobility program needs follow-up exercise designed to rewrite the movement engrams to incorporate the newfound movement.

Considering these muscles reveals the tremendous number of them that cross the hip joint. Collectively, they can create significant power, and they can direct force in a powerful manner in many directions. In many cases they are the major power source for functional performance. Specifically, they create and direct force while changing length at rapid speed. Many of them cross both the hip and knee joint, indicating that functional training must involve tasks that challenge both joints at different velocities. Further, this training must incorporate lateral motion and rotational motion in the transverse plane. All function involving power generation across the hip relies on the ability of the core to stiffen and stabilize. The stiffer the core, the greater the power transfer to the limbs.

Clinically Relevant Aspects of Pain and Anatomic Structure

Recall from the introduction to this book that tissue damage can alter the biomechanics of a spinal joint, and that once the biomechanics have changed, any innervated tissue can be the candidate for symptoms. Patients do not get used to pain, nor is it in their heads, as poorly trained doctors often claim. Pain biology clearly describes pathways beginning with mechanical factors such as disc bulges causing nerve root compression. Pain originates with the free nerve endings of the various pain receptors that typically form small nerve fibers. As Guyton (1981) noted, not all of the small fibers originate in pain receptors: some originate in organs sensitive to temperature, pressure, or other sensations. Pain also may be initiated at higher levels in the pain pathway: Howe, Loeser, and Calvin (1977) demonstrated that mechanical pressures on the dorsal root ganglion produce discharges for up to 25 minutes following the removal of the mechanical pressure. In addition, Cavanaugh (1995) showed that nerve endings are sensitive to chemical mediators released during tissue damage and inflammation. Some studies have attempted to examine inflammatory processes using the injection of chemicals. For example, Ozaktay and colleagues (1994) injected carrageenan into the region of nerve receptors around the facet joints of rabbits and reported that the discharge from the pressure-sensitive neurons lasted 3 hours. This finding suggests that tissue damage producing inflammatory processes may contribute to a long-lasting muscle spasm.

Direct mechanical contact and pressure on the nerve roots located at each level of the spine create changes that cause pain and pain sensitivity. Hubbard, Chen, and Winkelstein (2007) measured the allodynia (increase in pain sensitivity) from mechanical pressure applied to nerve roots simulating a disc bulge in an animal model (unaffected by compensation, an employer, an emotional state, and so on.). They investigated the increased neuropeptides that cause this disabling and disruptive pain and their link to load magnitude and duration. Others have documented mechanical compression causing edema inside the nerve root sheaths (Olmarker, Rydevik, and Holm, 1989; Winkelstein et al., 2001), which results in bilateral sensitivity (Hubbard and Winkelstein, 2005) and central sensitization. This is similar to hitting your thumb with a hammer. Your tissues are damaged and sensitized. Hit it again tomorrow and it will be more painful. In two days, hitting your thumb will be excruciating and disabling. This is what many back pain sufferers experience because of the way they load and move their spines, unknowingly hitting their spines with pain hammers.

Bogduk (1983) provided an excellent review of the innervation of lumbar tissues. For example, the facet is well innervated with a variety of low- and high-threshold nerves, suggesting both pain and nociceptive functions. Free nerve endings also have been observed around the superficial layers of intervertebral discs. With injury, nerves sprout into the damaged region. In summary, mechanical factors cause disabling pain that lasts. Addressing this pain by changing the mechanical cause is the only way to have success with many back pain cases.

Tissue-Specific Types of Pain

I have had some personal experience with direct mechanical irritation of specific low back tissues and the resultant pain. Admittedly, these results are limited, given the single subject (myself) and the subjective nature of the observations, but I believe they are worth reporting nonetheless. I obtained these insights from indwelling EMG experiments in which needles were used to implant fine wire EMG electrodes in the psoas, QL, multifidus, and three layers of the abdominal wall. Many people have experienced the burning sensation as the needle penetrates the skin. This is cutaneous pain, because the application of ice and a hot material feels similar. As the needle applies pressure to, and punctures through, the LDF, the pain is felt as a scraping sensation and sometimes as an electric current. The same sort of pain is felt as the needle progresses through the various sheaths between the layers of muscle of the abdominal wall. It is interesting to note that people with fibromyalgia sometimes report this scratchy type of muscularly located pain —it is very consistent with epimysium and fascia irritation. Once the needle was inside the muscle, no pain was perceived, just an occasional feeling of mechanical pressure. As the needle touched the peritoneum of the abdominal cavity in any location, a general intestinally sick feeling was produced in the abdominal region, focused anteriorly to a small area just below the naval. As the needle touched the bone of the vertebra, even with very light pressure, a very pointed and “boring” pain occurred, similar to the pain experienced on being kicked in the shins. Once again, these are the experiences of a single person. Nonetheless, they do provide crude qualitative insight into the types of pain produced in specific tissues.

Can Pain Descriptors Provide a Reliable Diagnosis?

Pain is clearly produced from tissue irritation, particularly mechanical overload. Some have argued that some tissues may or may not be candidates for sources of pain based on the presence or absence of nociceptive nerve endings. This may be a diversionary argument. If the overload is sufficient to damage tissue and produce biomechanical change in the joint, then the loading patterns of other tissues are disturbed. Thus, even though one tissue may not be capable of producing pain, if it is damaged sufficiently to shift load to another suitably innervated tissue, pain may result. For example, innervated annulus and disc end plates may be sources of pain as a consequence of end-plate fracture or annular herniation. But end-plate fracture can cause significant disc height loss, which can lead to nerve entrapment, complex joint instability, subsequent facet overload, and so on. Evidence from our lab (Freeman et al., 2013) showed that induced hip pain inhibited gluteal activation, which changes the loading on the spine and anterior hip joint capsule and labrum. Once the biomechanics of the joint have been altered, it is no longer fruitful to attempt to diagnose specific tissue damage; the picture is complex. Functional diagnosis is the only feasible option.

A Final Note

The discussion in this chapter assumes a rudimentary anatomical and biomechanical knowledge. Using this foundation, some anatomical features were reviewed that are not often considered or discussed in classic anatomical texts. A serious discussion of anatomy must involve function and, by extension, must consider biomechanics and motor control. Hopefully, the functional discussions throughout this chapter have stimulated you to give more consideration to the architecture of the lumbar spine. The challenge for the scientist and clinician alike is to become conversant with the functional implications of the anatomy, which will guide decisions to develop the most appropriate prevention programs for the uninjured and the best treatments for patients.

Chapter 4
Normal and Injury Mechanics of the Lumbar Spine

Chapter 3 describes the tissues, or the anatomical parts, and their role in function; this chapter describes the normal mechanics of the whole lumbar spine. Because most biomechanics texts provide descriptions of spine motion, I address that only briefly here. Instead, I focus on the functional implications of that motion, which are far more important. I also explain injury mechanisms and the changes that follow injury. Controversy remains as to whether these changes are a consequence of injury or in fact play a causative role. In addition, muscle activation challenges, spine loads, and in some cases stability are assessed over a variety of rehabilitation exercises and activities. This enables better decisions for therapeutic program design, because you will know what your clients should avoid and what they should perform.

Upon completion of this chapter, you will be able to explain the role of tissues in various tasks and consequently identify back-sparing techniques. In addition, you will understand the changes that follow injury, which have an impact on functional ability and rehabilitation decisions.

Kinematic Properties of the Thoracolumbar Spine

The ranges of thoracic and lumbar segmental motion about the three principal axes (shown in table 4.1) demonstrate the greater flexion, extension, and lateral bending capability of the lumbar region and the relatively greater twisting capability of the thoracic region. The segmental ranges shown in the table are population averages, but keep in mind that a large variability exists among people, among age groups (McGill, Yingling, and Peach, 1999), and among segments within an individual.

Table 4.1 Range of Motion of Each Spine Level (in Degrees)

Level	Flexion	Flexion and extension combined	Extension	Lateral bending	Axial twist
T1-2		4		6	9
T2-3		4		6	8
T3-4		4		6	8
T4-5		4		6	8
T5-6		4		6	8
T6-7		5		6	8
T7-8		6		6	8
T8-9		6		6	7
T9-10		6		6	4
T10-11		9		7	2
T11-12		12		9	2
T12-L1		12		8	2
L1-2	8		5	6	2
L2-3	10		3	6	2
L3-4	12		1	8	2
L4-5	13		2	6	2
L5-S1	9		5	3	5

All data are from White and Panjabi (1978), except flexion and extension lumbar data, which are from Pearcy, Portek, and Shepherd (1984) and Pearcy and Tibrewal (1984).

Joint stiffness values convey the amount of translational and rotational deformation of a spine section under the application of force or moment. The average stiffness values (shown in table 4.2) document the translational stiffness of the spine in a neutral posture; they indicate greater stiffness under compression than under shear loads. In rotational modes, greater stiffness occurs during axial torsion than during rotation about the flexion–extension and lateral bending axes. Although generally, the range of motion decreases with age, certain injuries, particularly to the disc, can increase the range of motion in bending and shear translation (Spencer, Miller, and Schultz, 1985). Kirkaldy-Willis and Burton (1992) implicated these large unstable movements in facet joint derangement. Recent data have quantified the increase in the range of motion about all three spine axes as disc degeneration proceeds from grade I to grades III and IV. Radial tears of the annulus are most prevalent in these stages. But this extra motion is replaced by extreme loss of motion in grade V discs, which are characterized by collapse and osteophyte formation (Tanaka et al., 2001).

Table 4.2 Average Stiffness Values for the Adult Human Spine

		>SHEAR		BENDING		
Spine level	Comp.	Ant./Post.	Lat.	Flex./Ext.	Lat.	Axial torsion
T1-T12	1,250	86/87	101	155/189	172	149
L1-L5	667	145/143	132	80/166	92	395
L5-S1	1,000	78/72	97	120/172	206	264

Compression and shear values are given in newtons per millimeter and bending and axial torsion in newton-meters per radian.

T1-T12 data are from White and Panjabi (1978). L1-L5 data are from Schultz et al. (1979) and Berkson, Nachemson, and Shultz (1979). L5-S1 data from McGlashen et al. (1987).

Motion Palpation—Pathology or Normal Asymmetry?

Clinical Relevance

When assessing a patient, stiffness asymmetries during bending to the right compared to the left and during twisting clockwise compared to counterclockwise at specific vertebral levels are not uncommon. This finding is of great importance to the clinician, who may suspect pathology at a specific location when what is present is simply normal anatomical asymmetry.

Our work (Ross, Bereznik, and McGill, 1999) exemplifies the peril in assuming that a joint with an asymmetrical feel upon palpation is pathological. Clinicians who hold to a typical motion palpation philosophy often identify an abnormal feeling at a specific spinal level as the target for therapy. Sometimes the asymmetrical stiffness is simply asymmetrical skeletal anatomy—perhaps a single facet with a unique angular orientation. Obviously, such a joint would be resistive to any mobilizing therapy.

As the spine moves in three dimensions (flexion–extension, lateral bend, and twist), the alignment of muscle vectors changes with respect to the vertebral orthopedic axes. This causes the role of the muscles to change. Sometimes their relative contribution to producing a specific moment changes along with the resultant joint compression and shear. Muscle lengths and their moment potentials as a function of spine posture are shown in table 3.2 in chapter 3 and table 4.3 here. A range of extreme postures is shown in figure 4.1. Some muscles close to the spine obviously do not undergo great length excursions.

Table 4.3 Moment Potential (N · m) of Some Representative Muscles in Postures Shown in Figure 4.1

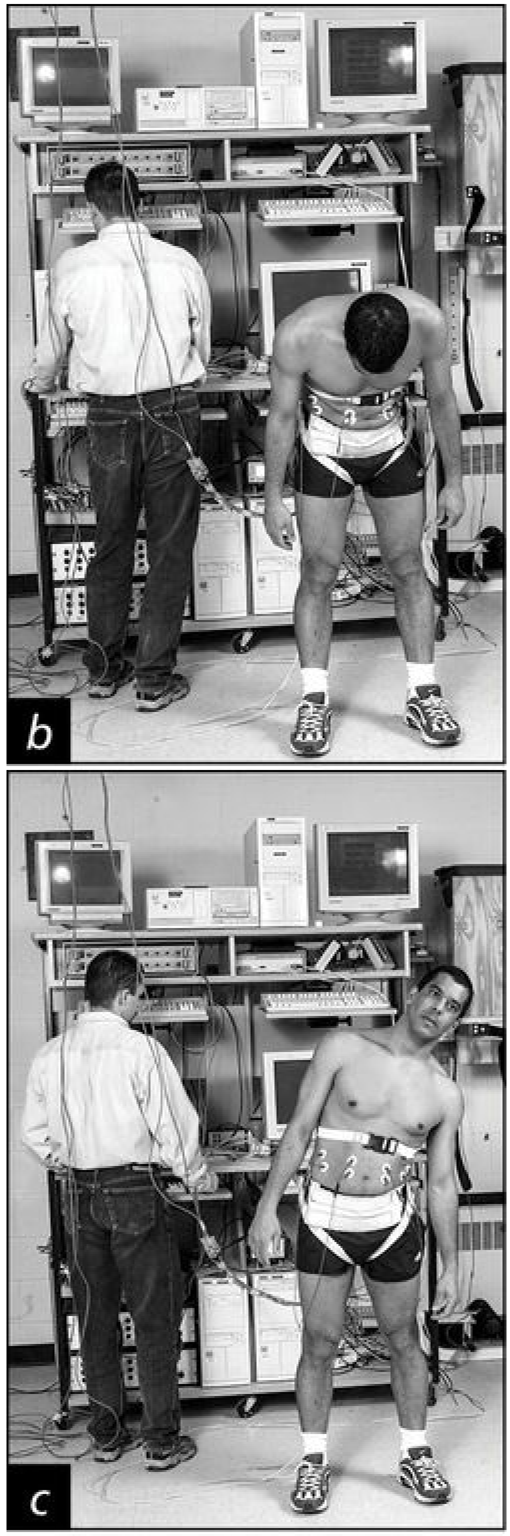
	FORCE	UPRIGHT STANDING			60° FLEXION			25° RIGHT LATERAL BENDING			10° TWIST			COMBINED[a]		
Muscle	(N)	F[b]	B[c]	T[d]	F	B	T	F	B	T	F	B	T	F	B	T
R rectus abdominis	350	−28	17	6	−35	17	12	−28	21	8	−29	15	4	−33	20	15
R external oblique	315	−7	27	9	−7	24	15	−7	28	11	−8	28	6	−1	20	22
R internal oblique (1)	280	−15	19	−16	−9	8	−31	−13	18	−20	−14	17	−18	−2	2	−33
R pars lumborum (L1)	455	36	23	9	35	23	8	36	24	9	3	24	5	38	23	7
R pars lumborum (L4)	595	23	23	−7	23	24	−4	23	23	−8	23	22	−8	23	25	−6
R iliocostalis lumborum	210	18	15	−1	18	14	2	18	15	−1	18	15	−2	18	15	1
R longissimus thoracis	280	24	10	−3	22	10	0	24	13	−3	24	12	−5	24	11	−1
R quadratus lumborum	175	5	11	5	7	14	1	5	12	6	6	13	4	7	14	0
R latissimus dorsi (L5)	140	9	5	−3	8	7	−4	8	6	−5	9	5	−5	8	7	−6
R multifidus	98	5	1	3	5	1	2	5	1	3	5	1	2	5	1	2
R psoas (L1)	154	1	9	3	-5	9	2	1	9	4	1	6	3	−4	8	4

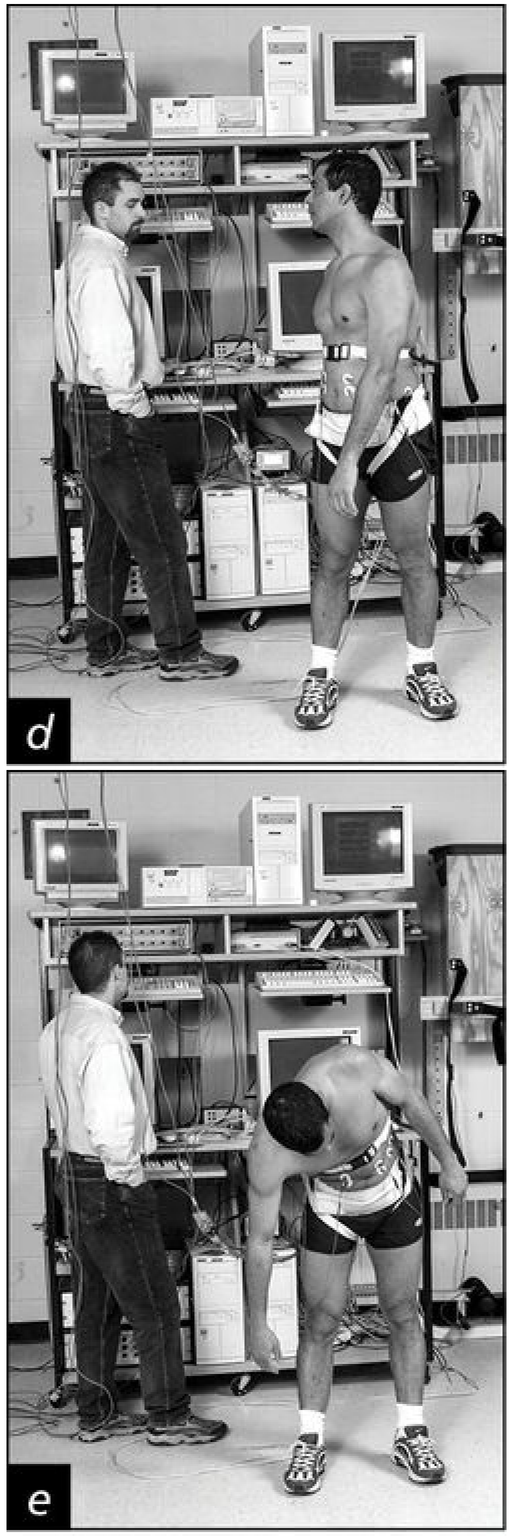
Only a portion of some muscles are represented; for example, the laminae of psoas to L1 is shown rather than the whole psoas.

[a]Combination of 60° flexion, 25° right lateral bending, and 10° counterclockwise twist in one posture; [b]F = flexion; [c]B = lateral bend; [d]T = axial twist.

Reprinted, by permission, from S.M. McGill, 1991, "Kinetic potential of the lumbar trunk musculature about three orthogonal orthopaedic axes in extreme positions," Spine 19(7): 809-815.

Figure 4.1 A range of extreme postures was chosen to assess muscle length changes (table 4.3) and their potential to produce three-dimensional moments (table 4.3). Postures depicted are (a) upright standing, (b) 60° flexion, (c) 25° right lateral bending, (d) 10° twist, and (e) combined.


b
c

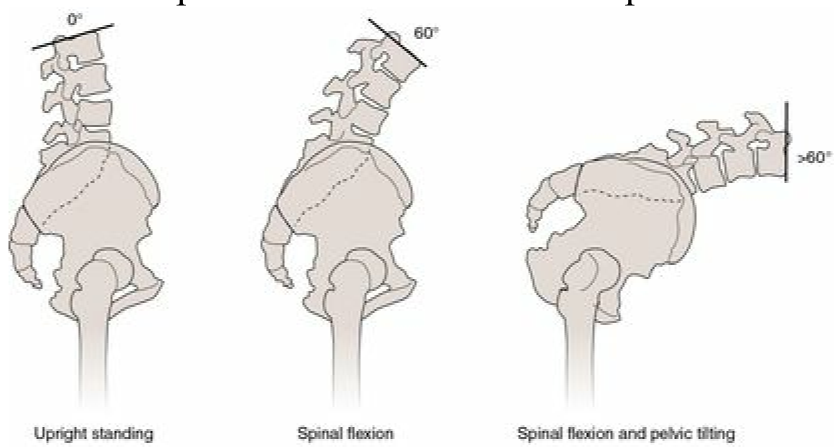


Photos from Stuart McGill

Lumbopelvic Rhythm: Questionable Assumptions

The typical description of torso flexion suggests that the first 60° takes place in the lumbar spine, whereas any further flexion is accomplished by flexion about the hips (see figure 4.2). Although this notion is very popular in clinical textbooks, we have never measured this strict sequence in anyone. In fact, Olympic weightlifters attempt to do the opposite—they lock the lumbar spine close to the neutral position and rotate almost entirely about the hips. When the lumbar spine and hip interplay is quantified in most people, it is apparent that torso flexion is accomplished with a combination of hip motion and lumbar spine flexion. In fact, given the ligament and annulus stresses associated with lumbar flexion, avoidance of full spine flexion is both prophylactic and therapeutic for most patients. The belief that the lumbopelvic rhythm (with distinct separation of spine and pelvic motion) is beneficial, as described in so many textbooks, appears to be a clinical myth and not the product of quantified spine and pelvis motion.

Figure 4.2 The lumbopelvic rhythm is the textbook description of how people bend over. It has been assumed that the first 60° takes place in the lumbar spine (flexing), and that further rotation of the torso is accomplished with rotation about the hip. We have never measured this sequence in anyone —from professional athlete to back patient.



Kinetics and Normal Lumbar Spine Mechanics

An interpretation of the function of the anatomical components of the lumbar spine requires the following:

- An analysis of their architecture and neural activation of muscles
- Knowledge of forces in the individual tissues (both active and passive) during a wide variety of tasks

This information is crucial for understanding how tissue overloading and injuries occur and also for optimizing treatment strategies for specific spine injury. Table 4.4 and figure 4.3 provide activation levels quantified with surface and intramuscular electromyography (EMG) for a variety of torso muscles and over a variety of activities. These are referred to throughout this book. This section addresses several issues and controversies about the functional interpretation of the thoracolumbar anatomy. Given the inability of the clinician and scientist to measure individual tissue forces in vivo, the only tenable option is to use sophisticated measurement techniques to collect biological signals from living subjects and integrate them with sophisticated modeling approaches to estimate tissue loads. Chapter 1 includes a brief description of the technique we used to assess the issues in this chapter (the virtual spine).

Table 4.4 Subject Averages of EMG Activation Normalized to Activity Observed During a Maximal Effort (100%)—Mean and (Standard Deviation in Parentheses)

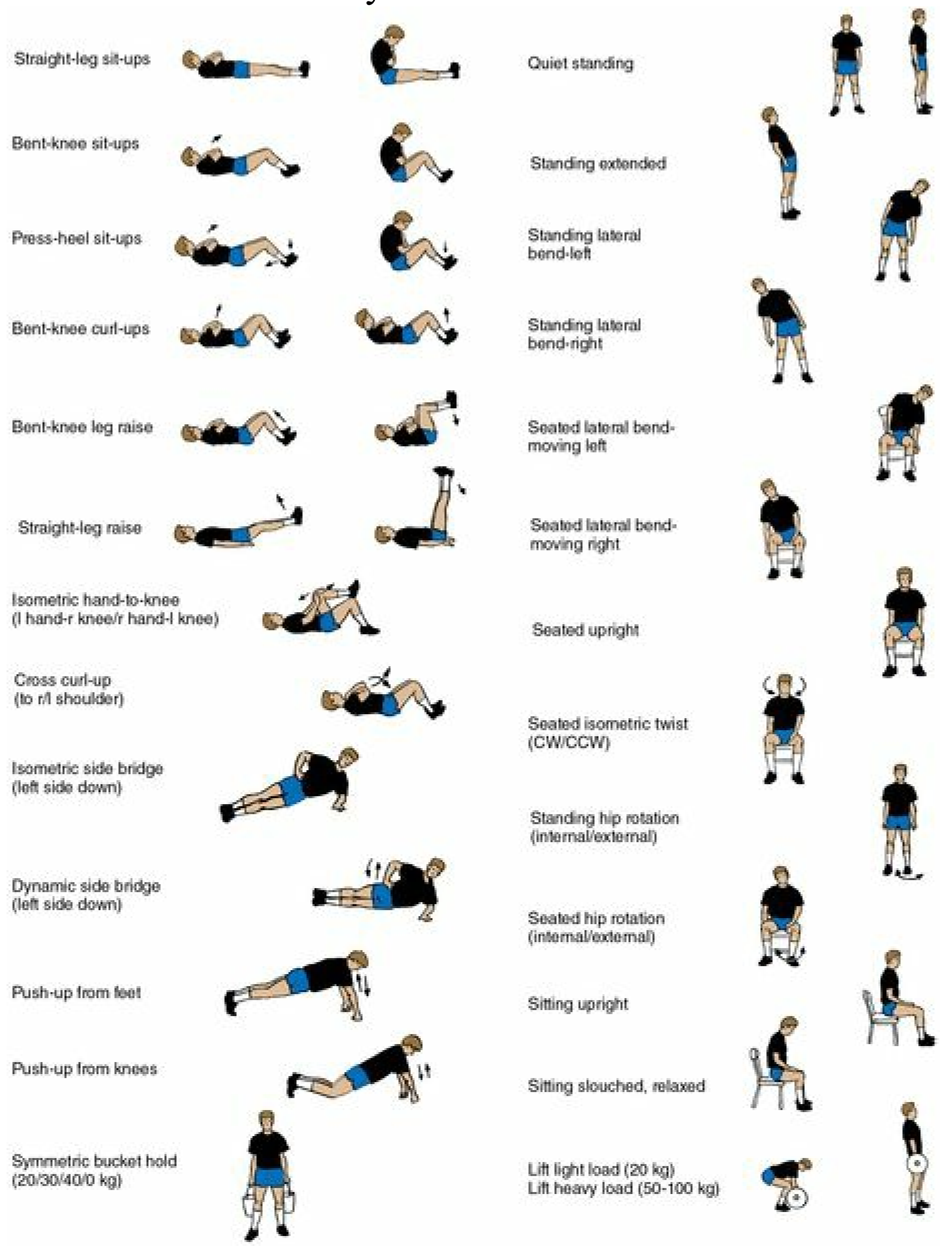
Task	Psoas\tab\1	Psoas\tab\2	EOi	IOi	TAi	RAs	RFs	ESs
Straight-leg sit-up	15 (±12)	24 (±7)	44 (±9)	15 (±15)	11 (±9)	48 (±18)	16 (±10)	4 (±3)
Bent-knee sit-up	17 (±10)	28 (±7)	43 (±12)	16 (±14)	10 (±7)	55 (±16)	14 (±7)	6 (±9)
Press-heel sit-up	28 (±23)	34 (±18)	51 (±14)	22 (±14)	20 (±13)	51 (±20)	15 (±12)	4 (±3)
Bent-knee curl-up	7 (±8)	10 (±14)	19 (±14)	14 (±10)	12 (±9)	62 (±22)	8 (±12)	6 (±10)
Bent-knee leg raise	24 (±15)	25 (±8)	22 (±7)	8 (±9)	7 (±6)	32 (±20)	8 (±5)	6 (±8)
Straight-leg raise	35 (±20)	33 (±8)	26 (±9)	9 (±8)	6 (±4)	37 (±24)	23 (±12)	7 (±11)
Isom. hand-to-knee (left hand → right knee, right hand → left knee)	16 (±16) 56 (±28)	16 (±8) 58 (±16)	68 (±14) 53 (±12)	30 (±28) 48 (±23)	28 (±19) 44 (±18)	69 (±18) 74 (±25)	8 (±7) 42 (±29)	6 (±4) 5 (±4)
Cross curl-up (right shoulder → across, left shoulder → across)	5 (±3) 5 (±3)	4 (±4) 5 (±5)	23 (±20) 24 (±17)	24 (±14) 21 (±16)	20 (±11) 15 (±13)	57 (±22) 58 (±24)	10 (±19) 12 (±24)	5 (±8) 5 (±8)
Isom. side support (left side down)	21 (±17)	12 (±8)	43 (±13)	36 (±29)	39 (±24)	22 (±13)	11 (±11)	24 (±15)
Dyn. side support (left side down)	26 (±18)	13 (±5)	44 (±16)	42 (±24)	44 (±33)	41 (±20)	9 (±7)	29 (±17)
Push-up from feet	24 (±19)	12 (±5)	29 (±12)	10 (±14)	9 (±9)	29 (±10)	10 (±7)	3 (±4)
Push-up from knees	14 (±11)	10 (±7)	19 (±7)	7 (±9)	8 (±8)	19 (±11)	5 (±3)	3 (±4)
Lift light load (20 kg, or 44 lb)	9 (±10)	3 (±4)	3 (±3)	6 (±7)	6 (±5)	14 (±21)	6 (±5)	37 (±13)
Lift heavy load (50-110 kg, or 110-243 lb)	16 (±18)	5 (±6)	5 (±4)	10 (±11)	10 (±9)	17 (±23)	6 (±5)	62 (±12)
Symmetrical bucket hold, 20 kg (44 lb) 40 kg (88 lb) 0 kg	2 (±4) 3 (±4) 3 (±5) 1 (±2)	1 (±1) 1 (±1) 1 (±1) 0 (±1)	7 (±4) 9 (±5) 10 (±6) 2 (±1)	5 (±3) 6 (±4) 8 (±6) 2 (±2)	5 (±1) 6 (±1) 6 (±2) 2 (±1)	10 (±7) 10 (±8) 10 (±8) 10 (±9)	3 (±3) 3 (±3) 3 (±3) 2 (±1)	3 (±6) 4 (±7) 3 (±2) 2 (±1)
Seated isom. twist CCW	30 (±20)	17 (±15)	18 (±8)	43 (±25)	49 (±35)	17 (±22)	7 (±4)	14 (±6)
Seated isom. twist CW	23 (±20)	11 (±8)	52 (±13)	15 (±11)	18 (±19)	13 (±10)	9 (±10)	13 (±8)
Standing hip internal rotation	21 (±18)	10 (±9)	18 (±12)	24 (±23)	33 (±20)	13 (±9)	9 (±7)	18 (±6)
Standing hip external rotation	27 (±20)	22 (±19)	17 (±13)	21 (±19)	31 (±17)	13 (±8)	19 (±11)	17 (±9)
Seated hip internal rotation	19 (±15)	21 (±18)	36 (±31)	30 (±30)	31 (±29)	18 (±8)	20 (±19)	12 (±8)
Seated hip external rotation	32 (±25)	25 (±20)	11 (±9)	15 (±17)	16 (±13)	15 (±9)	16 (±13)	8 (±8)
Seated upright	12 (±7)	7 (±5)	3 (±6)	3 (±3)	4 (±2)	17 (±9)	4 (±2)	5 (±8)
Seated slouched/relaxed	4 (±4)	3 (±3)	2 (±5)	2 (±2)	4 (±3)	17 (±11)	3 (±2)	5 (±8)
Quiet standing	2 (±1)	1 (±1)	3 (±4)	5 (±3)	4 (±2)	5 (±5)	3 (±3)	11 (±11)
Standing extended	3 (±2)	2 (±1)	12 (±9)	6 (±3)	5 (±3)	11 (±5)	4 (±3)	7 (±8)
Standing lateral bend, left	9 (±10)	1 (±2)	11 (±8)	18 (±14)	12 (±7)	13 (±7)	3 (±2)	11 (±13)
Standing lateral bend, right	6 (±5)	1 (±2)	19 (±18)	18 (±14)	25 (±20)	14 (±9)	3 (±1)	8 (±8)
Seated lateral bend, moving left	2 (±3)	1 (±1)	21 (±19)	7 (±7)	7 (±11)	13 (±8)	4 (±3)	6 (±7)
Seated lateral bend, moving right	18 (±12)	12 (±2)	15 (±26)	10 (±7)	12 (±7)	17 (±20)	5 (±4)	5 (±8)
Upright	14 (±9)	8 (±4)	6 (±4)	5 (±3)	5 (±5)	19 (±23)	5 (±3)	6 (±8)

Psoas channels, external oblique, internal oblique, and transverse abdominis are intramuscular electrodes, whereas rectus abdominis, rectus femoris, and erector spinae are surface electrodes.

EOi: external oblique (intramuscular); IOi: internal oblique (intramuscular); TAi: transverse abdominis (intramuscular); RAs: rectus abdominis; RFs: rectus femoris; ESs: erector spinae; ISOM: isometric; DYN: dynamic; CW: clockwise; CCW: counterclockwise

Reprinted, by permission, from D. Juker, S.M. McGill, S.M. Kropf, T. Steffen, 1998, "Quantitative intramuscular myoelectric activity of lumbar portions of psoas and the abdominal wall during a wide variety of tasks," *Medicine and Science in Sports and Exercise* 30(2): 301-310.

Figure 4.3 Schematic documenting tasks during which EMG signals were obtained. They are listed in table 4.4.



Reprinted, by permission, from D. Juker, S.M. McGill, S.M. Kropf, T. Steffen, 1998, "Quantitative intramuscular myoelectric activity of lumbar portions of psoas and the abdominal wall during a wide variety of tasks," Medicine and Science in Sports and Exercise 30(2): 301-310.

Loads on the Low Back During Functional Movements

Of course, no one can avoid performing countless functional movements every day. And if your patient's form is poor for any of those movements, he is exacerbating his low back problems simply by going about his business. Thus, you must be able to analyze how your patient moves in all sorts of ordinary situations so that you can identify and explain where his problems lie, and how to correct them.

Standing and Then Bending Forward

People with discogenic disorders often become symptomatic with prolonged static postures, standing being one of them. Is early onset of pain during standing a warning sign of impending symptoms? Nelson-Wong and Callaghan (2014) found that those without clinical signs who developed transient pain when standing developed clinical low back pain in the future. Several hypotheses could account for this.

Several studies over the years have shown the flexion–relaxation phenomenon, or the apparent myoelectric silence of the low back extensor muscles during a standing-to-full-flexion maneuver. The hypothesis has been that, as full flexion occurs, either the extensors shut down their neural drive by reflex or the passive tissues simply take the load as they strain under full flexion. A study by McGill and Kippers (1994) using the virtual spine approach described in chapter 1 quantified individual tissue forces, thus adding more insight into this task. As one bends forward, the spine flexes and the extensors undergo eccentric contraction. As full flexion is approached, the passive tissues rapidly take over moment production, relieving the muscles of this role and accounting for their myoelectric silence. Figure 4.4 shows the relative contribution of the muscles and the passive tissues (ligaments, disc, and gut) to the reaction moment throughout the movement. Table 4.5 documents the distribution of tissue forces and their moments and joint load consequence. Interestingly, the relaxation of the lumbar extensor muscles appeared to occur only in an electrical sense because they generated substantial force elastically during full spine flexion through stretching. Perhaps the term flexion–relaxation is inappropriate, particularly for those who may be attempting to minimize forces in the muscle in clinical settings. Furthermore, the shear loading is substantial (see chapter 3 for a discussion of the ligament and muscle directions and the loss of shear support in the extensors with full flexion) and would suggest caution for those with spondylolisthesis or other subtler shear instabilities. Clearly, straight-leg toe touches or knees-to-chest stretches would cause similar concern.

Figure 4.4 During standing to full forward flexion, then back to standing, the extensor muscles eccentrically contract but transfer their moment supporting role (MuscleTq) to the passive tissues at full flexion—the disc (DiscTq), the buckled gut (GutTq), and the ligaments (LigTq). Note that some force remains in the muscles with passive stretching.

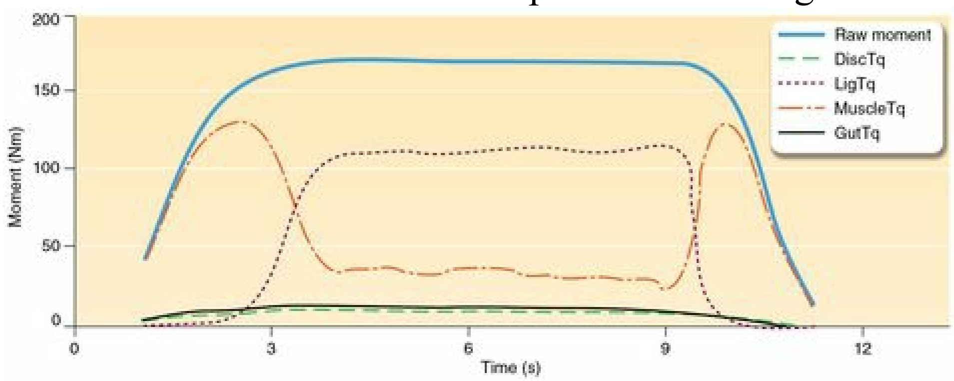


Table 4.5 Individual Muscle and Passive Tissue Forces and Moments During Full Flexion

	FORCE	MOMENT (NM)			COMPRESSION	SHEAR (N)	
	N	Flexion	Lateral	Twist	N	Anteroposterior	Lateral
MUSCLE							
R rectus abdominis	16	–2	1	1	15	5	–4
L rectus abdominis	16	–2	–1	–1	15	5	4
R external oblique 1	10	–1	1	1	8	7	–3
L external oblique 1	10	–1	–1	–1	8	7	3
R external oblique 2	7	–1	1	0	6	2	–3
L external oblique 2	7	–1	–1	0	6	2	3
R internal oblique 1	35	0	3	–2	21	–19	20
L internal oblique 1	35	0	–3	2	21	–19	–20
R internal oblique 2	29	–2	2	–3	8	–17	21
L internal oblique 2	29	–2	–2	3	8	–17	–21
R pars lumborum (L1)	21	2	1	0	21	6	2
L pars lumborum (L1)	21	2	–1	0	21	6	–2
R pars lumborum (L2)	27	2	1	0	26	8	2
L pars lumborum (L2)	27	2	–1	0	26	8	–2
R pars lumborum (L3)	31	1	1	0	29	–4	6
L pars lumborum (L3)	31	1	–1	0	29	–4	–6
R pars lumborum (L4)	32	1	1	0	30	–7	6
L pars lumborum (L4)	32	1	–1	0	30	–7	–6
R iliocostalis lumborum	58	5	4	1	57	14	–1
L iliocostalis lumborum	58	5	–4	–1	57	14	1
R longissimus thoracis	93	7	4	0	91	23	–6
L longissimus thoracis	93	7	–4	0	91	23	6
R quadratus lumborum	25	1	2	0	25	–1	1
L quadratus lumborum	21	1	–2	0	25	–1	–1
R latissimus dorsi (L5)	15	1	1	0	14	–1	–6
L latissimus dorsi (L5)	15	–1	1	0	14	–1	6
R multifidus 1	28	1	1	1	26	6	9
L multifidus 1	28	1	–1	–1	26	6	–9
R multifidus 2	28	1	1	0	28	6	0
L multifidus 2	28	1	–1	0	28	6	0
R psoas (L1)	25	1	2	0	24	9	6
L psoas (L1)	25	–1	–2	0	24	9	–6
R psoas (L2)	25	–1	2	0	24	9	6
L psoas (L2)	25	0	–2	0	24	9	–6
R psoas (L3)	25	0	1	0	24	9	7
L psoas (L3)	25	0	–1	0	24	9	–7
R psoas (L4)	25	0	1	1	24	9	8
L psoas (L4)	25	0	–1	–1	24	9	–8

	FORCE	MOMENT (NM)			COMPRESSION	SHEAR (N)	
	N	Flexion	Lateral	Twist	N	Anteroposterior	Lateral
MUSCLE							
Anterior longitudinal	0	0	0	0	0	0	—
Posterior longitudinal	86	2	0	0	261	44	—
Ligamentum flavum	21	1	0	0	21	2	—
R intertransverse	14	0	0	0	13	3	—
L intertransverse	14	0	0	0	13	3	—
R articular	74	2	1	1	65	40	—
L articular	74	2	–1	–1	65	40	—
R articular 2	103	3	2	2	84	–3	—
L articular 2	103	3	–2	–2	84	–3	—
Interspinous 1	301	18	0	0	273	142	—
Interspinous 2	345	14	0	0	233	268	—
Interspinous 3	298	10	0	0	194	238	—
Supraspinous	592	41	0	0	591	79	—
R lumbodorsal fascia	122	8	1	0	109	–1	—
L lumbodorsal fascia	122	8	–1	0	109	–1	—
PASSIVE							
Disc	—	9	0	0	—	—	—
Gut, etc.	—	11	0	0	—	—	—

The moment was 171 Nm (38 Nm by muscle, 113 Nm by ligaments, and 20 Nm by passive tissues, such as the disc, skin, and buckled viscera). The joint compression was 3,145 N, and shear was 1,026 N.

Loads on the Low Back During Lifting

During lifting, the muscle and ligament forces required to support the posture and facilitate movement impose mammoth loads on the spine. This is why lifting technique is so important to reduce low back moment demands and the risk of excessive loading. The following example demonstrates this concept.

A man is lifting 27 kg (59.5 lb) held in the hands using a squat lift style. This produces an extensor reaction moment in the low back of 450 Nm. The forces in the tissues that support this moment impose a compressive load on the lumbar spine of over 7,000 N. Table 4.6 details the contributions to the total extension moment and to the forces from the muscular components. These forces and their effects are predicted using the sophisticated modeling approach, which uses biological signals obtained directly from the subject (see chapter 1). It should be noted here that 7,000 N of compression begins to cause damage in very weak spines, although the tolerance of the lumbar spine in an average healthy young man probably approaches 12 to 15 kN (Adams and Dolan, 1995). In extreme cases, compressive loads on the spines of competitive weightlifters have safely exceeded 20 kN (Cholewicki, McGill, and Norman, 1991).

Table 4.6 Musculature Components for Moment Generation of 450 Nm During Peak Loading for a Squat Lift of 27 kg (59.5 lb)

Muscle	Force (N)	Moment (Nm)	Compression (N)	Shear (N)
Rectus abdominis	25	−2	24	5
External oblique 1	45	1	39	24
External oblique 2	43	−2	30	31
Internal oblique 1	14	1	14	−2
Internal oblique 2	23	−1	17	−16
Longissimus thoracis pars lumborum L4	862	35	744	−436
Longissimus thoracis pars lumborum L3	1,514	93	1,422	−518
Longissimus thoracis pars lumborum L2	1,342	121	1,342	0
Longissimus thoracis pars lumborum L1	1,302	110	1,302	0
Iliocostalis lumborum pars thoracis	369	31	369	0
Longissimus thoracis pars thoracis	295	25	295	0
Quadratus lumborum	393	16	386	74
Latissimus dorsi L5	112	6	79	−2
Multifidus 1	136	8	134	18
Multifidus 2	226	8	189	124
Psoas L1	26	0	23	12
Psoas L2	28	0	27	8
Psoas L3	28	1	27	6
Psoas L4	28	1	27	5

Negative moments correspond to flexion, whereas negative shear corresponds to L4 shearing posteriorly on L5.

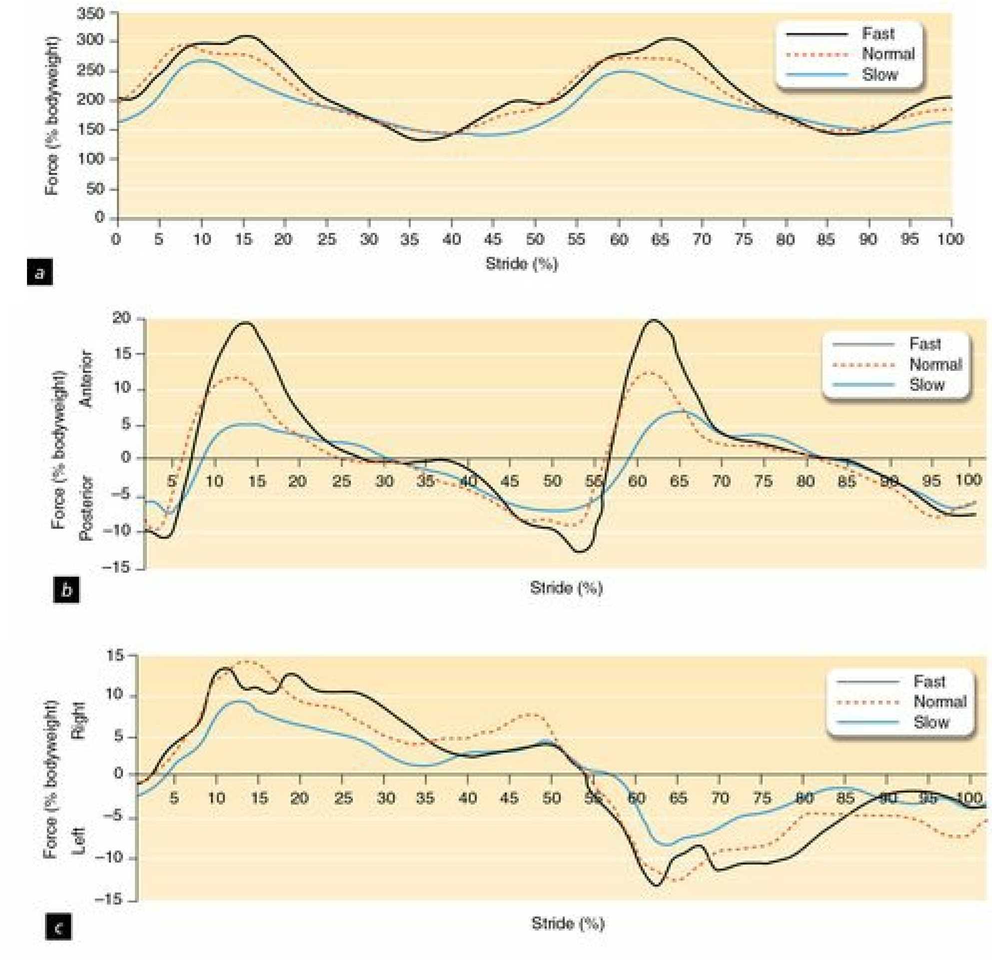
Understanding the individual muscle forces, their contributions to supporting the low back, and their components of compression and shear force that are imposed on the spine is very useful. In this particular example, the lifter avoided full spine flexion by flexing at the hip, minimizing ligament and other passive tissue tension and relegating the moment generation responsibility to the musculature. An example in which the spine is flexed is presented later in this chapter. As described in chapter 3, the pars thoracis extensors are very effective lumbar spine extensors, given their large moment arms. Also, because the lifter's upper body is flexed, large reaction shear forces on the spine are produced (the rib cage is trying to shear forward on the pelvis). These shear forces are supported to a very large degree by the pars lumborum extensor muscles. Furthermore, the abdominal muscles are activated but do not produce movement. Why are they active? These muscles are activated to stabilize the spinal column, although this mild abdominal activity imposes a compression penalty to the spine. A more robust explanation of stabilizing mechanics is presented in chapter 5.

The preceding example demonstrates how diffusely the forces are distributed and illustrates how proper clinical interpretation requires anatomically detailed free body diagrams that represent reality (such as those incorporated into the virtual spine model). I believe that oversimplified free body diagrams have resulted in clinicians' overlooking of the important mechanical compressive and especially shear components of muscular force. This has compromised the assessment of injury mechanisms and the formulation of optimal therapeutic exercise.

Loads on the Low Back During Walking

Thousands of low-level loading cycles are endured by the spine every day during walking. Although the small loads in the low back during walking suggest that the activity is safe and tolerable, clinicians have found that walking provides relief to some people but is painful to others. Recent work has suggested that walking speed affects spine mechanics and may account for these differences. During walking, the compressive loads on the lumbar spine of approximately 2.5 times body weight, together with the very modest shear forces, are well below any known in vitro failure load (see figure 4.5). Strolling reduces spine motion and produces almost static loading of tissues, however, whereas faster walking, with arms swinging, causes cyclic loading of tissues (Callaghan, Patla, and McGill, 1999) (see figure 4.6). This change in motion may begin to explain the relief experienced by some. Arm swinging while walking faster, with all other factors controlled, results in lower lumbar spine torques, muscle activity, and loading (see figure 4.7). In fact, we have observed up to 10% reduction in spine loads from arm swinging in some people. This may be because swinging the arms facilitates the efficient storage and recovery of elastic energy, reducing the need for concentric muscle contraction and the upper-body accelerations associated with each step. Interestingly, Kubo and colleagues (2006) reported higher torso stiffness with faster walking, which would further facilitate efficient energy recovery. Also interesting is the fact that fast walking has been shown to be a positive cofactor in the prevention of, and more successful recovery from, low back troubles (Nutter, 1988).

Figure 4.5 Loads on the lumbar spine (normalized to body weight) during three speeds of walking and with normal arm swing. The curves are normalized for one stride (beginning and ending with right heel contact). (a) L4-L5 compression, (b) anteroposterior shear forces in which positive indicates anterior shear of the superior vertebrae, (c) lateral shear force in which positive indicates right shear of the superior vertebrae.



Reprinted from Clinical Biomechanics, 14, J.P. Callaghan, A.E. Patla, and S.M. McGill, “Low back three-dimensional joint forces, kinematics and kinetics during walking,” 203-216, 1999, with permission from Elsevier Science.

Figure 4.6 Lumbar motion during three speeds of walking for one normal subject (beginning and ending with right heel contact): (a) lumbar flexion–extension in which extension is positive, (b) lateral bend in which positive indicates bending to the right, (c) axial twist in which positive indicates the upper body twisting to the right.

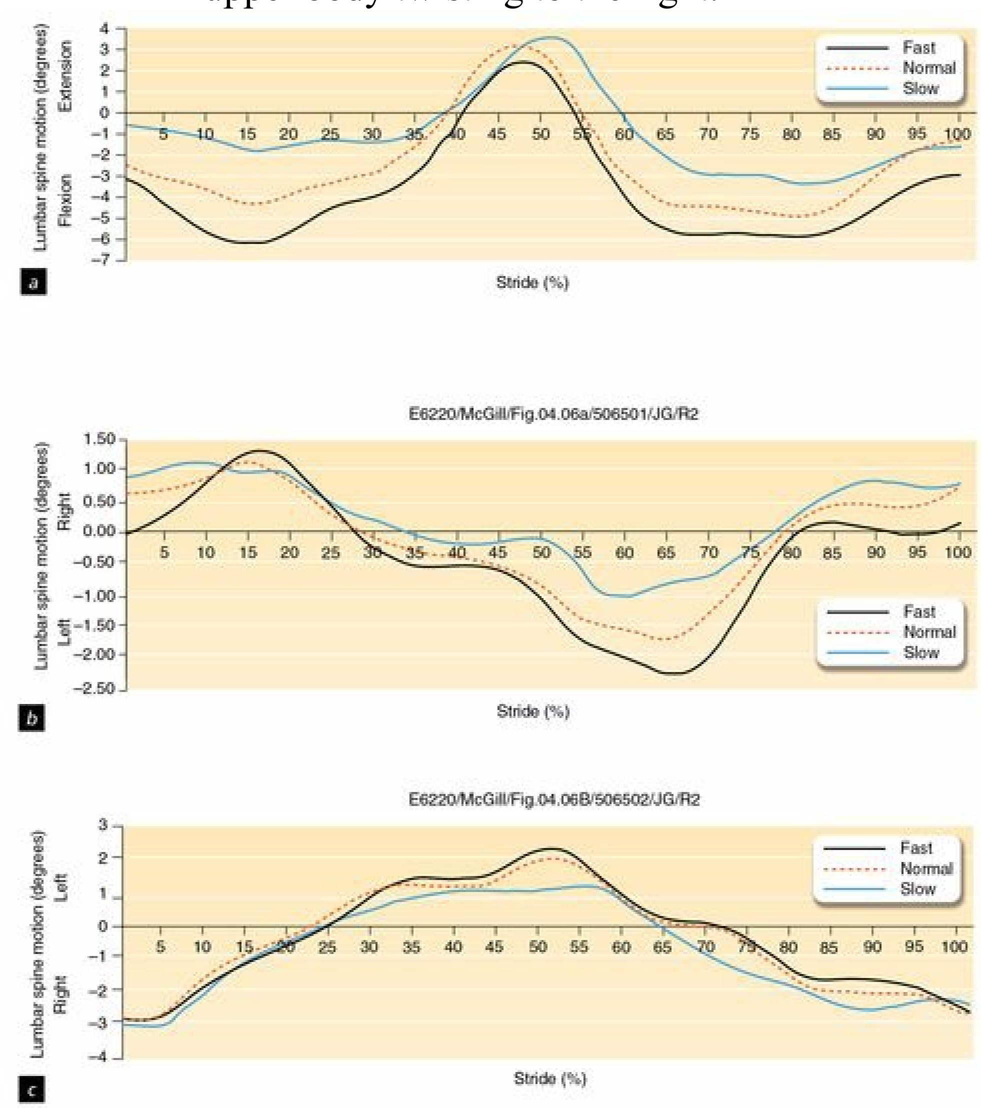
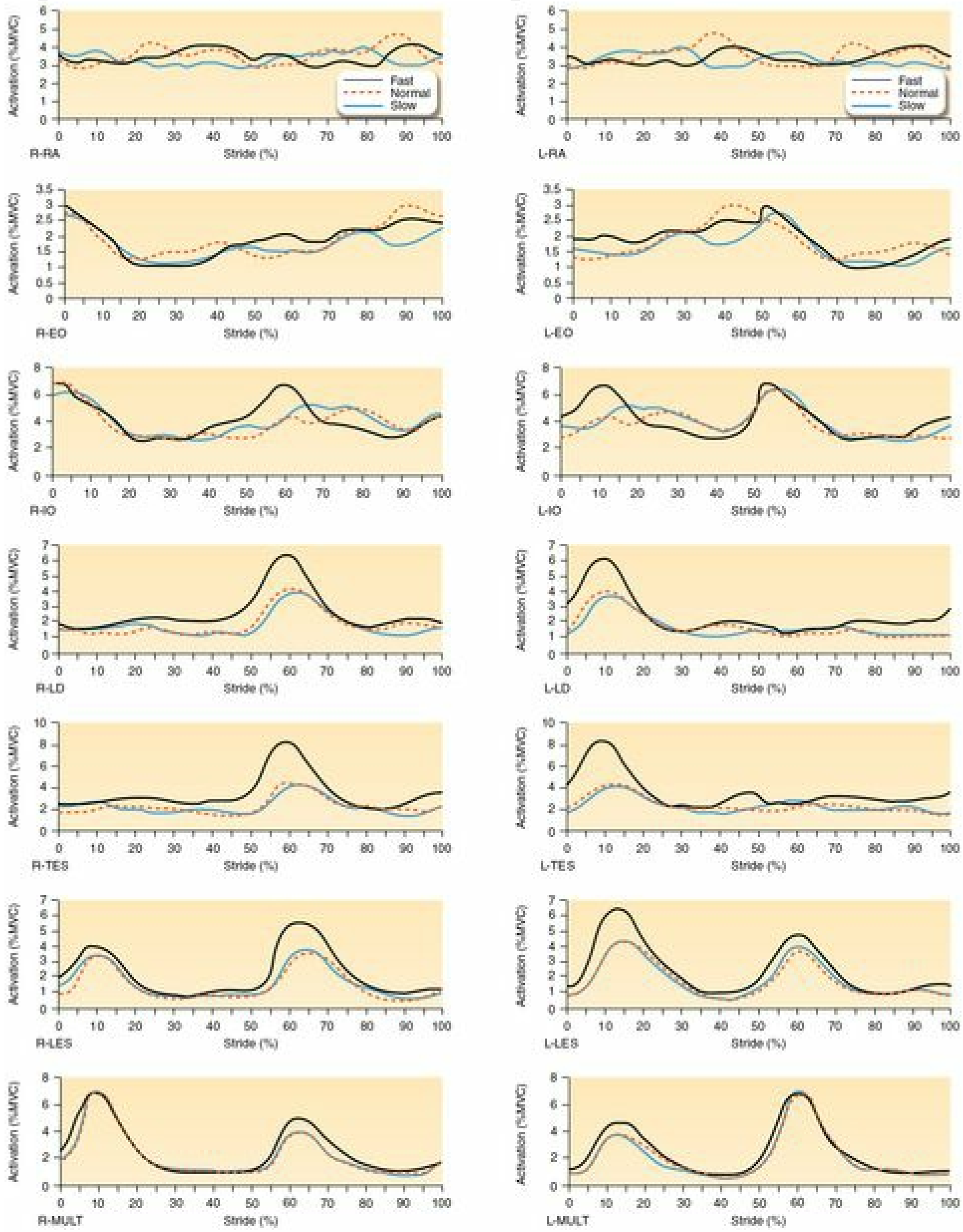


Figure 4.7 Activation profiles (EMG signals) from 14 torso muscles during three speeds of walking with normal arm swing (beginning and ending with right heel contact). The muscle pairs are (RA) rectus abdominis, (EO) external oblique, (IO) internal oblique, (LD) latissimus dorsi, (TES) thoracic erector spinae, (LES) lumbar erector spinae, and (MULT) multifidus.



Many patients are concerned about the implications of walking with shoes with elevated heels. Barton, Coyle, and Tinley (2009) documented the increased erector spinae muscle activity with heels elevated 20 cm (about 8 in.). Activity initially increased 20% immediately following heel strike, but after 2 days of accommodating to the extra heel height, it increased to 24%. Explanations included a pitched-forward torso and changes in the shock absorption of a more flexed subtalar joint.

Therapeutic Walking

Clinical Relevance

Fast Walking

Fast walking is generally therapeutic (Nutter, 1988). Several mechanisms appear to account for this: reciprocal muscle activation and tissue load sharing, gentle motion, and reduced spine loads with energy conservancy from arm swing. In contrast, these benefits do not occur during slow walking or mall strolling, which exacerbates symptoms in many because of the static loading that results. Finally, we have noted that people with pained backs exemplify the typical pained general flexor response in that they often tend to swing the arms primarily about the elbows. This should be corrected to arm swing about the shoulders to optimize the benefits of arm swing.

Aquatherapy

Many assume that walking in a pool (aquatherapy) unloads the joints because weight is supported with water buoyancy. With no movement, this is correct. But this is an inappropriate assumption because the moving limbs require much greater joint moments to propel them against the viscous resistance of the water. Kaneda and colleagues (2009) documented elevated leg and torso muscle activity during deep-water running and in-pool walking at three speeds compared with land walking—for example, erector spine activity during land walking of 11% of maximal voluntary contraction (MVC) versus 17% in the pool, and gluteus maximus activity on land of 9% versus 12% in the pool. There are other considerations and merits to aquatherapy, however. Water exercise assists in venous return of blood to the heart. The hydrostatic pressure of the water equals blood pressure at just over 1 m (3.3 ft) in depth so that the blood is returned to the heart "for free" with body parts submerged at these depths.

Loads on the Low Back During Sitting

Nachemson's classic work (1966), using intradiscal pressure measurements, documented the higher loads on the discs in various sitting postures compared to the standing posture. Normal sitting causes flexion in the lumbar spine, and people, if left alone, generally sit in a variety of flexed postures (Callaghan and McGill, 2001b). Sitting generally involves lower abdominal wall activity (particularly the deep abdominal muscles) compared to standing, and unsupported sitting generally involves higher extensor activity (see Callaghan, Patla, and McGill, 1999, for walking and Callaghan and McGill, 2001b, for sitting). Sitting slouched minimizes muscle activity, whereas sitting more upright requires higher activation of the psoas and the extensors (Juker, McGill, Kropf, and Steffen, 1998). Full flexion increases disc annulus stresses; although this posture has produced disc herniations in the lab (e.g., Wilder, Pope, and Frymoyer, 1988), substantial simultaneous load was needed. In fact, Kelsey (1975) discovered a specific link between prolonged sitting and the incidence of herniation. But association is not causation. We have not been able to initiate a herniation with sitting postures. Rather, the cumulative herniating trauma is caused in places such as the gym and as a result of poor lifting form throughout the day. Once the delaminations caused by exposure to flexion and load in the annulus have been initiated, then sitting adds to the trauma. Thus, those who cannot tolerate sitting for a period of time have already begun the cumulative trauma cascade. Paradoxically, those who have never stressed their spines usually find sitting comfortable.

Transitioning from a slouched to an upright posture is usually accomplished with two approaches (figure 4.8). Contrary to common perception, even when sitting upright, the L5/S1 joint is still quite flexed (60% of its neutral-to-full-flexion range, on average) (Dunk et al., 2009). When correcting this posture, some choose to lift the rib cage, hinging more at the thoracolumbar region with thoracic erector spinae activity. Not surprisingly, this strategy often results in midback pain and responds to corrective movement strategies such as the Lewit exercise (shown in chapter 10). Others instead focus more on flexing the hips, which aligns the spine above it. Comparing the two strategies, Casthanhero, Duarte, and McGill (2014) found roughly equivalent spine loading—thoracic erectors in the rib lift strategy and the psoas and hip flexors in the pelvic tilt strategy. A combination of the two was preferred by most people and resulted in the least spine loading. Thus, changing lumbar postures causes a migration of the loads from one tissue to another. Sitting upright relieves pain due to this load migration. Callaghan and McGill (2001b) suggested that no single, ideal sitting posture exists for any length of time; rather, a variable posture is recommended to minimize the risk of tissue overload. For example, Snijders, Hermens, and Kleinrensink (2006) suggested that a cross-legged sitting posture stabilizes the sacroiliac joints via passive tensioning of the iliolumbar ligament and piriformis muscle.

Figure 4.8 Sitting with (a) a flexion slouch is corrected by two common strategies. (b) Lifting the ribcage creates a hinge at the thoracolumar junction often creating pain or sensitivity at this level, and (c) flexing the hips to achieve a neutral posture. The least stress is achieved with a combination of the two.

Loads from Backpack Carriage

Backpacks come in various designs that affect low back loading. Generally, if rough terrain is anticipated, the load should be placed low in the pack to minimize the moment arm or distance to the low back. As the load is carried over rough ground, it accelerates and decelerates. The load placed closer to the low back reduces the torso forces needed to move the backpack load. On the other hand, if smooth ground is anticipated, carrying the load high in the pack, and over the fulcrum of the low back and hips, requires smaller torso muscle forces—and lower lumbar spine loads result.

Backpack Carrying

Clinical Relevance

Now for the curious situation in which backpacks can reduce spine loads

—and thus form one of our exercise-based therapy prescriptions. A person who is flexion intolerant, and also has posterior discogenic back pain exacerbated by prolonged sitting, generally has difficulty standing up. Upon standing, a forward torso angle (antalgic posture) remains. If this person can tolerate compression, we prescribe wearing a backpack with about 10 kg (22 lb) placed low in the backpack (about the level of the lumbar spine) and going for a walk over uneven ground. Wearing the backpack generates torso extensor moment, bringing the torso into an upright posture. This alleviates the spine extensors, which were previously contracted in the standing, but flexed, posture. Given their larger moment arm, this reduces the compressive load on the spine. In fact, recent observations by Rohlmann and colleagues (2014) confirmed that carrying 9 kg (20 lb) in a backpack did not increase the load on the lumbar spine of an older man, which was detected via an instrumented vertebral body replacement. The compression reduction from the muscles shutting down is larger than the extra compression from the additional load in the pack, resulting in a net reduction in total compression on the back. Walking over uneven ground provides gentle motion to the lumbar spine, which is therapeutic to the type of discogenic person we are describing here. Typically, the patient returns saying, “Thanks—that was amazing.” Thus, although some have blamed backpacks as a source of back troubles, they can actually be used therapeutically.

I recall a radio interview in which a spine expert was claiming that carrying backpacks over one shoulder was a serious problem for children. The interviewers phoned me for my on-air comment. I explained that, no doubt, some children will experience troubles, but there is also a training opportunity here. If the children were to switch shoulders frequently, this problematic task would become clever back training! The issue of the training load versus the dangerous load hinges on some subtle modulators. Perhaps the spine expert was right—that the children should carry their backpacks on both shoulders. But the technique of changing shoulders would have transformed a perceived danger into training for better health and performance.

Loads on the Low Back During Various Exercises

Because exercise is a crucial element of rehabilitation for low back problems, it is crucial that you understand the loads you are imposing on your patient's back when you prescribe an exercise. Otherwise, what was intended to be therapeutic may exacerbate her back troubles. Mastering the information in this section will increase your ability to tailor every exercise to each client's unique needs not only at the outset of treatment, but at every stage of progression toward better low back health.

Loads on the Low Back During Flexion Motion Exercises

Very few studies (only our own that we are aware of) have quantified the tissue loading on the low back tissues during various types of torso flexion exercises, although some have measured the EMG activity of selected muscles (e.g., Flint, 1965; Halpern and Bleck, 1979; Jette, Sidney, and Cicutti, 1984). This type of information alone can provide insight into relative muscle challenges, but it is restrictive for guiding exercise prescription decisions because the resultant spine load is unknown. The goal is to challenge muscle at appropriate levels but in a way that spares the spine. Too many exercises are prescribed that exceed the tolerance of back sufferers' compromised tissues. In fact, I believe that many commonly prescribed flexion exercises result in so much spine compression that these exercises, together with the flexion motion stresses, will ensure that the person remains a patient. For example, the traditional sit-up imposes approximately 3,300 N of compression on the spine (Axler and McGill, 1997). (Figure 4.9 illustrates psoas and abdominal muscle activation levels in a variety of flexion tasks. Figure 4.10 illustrates activation patterns with bent-knee sit-ups, and figure 4.11 illustrates activation patterns with bent-knee curl-ups.) Note that muscle activation levels are expressed in normalized units (%MVC). This means that the activity is expressed as a percentage of what would be observed during a maximal voluntary contraction (MVC), thus quantifying activity in a physiological and functional context. Further, the spine is very flexed during the period of this load (McGill, 1998). The National Institute for Occupational Safety and Health (NIOSH) (1981) in the United States has set the action limit for low back compression at 3,300 N; repetitive loading above this level is linked with higher injury rates in workers, yet this is imposed on the spine with each repetition of the sit-up!

Figure 4.9 Activation of the psoas and the abdominal muscles in a variety of flexion tasks from a group of highly trained subjects (five men and three women).

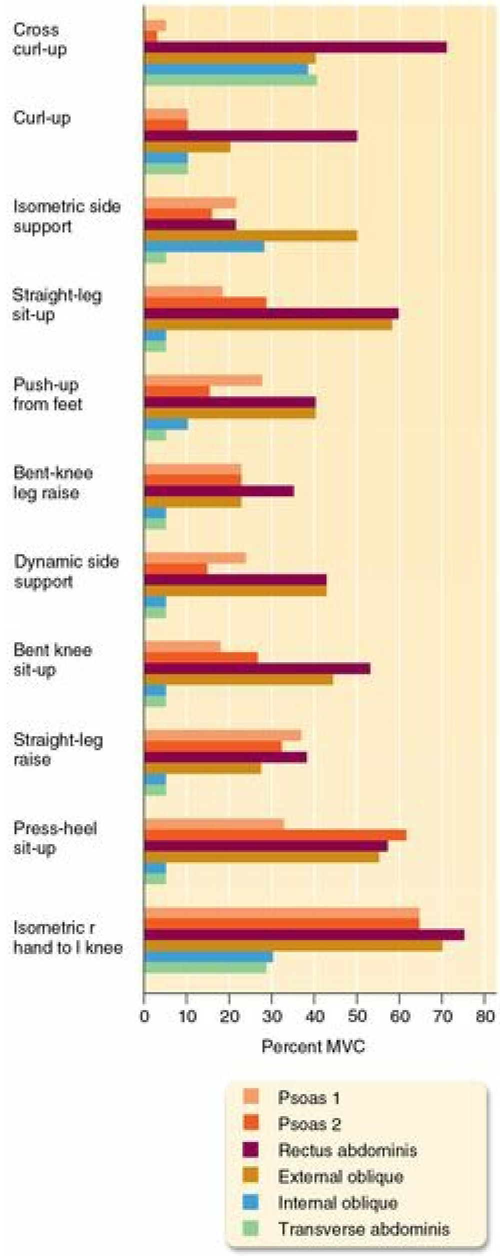


Figure 4.10 Activation-time histories of the same subjects as in figure 4.9 performing a bent-knee sit-up. Surface and indwelling electrodes are indicated.

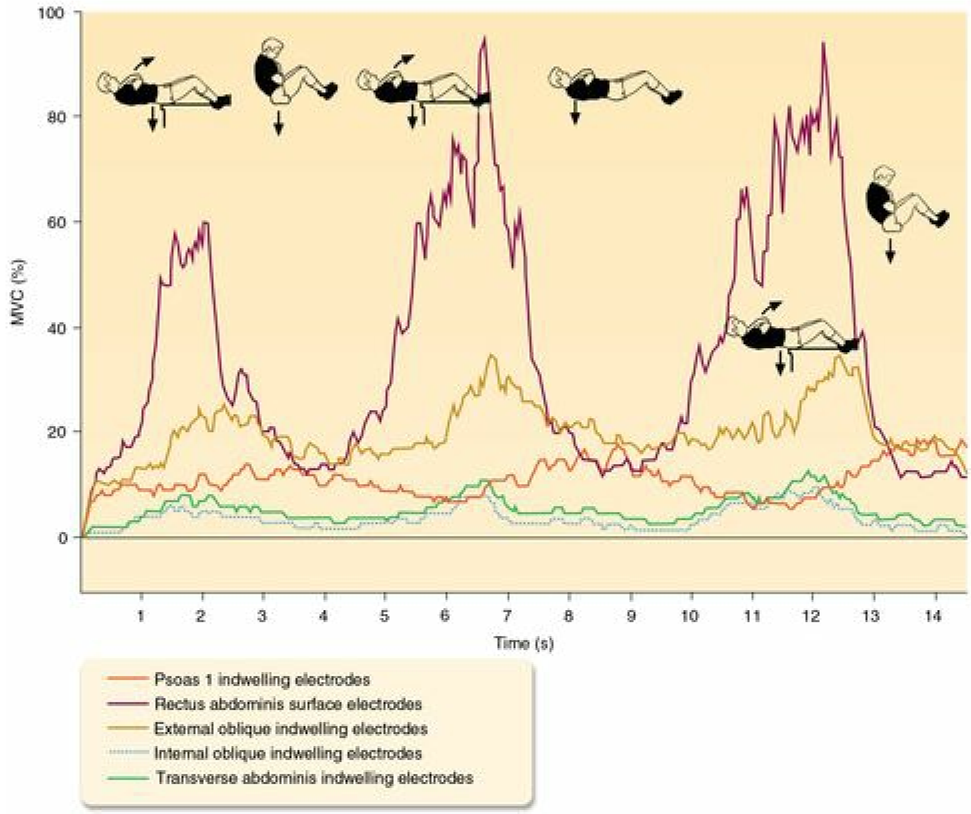
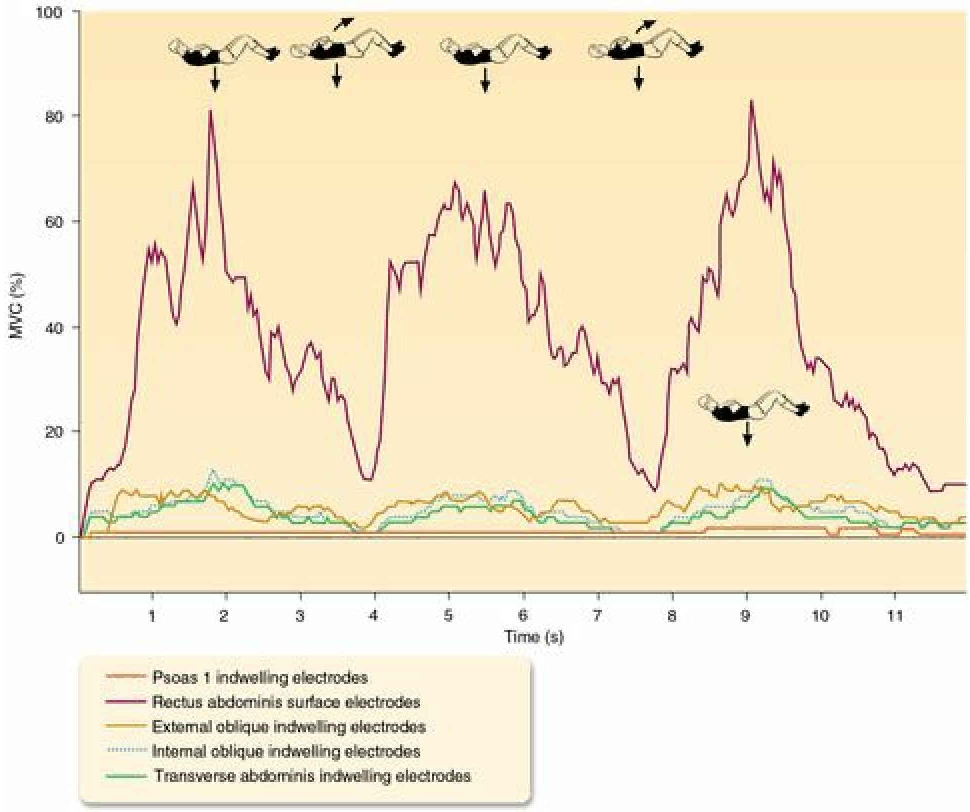


Figure 4.11 Activation-time histories for the same group performing bent-knee curl-ups.



Many recommend performing sit-ups with the knees bent, the theory being that the psoas is realigned to reduce compressive loading, or perhaps the psoas is shortened on the length–tension relationship so that the resulting forces are reduced. After examining both of these ideas, we found them to be untenable. We recruited a group of women who were small enough to fit into a magnetic resonance imaging (MRI) scanner. We placed each woman into the scanner and varied her knee and hip angles while she was supine (Santaguida and McGill, 1995). The psoas did not change its line of action, nor could it because it is attached to each vertebral body and transverse process (as the lumbar spine increases lordosis, the psoas follows this curve). The psoas does not change its role from a flexor to an extensor as a function of lordosis—this interpretation error occurred from models in which the psoas was represented as a straight-line puller. In fact, the psoas follows the lordotic curve as the lumbar spine flexes and extends. Further, it is true that the psoas is shortened with hip flexion, but its activation level is higher during bent-knee sit-ups (Juker, McGill, Kropf, and Steffen, 1998), not lower as has been previously thought. This is because the hip flexion torque must come from somewhere, and the shortened psoas must contract to higher levels of activation given its compromised length.

Given that the sit-up imposes such a large compression load on the spine, regardless of whether the leg is bent or straight, the issue is not which type of sit-up should be recommended. Rather, sit-ups should not be performed at all by most people. Far better ways exist to preserve the abdominal muscle challenge while imposing lower spine loads. Those who are training for health never need to perform a sit-up; those training for performance may get better results by judiciously incorporating them into their routine.

Part III of this book (Low Back Rehabilitation) offers specific preferred exercises and challenges to specific muscles, but a few flexion exercises are reviewed here. First, hanging with the arms from an overhead bar and flexing the hips to raise the legs is often thought to impose low spine loads because the body is hanging in tension—not compression. This is faulty logic. This hanging exercise generates well over 100 Nm of abdominal torque (Axler and McGill, 1997). This produces almost maximal abdominal activation, which in turn imposes compressive forces on the spine (see table 4.7). (Note: Hanging with bent knees resulted in higher average spine loads because the imprecise technique employed by many subjects creates substantial lumbar flexion. Further, few subjects were able to maintain form during the straight-leg hang. Good form is important but requires substantial athletic ability.) Similar activation levels can be achieved with the side bridge (shown later and discussed in detail) with lower spine loads.

This having been stated, those who are not interested in sparing their backs and are training with performance objectives may benefit from the high psoas challenge, together with rectus abdominis and oblique activity. Clearly, the curl-up primarily targets the rectus (both upper and lower), and generally, other exercises should be performed to train the obliques. Some have suggested a twisting curl-up to engage the obliques, but this results in a poor ratio of oblique muscle challenge to spine compression compared to the side bridge exercise (Axler and McGill, 1997)—making the side bridge a preferred exercise (table 4.7).

Table 4.7 Low Back Moment, Abdominal Muscle Activity, and Lumbar Compressive Load During Abdominal Exercises

	MUSCLE ACTIVATION			
	Moment (Nm)	Rectus abdominis (%MVC)*	External oblique	Compression (N)
Straight-leg sit-up	148	121	70	3,506
Bent-leg sit-up	154	103	70	3,350
Curl-up, feet anchored	**92**	**87**	**45**	**2,009**
Curl-up, feet free	**81**	**67**	**38**	**1,991**
Quarter sit-up	114	78	42	2,392
Straight-leg raise	**102**	**57**	**35**	**2,525**
Bent-leg raise	**82**	**35**	**24**	**1,767**
Cross-knee curl-up	112	89	67	2,964
Hanging, straight leg	**107**	**112**	**90**	**2,805**
Hanging, bent leg	**84**	**78**	**64**	**3,313**
Isometric side bridge	**72**	**48**	**50**	**2,585**

*MVC contractions were isometric. Activation values higher than 100% are often seen during dynamic exercise.

Practical Application: The Paradox of the Sit-Up as a Fitness Test

Schools and the military have long incorporated the sit-up as part of annual fitness testing. Curiously, one branch of the U.S. government (NIOSH) is warning against compressive loads exceeding 3,400 N because they document an increased risk of back injury. Yet another branch, the military services, is incorporating sit-ups as part of its annual fitness tests (recall that they exceed the NIOSH limit). Recognizing this paradox, coupled with the incidence of back injury, branches of the U.S. military have assigned task forces to come up with an alternative. Because measuring components of core fitness is worthwhile, the U.S. Navy, for example, is changing its standards to replace the sit-up with the plank exercise (Peterson, 2013). (They quote many of the norms we have published from various populations over the years: McGill et al., 2013a, 2013b, 2012, 2010).

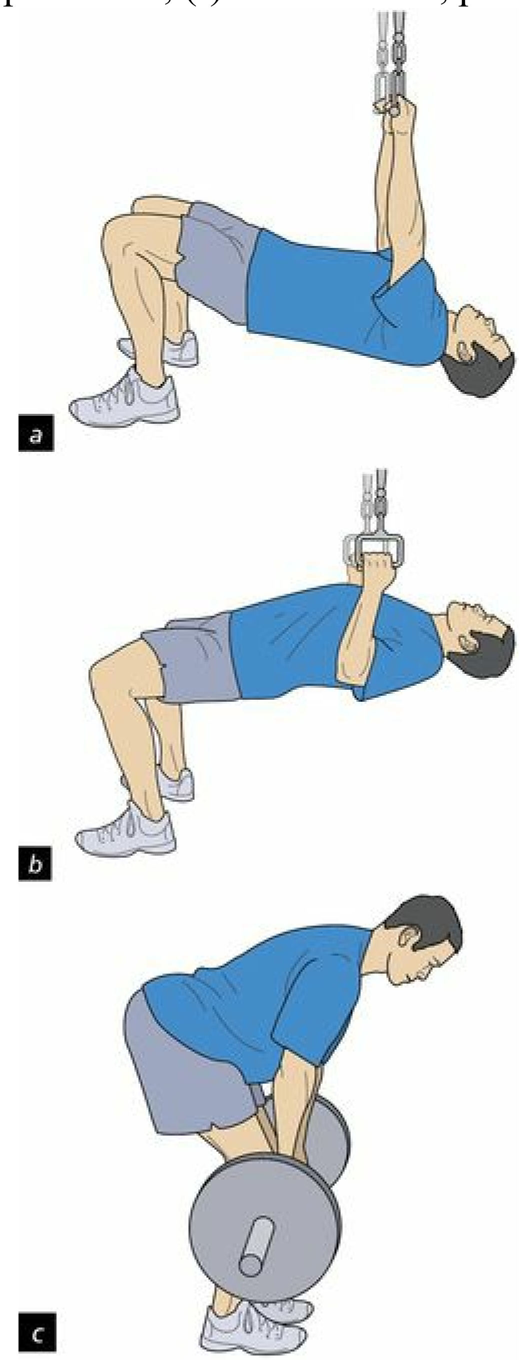
Loads on the Low Back During Pushing and Pulling

Our insights into the mechanics of pushing and pulling were obtained from our investigations originally intended to set safe occupational load limits (see Lett and McGill, 2006). Both novice (senior university students) and expert (firefighters) pushers/pullers were recruited. The expert firefighters experienced much lower spine forces. They cleverly used body mechanics with body lean to generate the driving force and to direct the hand forces through the low back, resulting in very small low back moments. The implication is that, once again, the motion and motor patterns that the firefighters elected to use resulted in their superior performance and safety. In this way the magnitude of the push/pull loads became much less important for the back. Technique was dominant!

The technique used in pushing and pulling is very dominant in determining the load on the back. The magnitude of the hand forces is almost irrelevant until extremely high pushing and pulling forces are required. Specifically, hand forces are skillfully directed through the low back. In this way they do not create a moment that would require extra muscle force to counterbalance. For athletes such as rugby players and strongman competitors pulling a bus, this same technique is used. Here the pushing or pulling force is directed through the lumbar spine. Skill is developed to enhance the foot grip, thereby ensuring minimal joint torque.

Pulling exercises generally engage the whole-body linkage with focused challenge to the posterior chain. For example, when standing and performing three variations of rowing exercises, the bent-over row focuses on the low back; the upright cable pull, on the upper back; and the standing one-arm cable pull, on the core stabilizers (Fenwick et al., 2009) (see figures 4.12 and 4.13). The use of suspension straps adds lability to pulling, which generally increases the amount of muscle activation (see figure 4.14 and table 4.8). Because tasks with suspension straps are more challenging, exercise choice would be guided by the person’s injury history, training goals, and current fitness level and tolerance.

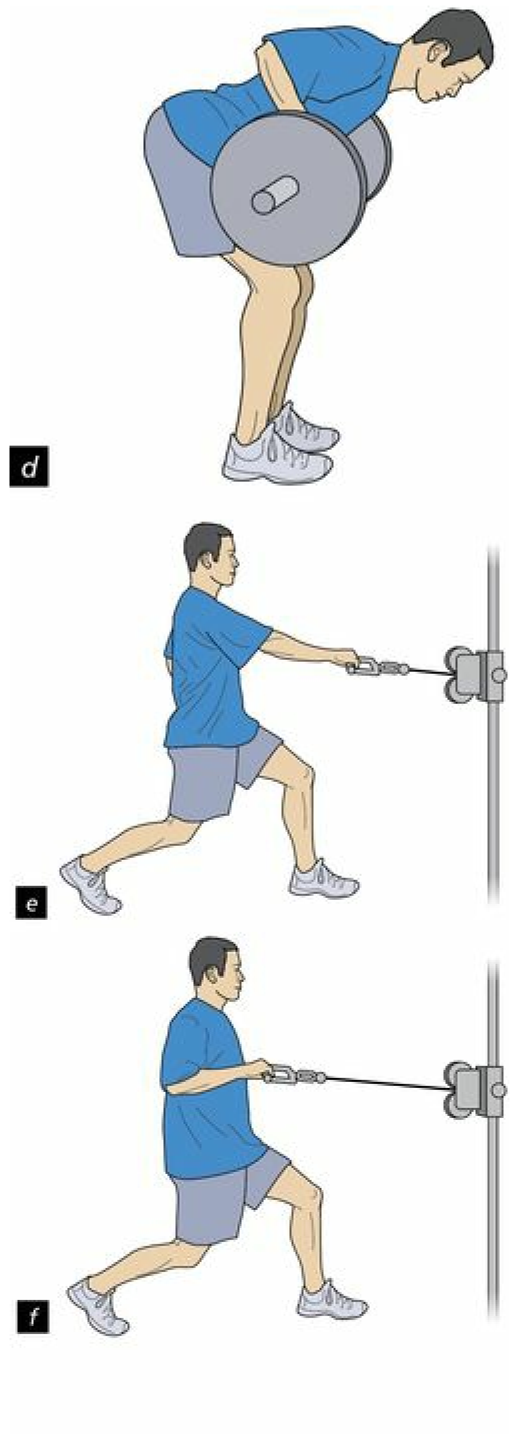
Figure 4.12 Three variations of pulling create different changes. The inverted row focuses on the upper back, the traditional row focuses on the low back, and the standing one-arm cable pull primarily challenges the antitwist mechanism of the core: (a) TRX pull up, position 1; (b) TRX pull up, position 2; (c) bentover row, position 1; (d) bentover row, position 2; (e) one-arm row, position 1; (f) one-arm row, position 2.



a

b

c



d e f

Figure 4.13 Average muscle activation of the figure 4.12 exercises.

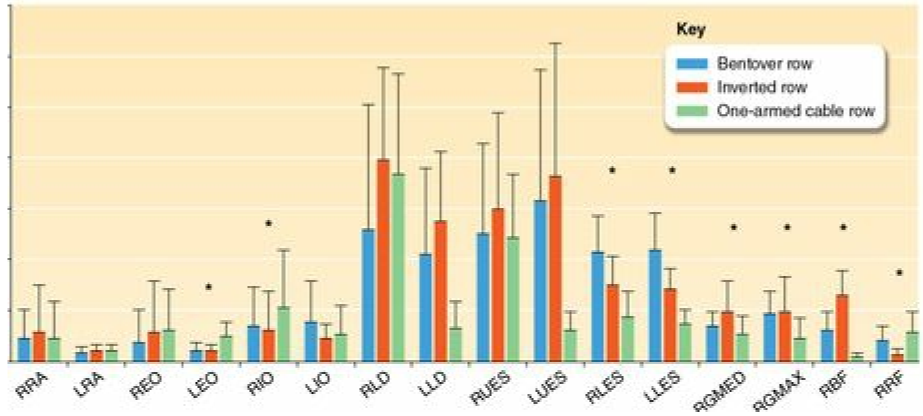


Figure 4.14 TRX suspension straps create a range of pulling and pushing exercise challenges simply by changing the angle of the body. Spine-conserving technique requires stiffening the core for pain control, centering the scapulae (i.e., the first part of the exercise is to depress the scapulae and activate the latissimus dorsi), and using a robust handgrip to radiate force up the arm linkage.

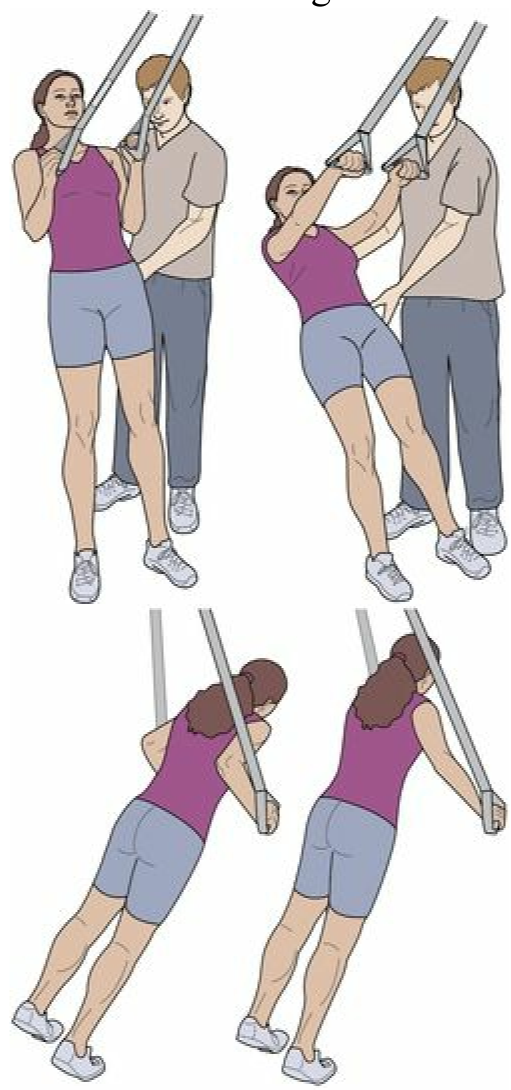


Table 4.8 Pulling Exercises Ranked on Compression Load Imposed on the Low Back

Exercise	Rank	Mean spine compression (N)	Standard deviation
Pull-up	1	2,852.3	1,339.1
Chin-up	2	2,679.8	1,327.0
TRX pull-up	3	2,626.6	1,181.5
Inverted row using body weight	4	2,294.3	767.0
TRX pull–angle 2	5	2,287.6	764.2
Stable shoulder retraction, coached	6	2,221.3	774.9
TRX pull—angle 1	7	2,041.8	715.1
Stable shoulder retraction, not coached	8	1,997.1	633.6
TRX shoulder retraction, coached	9	1,706.7	620.3

Rank of mean spine compression during the peak pulling phase of each pulling exercise. The TRX exercises use suspension straps adding lability to the exercise. For a complete description of each exercise, as well as a description of the muscle activation level, refer to McGill, Cambridge, and Andersen, 2014).

Loads on the Low Back During Pushing Exercises

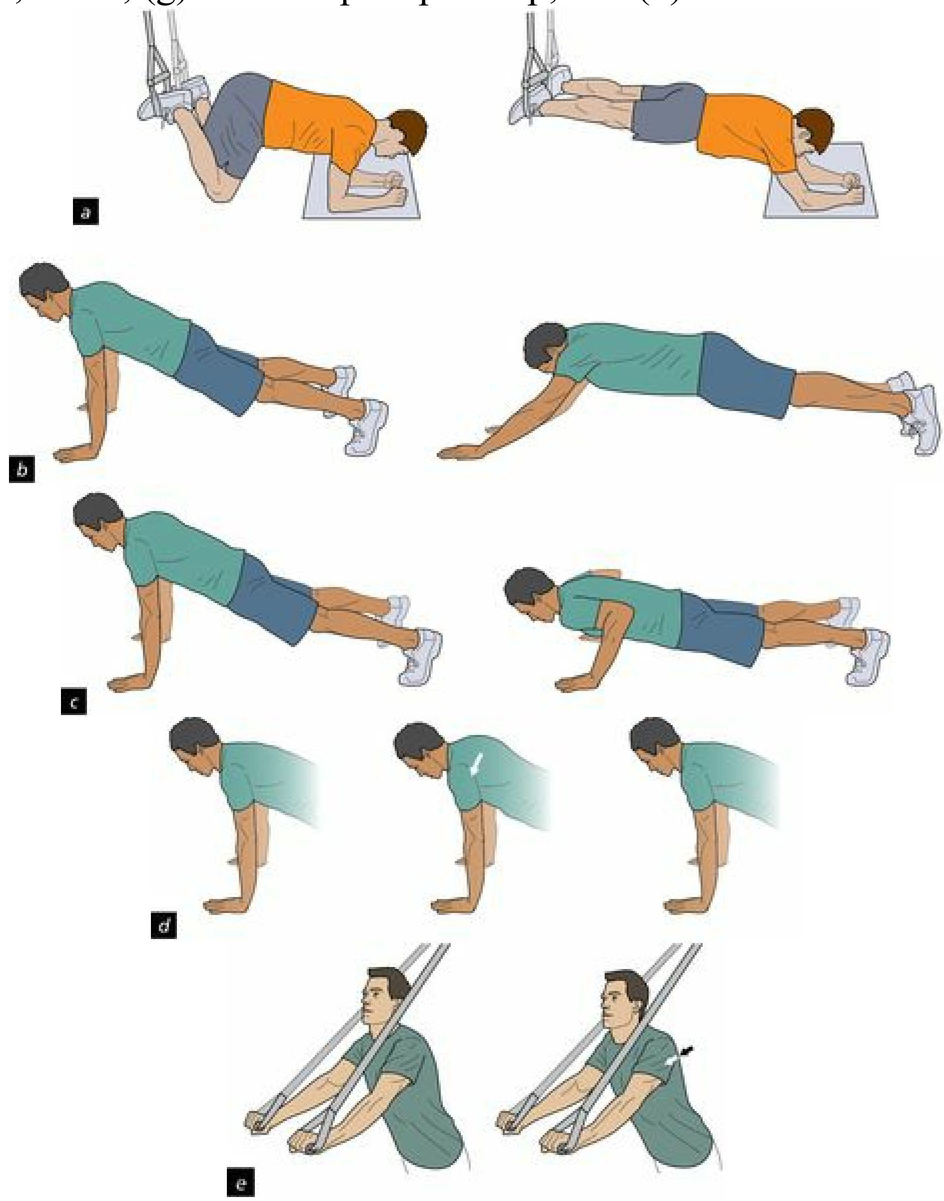
Many have recognized that many forms of spine stabilization exercise engage the abdominal hoop comprising rectus abdominis, the internal and external obliques, and transverse abdominis in an isometric contraction (McGill, 2006). For this reason, push-up exercises are sometimes used as torso training exercises. Clinical observation confirms that performing push-ups elicits back pain in some patients, yet others find push-ups relieving. In our quantification of push-up exercises, we examined styles ranging from traditional to placing the hands on labile surfaces (balls), staggered hand placement, one arm push-ups, and so on (Freeman et al., 2006). Although performing push-ups with the hands on a labile surface such as a ball has some effect on spine load, the one-arm and more ballistic forms of the exercise that require the hands to move are much more demanding on the spine (see table 4.9). Our recent work on performing pushes with suspension straps illustrates how muscle challenge and muscle demand can be influenced by this lability (McGill, Cannon, and Andersen, 2014a) and adjusted with the angle of the body (McGill et al., 2014b) (see figure 4.15 and table 4.10). On average, the hanging straight-leg raise created approximately 3,000 N of spine compression, whereas the body saw created less than 2,500 N. The hanging straight-leg raise created the highest challenge to the abdominal wall (>130% of MVC in rectus abdominis, 88% of MVC in external oblique). The body saw resulted in almost 140% of MVC activation of the serratus anterior. Those interested in challenging the abdominal obliques and steering the asymmetrical force from staggered hand placement through the torso will be interested in the quite modest increase in spine compression demand. Not surprisingly, the plyometric forms of the push-ups are much more muscularly demanding and therefore result in higher spine load. This may be a concern for those who are sensitive to spine compression during provocative diagnostic testing. Spinal loading during many forms of the push-up is substantial. There is little wonder that these exercises are problematic for some people with painful backs. On the other hand, they may be very appropriate as an abdominal plyometric exercise for high-performance athletes.

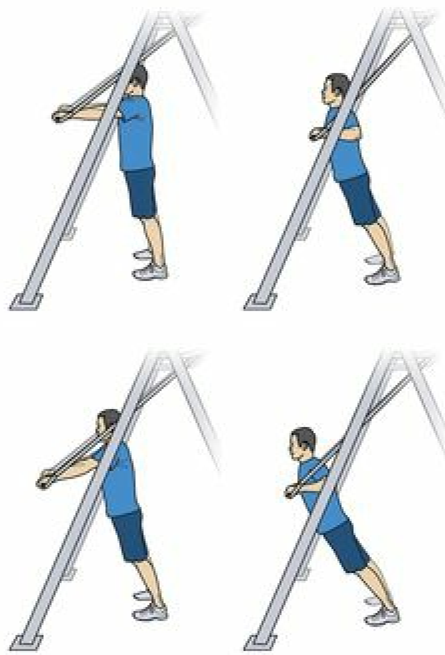
Table 4.9 Total Compression While Performing Various Styles of Push-Ups

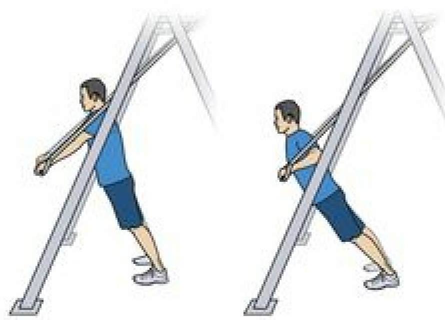
Exercise	Compression (N)
Standard	1,838
One-arm	5,848
Staggered hands (right forward)	2,532
Staggered hands (left forward)	2,337
Right hand on ball	2,315
Left hand on ball	2,295
Two hands on one ball	2,840
Hands on two balls (one on each)	2,829
Alternating	6,224
Clapping	4,699
Fast concentric	3,905
Slow eccentric	2,222

Adapted, by permission, from S. Freeman, A. Karpowicz, J. Gray, and S.M. McGill, 2006, "Quantifying muscle patterns and spine load during various forms of the pushup." *Medicine and Science in Sports and Exercise,* 38(3): 570-577.

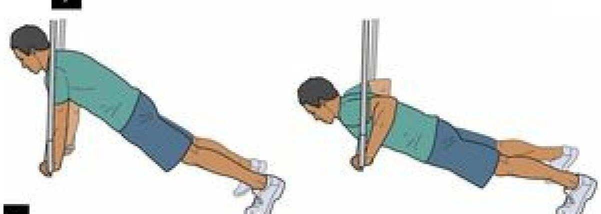
Figure 4.15 (a) Body saw; (b) walkout; (c) standard push-up; (d) stable shoulder protraction; (e) TRX shoulder protraction; (f) TRX pushes at angles 1, 2, and 3; (g) TRX scapula push-up; and (h) chest-arm flies.



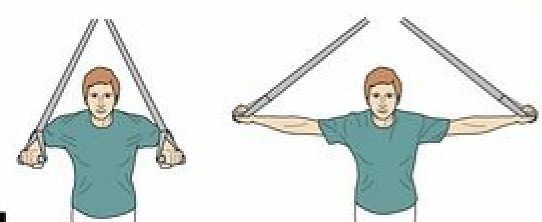




f



g



h

Table 4.10 Push Exercises Ranked on Compression Load Imposed on the Low Back

Exercise	Rank	Mean spine compression N)	Standard deviation
TRX push—angle 3	1	1,838.9	852.9
TRX push-up	2	1,653.4	759.8
TRX push—angle 2	3	1,631.1	712.0
Stable shoulder protraction, coached	4	1,582.7	717.3
TRX scapula push-up	5	1,581.0	505.6
TRX shoulder protraction, coached	6	1,528.2	707.7
TRX push—angle 1	7	1,484.9	638.3
TRX shoulder protraction, not coached	8	1,449.5	566.8
Standard push-up	9	1,399.2	716.7
Stable shoulder protraction, not coached	10	1,381.0	515.3

Rank of mean spine compression at the peak force phase of each push exercise. We also tested whether coaching shoulder centration patterns during pushing reduced joint load.

Loads on the Low Back During Extension Exercises

As with the flexion exercises discussed previously, plenty of EMG-based studies have explored extension exercises, but only one attempted to quantify the resulting tissue loads. Exercise prescriptions will not be successful if the spine loading is not constrained for those with bad backs. Using the virtual spine approach, Callaghan, Gunning, and McGill (1998) attempted to rank extension exercises on the muscle challenge, the resultant spine load, and their optimal ratio. The key to preserving a therapeutic muscle activation level while minimizing the spine load is to activate only one side of the spine musculature at a time. The muscle anatomy section in chapter 3 describes the functional separation of the thoracic and lumbar portions of the longissimus and iliocostalis. For the purposes of this discussion, we can think of the extensors in four sections—right and left thoracic portions and right and left lumbar portions. The common extension task of performing torso extension with the legs braced and the cantilevered upper body extending over the end of a bench or Roman chair (figure 4.16a) activates all four extensor groups and typically imposes over 4,000 N of compression on the spine. Even worse is the commonly prescribed back extension task in clinics, in which the patient lies prone and extends the legs and outstretched arms; this again activates all four extensor sections but imposes up to 6,000 N on a hyperextended spine (figure 4.16b). This is not justifiable for any patient!

Figure 4.16 Specific extension exercises quantified for muscle activation and the resultant spine load (shown in table 4.11): (a) trunk extension, (b) prone leg and trunk extension, (c) single-leg extension, and (d) single-leg and contralateral arm extension (bird dog).

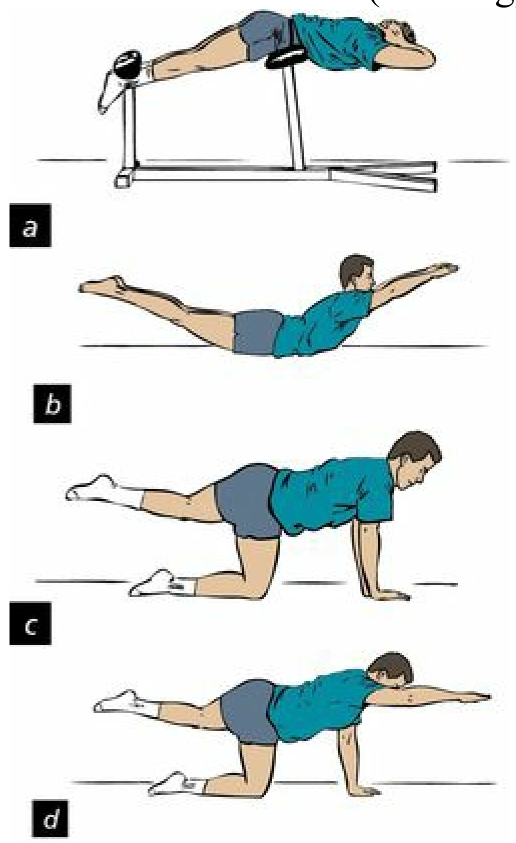


Table 4.11 Mean Activation Levels (±1 SD) of 14 EMG Channels for 13 Subjects Performing a Variety of Extensor-Dominant Exercises (Expressed as %MVC)

	EXTENSION						
Electromyographic channel*	Right leg	Left leg	Right leg and left arm	Left leg and right arm	Trunk and legs	Trunk	Calibration posture
Right RA X	3.3	2.7	4.0	3.5	4.7	3.1	1.4
SD	2.4	1.9	2.0	2.0	2.2	1.8	1.0
Right EO X	8.4	4.9	16.2	5.2	4.3	3.7	1.0
SD	4.9	1.5	6.0	2.3	2.5	1.7	0.6
Right IO X	12.0	8.2	15.6	12.0	12.1	12.7	1.9
SD	6.8	2.5	8.2	4.2	10.1	10.8	1.2
Right LD X	8.1	5.8	12.0	12.5	11.2	6.5	5.9
SD	5.4	3.5	9.6	6.2	4.3	4.0	8.5
Right TES X	5.7	13.7	11.5	46.8	66.1	45.4	21.0
SD	2.0	7.5	6.6	29.3	18.8	10.6	9.0
Right LES X	19.7	11.7	28.4	19.4	59.2	57.8	21.3
SD	9.1	4.9	10.2	11.0	11.7	8.5	4.6
Right MF X	21.9	10.8	31.5	16.1	51.9	47.5	16.4
SD	6.3	6.0	8.2	12.0	14.7	12.3	5.6
Left RA X	4.3	3.6	4.4	4.2	6.5	3.7	2.2
SD	3.4	3.6	3.8	3.9	3.4	2.4	2.1
Left EO X	5.4	9.0	6.2	15.9	6.3	5.2	1.8
SD	2.0	3.8	2.5	6.6	3.2	5.2	1.0
Left IO X	16.0	11.3	22.6	15.2	11.0	12.5	1.6
SD	8.6	7.0	9.2	6.7	5.9	6.1	1.3
Left LD X	4.5	5.0	10.7	6.2	9.2	5.1	6.1
SD	4.3	4.5	18.2	4.4	5.1	4.1	8.5
Left TES X	15.0	4.5	42.9	10.5	63.6	41.6	21.2
SD	7.5	2.0	20.5	5.9	22.7	10.0	9.8
Left LES X	11.3	16.8	19.5	25.5	56.8	57.0	23.3
SD	6.6	4.5	7.4	7.3	14.5	14.7	8.4
Left MF X	11.9	22.3	16.6	33.8	57.3	53.3	18.7
SD	7.0	6.1	7.2	6.7	11.4	12.0	4.3

*Electromyographic channel: RA = rectus abdominis muscle; EO = external oblique muscle ; IO = internal oblique muscle ; LD = latissimus dorsi muscle; TES = thoracic erector spinae muscle; LES = lumbar erector spinae muscle; MF = multifidus muscle. Calibration posture: standing, trunk flexed to 60°, neutral lumbar posture, 10 kg (22 lb) held in hands with arms hanging straight down.

Several variations of exercise technique can preserve activation in portions of the extensors and greatly spare the spine of high load. For example, kneeling on all fours and extending one leg at the hip generally activates one side of the lumbar extensors to over 20% of maximum and imposes only 2,000 N of compression (figure 4.16c). Performing the bird dog, in which the opposite arm is extended at the shoulder while the leg is raised (figure 4.16d), adds activity to one side of the thoracic extensors (generally around 30 to 40% of maximum) and contains the spine load to about 3,000 N. In addition, the special techniques shown for this exercise in chapter 10 attempt to enhance the motor control system to groove stabilizing patterns. For data describing these exercises, see table 4.11.

Loads on the Low Back During Gait: Walking, Carrying, and Using

Elliptical Trainers

Walking has already been discussed and forms a basic pattern for the ambulation tasks. When carrying a handheld load while walking, what is the most spine-friendly tactic? Carrying loads in one hand causes more spine load than doubling the load by carrying the same load in each hand. For example, when carrying 30 kg (66 lb) in one hand, the low back compression exceeded 2,800 N; however, splitting the load between hands reduced low back compression to 1,570 N (a reduction of 44%). Doubling the total load by carrying 30 kg in each hand (total of 60 kg) actually produced lower spine compression than carrying 30 kg in one hand. This is due to the additional muscle activity required to support the lateral bending moment with uneven frontal plane loading (McGill, Marshall, and Andersen, 2013). The message for those with back pain is to split the load between the hands (figure 4.17).

Other carrying exercises such as the kettlebell carry, known as bottoms-up, change the stability requirements which the motor control scheme addresses by using more bilateral torso muscle coactivation (figure 4.18). Such activities are modest and conservative in terms of influencing spine load (McGill and Marshall, 2012).

Figure 4.17 Carrying loads in one hand or splitting the load between two hands greatly unloads the spine. This effect is greater with heavier loads as they dominate the effect of body mass.

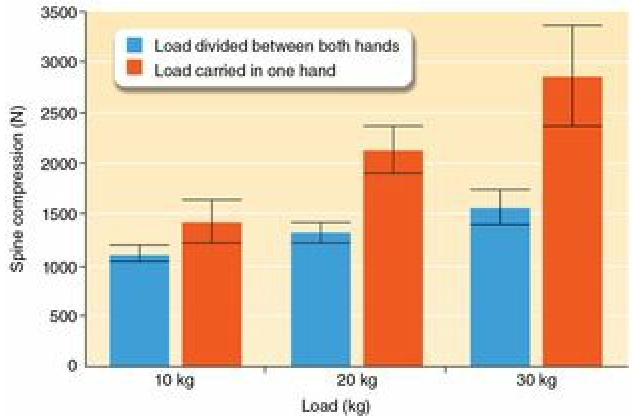


Figure 4.18 The kettlebell bottoms-up carry enhances the control requirements while restricting the amount of load that can be carried and the torso muscle activity.

Walking and exercising on an elliptical trainer require quite different body linkage mechanics. Our study was motivated by the observation that some people reported back pain relief with elliptical trainer exercise, whereas others reported that their pain was exacerbated. Those with tight hips and normal hip range of motion were recruited. First, the elliptical trainer requires more hip motion than normal walking (Moreside and McGill, 2012). On the other hand, elliptical trainers result in much higher gluteal muscle activation, which may be a training target for some people. Those with tight hips endured more spine motion and a more forward leaning torso, increasing the spine load. Thus, we recommend hip screening and pain provocation tests, as well as a pain history, before suggesting elliptical training.

Loads on the Low Back During Standing Exercises

Many exercises, and combinations of exercises, are performed in a standing posture. Given the variety of loads and technique, I refer you to our original studies:

- Standing torso exercises: McGill, S.M., Karpowicz, A., and Fenwick, C. (2009b) Exercises for the torso performed in a standing posture: Motion and motor patterns. Journal of Strength and Conditioning Research 23 (2): 455-464.
- Standing body blade exercises: Moreside, J.M., Vera-Garcia, F.J., and McGill, S.M. (2007) Trunk muscle activation patterns, lumbar compressive forces and spine stability when using the body blade. Physical Therapy, 87 (2): 153-163.
- Ski simulators: Banerjee, P., Brown S., Howarth, S., and McGill, S.M. (2009) Torso and hip muscle activity and resulting spine load and stability while using the Profitter 3-D Cross Trainer. Journal of Applied Biomechanics, 25: 73-84.
- Hula hooping: McGill, S.M., Cannon, J., and Andersen, J. (Submitted) Physiological and biomechanical mechanisms in hula hooping: Caloric expenditure and spine loads.

Loads on the Low Back During Coitus

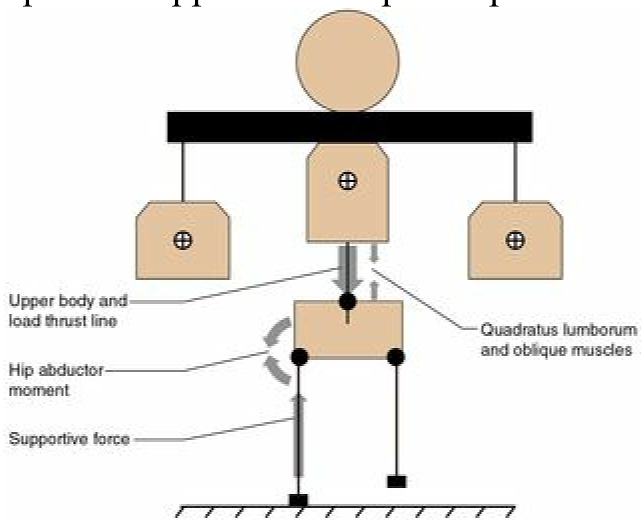
Every primary care clinician reports being often asked by couples with back pain about less-painful or pain-free sexual positions. Until now, clinicians had no scientific foundation to guide them. We appear to have been the first group to document spine and hip motion, and muscle activation levels, during coitus. Given the variety of positions and intensity of effort, we simply direct you to our recent medical publications to appreciate the spectrum of loads: Sidorkewicz and McGill (2014a and 2014b) and McGill (2015).

Loads on the Low Back During More Demanding Forms of Exercise

Kettlebell swings have gained popularity; some athletes claim that the exercise was vital in their rehabilitation following disc injury. Yet other very athletic people claim that swings are the one exercise that instigates pain. This motivated our investigation, in which we found that kettlebell swings create a desirable hip-hinge squat pattern characterized by rapid muscle activation and relaxation cycles of substantial magnitude (50% of MVC for the low back extensors and 80% of MVC for the gluteal muscles with a 16 kg [35 lb] kettlebell), which results in about 3,200 N of low back compression. Some unique loading patterns discovered during the kettlebell swing included the posterior shear of the L4 vertebra on L5, which is opposite in polarity to a traditional lift. Thus, the quantitative analysis provided an insight into why many people credit kettlebell swings with restoring and enhancing back health and function, although a few find that they irritate tissues. It appears that those with sufficient shear stability of the lumbar joints may do well with swings, whereas those deficit in shear stability should perhaps perform the shear instability tests described in this book.

In one study we measured competitive strongman athletes as they performed events such as the farmer's walk, super yoke (figure 4.19), Atlas stone lift, suitcase carry, keg walk, tire flip, and log lift (McGill, McDermott, and Fenwick, 2009). The results document the unique demands of these whole-body events and, in particular, the demands on the back and torso. For example, the very large moments required at the stance-leg hip for abduction when performing a yoke walk exceed the strength capability of the hip. Here, muscles such as quadratus lumborum made up for the strength deficit by generating frontal plane torque to support the torso and pelvis. In this way, the stiffened torso acts as a source of strength to allow joints with insufficient strength to be buttressed, resulting in successful performance. We are often asked why we study world-class performance. Here we see a wonderful example of obtaining insight that explains many lower-level rehabilitation challenges. Children with a paralyzed quadratus lumborum muscle have extreme difficulty walking. Most gait biomechanists have concluded that frontal plane support of the pelvis is supplied by the hip abductor muscles. The insight obtained here demonstrated that the opposite core muscles must function with the gluteal muscle to create walking proficiency.

Figure 4.19 Measuring world-class strongmen revealed how they carried loads that exceeded their strength. We measured a hip abduction strength of approximately 500 Nm, yet 750 Nm of strength was required on the support hip to complete the super yoke carry. The missing strength appears to have been supplied by the contralateral quadratus lumbarum and abdominal obliques to support a level pelvic platform.



Future Trends for Linking Back Load and Exercise for People With Osteoporosis

Matching exercise with the specific load-bearing tolerance of a person is critical for fragile osteoporotic spines and otherwise injured backs. However, metrics such as vertebral bone density (measured with DEXA, for example) have not proven to be very accurate in representing compressive strength. Advances by Professor Keaveney's group (e.g., Wang, Videman, and Battié, 2012) have resulted in a CT-based bone quality index that incorporates density with the connectivity of the trabecular bone—resulting in a better prediction of strength. Our work has identified spine loads with various activities. Here we put the two approaches together to create a rough guide for selecting activity based on bone quality and load-bearing ability (see table 4.12). For example, a person with a bone quality index of 0.7 can walk safely without risk of further fracture. Having a quality index of 0.8 would allow the person to add one-legged quadruped exercises and retain a margin of safety.

Table 4.12 Ranking of Activities Based on Estimates of Spine Load and Keaveney's Bone Quality Index

Minimum	aBMD (g/cm^3)	Activity
0.7		Walking (swing arms)
0.8	Add	Curl-up, one-leg quadruped
0.9		
1.0	Add	Push-up
1.1	Add	Sit-up, kettlebell swing with <16 kg (35 lb)
1.2	Add	Superman, suspended push-up
1.3	Add	Rowing (in boat)
1.4		
1.6	Add	Golf, one-arm push-up, box lift 50 lb (22.7 kg) (knee to waist)

This index is linked with our many studies quantifying joint load to create this ranked listing of activities that, on average, should be tolerable. We have incorporated a safety factor of a spine load that is twice the tolerance load.

For those with quantifiable osteoporosis, we can use their bone quality score to match tolerable exercise and activity. Keaveney's work can guide this exercise selection while also building in a predetermined margin of safety. We chose half of the tolerable load as a reasonable compromise between promoting a variety of activities and safety.

Dubious Lifting Mechanisms

In the 1950s and 1960s, spine biomechanists faced a paradox. The simple spine models of the day predicted that the spine would be crushed to the point of injury during certain lifting tasks, yet when people performed those tasks, they walked away uninjured. This motivated several research groups to theorize about mechanisms that could unload compressive stresses from the spine. Researchers proposed three major mechanisms: the intra-abdominal pressure mechanism, the lumbodorsal fascia mechanism, and the hydraulic amplifier. Although none has survived scrutiny, clinical belief in these mechanisms remain. Nonetheless, some components of these mechanisms provided insight for subsequent studies and led to the understanding that we have today. For this reason they are reviewed briefly here.

Intra-Abdominal Pressure

Does intra-abdominal pressure (IAP) play an important role in the support of the lumbar spine, especially during strenuous lifting, as has been claimed for many years? Anatomical accuracy in representation of the involved tissues has been influential in this debate. Further, research on lifting mechanics has formed a cornerstone for the prescription of abdominal belts for industrial workers and has motivated the prescription of abdominal strengthening programs. Many researchers have advocated the use of IAP as a mechanism to directly reduce lumbar spine compression (Bearn, 1961; Thomson, 1988). However, some researchers believe that the role of IAP in reducing spinal loads has been overemphasized (Grew, 1980; Krag et al., 1986).

Anatomical Consistency in Examining the Role of Intra-Abdominal Pressure

Morris, Lucas, and Bresler (1961) first operationalized the mechanics of the original proposal into a model and described it as follows. Pressurizing the abdomen by closing the glottis and bearing down during an exertion exerts hydraulic force down on the pelvic floor and up on the diaphragm, creating a tensile effect over the lumbar spine or at least alleviating some of the compression. Missing in the early calculations of this hydraulic potential was the full acknowledgment of the necessary abdominal activity (contracting the abdominal wall imposes extra compression on the spine). But an evaluation of the trade-off between the extra abdominal muscle compression and the hydraulic relief depended on the geometrical assumptions made. Some of these assumptions appear to be outside of biological reality. In fact, experimental evidence suggests that somehow, in the process of building up IAP, the net compressive load on the spine is increased! Krag and coworkers (1986) observed increased low back EMG activity with increased IAP during voluntary Valsalva maneuvers. Nachemson and Morris (1964) and Nachemson, Andersson, and Schultz (1986) showed an increase in intradiscal pressure during a Valsalva maneuver, indicating a net increase in spine compression with an increase in IAP, presumably a result of abdominal wall musculature activity.

In our own investigation, in which we used our virtual spine model, we noted that net spine compression was increased from the necessary concomitant abdominal activity to increase IAP. Furthermore, the size of the cross-sectional area of the diaphragm and the moment arm used to estimate force and moment at the lower lumbar levels, produced by IAP, have a major effect on conclusions reached about the role of IAP (McGill and Norman, 1987). The diaphragm surface area was taken as 243 cm^2, and the centroid of this area was placed 3.8 cm anterior to the center of the T12 disc (compare these values with those used in other studies: 511 cm^2 for the pelvic floor, 465 cm^2 for the diaphragm, and moment arm distances of up to 11.4 cm, which is outside the chest in most people). During squat lifts, the net effect of the involvement of the abdominal musculature and IAP seems to be to increase compression rather than alleviate joint load. (A detailed description and analysis of the forces are in McGill and Norman, 1987.) This theoretical finding agrees with the experimental evidence of Krag and colleagues (1986), who used EMG to evaluate the effect of reducing the need for the extensors to contract (they didn't), and of Nachemson, Andersson, and Schultz (1986), who documented increased intradiscal pressure with an increase in IAP.

Role of IAP During Lifting

The generation of appreciable IAP during load-handling tasks is well documented. The role of IAP is not. Farfan (1973) suggested that IAP creates a pressurized visceral cavity to maintain the hooplike geometry of the abdominal muscles. In recent work in which they measured the distance of the abdominal muscles to the spine (moment arms), McGill, Juker, and Axler (1996) were unable to confirm substantial changes in geometry when the abdominal muscles were activated in a standing posture. However, the compression penalty of abdominal activity cannot be discounted. The spine appears to be well suited to sustain increased compression loads if intrinsic stability is increased. An unstable spine buckles under extremely low compressive loads (e.g., approximately 20 N) (Lucas and Bresler, 1961). The geometry of the spinal musculature suggests that individual components exert lateral and anteroposterior forces on the spine that can be thought of as guy wires on a mast to prevent bending and compressive buckling (Cholewicki and McGill, 1996). Moreover, activated abdominal muscles create a rigid cylinder of the trunk, resulting in a stiffer structure. Both Cholewicki and colleagues (1999) and Grenier and McGill (2007, 2008) documented increased torso stiffness during elevated IAP even when accounting for similar abdominal wall contraction levels. It also appears that IAP can influence pelvic mechanics and pain. Mens and colleagues (2006) noted higher pelvic ring forces with elevated IAP, which may stabilize some yet destabilize others—depending on the nature of tissue compromise. The clinical solution for this divergence is to perform provocative testing to reveal whether the patient's problem is helped or exacerbated. Thus, although the increased IAP commonly observed during lifting and in people experiencing back pain does not have a direct role in reducing spinal compression or in adding to the extensor moment, it does stiffen the trunk and prevent tissue strain or failure from buckling.

Lumbodorsal Fascia

Various mechanical roles have been attributed to the lumbodorsal fascia (LDF). In particular, some have suggested that the LDF reduces spine loads —solving the paradox noted earlier. In fact, some have recommended lifting postures based on various interpretations of the mechanics of the LDF. Gracovetsky, Farfan, and Lamy (1981) originally suggested that lateral forces generated by internal oblique and transverse abdominis are transmitted to the LDF via their attachments to the lateral border. They also claimed that the fascia could support substantial extensor moments. Further, lateral tension from abdominal wall attachments was hypothesized to increase longitudinal tension from Poisson's effect, causing the posterior spinous processes to move together resulting in lumbar extension. This sequence of events formed an attractive proposition because the LDF has the largest moment arm of all the extensor tissues. As a result, any extensor forces within the LDF would impose the smallest compressive penalty to vertebral components of the spine.

Three independent studies, however, examined the mechanical role of the LDF and collectively questioned the idea that the LDF could support substantial extensor moments (Macintosh and Bogduk, 1987; McGill and Norman, 1988; Tesh, Dunn, and Evans, 1987). As previously noted, regardless of the choice of LDF activation strategy, the LDF contribution to the restorative extension moment was negligible compared with the much larger low back reaction moment required to support a load in the hands. Its function may be that of an extensor muscle retinaculum (Bogduk and Macintosh, 1984). Hukins, Aspden, and Hickey (1990) proposed on theoretical grounds that the LDF acts to increase the force per unit of cross-sectional area that muscle can produce by up to 30%. They suggested that this increase in force is achieved by constraining the bulging of the muscles when they shorten. This contention remains to be proven. Tesh, Dunn, and Evans (1987) suggested that the LDF may be important for supporting lateral bending. Furthermore, there is no question that the LDF is involved in enhancing the stability of the lumbar column. No doubt, a complete assessment of these notions will be pursued in the future.

Hydraulic Amplifier

The final mechanism hypothesized to unload compressive stresses from the spine was the hydraulic amplifier. This hybrid mechanism depends on three notions. First, the elevated IAP preserves the hooplike geometry of the abdominal wall during exertion. The IAP must also exert hydraulic pressure posteriorly over the spine and presumably through to the underside of the LDF. Finally, as the extensor muscle mass contracts, it was proposed to bulk upon shortening, again increasing the hydraulic pressure under the fascia. The biomechanical attraction of the pressure under the fascia is that any longitudinal forces generated in the fascia reduce the need for the underlying muscles to contribute extensor forces, thereby lowering the compressive load on the spine. Both of these proposals were dismissed. Given the size of the fascia, hydraulic pressures would have to reach levels of hundreds of mmHg. Pressures of this magnitude simply are not observed during recording (Carr et al., 1985). Moreover, the presence or absence of IAP makes little difference on the hooplike geometry of the abdominal wall (McGill, Juker, and Axler, 1996), because this is more modulated by the posture.

IAP, LDF, and Hydraulic Amplifier: A Summary

IAP, the mechanical role of the LDF, and the existence of the hydraulic amplifier were proposed to account for the paradox that people were able to perform lifts that the simple models suggested would crush their spines. Yet, although both IAP and the LDF appear to play some role in lifting, none of the three proposed mechanisms was a tenable explanation for the paradox of uncrushed spines under heavy loading, whether considered separately or combined with the other two. The problem lay in the simple models of three and four decades ago. Not only were the rather complex mechanics not represented with the necessary detail, but also the strength of the tissues to bear load was also quite underestimated in the early tests that used old cadaveric samples that were crushed underneath artificially stiff rams of materials-testing machines that caused failure too early. Vestiges of these old notions remain today.

Other Important Mechanisms of Normal Spine Mechanics

Several other features of spine mechanics influence function and ultimately underpin strategies for injury prevention and rehabilitation. The most important are presented in this section.

Biomechanics of Diurnal Spine Changes

Most people have experienced the ease of taking off their socks at night compared to putting them on in the morning. The diurnal variation in spine length (the spine being longer after a night's bed rest), together with the ability to flex forward, has been well documented. Reilly, Tynell, and Troup (1984) measured losses in sitting height over a day of up to 19 mm (0.7 in.). They also noted that approximately 54% of this loss occurred in the first 30 minutes after rising. Over the course of a day, hydrostatic pressures cause a net outflow of fluid from the disc, resulting in narrowing of the space between the vertebrae, which in turn reduces tension in the ligaments. When a person lies down at night, osmotic pressure in the disc nucleus exceed the hydrostatic pressure, causing the disc to expand. Adams, Dolan, and Hutton (1987) noted that the range of lumbar flexion increased by 5 to 6° throughout the day. The increased fluid content after rising from bed caused the lumbar spine to be more resistant to bending, although the musculature did not appear to compensate by restricting the bending range. Adams and colleagues estimated that disc-bending stresses were increased by 300% and ligament stresses by 80% in the morning compared to the evening; they concluded that there is an increased risk of injury to these tissues during bending forward early in the morning. Subsequently, Snook and colleagues (1998) demonstrated that simply avoiding full lumbar flexion in the morning reduced back symptoms. We are beginning to understand the mechanism.

Early-Morning Exercise

Clinical Relevance

People should not undertake spine exercises—particularly those that require full spine flexion or bending—just after rising from bed, given the elevated tissue stresses that result. This would hold true for any occupational task requiring full spine range of motion. Waiting an hour would be wise. Going for a walk would reduce this length of time.

Spinal Memory

The function of the spine is modulated by previous activity. This occurs because the loading history determines disc hydration (and therefore the size of the disc space and disc geometry), which in turn modulates ligament rest length, joint mobility, stiffness, and load distribution. Consider the following scenario: McKenzie (1979) proposed that the nucleus within the annulus migrates anteriorly during spinal extension and posteriorly during flexion. McKenzie's program of passive extension of the lumbar spine (which is currently popular in physical therapy) was based on the supposition that an anterior movement of the nucleus would decrease pressure on the posterior portions of the annulus, which is the most problematic site of herniation. Because of the viscous properties of the nuclear material, such repositioning of the nucleus is not immediate after a postural change, but rather, takes time. Krag and coworkers (1987) observed anterior movement of the nucleus during lumbar extension, albeit quite minute, from an elaborate experiment that placed radio-opaque markers in the nucleus of cadaveric lumbar motion segments. Whether this observation was caused simply by a redistribution of the centroid of the wedge-shaped nuclear cavity moving forward with flexion or was a movement of the whole nucleus remains to be determined. Nonetheless, hydraulic theory would suggest lower bulging forces on the posterior annulus if the nuclear centroid moved anteriorly during extension. If compressive forces were applied to a disc in which the nuclear material was still posterior (as in lifting immediately after a prolonged period of flexion), a concentration of stress would occur on the posterior annulus.

Although this area of research needs more development, a time constant seems to be associated with the redistribution of nuclear material. It appears unwise to lift an object immediately following prolonged flexion, such as sitting or stooping (e.g., a stooped gardener should not stand erect and immediately lift a heavy object). Furthermore, Adams and Hutton (1988) suggested that prolonged full flexion may cause the posterior ligaments to creep, which may allow damaging flexion postures to go unchecked if lordosis is not controlled during subsequent lifts. In a study of posterior passive tissue creep during sitting in a slouched posture, McGill and Brown (1992) showed that over the 2 minutes following 20 minutes of full flexion, subjects regained only half of their intervertebral joint stiffness. Even after 30 minutes of rest, some residual joint laxity remained. This finding is of particular importance for people whose work is characterized by cyclic bouts of full-end-range-of-motion postures followed by exertion. Before lifting following a stooped posture or after prolonged sitting, a case could be made for standing or even consciously extending the spine for a short period. Allowing the nuclear material to equilibrate, or move anteriorly to a position associated with normal lordosis, may decrease forces on the posterior nucleus in a subsequent lifting task. Ligaments will regain some protective stiffness during a short period of lumbar extension. In conclusion, the anatomy and geometry of the spine are not static. Much research remains to be done to understand the importance of tissue loading history on subsequent biomechanics, rehabilitation therapies, and injury mechanics.

Functional Significance of Spinal Memory

Clinical Relevance

It would appear protective to avoid loading immediately after a bout of prolonged flexion. In the occupational world this has relevance to ambulance drivers, for example, who drive to an accident scene without the luxury of time to warm up (or reset the passive tissues) before lifting. They would be wise to sit with a lumbar pad to avoid lumbar flexion and the associated creep. The athletic world provides good examples as well, such as sitting on the bench before engaging in play. Those with sensitive backs would do well to avoid sitting on the bench with a flexed lumbar spine while waiting to perform. We recently quantified the loss of compliance in the lumbar spine with bench sitting between bouts of athletic performance (Green, Grenier, and McGill, 2002) in elite volleyball players. Viscosity is also a consideration in prolonged postures because internal friction increases with prolonged static postures. Sitting in this way, and the associated changes in stiffness and viscosity, are detrimental to athletes' performance and increase their risk of injury. We have been successful in reducing pain in professional basketball players and people in occupational groups by providing lumbar supports and chair seat pan adjustments. We address this issue more completely in chapter 11.

Anatomical Flexible Beam and Truss: Muscle Cocontraction and Spine Stability

The osteoligamentous spine is somewhat of an anatomical paradox: it is a weight-bearing, upright, flexible rod. Observationally, the ability of the joints of the lumbar spine to bend in any direction is accomplished with large amounts of muscle coactivation. Such coactivation patterns are counterproductive to generating the torque necessary to support the applied load. Coactivation is also counterproductive to minimize the load penalty imposed on the spine from muscle contraction. Researchers have postulated ideas to explain muscular coactivation, including that abdominal muscles are involved in the generation of IAP (Davis, 1959) and that they provide support forces to the lumbar spine via the LDF (Gracovetsky, Farfan, and Lamy, 1981). These ideas have not been without opposition (see previous sections).

Another explanation for muscular coactivation is tenable. A ligamentous spine will fail under compressive loading in a buckling mode, at about 20 N (Lucas and Bresler, 1961). In other words, a bare spine is unable to bear compressive load! The spine can be likened to a flexible rod that will buckle under compressive loading. However, if the rod has guy wires connected to it, like the rigging on a ship's mast, although more compression is ultimately experienced by the rod, it is able to bear much more compressive load because it is stiffened and therefore more resistant to buckling. The cocontracting musculature of the lumbar spine (the flexible beam) can perform the role of stabilizing guy wires (the truss) to each lumbar vertebra, bracing it against buckling. Collective work by Crisco and Panjabi (1990); Cholewicki and McGill (1996); Cholewicki, Juluru, and McGill (1999); and Gardner-Morse, Stokes, and Laible (1995) has quantified the influence of muscle architecture and the necessary coactivation on lumbar spine stability. The architecture of many torso muscles is especially suited for the role of stabilization (Kavcic, Grenier, and McGill, 2004; Macintosh and Bogduk, 1987; McGill and Norman, 1987).

Injury Mechanisms

Many clinicians, engineers, and ergonomists believe that reducing the risk of low back injury requires the reduction of applied loads to the anatomical components at risk of injury. Without question, reduction of excessive loads is beneficial, but this is an overly simplistic view. Optimal tissue health requires just the right amount of loading, not too much and not too little. Although some occupations require lower loads to reduce the risk, in sedentary occupations the risk can be better reduced with more loading and varying the nature of the loading. To decide which is better, the clinician must have a thorough understanding of the biomechanics of injury, which is addressed in two parts in this section: a brief review of the injury mechanisms of individual tissues, and a description of the injury pathogenesis and several injury scenarios. Also needed is an understanding of generic situations that result in low back tissue damage, as described in the tissue injury primer at the end of chapter 1.

Muscular Cocontraction

To invoke the antibuckling and stabilizing mechanism during lifting, one could justify lightly cocontracting the musculature to minimize the potential of spine buckling.

Summary of Tissue Injury Mechanisms

This section provides a very brief description of damage from excessive load. All injuries noted are known to be accelerated with repetitive loading.

- End plates. Schmorl's nodes are thought to be healed end-plate fractures (Vernon-Roberts and Pirie, 1973) and pits that form from localized underlying trabecular bone collapse (Gunning, Callaghan, and McGill, 2001), and are linked to trauma (Aggrawall et al., 1979). In fact, Kornberg (1988) documented (via MRI) traumatic Schmorl's node formation in a patient following forced lumbar flexion that resulted in an injury. People apparently are not born with Schmorl's nodes; their presence is associated with a more active lifestyle (Hardcastle, Annear, and Foster, 1992) and demanding occupational work (Wang, Videman, and Battié, 2012). Under excessive compressive loading of spinal units in the laboratory, the end plate appears to be the first structure to be injured (Brinckmann, Biggemann, and Hilweg, 1988; Callaghan and McGill, 2001a). Studies have revealed end-plate avulsion under excessive anteroposterior shear loading.

- Vertebrae. Vertebral cancellous bone is damaged under compressive loading (Fyhrie and Schaffler, 1994) and often accompanies disc herniation and annular delamination (Balkovec et al., 2014; Gunning, Callaghan, and McGill, 2001). The site of trabecular bone fracture is greatest around any resorption pits in the patient with osteoporosis (Slyfield et al., 2012), or around any pits subsequent to Modic changes associated with end-plate fracture inflammation. Cancellous damage is also more likely along the growth plate in the adolescent spine (Balkovec et al., 2014).
- Disc annulus. Several types of damage to the disc annulus appear to occur. Classic disc herniation appears to be associated with repeated flexion motion with only moderate compressive loading required (Callaghan and McGill, 2001a) and with full flexion with lateral bending and twisting (Adams and Hutton, 1985; Gordon et al., 1991). Yingling and McGill (1999a, 1999b) documented avulsion of the lateral annulus under anteroposterior shear loading.
- Disc nucleus. Although Buckwalter (1995), when referring to the disc nucleus, stated that "no other musculoskeletal soft tissue structure undergoes more dramatic alterations with age," the relationships among loading, disc nutrition, decreasing concentration of viable cells, accumulation of degraded matrix molecules, and fatigue failure of the matrix remain obscure. However, Lotz and Chin (2000) documented that cell death (apoptosis) within the nucleus increases under excessive compressive load. Interestingly, these changes are generally not detectable or diagnosable in vivo. However, apoptosis appears reduced once the load is removed (Chin et al., 2007).
- Neural arch (posterior bony elements). Spondylitic fractures are thought to occur from repeated stress–strain reversals associated with cyclic full

flexion and extension (Burnett et al., 1996; Hardcastle, Annear, and Foster, 1992). Cripton and colleagues (1995) and Yingling and McGill (1999a) also documented that excessive shear forces can fracture parts of the arch.

- Ligaments. Ligaments seem to avulse at lower load rates but tear in their midsubstance at higher load rates (Noyes, De Lucas, and Torvik, 1994). McGill (1997) hypothesized that landing on the buttocks from a fall will rupture the interspinous complex given the documented forces (McGill and Callaghan, 1999) and joint tolerance. Falling on the behind increases the risk for prolonged disability (Troup, Martin, and Lloyd, 1981), which is consistent with the prolonged length of time it takes for ligamentous tissue to regain structural integrity when compared with other tissues (Woo, Gomez, and Akeson, 1985). Logically, damage to the vertebrae appears to increase the risk of subsequent ligament damage via altered stress (Wagnac et al., 2012).

Reducing Tissue Damage

Clinical Relevance

Summarizing injury pathways from in vitro testing, evidence suggests that reduction in specific tissue damage could be accomplished by doing the following:

- Reducing peak (and cumulative) spine compressive loads to reduce the risk of end-plate fracture
- Reducing repeated spine motion to full flexion to reduce the risk of disc herniation (reducing spine flexion in the morning reduces symptoms)
- Reducing repeated full-range flexion to full-range extension to reduce the risk of pars (or neural arch) fracture
- Reducing peak and cumulative shear forces to reduce the risk of facet and neural arch damage and painful discs.
- Reducing slips and falls to reduce the risk to passive collagenous tissues such as ligaments
- Reducing the length of time sitting, particularly exposure to seated vibration, to reduce the risk of disc herniation or accelerated degeneration

- Increasing appropriate loads to stimulate tissue adaptation (this is person and situation specific)
- Allowing sufficient recovery to promote tissue adaptation

Injury Mechanics Involving the Lumbar Mechanism

Many researchers have established that too great a load placed on a tissue will result in injury. Epidemiological studies (Hilkka et al., 1990; Marras et al., 1993; Norman et al., 1998; Videman, Nurminen, and Troup, 1990; Wang et al., 2012) have proven this notion by identifying peak loading measures (i.e., shear, compression, trunk velocity, extensor moment, heavy work, etc.) as factors that explain the frequency and distribution of reporting of back pain or increased risk of back injury. However, the search for direct evidence that links spine load with occupational low back disorders (LBDs) may have been hampered by focusing on too narrow a range of variables. Researchers have paid a massive amount of attention, for example, to a single variable—namely, acute, or single maximal exposure to, lumbar compression. A few studies have suggested that higher levels of compression exposure increased the risk of LBD (e.g., Herrin, Jaraiedi, and Anderson, 1986), although the correlation was low. Yet some studies show that higher rates of LBDs occur when levels of lumbar compression are reasonably low.

In contrast, Hadler (1991) claimed that the incidence of back injury had not declined over the past 25 years (from the date he published his opinion), even after increased research and resources had been dedicated to the area over that time frame. Hadler suggested that the focus be turned from biomechanical causes of injury to developing more "comfortable" workplaces. However, the research described thus far in this text has clearly documented links to mechanical variables. Clearly, LBD causality is often extremely complex with all sorts of factors interacting. We consider some of those factors in the following sections.

Staying Within the Biomechanical Envelope

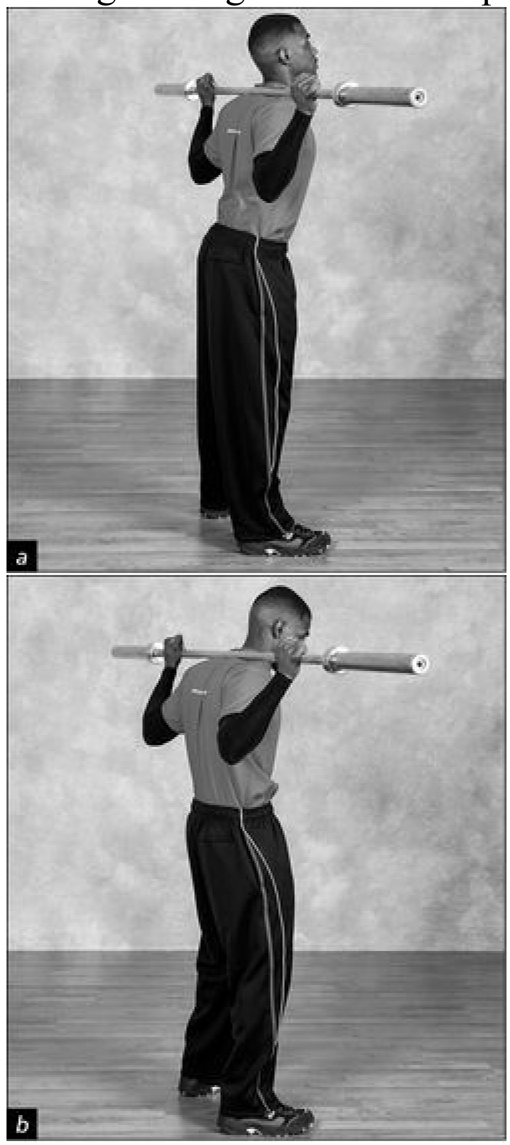
Work to understand the risk of back injury in occupational contexts has had carryover for training. What other mechanical variables modulate the risk of LBDs? As noted in chapter 2, they are as follows:

- Too many repetitions of force and motion or prolonged postures and loads have also been indicated as potential injury- or pain-causing mechanisms.
- Cumulative loading (i.e., compression, shear, or extensor moment) has been identified as a factor in the reporting of back pain (Kumar, 1990; Norman et al., 1998).
- Cumulative exposure to unchanging work has been linked to the reporting of low back pain (Holmes, Hukins, and Freemont, 1993) and intervertebral disc injury (Videman, Nurminen, and Troup, 1990).
- Many personal factors appear to affect spine tissue tolerance—for example, age and gender. In a compilation of the available literature on the tolerance of lumbar motion units to bear compressive load, Jager and colleagues (1991) noted that when males and females are matched for age, females are able to sustain only approximately two-thirds of the compressive loads of males. Furthermore, the data of Jager Luttman, and Laurig (1991) showed that within a given gender, a 60-year-old was able to tolerate only about two-thirds of the load tolerated by a 20-year-old. These data are helpful in determining optimal training loads.

To complicate the picture, Holm and Nachemson (1983) showed that increased levels of motion are beneficial in providing nutrition to the structures of the intervertebral disc, whereas much of our lab's research has demonstrated that too many motion cycles (to full flexion) resulted in intervertebral disc herniation. Buckwalter (1995) associated intervertebral disc degeneration with decreased nutrition. Meanwhile, Videman, Luttman, and Laurig (1990) showed that too little motion from sedentary work resulted in intervertebral disc injury. Although workers who performed heavy work were also at increased risk of developing back troubles, workers who were involved in varied types of work (mixed work) had the lowest risk of developing a spine injury (Videman, Luttman, and Laurig, 1990). Of course, rest allows adaptation and has been shown to be necessary for maintaining tissue strength and load-bearing ability (Marras et al., 2013). This presents the idea that too little motion or load, or too much motion or load, with insufficient rest, can increase the risk of spinal injury.

A simple experiment can be revealing. A number of years ago we asked a group of athletes to stand with a barbell on their shoulders. We were measuring spinal microshrinkage. Then we asked them to roll the pelvis anteriorly and posteriorly to impart some gentle motion to the lumbar region (see figure 4.20). They remained standing upright. We had to stop the experiment because of the pain reported by the first few subjects. Training spine motion under load requires caution. No specific guidelines exist for determining training loads—nor can such guidelines be developed that work for everyone. The point is that these notions must be acknowledged and considered on an individual basis.

Figure 4.20 Standing with a bar on the shoulders and (a) cyclically flexing and (b) extending the lumbar spine is painful and disarming—proof that training the loaded spine through a range of motion requires extreme caution.



Stoop Versus Squat in Lifting Injury Risk

Although the scientific method can prove that a phenomenon is possible if observed, failure to observe the expected result does not eliminate the possibility that it exists. One may only conclude that the experiment was insensitive to the particular phenomenon. The following discussion, like many in this book, is an attempt to incorporate this limitation and temper it with clinical wisdom.

A previous section addressed lifting with the torso flexing about the hips rather than flexing the spine. Specifically, the lifter elected to maintain a neutral lumbar posture rather than allowing the lumbar region to flex. Here we reexamine the lifting exercise, but the lifter flexes the spine sufficiently to cause the posterior ligaments to strain. This lifting strategy (spine flexion) has quite dramatic effects on shear loading of the intervertebral column and the resultant injury risk. The dominant direction of the pars lumborum fibers of the longissimus thoracis and iliocostalis lumborum muscles when the lumbar spine remained in neutral lordosis caused these muscles to produce a posterior shear force on the superior vertebra. In contrast, with spine flexion, the strained interspinous ligament complex generates forces with the opposite obliquity and therefore imposes an anterior shear force on the superior vertebra (see figures 4.21 and 4.22, a and b).

Figure 4.21 This gardener appears to be adopting a fully flexed lumbar spine. Is this a wise posture? The force analysis in figure 4.22 suggests that it is not.

Photos from Stuart McGill

Figure 4.22 These original computer image bitmaps from the experiment conducted around 1987 illustrate (a) the fully flexed spine that is associated with myoelectric silence in the back extensors and strained posterior passive tissues and high shearing forces on the lumbar spine (from both reaction shear on the upper body and interspinous ligament strain. (b) A more neutral spine posture recruits the pars lumborum muscle groups and aligns the fibers to support the shear forces (see figure 3.34). In this example, posture a resulted in 1,900 N of shear load on the lumbar spine, whereas posture b reduced the shear load to about 200 N!

Reprinted from Journal of Biomechanics, 30(5), S.M. McGill, Invited paper: Biomechanics of low back injury: Implications on current practice and the clinic, 465-475, 1997, with permission from Elsevier Science.

Let's examine the specific forces that result from flexing the lumbar spine. The recruited ligaments appear to contribute to the anterior shear force so that shear force levels are likely to exceed 1,000 N. Such large shear forces are of great concern from an injury risk viewpoint. However, when a more neutral lordotic posture is adopted, the extensor musculature is responsible for creating the extensor moment and at the same time provides a posterior shear force that supports the anterior shearing action of gravity on the upper body and handheld load. The joint shear forces are reduced to about 200 N. Thus, using muscle to support the moment in a more neutral posture, rather than being fully flexed with ligaments supporting the moment, greatly reduces shear loading (see table 4.13).

Table 4.13 Individual Muscle and Passive Tissue Forces During Full Flexion and in a More Neutral Lumbar Posture Demonstrating the Shift From Muscle to Passive Tissue

	Fully flexed lumbar spine force (N)	Neutral lumbar spine force (N)
MUSCLE		
R rectus abdominis	16	39
L rectus abdominis	16	62
R external oblique 1	10	68
L external oblique 1	10	40
R external oblique 2	7	62
L external oblique 2	7	31
R internal oblique 1	35	130
L internal oblique 1	35	102
R internal oblique 2	29	88
L internal oblique 2	29	116
R pars lumborum (L1)	21	253
L pars lumborum (L1)	21	285
R pars lumborum (L2)	27	281
L pars lumborum (L2)	27	317
R pars lumborum (L3)	31	327
L pars lumborum (L3)	31	333
R pars lumborum (L4)	32	402
L pars lumborum (L4)	32	355
R iliocostalis lumborum	58	100
L iliocostalis lumborum	58	137
R longissimus thoracis	93	135
L longissimus thoracis	93	179
R quadratus lumborum	25	155
L quadratus lumborum	25	194
R latissimus dorsi (L5)	15	101
L latissimus dorsi (L5)	15	115
R multifidus 1	28	80
L multifidus 1	28	102
R multifidus 2	28	87
L multifidus 2	28	90
R psoas (L1)	25	61
L psoas (L1)	25	69
R psoas (L2)	25	62
L psoas (L2)	25	69
R psoas (L3)	25	62
L psoas (L3)	25	69
R psoas (L4)	25	61
L psoas (L4)	25	69

	Fully flexed lumbar spine force (N)	Neutral lumbar spine force (N)
LIGAMENT		
Anterior longitudinal	0	0
Posterior longitudinal	86	0
Ligamentum flavum	21	3
R intertransverse	14	0
L intertransverse	14	0
R articular	74	0
L articular	74	0
R articular 2	103	0
L articular 2	103	0
Interspinous 1	301	0
Interspinous 2	345	0
Interspinous 3	298	0
Supraspinous	592	0
R lumbodorsal fascia	122	0
L lumbodorsal fascia	122	0

The extensor moment with full lumbar flexion was 171 Nm producing 3,145 N of compression and 954 N of anterior shear. The more neutral posture of 170 Nm produced 3,490 N of compression and 269 N of shear.

Putting Knowledge Into Practice

Clinical Relevance

Although the notion that too much of any activity (whether it be sitting at a desk or loading heavy boxes onto pallets) can be harmful is widely accepted, it is rarely taken into account in practice. For example, industrial ergonomics has not yet wholeheartedly embraced the idea that not all jobs need to be made less demanding, that some jobs need much more variety in the patterns of musculoskeletal loading, or that there is no such thing as a best posture for sitting. It is time for the profession as a whole to remember that in any job, the order and type of loading should be considered and the demand on tissues should be varied. All sedentary workers should be taught, for example, to adopt a variable posture that causes a migration of load from tissue to tissue to reduce the risk of troubles.

Quantification of the risk of injury requires a comparison of the applied load to the tolerance of the tissue. Cripton and colleagues (1995) found the shear tolerance of the spine to be in the neighborhood of 2,000 to 2,800 N in adult cadavers, for one-time loading. Work by Yingling and McGill (1999a and 1999b) and Gallagher, Howarth, and Callaghan (2010) on pig spines has shown that load rate is not a major modulator of shear tolerance unless the load is very ballistic, such as what might occur during a slip and fall. This example demonstrates that the spine is at much greater risk of sustaining shear injury (>1,000 N applied to the joint) than compressive injury (3,000 N applied to the joint) simply because the spine is fully flexed. (For a more comprehensive discussion, see McGill, 1997.) The margin of safety is much larger in the compressive mode than in the shear mode because the spine can safely tolerate well over 10 kN in compression, but 1,000 N of shear causes injury with cyclic loading. This example also illustrates the need for clinicians and ergonomists to consider other loading modes in addition to simple compression. In this example the real risk is anteroposterior shear load. Interestingly, Norman and colleagues' 1998 study showed joint shear to be very important as a metric for risk of injury of auto plant workers, particularly cumulative shear (high repetitions of subfailure shear loads) over a workday.

Stoop Versus Squat

Clinical Relevance

Much has been written on the stoop style versus the squat style of lifting. Typically, conclusions were based on very simple analyses that measured only the low back moment. The reaction moment is a function of the size and position of the load in the hands and the position of the center of mass of the upper body. As the previous example demonstrated, the issue is actually much more complex—the lumbar spine curvature determines the sharing of the load between the muscles and the passive tissues. In addition, each person elects the way agonists and antagonists are coactivated. Thus, the spine kinematic motion patterns, together with the muscle activation patterns, heavily influence the resulting spine load and the ability of the spine to bear load without damage. The risk of injury is the real issue that motivated most of these types of analyses, yet it was not addressed with sufficient detail to lead to a correct conclusion.

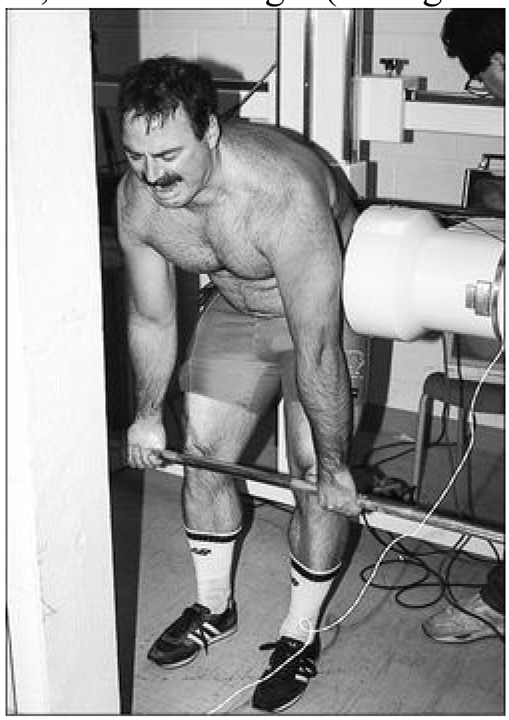
Yet another consideration impinges on the interpretation of injury risk. The ability of the spine to bear load is a function of the curvature of the spine in vivo. For example, Adams and colleagues (1994) suggested that a fully flexed spine is weaker than one that is moderately flexed. In a subsequent study, Gunning, Callaghan, and McGill (2001) showed that a fully flexed spine (using a controlled porcine spine model) is 20 to 40% weaker than if it were in a neutral posture.

Motor Control Errors and Picking Up Pencils

Although clinicians often hear patients report injuries from seemingly benign tasks (such as picking up a pencil from the floor), this phenomenon is not found in the scientific literature. Because such an injury would not be deemed compensable in many jurisdictions, medical personnel rarely record this type of event. Instead, they attribute the cause elsewhere. Moreover, although injury from large exertions is understandable, explaining injury that occurs during performance of such light tasks is not. The following is worth considering.

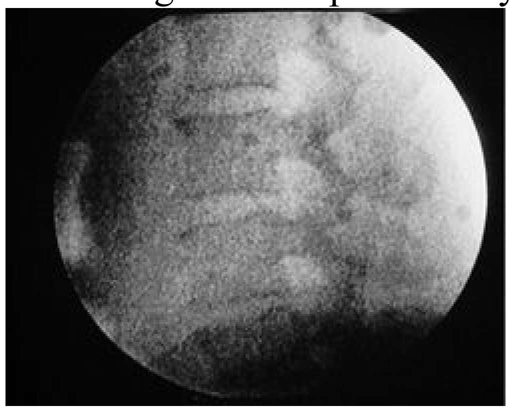
A number of years ago, using video fluoroscopy for a sagittal view of the lumbar spine, we investigated the mechanics of powerlifters' spines while they lifted extremely heavy loads (Cholewicki and McGill, 1992). The range of motion of the lifters' spines was calibrated and normalized to full flexion by first asking them to flex at the waist and support the upper body against gravity with no load in the hands. During the lifts, although the lifters appeared outwardly to have a very flexed spine, in fact the lumbar joints were 2 to 3° per joint from full flexion (see figures 4.23 and 4.24). This explains how they could lift such magnificent loads (up to 210 kg, or approximately 462 lb) without sustaining the injuries that are suspected to be linked with full lumbar flexion.

Figure 4.23 A group of powerlifters lifted very heavy weights while their lumbar vertebrae motion patterns were quantified. Each joint was skillfully controlled to flex but not fully flex. Each joint was 2 to 3° away from the fully flexed, calibrated angle (see figure 4.24).



Photos from Stuart McGill

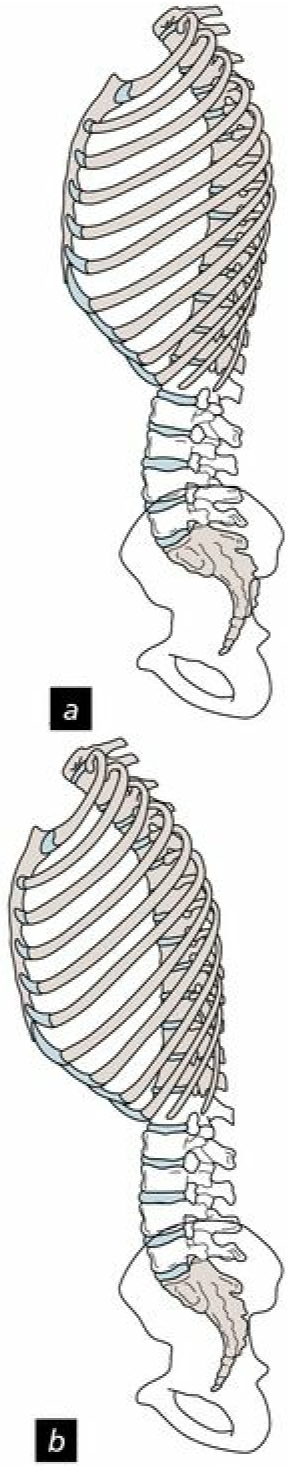
Figure 4.24 This fluoroscopy image of the powerlifter's lumbar spine shows how individual lumbar joint motion can be quantified in vivo. While recording, an injury occurred in one lifter when the vertebra at just one joint went to its full-flexion angle and surpassed it by about 0.5°.

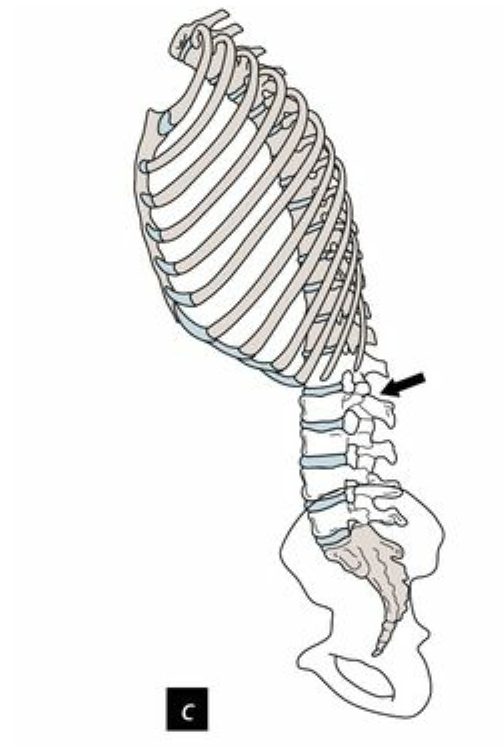


Photos from Stuart McGill

However, during the execution of a lift, one lifter reported discomfort and pain. Examination of the video fluoroscopy records showed that one of the lumbar joints (specifically, the L2-L3 joint) reached the full-flexion calibrated angle while all other joints maintained their static position (2 to 3° short of full flexion). The spine buckled and caused injury (see figure 4.25). This is the first observation we know of reported in the scientific literature documenting proportionately more rotation occurring at a single lumbar joint; this unique occurrence appears to have been due to an inappropriate sequencing of muscle forces (or a temporary loss of motor control wisdom). This motivated the work of my colleague and former graduate student Professor Jacek Cholewicki to investigate and continuously quantify the stability of the lumbar spine throughout a wide variety of loading tasks (Cholewicki and McGill, 1996). Generally speaking, the occurrence of a motor control error that results in a temporary reduction in activation to one of the intersegmental muscles (perhaps, for example, a lamina of longissimus, iliocostalis, or multifidus) may allow rotation at just a single joint to the point at which passive or other tissues could become irritated or injured.

Figure 4.25 (a) The powerlifter flexed to grab the weight bar and (b) began to extend the spine during the lift. (c) When the weight was a few inches (or about 7 cm) from the floor, a single joint (L2-L3) flexed to the full-flexion angle, and the spine buckled, indicated by the arrow.





Cholewicki noted that the risk of such an event was greatest when high forces were developed by the large muscles and low forces by the small intersegmental muscles (a possibility with our powerlifters), or when all muscle forces were low such as during a low-level exertion. Thus, injury from quite low-intensity bending is possible. Adams and Dolan (1995) noted that passive tissues begin to experience damage with bending moments of 60 Nm. This can occur simply from a temporary loss of muscular support while bending over. This mechanism of motor control error resulting in temporary inappropriate neural activation explains how injury might occur during extremely low-load situations—for example, picking up a pencil from the floor following a long day of performing a very demanding task.

Sources of Motor Control Errors

One must consider another issue when dealing with the possibility of buckling that results from specific motor control errors. In our clinical testing we observed similarly inappropriate motor patterns in some men who were challenged by holding a load in the hands while breathing 10% CO_2 to elevate breathing. (Challenged breathing causes some of the spine-supporting musculature to drop to inappropriately low levels in some people; see McGill, Sharratt, and Seguin, 1995.) These deficient motor control mechanisms heighten biomechanical susceptibility to injury or reinjury (Cholewicki and McGill, 1996). My work on large-scale longitudinal investigations to assess the power of these motor control parameters together with some personal performance variables and their role in causing back injury over a multiyear period were reviewed in chapter 2.

Sacroiliac Pain—Is It From the Joint?

Bogduk and colleagues (1996) proved that some low back pain is from the sacroiliac joint itself. The following discussion offers other considerations. The work that many have reported over recent years has demonstrated the extraordinary magnitudes of forces within the torso extensor musculature even during nonstrenuous tasks. Although these forces have been examined for their mechanical role, clinicians have expressed interest in their potential to cause injury. One possibility worth considering is that the high muscular forces may damage the bony attachments of the corresponding muscle tendons. Such damage has perhaps been wrongfully attributed to alternate mechanisms. One example follows.

Pain in the sacroiliac region is common and often attributed to disorders of the sacroiliac (SI) joint itself or the iliolumbar ligament. For this reason, the role of the musculature may have been neglected. It is known that a large proportion of the extensor musculature obtains its origin in the SI and posterior superior iliac spine (PSIS) region (Bogduk, 1980). The area of tendon–periosteum attachment and extensor aponeurosis is relatively small in relation to the volume of muscle in series with the tendon complex. From this, a hypothesis evolved that the seeming mismatch of large muscle tissue to small attachment area for connective tissue places the connective tissue at high risk of sustaining microfailure, resulting in pain (McGill, 1987). Knowledge of the collective muscle force–time histories enables speculation about one-time failure loads and cumulative trauma. For example, if the forces of muscles that originate in the SI region are tallied for the trial illustrated in table 4.4, then the total force transmitted to the SI region during peak load would exceed 5.6 kN. Such a load would lift a small car off the ground!

The failure tolerance of these connective tissues is not known, which makes speculation about the potential for microfailure difficult. No doubt the risk of damage must increase with the increase in the extremely large loads observed in the extensor musculature and with the frequency of application. Industrial tasks comparable to lifting three containers in excess of 18 kg (40 lb) per minute over an 8-hour day are not unusual, suggesting that the potential for cumulative trauma is significant.

This mechanical explanation may account for local tenderness on palpation associated with many SI syndrome cases. As well, muscle strain and spasm often accompany SI pain. Nonetheless, treatment is often directed toward the articular joint (guidance in diagnosing the joint as the primary source of pain is given in chapter 9). Although a reduction of spasm through conventional techniques would reduce the sustained load on the damaged connective fibers, patients should be counseled on techniques to reduce internal muscle loads through effective lifting mechanics. Interestingly, treatment of pain truly from the joint begins with the same strategies to reduce the irritating load. This is a single example, of which there may be several, in which knowledge of individual muscle force–time histories would suggest a mechanism for injury for which a specific—and possibly atypical—treatment modality could be prescribed.

Bed Rest and Back Pain

Although bed rest has fallen from favor as therapy, given the generally poor patient outcome, the mechanism for poor outcome is just now starting to be understood. Bed rest reduces the applied load (hydrostatic) below the disc osmotic pressure, resulting in a net inflow of fluid (McGill, van Wijk, et al., 1996). McGill and Axler (1996) documented the growth in spine length over the usual 8 hours of bed rest and then over continued bed rest for another 32 hours. This is unusual sustained pressure and is suspected to cause backache. Oganov and colleagues (1991) documented increased mineral density in the vertebrae following prolonged space travel (weightlessness); this finding is notable because other bones lost mineral density. It indicates that the spine was stimulated to lay down bone in response to higher loads; in this case, the higher load was due to the swollen discs. The analog on earth is lying in bed for periods longer than 8 hours—it actually stresses the spine!

Bending and Other Spine Motion

Although some researchers have produced disc herniations under controlled conditions (e.g., Gordon et al., 1991), Callaghan and McGill (2001a) and Balkovec and McGill (2012) have been able to consistently produce disc herniations by mimicking the spine motion and load patterns seen in workers. Specifically, only a very modest amount of spine compression force seems to be required (only 800-1,000 N), but the spine specimen must be repeatedly flexed, mimicking repeated torso–spine flexion from continual bending to a fully flexed posture. In these experiments, we documented the progressive tracking of disc nucleus material traveling posteriorly through the annulus of the disc with final sequestration of the nucleus material. This was noted to occur around 18,000 to 25,000 cycles of flexion with low levels of spine compression (about 1,000 N), but it would occur with much fewer cycles of bending with higher simultaneous compressive loads (about 5,000 cycles with 3,000 N of compression). Most interesting was the fact that the tracking of nuclear material began from the inside and slowly worked radially outward, suggesting that, had this been a living worker, there may have been little indication of cumulative damage until the culminating event.

Bending patterns appear to heavily influence the patient's pain pattern. Van Wingerden's group (e.g., Van Wingerden, Vleeming, and Ronchetti, 2008) quantified the bending patterns of various types of people with back pain; for example, lumbar pain versus pelvic girdle pain that may radiate. The two groups initiate bending motion differently. It is not clear whether this is adaptive and subsequent to the pain or pain-causative. In either case the tissue stress distribution is associated with the pain such that altering the pattern can reduce pain, sometimes immediately (Ikeda and McGill, 2012).

Moderation in All Things

Clinical Relevance

The phrase “moderation in all things” appears to encapsulate a lot of spine biomechanical wisdom; varying loading through constant change in activity is justifiable. Obviously, overload is problematic, but the Bed Rest and Back Pain section shows that even lying in bed too long is problematic—it increases annulus, end-plate, and vertebral stress!

Confusion Regarding Spine Flexion

This short section addresses the confusion regarding the issue of spine flexion and the misquoting of our work on this topic.

There is confusion between the terms flexion movement and flexion moment. Flexion movement defines the act of bending the spine forward, flexing the spine. This is the kinematic term. Flexion moment refers to the act of creating flexion moment, or torque. This is the kinetic term. This is independent of whether movement occurs. Standing and pushing a load requires the spine to stiffen with anterior muscle activation; hence, flexion moment occurs, which requires abdominal muscle strength but not movement.

Flexion movement of the spine strains the layers of collagen in the spinal discs and, if excessive, will lead to herniation. When loads on the spine are small, movement is healthy. Several other events occur depending on the amount of stretch on the spine ligaments at the end range of flexion. For example, cytokines linked to acute and chronic inflammation accumulate with repeated full-flexion motion exposure (D'Ambrosia et al., 2010).

As noted, many variables influence the rate of the herniation process. For example, the thickness of the spine also influences the rate of gradual herniation: thicker spines have higher bending stress and herniate faster with flexion cycles. For example, an NFL linebacker must have larger-diameter discs to survive the compressive loading, but will not do well performing 1,000 sit-ups. In contrast, a fellow in Brazil, featured on YouTube, does 1,000 sit-ups every day. He has a very slender spine, so the bending stresses are small. However, his spine would not survive the loading of a single NFL game. Different spines mean different injury mechanisms, different resiliencies to motion, and different training approaches. Choose your parents; then choose your best way to train!

Why is flexion exercise such a passionate issue? Core training, abdominal training, and core stiffness and stability are all essential components for performance enhancement and injury resilience. But the specific issue here is whether the spine needs flexion movement training or flexion moment training. The foundation for function has four components: (1) Proximal stiffness (meaning the lumbar spine and core) enhances distal athleticism and limb speed; (2) a muscular guy wire system is essential for the flexible spine to successfully bear load; (3) muscular coactivation creates stiffness to eliminate micromovements in the joints that lead to pain and tissue degeneration; and (4) abdominal armor is necessary for some combative and impact athletes. Logically, we must now discuss the priority for flexion movement or moment. These are addressed in chapter 5, Myths and Realities of Lumbar Spine Stability.

Some people claim that their sport or occupation is flexion movement–based and they must train flexion movement—such as jiu-jitsu athletes. I have consulted with several top jiu-jitsu athletes. They were not able to train because of the back pain they induced over time training flexion cycles. With no capacity to train, they were finished. We changed the training from a flexion movement to a flexion moment approach, thus restoring pain-free spine flexion ability but saving the spine flexion for the ring and octagon. Their capacity to train was restored. Some careers were salvaged and indeed flourished.

How are these thoughts put into practice? First, if the spine is under load, it is best not to move it—that is, to keep it stiff. This principle is not contestable. However, if the spine must flex such as when a strongman event competitor lifts an atlas stone, it should be held in an isometrically flexed posture. The stone is hooked by the thighs, arms, and pectoralis muscles as the spine curls over the stone. The spine does not move because the motion is focused about the hip joints until the final "hoik." Thus, the spine is flexed while under load, but it does not move. The worst thing to do would be to move the spine into flexion, over and over; the combination of load with motion would slowly and cumulatively delaminate the disc collagen. Here the spine is fatigued before the muscles. Training volume is compromised. From the perspective of injury mechanics and exercise prescription, the flexion issue is complex. The approach should not be guided by a single study, not one on pigs, humans, or within institutions such as the military. But together, all of these studies are important.

Confusion Over Twisting

Although twisting has been named in several studies as a risk factor for low back injury, the literature does not distinguish between the kinematic variable of twisting and the kinetic variable of generating twisting torque (similar to flexion movement and moment as discussed earlier). Many epidemiological surveillance studies link a higher risk of LBD with twisting, but twisting with low twist moment demands results in lower muscle activity and lower spine load (McGill, 1991). Further, passive tissue loading is not substantial until the end of the twist range of motion (Duncan and Ahmed, 1991). However, developing twisting moment places very large compressive loads on the spine because of the enormous coactivation of the spine musculature (McGill, 1991). This can also occur when the spine is not twisted but in a neutral posture in which the ability to tolerate loads is higher. Either single variable (the kinematic act of twisting or generating the kinetic variable of twist torque while not twisting) seems less dangerous than epidemiological surveys suggest. However, very high tissue loading may pose an elevated risk when the spine is fully twisted and there is a need to generate high twisting torque (McGill, 1991). Several studies, then, have suggested that compression on the lumbar spine is not the sole risk factor. Both laboratory tissue tests and field surveillance suggest that shear loading of the spine, together with large twists coupled with twisting torque, increases the risk of tissue injury.

Repeated Spine Flexion and Bending

Clinical Relevance

Although much ergonomic effort has been devoted to reducing spine loads, it is becoming clear that repeated spine flexion—even in the absence of moderate load—will lead to discogenic disorders. Furthermore, evidence shows that the direction of the bending determines the location of the annulus damage (Aultman, Scannell, and McGill, 2005). Ergonomic guidelines will be more effective once factors for flexion repetition and direction are included.

Is Viral or Bacterial Involvement Possible in Low Back Pain?

Research has suggested that the incidence of some musculoskeletal disorders of the upper extremity is elevated following exposure to a viral infection. Could viral or bacterial infections also be responsible for LBDs? Although this scenario is possible in individual cases, it could only account for some. Because animal model studies use animals that are screened for disease, with only approved specimens selected for testing, controlled mechanical loading would seem to account for the significant findings. Viral infections have been linked to an increased probability of developing such diseases as carpal tunnel syndrome and arthritis (Mody and Cassim, 1997; Phillips, 1997; Samii et al., 1996). However, Rossignol and colleagues (1997) found that work-related factors accounted for the majority of the causes influencing the development of carpal tunnel syndrome. Similar conclusions have been reached for back pain. For example, Albert and colleagues (2013) reported that patients with longer-term back pain following disc herniation with subsequent Modic type 1 changes did better with antibiotics than without. Thus, antibiotics may help some patients. For these patients, only mechanical causes created the original disc herniations, and the associated Modic changes are evidence of an inflammatory response. Inflammation is a natural and common response when the body perceives herniated material as a foreign body. The subsequent mechanical changes from the disc herniation may also enhance the bone bruise link to Modic changes noted in chapter 3. Bacterial activity may be present over the longer course in these patients. Thus, bacterial links may be involved in some cases, but the instigating mechanical cause must not be downplayed.

Sciatic Symptoms

Sciatica can be caused by tension of the sciatic nerve, or of the lumbar nerve roots, or within the cauda equina. It also can be caused by pinching at numerous locations along its length or by irritation by rough surfaces such as arthritic bone or extruded disc material. The symptoms range from radiating pain to sensations in the leg or foot. Back pain may or may not be present. Tragically, cases of foot pain, ankle pain, or leg pain caused by lumbar nerve compromise are often treated with stretching in the belief that muscle tightness rather than neural tension is the culprit. This just keeps the nerve angry. We take two approaches. First, spine-sparing motions are adopted to avoid end range of motion and associated nerve tension and to reduce possible disc bulging. The second approach involves nerve flossing (nerve flossing technique and qualifying tests for patient selection are explained in part III of this book).

Section Summary

From this description of injury mechanisms with several modulating factors, it is clear that multifactorial links exist. The interpretation of these links will enhance injury prevention and rehabilitation efforts. In fact, effective interventions will not occur without an understanding of how the spine works and how it becomes injured.

Biomechanical and Physiological Changes Following Injury

Although the foundation for good clinical practice requires an understanding of the mechanism for injury, an understanding of the lingering consequences is also helpful.

Tissue Damage Pathogenesis, Pain, and Performance

Many discuss spine injury as though it were a static entity; that is, by focusing on specific tissue damage. However, because tissue damage causes changes to the joint biomechanics, other tissues will be affected and drawn into the clinical picture. Understanding the links in this issue begins with the concept of pain.

Tissue damage causes pain. Some have said that there is no proof that this statement is true because pain is a perception and no instrument can measure it directly. This notion ignores the large body of pain literature (a review of which is well outside the scope of this chapter), which has motivated a recent proposal from a diverse group of pain specialists to classify pain by mechanism—specifically, transient pain (which does not produce long-term sequelae) and tissue injury and nervous system injury pain, both of which have complex organic mechanisms (Woolf et al., 1998).

Siddall and Cousins (1997) wonderfully summarized the great advances made in the understanding of the neurobiology of pain—in particular, the long-term changes from noxious stimulation known as central sensitization. Briefly, tissue damage directly affects the response of the nociceptor to further stimulation; over longer periods of time, both increase the magnitude of the response, sensitivity to the stimulus, and size of the region served by the same afferents. Some clinicians have used this as a rationale for the prescription of analgesics early in acute low back pain. Furthermore, some have based arguments on whether a certain tissue could initiate pain from the presence or absence of nociceptors or free nerve endings, requiring proof of damage to the specific tissue in question. But tissue damage (along a continuum from cell damage to macrostructural failure) changes the biomechanics of the spine. Not only may this change cause pain, but it also appears to initiate a cascade of change that can cause disruptions to the joint and continual pain for years leading to conditions such as facet arthritis, accelerated annular degeneration, and nerve root irritation, to name a few. (See Kirkaldy-Willis and Burton, 1992, for an excellent review of the cascade of spine degeneration initiated by damage.) Butler and colleagues (1990) documented that disc damage nearly always occurs before facet arthritis is observed. Injury and tissue damage initiate joint instabilities, causing the body to respond with arthritic activity to finally stabilize. This results in a loss of range of motion and, no doubt, pain (Brinckmann, 1985).

Injury Process: Motor Changes

It is conclusive that patients reporting debilitating low back pain suffer simultaneous changes in their motor control systems. Recognizing these changes is important because they affect the stabilizing system. Richardson and coworkers (1999) produced quite a comprehensive review of this literature and made a case for targeting specific muscle groups during rehabilitation. Specifically, their objective is to reeducate faulty motor control patterns postinjury. The challenge is to train the stabilizing system during steady-state activities, and also during rapid voluntary motions, to withstand sudden surprise loads.

Among the wide variety of motor changes researchers have documented, they have paid particular attention to the transverse abdominis and multifidus muscles. For example, during anticipatory movements such as sudden shoulder flexion, the onset of transverse abdominis has been shown to be delayed in a few subjects with back pain (Hodges and Richardson, 1996; Richardson et al., 1999). Although the Queensland group led by Professors Hodges and Richardson developed a rehabilitation protocol specifically intended to reeducate the motor control system for involvement of the transverse abdominis, many other muscles exhibit motor control perturbations.

A number of laboratories have also documented changes to the multifidus complex. Further, in a very nice study of 108 patients with histories of chronic LBD ranging from 4 months to 20 years, Sihvonen and colleagues (1997) noted that 50% had disturbed joint motion and that 75% of those with radiating pain had abnormal EMGs to the medial spine extensor muscles.

Interestingly, in the many studies on the multifidus, EMG abnormalities in the more lateral longissimus seem to appear along with those in multifidus in people with back pain (Haig, LeBreck, and Powley, 1995). Jorgensen and Nicolaisen (1987) associated lower endurance in the spine extensors in general in those with back pain, whereas Roy and colleagues (1995) established faster fatigue rates in the multifidus in those with low back troubles. In addition, changes in torso agonist–antagonist activity have been documented during gait (Arendt-Nielson et al., 1995; Vogt, Pfeifer, and Banzer 2003), particularly in the gluteals, back extensors, and hamstrings during walking. In addition, asymmetrical extensor muscle output has been observed during isokinetic torso extensor efforts (Grabiner, Koh, and Ghazawi, 1992), which alters spine tissue loading.

Further, evidence indicates that the structure of the muscle itself experiences change following injury or pain episodes. Anatomical changes following low back injury include asymmetrical atrophy in the multifidus (Hides, Richardson, and Jull, 1996), although this is controversial (see chapter 3), and fiber type changes in multifidus even 5 years after surgery (Rantanen et al., 1993). Long-term outcome was associated with certain composition characteristics. Specifically, good outcomes was associated with normal fiber appearance, whereas poor outcome was associated with atrophy in the Type II fibers and a moth-eaten appearance in Type I fibers. Moreover, even after symptoms had resolved, Hides, Richardson, and Jull (1996) documented a smaller multifidus and suggested impaired reflexes as a mechanism. This theory appears tenable, given documented evidence of this at other joints, particularly at the knee (Jayson and Dixon, 1970; Stokes and Young, 1984).

Once again, the reason for the clinical emphasis on the multifidus may well be that the bulk of the research has been performed on this muscle. Interestingly, although static low-level motor control approaches have been suggested to address the multifidus, Danneels and colleagues (2001) found that they did not restore multifidus bulk. Only more rigorous exercises accomplished this, such as the leg lift, bird dog, body extension lift, and leg lift from a prone posture. Despite the focus on the multifidus, researchers who have examined other muscles have observed similar changes in unilateral atrophy—for example, in the psoas (Dangaria and Naesh, 1998). Wise clinicians consider all muscles, not just those that have been chosen for study.

Function, Rehabilitation, and Pain

Clinical Relevance

An interesting point is that back pain can be both a blessing and a curse. Working through pain (assuming muscular pain) is often a rehabilitation and training necessity. But pain from tissue damage is another story. Pain inhibits normal motor patterns. Furthermore, it is well established that one does not get used to pain from tissue damage; rather, the process of central sensitization ensures that the person becomes even more sensitive to the pain. For many people, addressing proper mechanics to spare the damaged tissues must precede rigorous training. How this is done is explained in part III of this book.

Specific Patterns of Muscle Inhibition Following Injury

As stated in the previous section, pain inhibits normal motor patterns and is associated with several other characteristics. More insight into this issue is provided in this section.

Crossed-Pelvis Syndrome and Gluteal Amnesia

Dr. Vladamir Janda (see figure 4.26) proposed the crossed-pelvis syndrome in which those with a history of chronic low back troubles displayed characteristic patterns of what he referred to as weak and tight muscles. Specifically, he described the features of the crossed-pelvis syndrome as including a weak gluteal and abdominal wall complex with tight hamstrings and hip flexors. He developed a technique to correct this aberrant pattern. Although I have difficulty integrating the terms weak and tight from a scientific point of view, Janda's insights were generally true. Measuring groups of men with chronic back troubles during squatting types of tasks revealed that they try to accomplish this basic motion and motor pattern of hip extension emphasizing the back extensors and the hamstrings—they appear to have forgotten how to use the gluteal complex. Noticeable restrictions in the hip flexors may or may not be present, but without question the gluteals are not recruited to levels that are necessary to both spare the back and foster better performance. I refer to this as gluteal amnesia. Our recent work has proven that gluteal inhibition is caused by pain. We induced hip pain with intra-articular pressurization and documented the lower gluteal activation in standardized hip extension tests. Releasing the pressure restored the gluteal activation (Freeman et al., 2013).

Figure 4.26 I worked in several clinical workshops with Dr. Janda prior to his death. He taught me much clinical wisdom that we were able to evaluate and quantify. Here he is demonstrating assessing my gluteal activation while apparently providing mild entertainment to the audience.

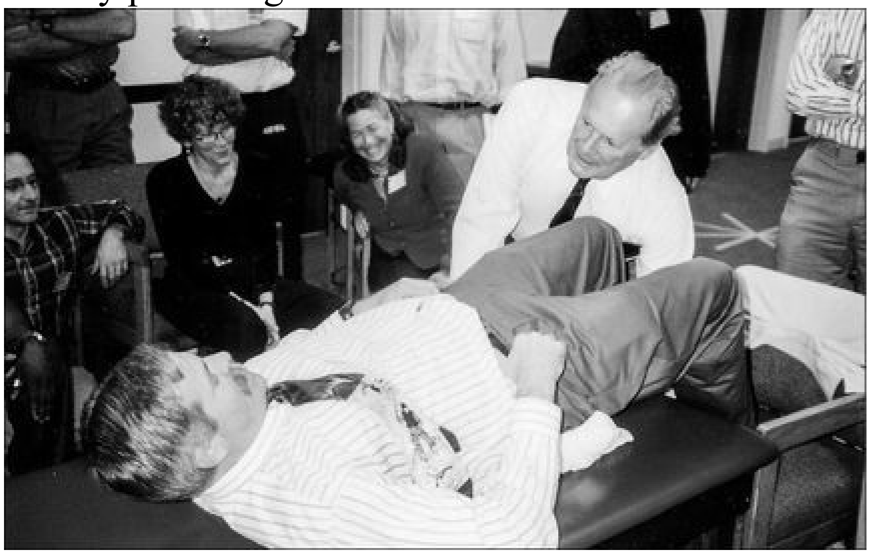


Photo from Stuart McGill

It is common for both patients and athletes to arrive at our research clinic with chronic back troubles and a crossed-pelvis overlay. Traditional strength approaches to rehabilitating their backs have failed because strength squat patterns were attempted on aberrant motor patterns. Specifically, the gluteal complex was not able to contribute its share to hip extension, which in turn loaded up the back as the erector spinae crushed the spine. Sparing the back during hip extensor training demands healthy gluteals. Specific training to reprogram gluteal integration is described and demonstrated in part III of this book.

Lingering Deficits Following Back Injury

Clinical Relevance

In our study (McGill et al., 2003), we extensively tested 72 workers. Of those workers, 26 had a history of back troubles sufficient to result in lost work time, 24 had some back troubles but not severe enough to ever lose work time, and the rest had never had any back complaints. All were asymptomatic and back to work at the time of testing. The litany of differences among the groups is explained elsewhere but included several motor control deficits. Our findings can be summarized as follows:

- Having a history of low back troubles was associated with a larger waist girth, a greater chronicity potential as predicted from psychosocial questionnaires, and perturbed flexion-to-extension strength and endurance ratios, among other factors.
- Those who had a history of back troubles had a lack of muscle endurance—specifically, a lack of balance of endurance among torso muscle groups. Absolute torso strength was not related, although the ratio of flexor to extensor strength was important.
- Those with a history of low back trouble had diminished hip extension and internal rotation, suggesting psoas involvement.
- Those with a history of back troubles had a wide variety of motor control deficits including deficits during challenged breathing (as would occur during challenging work), balancing, and enduring surprise loading. Generally, those who were poor in one motor task were likely to be poor in another.

Given that those workers who had missed work because of back troubles were measured 261 weeks, on average, after their last disabling episode, the multiple deficits appear to remain for very lengthy periods. The broad implication of this work is that having a history of low back troubles, even after a substantial amount of time has elapsed, is associated with a variety of lingering deficits such that a multidisciplinary training intervention approach would be required to diminish their presence. This collection of evidence strongly supports exercise prescriptions that promote the patterns of muscular cocontraction and movement patterns observed in fit spines and quite powerfully documents pathoneural-mechanical changes associated with chronic LBD. Once again, these changes are lasting years—not 6 to 12 weeks! The evidence presented here collectively justifies the training regimens described in part III of this book. It is critical to address basic motion and motor patterns first, before serious back training begins in earnest. This is the way to establish the foundation for ultimate performance, and to do it so as to break chronicity in those with a history of back troubles.

A Final Note

Some experts state that we don’t know what causes back pain. This implies that back pain just happens, leaving clinicians to administer their pain medications or surgeries as the treatment. I hope I have revealed their selective ignoring of evidence. Linking specific causes will guide specific strategies to avoid the cause and provide specific treatment.

Chapter 5
Myths and Realities of Lumbar Spine Stability

Stability is a popular term in discussions of the low back, but it may be widely misunderstood and inappropriately used. It has instigated passionate debate, and I have commented on some perceived biases and misunderstandings (McGill, 2011, 2013; Hodges et al., 2013b). In previous chapters we established several relevant facts. First, all sorts of tissue damage result in joint laxity, which in turn can lead to instability. For example, strained or failed ligaments cause joint laxity and unstable motion under load. End-plate fractures with loss of disc height are another example of tissue damage that results in unstable joint behavior. Clearly, joint instability is a consequence of tissue damage (this is nicely summarized by Oxland et al., 1991). A fundamental tenet is that lost mechanical integrity in any load-bearing tissue will result in stiffness losses and an increased risk of unstable behavior. We also saw in chapter 4 that during an event in which instability was observed (the buckling spine of the powerlifter), injury resulted. So, instability can both cause and be the result of injury. Finally, overlaying the tissue-based aspects of stability are the motor control aspects because coordinated contraction stiffens the joints and ultimately determines joint stability.

This chapter provides a definition of stability and an understanding of how it is increased or decreased. This is a critical foundation for those prescribing stabilization exercise or recommending strategies to prevent injury. Attempts to enhance stability and prevent instability are compromised without an understanding of the influencing factors. To quantify those factors, however, we must agree on definitions. What exactly do we mean when we use the terms spine stability, core stability, and stabilization exercise? Often, the meanings depend on the background of the person. To the biomechanist the terms pertain to a mechanical structure that can become unstable when a critical point is reached; a surgeon may view abnormal joint motion patterns as unstable but correctable through changing the anatomy; and the manual medicine practitioner may interpret patterns of muscle coordination and posture as indicative of instability and attempt to alter one, or a few, muscle activation profiles. Several groups have made contributions to the stability issue, but only a very few have attempted to actually quantify stability. This critical issue is addressed here.

Upon completion of this chapter, you will understand stability and its importance in injury prevention and rehabilitation. Furthermore, you will understand why certain approaches are preferable for achieving sufficient stability.

Why Spine Stability Is Important for Everyone

Why do so many feel so passionately about spine stability, core stability, and stabilization exercise? Core training, abdominal training, and core stiffness and stability are all essential components for pain control, performance enhancement, and injury resilience. The explanation has four components: (1) Proximal stiffness (meaning the lumbar spine and core) enhances distal athleticism and limb speed; (2) a muscular guy wire system is essential for the flexible spine to successfully bear load; (3) muscular coactivation creates stiffness to eliminate micromovements in the joints that lead to pain and tissue degeneration; and (4) abdominal armor is necessary for people in some occupational, combative, and impact situations.

How does core stiffness enhance limb speed and strength? Consider an example with the pectoralis major (pec) muscle, which attaches to the rib cage at its proximal end, crosses the shoulder joint, and attaches to the humerus at its distal end. When muscles contract, they try to shorten. Consider the specific action here: the arm flexes around the shoulder joint moving the arm from muscle shortening. But the same shortening also bends the rib cage toward the arm at the proximal end of the muscle. Thus, using just the pec muscle would not result in a fast or forceful push or punch. If the proximal end of the pec muscle attachment were stiffened, however—that is, the rib cage couldn't move—100% of pec muscle shortening would be directed to action at its distal end, producing fast and forceful motion in the arm. In the same way a stiffened core locks down the proximal ends of the hip muscles, thus producing faster leg motion. A loss of core stiffness causes the torso to bend when sprinting, resulting in a loss of speed: some force was robbed that should have been expressed in leg velocity. The same occurs during lifting, stair climbing, carrying, and chopping firewood. Thus, a universal law of human movement is established: Proximal stiffness enhances distal mobility and athleticism. This requires core stiffness training, not movement.

The second reason core training is essential is due to the fact that the spine is a stack of vertebrae that is called upon to bear loads, yet it is flexible. An engineer cannot design a structure to be good at both. A steel beam that is straight and stood on its end is stiff and can bear loads that try to compress, shear, and twist it. The beam can bear load, but it can't move. A flexible rod that allows movement will bend and buckle under load, but absorbs shock. Our spines do it all—they bend and allow the lungs to fill with air, and even allow us to dance. The spine is this beautiful structure that is flexible and allows flowing movement, but it requires a three-dimensional guy wire system to stiffen and stabilize it when it is required to bear loads. An analysis of the muscular system, together with its associated fascia sheets, reveals a clever guy wire system that creates balanced stiffness, eliminating the possibility of buckling and injury. The concern is that modern living does not tune and train this guy wire system. In many people it lapses into complacency. This requires core stiffness training, not movement.

The third reason core work is so important is that back injury causes tissue and joint laxity. Injury to the disc causes it to lose height, allowing aberrant micromovements. The micromovements irritate sensory nerves, which results in back pain and radiating pains. Spine stiffness from cocontracted torso muscles minimizes micromovements and controls pain. Again, this requires core stiffness training, not movement.

Fourth, some workers (e.g., fishermen, oil roughnecks, soldiers, firefighters) and combative and impact athletes require abdominal armor. Top combative athletes seek my consulting expertise for back pain. Typically, they have trained high repetition sit-ups to build armor, but eventually develop back pain that ends their careers. I change their flexion movement approach to flexion moment by having them perform exercises such as stir the pot (see chapter 11). No motion occurs in the spine. As a result, their ability to train pain-free is restored, and their careers are salvaged.

Finally there is practical, or applied, evidence from group trials regarding the value of core training. For example, military groups have made speed sit-ups mandatory for annual fitness tests. Recognizing their unacceptable low back injury statistics, some replaced sit-ups with planks and the stir the pot exercise, which are consistent with core stiffening training (not movement) (Childs et al., 2010). The groups using moment training performed better in the sit-up tests than the groups training with sit-ups, even though they did not train them. The reason is that they had healthier backs.

The big three exercises described in this book have been proven to enhance stiffness (Lee and McGill, 2015), tune the guy wire support loads, and eliminate the micromovements that cause pain (Ikeda and McGill, 2013).

Stability: A Qualitative Analogy

The following demonstration of structural stability illustrates key issues. Suppose a fishing rod is placed upright and vertical with the butt on the ground. If the rod were to have a small load placed in its tip (e.g., 1-2 lb, or 0.5-0.9 kg), it would soon bend and buckle. Now suppose that the same rod has guy wires attached at various levels along its length and that those wires are also attached to the ground in a circular pattern (see figure 5.1, a and b). Each guy wire is pulled to the same tension (this is critical). Now if the tip of the rod is loaded as before, the rod can sustain the compressive forces. If you reduce the tension in just one of the wires, the rod will buckle at a reduced load; we could actually predict the node, or locus, of the buckle.

Figure 5.1 (a) The spine is analogous to a fishing rod placed upright with the butt on the ground. When compressive load is applied downward to the tip, it buckles quickly. (b) Attaching guy wires at various levels and in different directions and, most important, tensioning each guy wire to the same tension ensures stability even with massive compressive loads. Note that the guy wires need not have high tension forces, but that the tensile forces must be of roughly equal magnitude. This is the role of the musculature in ensuring sufficient spine stability.

Photos from Stuart McGill

Compressive loading similar to this has been performed on human lumbar spines. Typically, an osteoligamentous lumbar spine from a cadaver with muscles removed (and no guy wires) will buckle under approximately 90 N of compressive load (first noted by Lucas and Bresler, 1961). This is all a spine can withstand!

This analogy demonstrates the critical role of the muscles (the guy wires) to first ensure sufficient stability of the spine so that it is prepared to withstand loading and sustain postures and movement. Also demonstrated with this example is the role of the motor control system, which ensures that the tensions in the cables are proportional so as to not create a nodal point at which buckling will occur. Revisiting the buckling injury that we observed fluoroscopically in the powerlifter (figure 4.24 in chapter 4), we would hypothesize that it was caused by a motor control error in which possibly one muscle reduced its activation or, from the previous analogy, lost its stiffness. The synchrony of balanced stiffness produced by the motor control system is critical. Now we can address how stability is quantified and modulated.

Quantitative Foundation of Stability

This section quantifies the notion of stability from a spine perspective. During the 1980s, Professor Anders Bergmark of Sweden very elegantly formalized stability in a spine model with joint stiffness and 40 muscles (Bergmark, 1987). In this classic work he was able to represent mathematically the concepts of energy wells, stiffness, stability, and instability. For the most part, this seminal work went unrecognized largely because the engineers who understood the mechanics did not have the biological or clinical perspective, and clinicians were hindered in the interpretation and implications of the engineering mechanics. This section synthesizes Bergmark's pioneering effort as well as its continued evolution in the work of several others and attempts to encapsulate the critical notions without mathematical complexity. If you are mathematically inclined, see Bergmark's original work or its formalization by Cholewicki and McGill (1996).

Potential Energy as a Function of Height

The concept of stability begins with potential energy, which for our purposes here is of two basic forms. In the first form, objects have potential energy (PE) by virtue of their height above a datum.

$$PE = \text{mass} \times \text{gravity} \times \text{height}$$

Critical to measuring stability are the notions of energy wells and minimal potential energy. A ball in a bowl is considered stable because if a force (or a perturbation) were applied to it, it would rise up the side of the bowl but then come to rest again in the position of least potential energy at the bottom of the bowl (the energy well) (see figure 5.2, a-d). As noted by Bergmark, "stable equilibrium prevails when the potential energy of the system is minimum." The system is made more stable by deepening the bowl or by increasing the steepness of the sides of the bowl, or both (see figure 5.3). Thus, the notion of stability encompasses the unperturbed energy state of a system and a study of the system following perturbation. If the joules of work done by the perturbation are less than the joules of potential energy inherent to the system, then the system will remain stable (i.e., the ball will not roll out of the bowl). The corollary is that the mechanical system will become unstable and possibly collapse if the applied load exceeds a critical value (determined by potential energy and stiffness).

Figure 5.2 The continuum of stability. (a) The deepest bowl is most stable, and (d) the hump is least stable. The ball in the bowl seeks the energy well, or position of minimal potential energy (m · g · h). Deepening the bowl or increasing the steepness of the sides increases the ability to survive perturbation. This increases stability.

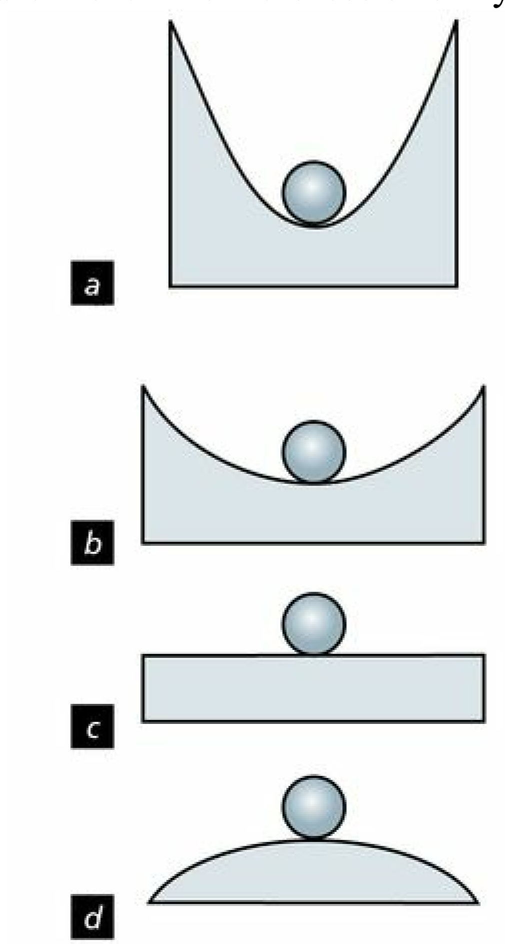
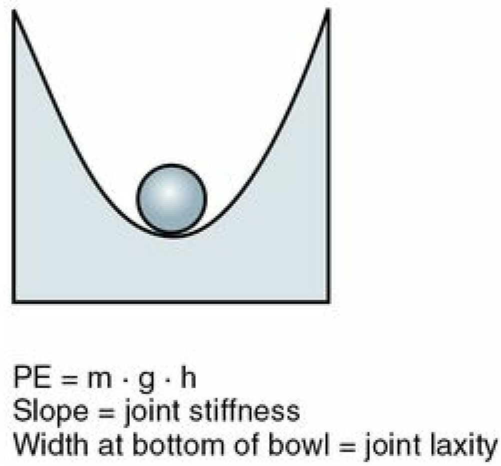


Figure 5.3 The steepness of the sides of the bowl corresponds to the stiffness of the passive tissues of the joint, which creates the mechanical stop to motion. The width of the bottom of the bowl corresponds to joint laxity. For example, a positive drawer test of the knee would be represented by a flattened bottom of the curve in which small applied forces produce large unopposed motion.



The previous ball analogy is a two-dimensional example. This would be analogous to a hinged skeletal joint that has the capacity only for flexion–extension. Spinal joints can rotate in three planes and translate along three axes, requiring a six-dimensional bowl for each joint. Mathematics enables us to examine a 36-dimensional bowl (6 lumbar joints with 6° of freedom each) representing the whole lumbar spine. If the height of the bowl were decreased in any 1 of these 36 dimensions, the ball could roll out. In clinical terms, a single muscle having an inappropriate force (and thus stiffness) or a damaged passive tissue that has lost stiffness can cause instability that is both predictable and quantifiable.

Some clinicians have confused spine stability with whole-body balance and stability, which involves the center of mass and base of support in the context of falling over; this is quite different from spine stability. Figure 5.4, a and b, illustrates the mechanics of whole-body balance and stability. Figure 5.5, a and b, shows the clinical practice of prodding patients. This approach is misguided for enhancing spine stability.

Figure 5.4 Another type of stability, often confused with spinal column stability, involves the center of mass and base of support in the context of falling over. (a) This triangle is stable because a small perturbation to its top would not cause it to fall. The size of this stability can be quantified, in part, by the size of the angle θ. (b) As the center of the mass approaches a vertical line drawn from the base of support, a smaller perturbing force would be required for it to fall, demonstrating that it is stable but less so.

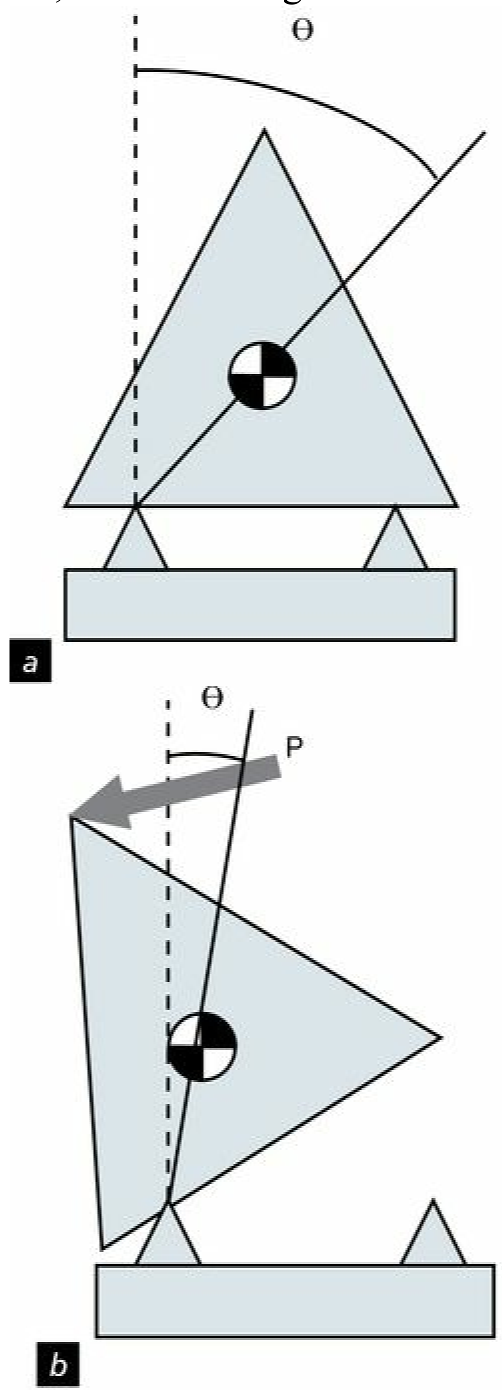


Figure 5.5 (a, b) Misunderstanding stability, illustrated in the examples given in figure 5.4, has led clinicians to try to enhance spine stability by prodding patients and attempting to knock them off balance. This is not spine stability but whole-body stability.

Photos from Stuart McGill

Potential Energy as a Function of Stiffness and Elastic Energy Storage

Although potential energy by virtue of height is useful for illustrating the concept, potential energy as a function of stiffness and storage of elastic energy is actually used for musculoskeletal application. Elastic potential energy is calculated from stiffness (k) and deformation (x) in the elastic element, as shown here:

$$PE = \frac{1}{2} \times k \times x^2$$

We will use an elastic band as an example. Stretching the band with a stiffness (k) and a distance x, it will store energy (PE). The stretched band has the potential to do work when stretched. In other words, the greater the stiffness (k), the greater the steepness of the sides of the bowl (from the previous analogy), and the more stable the structure. Thus, stiffness creates stability (see figure 5.6). More specifically, symmetrical stiffness creates even more stability. (Symmetry in stiffness is achieved by virtually all muscles of the torso; see figure 5.7, a and b.) If more joules of work are performed on the spine than there are joules of potential energy due to stiffness, the spine will become unstable (see figure 5.8).

Figure 5.6 Increasing the stiffness of the cables (muscles) increases the stability (or deepens the bowl) and increases the ability to support larger applied loads (P) without failing. But most important, ensuring that the stiffness is balanced on each side enables the column to survive perturbation from either side. Increasing stiffness of just one spring will actually decrease the ability to bear compressive load (lowering PE in one direction and mode), illustrating the need for balancing stiffness for a given posture and moment demand.

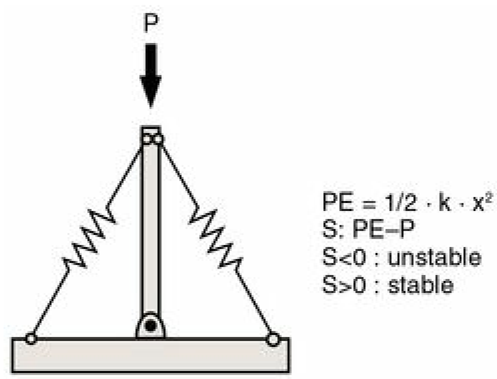


Figure 5.7 (a) Spine stiffness (and stability) is achieved by a complex interaction of stiffening structures along the spine and (b) those forming the torso wall. Balancing stiffness on all sides of the spine is more critical to ensuring stability than having high forces on a single side.

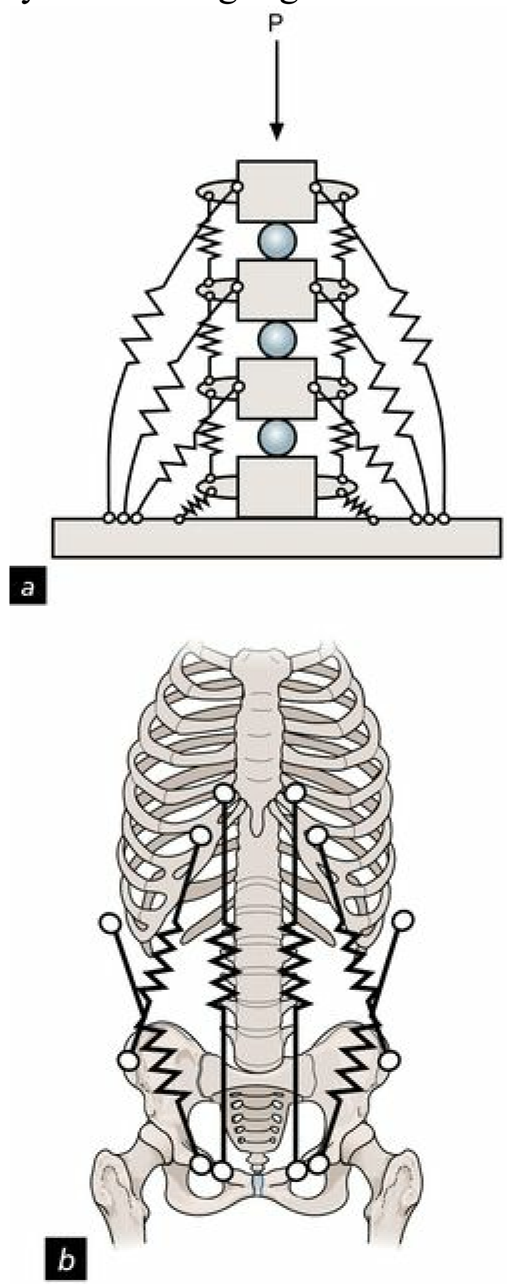
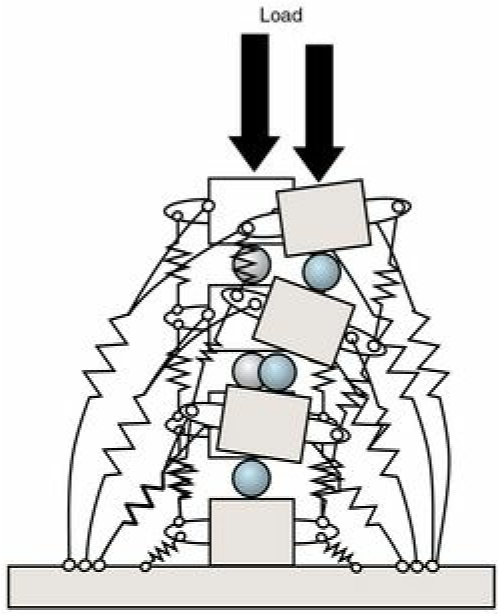


Figure 5.8 If more joules of work are performed on the spine than there are joules of potential energy due to stiffness, the spine will become unstable. In this case, the instability mode is buckling.



Active muscle acts like a stiff spring; and in fact, the greater the activation of the muscle, the greater this stiffness. Hoffer and Andreassen (1981) showed that joint stiffness increases rapidly and nonlinearly with muscle activation such that only very modest levels of muscle activity create sufficiently stiff and stable joints. Brown and McGill documented the presence of this phenomenon in the muscularly stiffened spine (Brown and McGill, 2008, 2009, 2010). Furthermore, joints possess inherent joint stiffness because the passive capsules and ligaments contribute stiffness, particularly at the end range of motion. The motor control system is able to control the stability of the joints through coordinated muscle coactivation and to a lesser degree by placing joints in positions that modulate passive stiffness contribution. However, a faulty motor control system can lead to inappropriate magnitudes of muscle force and stiffness, allowing a valley for the ball to roll out or, clinically, for a joint to buckle or undergo shear translation. However, we are limited in our ability to analyze the local stability of mechanical systems and particularly musculoskeletal linkages, because the energy wells are not infinitely deep and the many anatomical components contribute force and stiffness in synchrony to create surfaces of potential energy in which many local wells exist. Thus, we locate local minima by examining the derivative of the energy surface. (See Bergmark, 1987; Cholewicki and McGill, 1996; and Ikeda and McGill, 2013, for mathematical detail.)

We quantify spine stability, then, by forming a matrix in which the total stiffness energy for each degree of freedom of joint motion is represented by a number (or eigenvalue); the magnitude of that number represents its contribution to forming the height of the bowl in that particular dimension. Eigenvalues less than zero indicate the potential for instability. The eigenvector (different from the eigenvalue) can then identify the mode in which the instability occurred, whereas a sensitivity analysis is used to reveal the possible contributors to unstable behavior. Gardner-Morse, Stokes, and Laible (1995) initiated interesting investigations into eigenvectors by predicting patterns of spine deformation due to impaired muscular intersegmental control. Their question was Which muscular pattern would have prevented the instability? Crisco and Panjabi (1992) began investigations into the contributions of the passive tissues. Our group has investigated the eigenvector by systematically adjusting the stiffness of each muscle and assessing stability in a variety of tasks and exercises. The contributions of individual muscles to stability are shown later in figure 5.9.

Activating a group of muscle synergists and antagonists in the optimal way now becomes a critical issue. In clinical terms, the full complement of the stabilizing musculature must work harmoniously to ensure stability, the generation of the required moment, and the desired joint movement. But only one muscle with inappropriate activation amplitude may produce instability, or at least unstable behavior could result from inappropriate activation at lower applied loads.

Muscles Create Force and Stiffness

When a muscle contracts, it creates both force and stiffness. Although stiffness is always stabilizing, force may stabilize or destabilize. The relationship between increasing force in a muscle and the corresponding stiffness is quite nonlinear (Brown and McGill, 2010), given that large increases in stiffness occur quite early as activation begins (Brown and McGill, 2008). Thus, as a muscle becomes more active, it usually adds to spine stability; but if the force keeps rising, little additional stiffness is created and the force of the muscle becomes large enough to actually induce buckling of the spine (Brown and McGill, 2005). This is more evidence for the wisdom of clinically enhancing spine stability with the objective of balance in stiffness and force in all contributing muscles rather than focusing on a single muscle group.

Sufficient Stability

How much stability is necessary? Obviously, insufficient stiffness renders the joint unstable, but too much stiffness and coactivation imposes massive load penalties on the joints and prevents motion. We can define sufficient stability as the muscular stiffness necessary for stability, with a modest amount of extra stability to form a margin of safety. Interestingly, given the rapid increase in joint stiffness with modest muscle force, large muscular forces are rarely required.

Cholewicki's work (Cholewicki and McGill, 1996; Cholewicki, Simons, and Radebold, 2000) demonstrated that, in most people with an undeviated spine, modest levels of coactivation of the paraspinal and abdominal wall muscles result in sufficient stability of the lumbar spine. This means that people, from patients to athletes, must be able to maintain sufficient stability in all activities—with low but continuous muscle activation. Thus, maintaining a stability margin of safety when performing tasks, particularly the tasks of daily living, is not compromised by insufficient strength but probably by insufficient endurance. We are now beginning to understand the mechanistic pathway of those studies, which show the efficacy of endurance training for the muscles that stabilize the spine. Having strong abdominal muscles does not necessarily provide the prophylactic effect that many hoped for. However, several works suggest that muscular endurance reduces the risk of back troubles (Biering-Sorensen, 1984). Finally, the Queensland group (e.g., Richardson et al., 1999) and several others (e.g., O'Sullivan, Twomey, and Allison, 1997; Silfies et al., 2009) noted the disturbances in the motor control system following injury (detailed in chapter 4). These disturbances are not primarily in one muscle but are widespread and compromise the ability to maintain sufficient stability. In summary, stability comes from stiffness, passive stiffness is lost with tissue damage, and active stiffness throughout the range of motion is lost with perturbed motor patterns following injury.

Stability Myths, Facts, and Clinical Implications

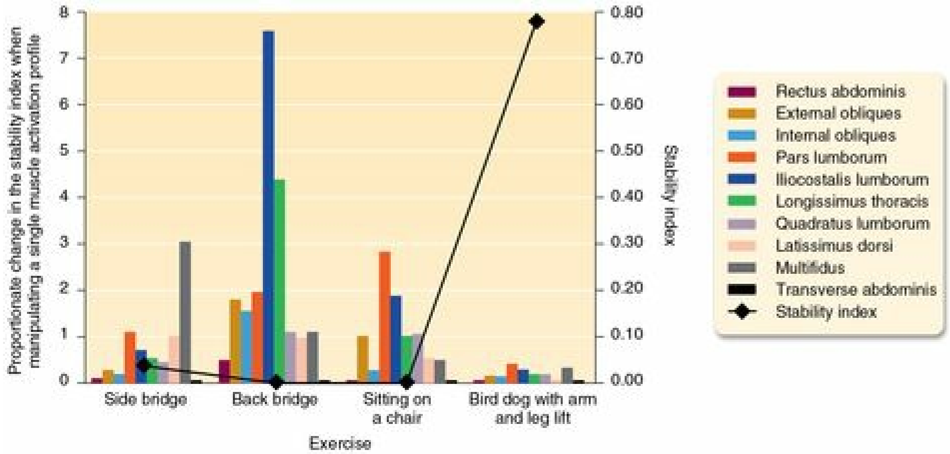
Having explored some of the issues of spine stability, we now consider a few crucial questions that will enhance clinical decisions.

- How much muscle activation is needed to ensure sufficient stability? The amount of muscle activation needed to ensure sufficient stability depends on the person and the task. For normal people walking, the normal muscular patterns are sufficient. A person in pain may need additional muscular stiffness to control the joint micromovements. Generally, for most tasks of daily living (e.g., holding and carrying loads), very modest levels of abdominal wall cocontraction (activation of about 10% of maximal voluntary contraction or even less) are sufficient. Again, depending on the task, cocontraction with the extensors (this also includes the quadratus) will ensure stability. However, if a joint has lost stiffness because of damage, more cocontraction and stiffness is needed to control pain-inducing joint micromovements. A specific example is shown in chapter 10.
- Is any single muscle most important? Several clinical groups have suggested focusing on one or two muscles to enhance stability. This would be similar to emphasizing a single guy wire in the fishing rod example. Rarely would it help; rather, it would be detrimental to achieving the balance in stiffness needed to ensure stability throughout the changing task demands. In particular, clinical groups have emphasized the multifidus and transverse abdominis. The Queensland group performed some of the original work emphasizing these two muscles. This was based on their research noting motor disturbances in these muscles in some people following injury. In fact, they developed a tissue damage model suggesting that chronically poor motor control (and motion patterns) initiates microtrauma in tissues that accumulates, leading to symptomatic injury (Richardson et al., 1999). Further, according to the Queensland model, injury leads to further deleterious changes in motor patterns such that chronicity can be broken only with specific techniques to reeducate the local muscle–motor control system. The intentions of the Queensland group were to address the documented motor deficits and attempt to reduce the risk of aberrant motor patterns

that could lead to the pathology-inducing patterns in their damage model. In other words, their recommendations appear to be directed toward reeducating the faulty motor patterns. However, many clinical groups have interpreted this approach to mean that these two muscles should be the specific targets when teaching stability maintenance over all sorts of tasks. This is problematic.

Hopefully, the understanding obtained from the stability explanation provided earlier will underscore the folly of this unidimensional emphasis. In fact, the multifidus or any other muscle can be the most important stabilizer at an instance in time. Four exercises are presented as an example (see figure 5.9) in which the contributors to stability are shown. The importance of individual muscles will change and adjust with subtle changes in relative muscle activation. In these examples the adjustments of all the laminae in each whole muscle are averaged to evaluate the entire muscle's role. Obviously, adjustments in a single laminae of some muscles would result in yet a different distribution of contributions to stability. Evaluating a wide variety of tasks and exercises is most revealing. The muscular and motor control system must satisfy requirements to sustain postures, create movements, brace against sudden motion or unexpected forces, build pressure, and assist challenged breathing, all the while ensuring sufficient stability. Virtually all muscles play a role in ensuring stability, but their importance at any point in time is determined by the unique combination of the demands just listed (Kavcic and McGill, 2004). Every study that has actually quantified spine stability has reached the same conclusion—all muscles are important. This implies that a clinical focus on one muscle and not the whole system is detrimental. In some instances, the performance of one muscle enhances another (e.g., the rectus abdominis assists the obliques for stabilization). Further, evidence has demonstrated that endurance in these muscles is necessary to facilitate the continuous low-level stiffening contractions required for the maintenance of sufficient stability (e.g., Wagner et al., 2005).

Figure 5.9 Measuring individual muscle influence on lumbar spine stability during the performance of four stabilization exercises demonstrates that all muscles are important and that their relative importance changes with the task. Total stability is also indicated by the determinant, demonstrating how stability can change between tasks. (This is an example from a single subject. Typically, group means indicate a different stability ranking of exercises.)



Based on N. Kavcic, S. Grenier, and S.M. McGill, 2004, "Determining the stabilizing role of individual torso muscles during rehabilitation exercises," Spine 29(11): 1254-1265.

As noted elsewhere in this book, focus on a single muscle such as transverse abdominis (TvA) does not enhance stability or affect many people's back pain There is a disconnect between the science and clinical practice regarding this muscle. Many clinicians have been taught to have all patients with back pain begin isolation training of TvA. Some patients may have very recognizable disturbances in motor patterns, yet these are ignored with an overzealous focus on TvA. For example, a patient may present with poor posture causing chronic muscle activity when loading the spine in compression. He may be bending painful discs because of his movement choices. Yet some clinicians fail to see these overt pain mechanisms and begin all back pain patients with isolated TvA training. Simple movement and posture training could have relieved the pain immediately. Perhaps even more disturbing is the number of high-performance coaches from around the world who instruct their athletes to draw their navels toward their spines to activate TvA, which they claim enhances stability while training or performing. Drawing the navel toward the spine reduces stability; increasing the distance between the abdominal wall and the spine enhances stability. A major question that must be asked is this: Is there even a disturbance in TvA?

In most people with back pain, I believe many other more important disturbances should be addressed. Efficacy studies support this view (e.g., Allison, 2012; Brooks, Kennedy, and Marshall, 2012; Hebert et al., 2010; Unsgaard-Tondel et al., 2012; Vasseljen et al., 2012; Wong et al., 2013).

Although much of the TvA focus was motivated by reports of onset delays in rapid arm movements, finding patients with a delayed TvA is very difficult. Several studies have failed to find any systematic TvA delay in patients with nonspecific back pain (Gubler et al., 2010). The paradigm to detect a delay is a rapid arm raise. Finding delays in tasks more common to daily living would be helpful in understanding the motor control and biomechanical importance. For example, other variables of motor control disturbances are very blatant and easily measured in patients. However, the clinical community does not seem to collectively appreciate that many muscles display delays in people with back pain and that the patterns of delay among different muscles are helpful in classifying the nature of the link between motor control and pain. Silfies and colleagues (2009) showed delays in several muscles and in fact could categorize subpopulations of pained patients based on this criterion.

It is possible that some patients benefit from specific TvA training. I suspect that these are the "abdominal sloths" who do not contract their abdominal wall muscles when defecating or when performing functional tasks such as lifting or carrying. TvA training would enhance their awareness of the existence of the abdominal wall, and I have seen this and appreciate the research. We prefer teaching bracing to encourage activation of all components of the abdominal wall; this immediately reduces pain in many patients (Ikeda and McGill, 2012). It is very stabilizing. Consider the heel drop test that initiates pain. Repeating with a tuned abdominal brace often eliminates pain. Obviously, the brace contraction is then tuned to optimize stability, reduce spine load, and reduce pain. The work of Koumantakis, Watson, and Oldham (2005) suggested that in the average painful back, specific TvA training delayed recovery until full abdominal wall work was undertaken. We suggest instead that patients simply perform stabilization training of the many muscles around the torso.

One final issue is whether the mechanics of TvA should be documented with ultrasound-derived data, as shown on EMG. Our data suggest that they should not. The link between TvA muscle thickening and activation is obscured by the composite structure of the three layers of the abdominal wall

(Brown and McGill, 2008, 2010) such that only EMG data can link activation with force and, with additional analysis, stiffness.

Some clinical issues remain and are addressed now:

- Do local and global or intrinsic and extrinsic stabilizers exist? From the classic definition and quantification of stability, the answer is no. Stability results from the stiffness at each joint in a particular degree of freedom. The relative contribution from every muscle source is dynamically changing depending on its need to contract for other purposes. The way the various contributors to stiffness add up, however, is important. In some instances, removing a muscle from the analysis has very little effect on joint stability. Once again, it depends on the demands and constraints unique to the task at that instant. The point is that all contributors are important for some tasks and should be recognized as potentially important in any prevention or rehabilitation program. Conversely, conceiving of stabilizers as intrinsic or extrinsic may offer no benefit for clinical decision making.
- What are stabilization exercises? Stabilization exercises and a stable core are often discussed in exercise forums. What are stabilization exercises? The fact is that any exercise can be a stabilization exercise depending on how it is performed. Sufficient joint stiffness is achieved through the creation of specific motor patterns. To adapt a phrase popular in motor control circles, Practice does not make perfect; it makes permanent. Ideally, good stabilization exercises that are performed properly groove motor and motion patterns that ensure stability while satisfying all other demands. Thus, an exercise repeated in a way that grooves motor patterns and ensures a stable spine constitutes a stabilization exercise (McGill, 2001). However, some stabilization exercises are better than others; again, it depends on the objectives. For example, the resultant load on the spine is rarely considered. One key to improving painful backs is to select stabilization exercises that impose the lowest load on the damaged spine. Chapter 4 provides a ranking of such exercises. The big three exercises have been shown to create a lasting stiffness and stability (Lee and McGill, 2014a, 2014b) in a spine-sparing way. On the other hand, the best stabilization exercise for a high-performance athlete will involve the establishment of dynamic and complex motion patterns, all the while ensuring sufficient

spine stability. An important requirement for many athletes is to maintain a stable spine while breathing hard, such as when playing high-intensity sports. This is discussed in chapter 11.

- Can clinicians identify those who are poor stabilizers? From a qualitative perspective, the answer is yes, as long as the clinician is cued into seeing and feeling compromised motion and muscle activation patterns. From a quantitative perspective, we have used modeling analysis to find ways to identify those who compromise their lumbar stability via specific motor control errors. We observed such inappropriate muscle sequencing in men who were challenged by holding a load in the hands while breathing 10% CO_2 to elevate breathing. On one hand, the muscles must cocontract to ensure sufficient spine stability; but on the other, challenged breathing is often characterized by a rhythmic contraction–relaxation of the abdominal wall (McGill, Sharratt, and Seguin, 1995). Thus, the motor system is presented with a conflict: Should the torso muscles remain active isometrically to maintain spine stability, or will they rhythmically relax and contract to assist with active expiration (but sacrifice spine stability)? Fit motor systems appear to meet the simultaneous breathing and spine support challenge; unfit ones may not (Grenier and McGill, 2007, 2008; Wang and McGill, 2008). All of these deficient motor control mechanisms heighten biomechanical susceptibility to injury or reinjury (Cholewicki and McGill, 1996). We are currently using this paradigm in a longitudinal study to see whether those who border on instability during this challenge will be the ones who develop low back troubles. Some other tests to detect functional instability are described in chapter 9.

A Final Note

In summary, achieving stability is not just a matter of activating a few targeted muscles, be they the multifidus, transverse abdominis, or any other. Sufficient stability is a moving target that continually changes as a function of the three-dimensional torques needed to support postures. It involves achieving the stiffness needed to endure unexpected loads, preparing for moving quickly, and ensuring sufficient stiffness in any degree of freedom of the joint that may be compromised from injury. Thus, this stiffness is tuned to the situation with a skilled level of coordinated muscular cocontraction. Motor control fitness is essential for achieving the stability target under all possible conditions for quality performance and injury avoidance. Finally, the terms stability and stabilization exercise have been used by many to mean different things. When one trial reports no efficacy for stabilization exercise, beware, read carefully, and determine whether the exercises were truly consistent with bonafide stability.

Part II
Injury Prevention

In part I we went back to school for an update on lumbar function. In part II this foundation is used to justify the best injury prevention approaches. The next two chapters discuss multiple models for risk assessment and ways to reduce the risk of low back injury.

Chapter 6
LBD Risk Assessment

Two types of physical risk factors predispose people to developing low back disorders (LBD): those linked to the person (e.g., movement competency and muscle endurance or the lack thereof) and those linked to the demands of performing a certain task (e.g., applied loads). This chapter describes how to assess the risk of back disorders that result from task demands.

Upon completion of this chapter, you will understand the variables that are important to include for risk assessment and be familiar with tools used to assess the risk. These tools are the NIOSH approach, the Snook psychophysical approach, the lumbar motion monitor (LMM) approach, and the Ergowatch approach.

Tissue overload causes damage and subsequent low back troubles. Assessing the risk involves comparing applied loads with some form of load-bearing tolerance value. Chapter 4 included examples of applied loads, tissue loads, and injury scenarios. Although direct measurement of tissue loads would be ideal, this remains impractical for large numbers of people performing a wide variety of occupational tasks and activities of daily living. Researchers have developed modeling approaches to predict these loads, but they remain methodologically complex. For this reason, surrogates for tissue load that are linked with posture, applied load, and motion have been proposed. These were introduced in chapter 2. The first section of this chapter, Brief Review of the Risk Factors for LBD, provides a list of known risk factors that includes descriptions and critiques of several approaches to assessing them.

Four risk assessment approaches are presented more or less from least complex to most complex. The simplest approaches use metrics that are the easiest and cheapest to employ for injury risk assessment. However, these are compromised by a lack of accuracy and specificity and are not sensitive to individual worker or person techniques. The more complex approaches involve more sophisticated methods and are more expensive to conduct, but they are more robust in their sensitivity to specific tasks and the unique ways people elect to perform them.

Brief Review of the Risk Factors for LBD

Here are the risk factors for LBD that have been identified from epidemiological approaches:

- Static work postures, specifically prolonged trunk flexion and a twisted or laterally bent trunk posture
- Seated working postures
- Frequent torso motion, high spine rotational velocity, and spine rotational deviations
- Frequent lifting, pushing, and pulling
- Vibration exposure, particularly seated whole-body vibration
- Peak and cumulative low back shear force, compressive force, and extensor moment
- Slips and falls

The following risk factors have been identified from tissue-based studies:

- Repeated full lumbar flexion
- Time of day (or time after rising from bed)
- Excessive magnitude and repetition of compressive loads, shear loads, and torsional displacement and moments
- Insufficient loading so that tissue strength is compromised
- Rapid ballistic loading (such as that resulting during landing from a fall)

And finally, these personal variables have been identified as risk factors:

- Increased spine mobility (range of motion)
- Lower torso muscle endurance
- Perturbed or awkward motor control patterns
- Age
- Gender
- Abdominal, or torso, girth

NIOSH Approach to Risk Assessment

For about the past 40 years both field surveillance and laboratory studies have focused on the relationship between back injury and compressive forces experienced by the lumbar spine. For example, in 1981 the National Institute for Occupational Safety and Health (NIOSH) in the United States published guidelines for maximal compressive loading of the lumbar spine (both an action limit of 3,400 N and a maximal permissible limit of 6,300 N) based on some earlier evidence. In 1994 Leamon questioned the use of compression when he quite correctly stated that the supporting evidence for compression as a metric, or index of risk, was sparse. However, since that time, several good data sets have shown that restricting the amount of low back compression (both peak and cumulative) is one valid approach to reducing the risk of low back injury (e.g., data sets in the studies by Norman et al., 1998, and Marras et al., 1995). In addition, NIOSH has also proposed lifting guidelines to limit the amount of load lifted in the hands. These limits remain popular and are described in the following sections.

The influence of NIOSH has been far-reaching; many groups have used these values to reduce the risk in workers. In fact, the NIOSH approach continues to be widely used today primarily because of its ease of use.

1981 Guideline

The first lifting guide (NIOSH, 1981) was restricted to lifts in the sagittal plane and lifts that involve only slow and smooth motions. Predictions of the load lifted in the hands were based on some rudimentary distances to characterize the kinematics of the lift and load moment at the low back. Two limits were defined: the action limit (AL), which, if exceeded, triggered action to apply engineering and administrative controls, and the maximal permissible limit (MPL) for the load lifted, above which the risk is too high and not permissible. Weights lighter than the AL are considered safe. Those with biomechanical, physiological, and psychophysical expertise chose the magnitude of these limits and the variables needed to compute them. The general form of the formula used to compute the AL for weight in the hands is as follows:

$$AL\ (kg) = 40\ (HF)(VF)(DF)(FF)$$

where

- HF = horizontal factor, or the horizontal distance (H) from a point bisecting the ankles to the center of gravity of the load at the lift origin. This was defined as (15 / H).
- VF = vertical factor, or the height (V) of the load at lift origin. This was defined as (0.004) / (V – 75).
- DF = distance factor, or the vertical travel distance (D) of the load. This was defined as 0.7 + 7.5 / D.
- FF = frequency factor, or the lifting rate defined as 1 – F / Fmax.
- F = average frequency of the lift, whereas Fmax is obtained from tabulated data.

The logic of the equation is to set a maximal load of 40 kg (88 lb) and multiply this value against variables that act as discount factors. Thus, the maximal lift is 40 kg (88 lb) under optimal conditions and when all discount variables equal unity (1). Suboptimal lifting conditions cause the discount multipliers to drop to smaller values, reducing the safe load. The MPL is computed as three times the AL for a particular set of lifting circumstances:

$$MPL = 3(AL)$$

1993 Guideline

The revised NIOSH equation (Waters et al., 1993) was introduced to address those lifting tasks that violated the sagittally symmetrical lifting task restriction of the earlier equation. In addition, the concepts of the AL and MPL were replaced with a recommended weight limit (RWL) for a particular situation. If the actual load to be lifted exceeds the RWL, the risk of developing an LBD is elevated. Although the RWL equation is similar to the 1981 equation, two additional factors were incorporated: an asymmetrical variable for nonsagittal lifts and a factor for whether the object has handles. Note that the variable weightings also changed from the 1981 equation form.

$$RWL\ (kg) = 23(25 / H)[1 - (0.003 / V - 75)]$$

$$3\ [0.82 + 4.5 / D])(FM)[1 - (0.0032A)](CM)$$

where

- H = horizontal location forward of the midpoint between the ankles at the origin of the lift. If significant control is required at the destination, then H should be measured both at the origin and destination of the lift.
- V = vertical location at the origin of the lift.
- D = vertical travel distance between the origin and destination of the lift.
- FM = frequency multiplier is obtained from a table supplied by NIOSH.
- A = angle between the midpoint of the ankles and the midpoint between the hands at the origin of the lift.
- CM = coupling multiplier ranked as good, fair, or poor. It is obtained from a table.

The 1993 equation predicts smaller loads that can be lifted safely, when compared to the 1981 equation, and is thus more conservative. Interestingly, NIOSH offered no provision for the difference in capacities of men and women; they are treated similarly largely for political reasons. (I discuss this issue of discrimination and the impact on protecting vulnerable workers in chapters 1 and 2.) Further, the approach ignored any individual differences in body mechanics used during lifting. In addition, some have suggested that the handle factors may not be consistent with the subsequent forces endured by the body. For example, although NIOSH assumed that having handles on the lifted object is better, our work has shown that handles allow the lifter to apply even more force, resulting in subsequently higher back loads (Honsa et al., 1998)!

Nonetheless, the equations form a rudimentary approach to risk assessment that is easy to conduct. Marras and colleagues (1999) concluded that the 1981 NIOSH guide identified low-risk jobs well (specificity of 91%) but did not predict the high-risk jobs well. The 1993 guide correctly identified 73% of the high-risk jobs but did not identify the low- and medium-risk jobs well. Overall, both NIOSH guides predicted jobs resulting in LBD with odds ratios between 3.1 and 4.6. Marras and colleagues (1999) noted that the most powerful variable was average low back moment (load in hands and the horizontal distance between the anteriorly placed load from the spine).

Subsequent work from the Marras lab (Ferguson, Marras, and Burr, 2005) has added perspective to the interpretation of the NIOSH approach. The researchers measured populations of workers with low back pain and age-matched controls and noted that lifting from the floor is problematic for many workers, in particular those with back pain. This has implications for return-to-work programs for people with injured backs. Further, their data have strengthened the growing impression that shear forces on the spine are probably more important than compression in determining injury risk. In fact, 74% of their high-risk tasks were categorized based on excessive lumbar shear. Norman and colleagues (1998) made similar observations.

Potvin (2014) revisited the original criteria on which the revised equation was based and concluded that the equation is extremely conservative. For example, the RWL results in lumbar compressive forces that are far too low when lifting above knuckle height. Similarly, the equation RWLs are acceptable to 95% of females, which far exceeds the stated objectives when originally proposed. In summary, he found the RWL to be an average of 63% of the calculated composite acceptable loads based on the integration of the psychophysical and physiological criteria. Put another way, one would have to multiply the RWL by 1.65 to meet the original criteria put forth by NIOSH.

Snook Psychophysical Approach

The psychophysical approach to setting load, or work limits, is based on people's beliefs of what constitutes a tolerable work rate. The main criticism of this approach is that most people perceive physiologically related discomfort and not the internal loading on tissues that actually causes damage. For example, Dul, Douwes, and Smitt (1994) made a case that muscle discomfort, which is readily perceived, may not correlate with actual tissue loads. Workers may not be cognizant of the tissue loads as they reach damaging levels (McGill, 1997). Furthermore, Karwowski and colleagues (Karwowski, 1991, 1992; Karwowski and Pongpatanasuegsa, 1989) noted in a series of studies that the general psychophysical approach is very dependent on such factors as the instructions provided to the experimental subjects and the color of the object being lifted, to name a couple. For example, the Snook studies (1978) advised the workers to determine a work rate and load to be moved based on the instruction not to become unduly fatigued. The workers selected the loads they perceived not to be fatiguing. Yet when Karwowski repeated the experiments but changed the instruction to lift loads so that subjects would not become injured, the acceptable loads changed (they chose smaller loads). Karwowski and Pongpatanasuegsa (1989), when evaluating the effect of the color of the boxes being lifted, noted that workers would lift more when the boxes were white, because they perceived the black boxes as being denser. Recent work by my colleague Mardy Frazer (unpublished) suggests that females base their perceptions of task-limiting loads more on shoulder discomfort than on spinal indicators. Males, on the other hand, base their perception on low back limitations. The psychophysical approach appears to depend on many factors that modulate perception.

The work of Snook (Snook, 1978; Snook and Ciriello, 1991) is probably the most widely recognized in the psychophysics area. The investigators experimentally controlled variables such as object size, lift height, and movement distance in lifting and pushing and pulling tasks for both men and women. They constructed tables compiling the acceptable loads for both men and women over a variety of tasks. On one hand, this approach inherently incorporates the dynamics of the tasks, whereas on the other hand, the concerns raised in the previous paragraph are worth considering. Nonetheless, the Snook approach remains popular for its ease of implementation and because it is one of the few assessment methods for pushing and pulling tasks.

Recent analysis has expanded the utility and interpretation of the Snook tables. Potvin (2012a) assessed the existing psychophysical data in a meta-analysis and noted that a strong, negative, exponential relationship exists between duty cycle and maximal acceptable effort (normalized to maximal voluntary effort). This means that the more time spent performing a repetitive task without rest greatly reduced perceived tolerance. No agreement exists on constant contractions with no rest. The equation originally developed for upper-extremity work, and subsequently confirmed for lifting and lowering tasks (hence the back; Potvin, 2012b), was as follows:

$$MAE = 1 - [DC\text{-}1 / 28{,}800]^{0.24}$$

where

- MAE = average maximum acceptable effort, where 1.0 represents 100% MVE.
- DC = duty cycle, where 1.0 represents 100% of the cycle.
- 28,800 = number of seconds in 8 hours (its inverse is 0.0000347 and, when subtracted from DC, results in MAE = 1.0(100%) for all DC $\leq$ 1 s/workday).

The best fit exponent of 0.24 well predicts the MAE ($r^2 = 0.87$).

Lumbar Motion Monitor (LMM)

Several studies have documented the links between spine motion and the development of LBD, but none more thoroughly than those of Marras and colleagues (Marras, 2008; Marras et al., 1993, 1995). They developed several types of regression models that demonstrated that spine motion and, in particular, the velocity of motion and the range of motion are important predictors of those jobs with high rates of disorders (in fact, some odds ratios exceeded 10). The lumbar motion monitor is a three-dimensional goniometer that measures the three-dimensional kinematics of the lumbar spine (see figure 6.1). Marras and colleagues showed that the kinematic variables obtained from workers wearing the LMM, when combined with simple measurements of lift frequency or motion duty cycle and load moment (the load magnitude multiplied by the distance to the low back), provide impressive risk predictions. Using the LMM on people while they are performing real on-site tasks and not laboratory mock-ups is relatively easy to do. In addition, the LMM captures the unique ways people use and move their spines. See chapter 4 for a discussion of the mechanisms of injury that depend on spine posture and motion.

Figure 6.1 Wearing the lumbar motion monitor to record the three-dimensional kinematics of the lumbar spine during the performance of industrial tasks.

Courtesy of my good friend Professor Bill Marras.

Ergowatch

For approximately four decades, biomechanical models have been used to estimate loads in the low back tissues and identify high-risk jobs. Some models were intended as simple tools for health and safety personnel to use to approximate the index of injury risk on the plant floor. Other models were designed to be more robust in illustrating injury mechanisms while being sensitive to worker variance. Decisions as to which model to use boil down to the purpose and complexity of the model.

The better simple models need the complex models to assess the many simplifying assumptions that affect accuracy and output validity, which in turn depend on the type of application. Further, in many workplaces, the most blatant or overt ergonomic injury risks have been addressed, and only the more subtle risks remain. Ergonomists need simple models but also must be conversant with the more complex models that will assist in rectifying the subtler injury risks and in developing more effective intervention strategies.

Although simple biomechanically based models designed to obtain quick estimates of low back compression exist, risk assessment for most jobs requires more complex metrics and analysis approaches. To optimize biofidelity and industrial utility, Bob Norman, Mardy Frazer, Rich Wells, and I developed the software package Ergowatch. It uses the three-dimensional moments computed at joints while workers perform three-dimensional postures or tasks together with the fourth dimension of time and the effect of repetition on determining the safe load. (It is available by contacting Bbutler@uwaterloo.ca). The package uses the detailed output of the virtual spine (described in chapter 1) as a foundation, but makes assumptions to simplify data collection and facilitate routine analysis. In this way, individual worker behavior and spine mechanics are quantified but simplified into average muscular responses seen in workers performing similar tasks. This greatly simplifies data collection and preserves the benefits of better anatomical representation and more valid predictions of low back loads. Furthermore, by incorporating the injury data obtained from samples of workers, Ergowatch quantifies the risk of back injury during fully three-dimensional tasks and postures. The fourth dimension is time, in this case a variable needed to take into account the repetition of tasks over a work shift.

To use the Ergowatch, the user enters joint coordinate data manually or manipulates an on-screen mannequin into the work posture of interest. The software executes the difficult task of determining the three-dimensional joint moments by computing the Euler angles and transforming them into moments about the orthopedic axes of each joint. In this way, the package is capable of calculating joint loads in any posture and for any combination of lift, lower, push, or pull task. It also incorporates algorithms that capture the average muscular response measured from workers to support three-dimensional spine moments of force together with strength data for both men and women. As well, the model is sensitive so spine posture in that ligament and passive tissue forces are accounted for during fully flexed spinal postures. Thus, the model accurately predicts low back shear forces supported by the spine (as compared to reaction shear forces predicted by other models), as well as compression forces that result from the load and the many torso muscles contracting to support a particular set of three-dimensional moments (see figure 6.2, a and b) (note that shear more often is the variable closest to tolerance). Although the upper limit for shear has been suggested to be 1,000 N, the equivalent action limit for repeated loading has been set at 500 N (McGill et al., 1998, from tissue-based data; and Norman et al., 1998, using industrial injury reporting data). Finally, the risk is also calculated from accumulated loads during repetitive work via comparison of the load–time integrals with epidemiological data obtained from a large surveillance study conducted in an automotive assembly plant (Norman et al., 1998). (See figure 6.3, a-d.)

Figure 6.2 Ergowatch computes lumbar compressive load from an algorithm representing the average response of the many muscles, measured from real workers, that combine to support the infinite combinations of three low back moments: flexion–extension, lateral bend, and axial twist. Because only three dimensions can be graphed at one time, (a) shows the surface of lumbar compression from combinations of flexion–extension and twist moments, and (b) shows the surface of compression from flexion–extension and lateral bend.

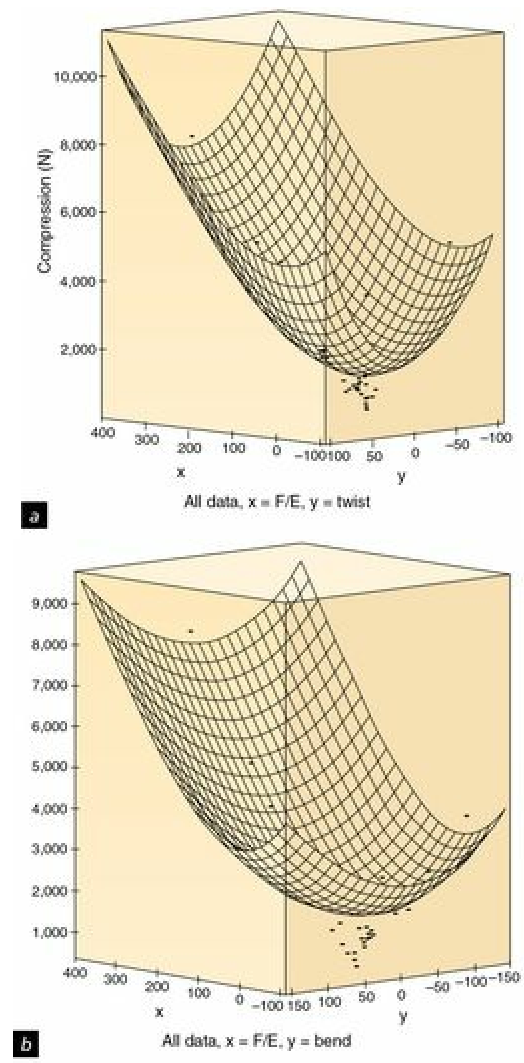
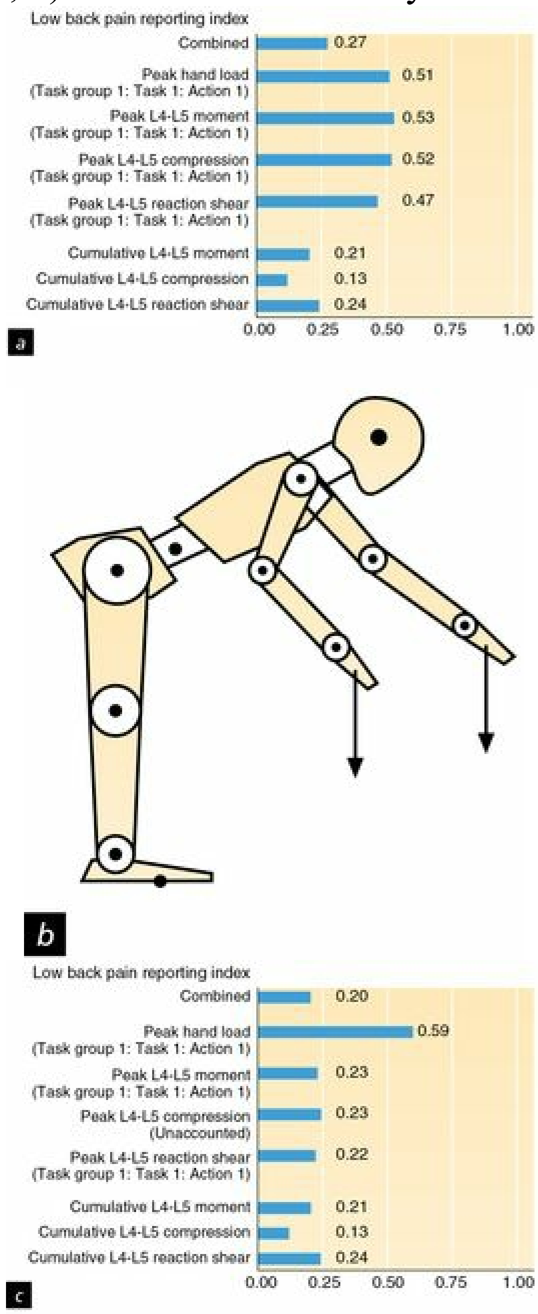
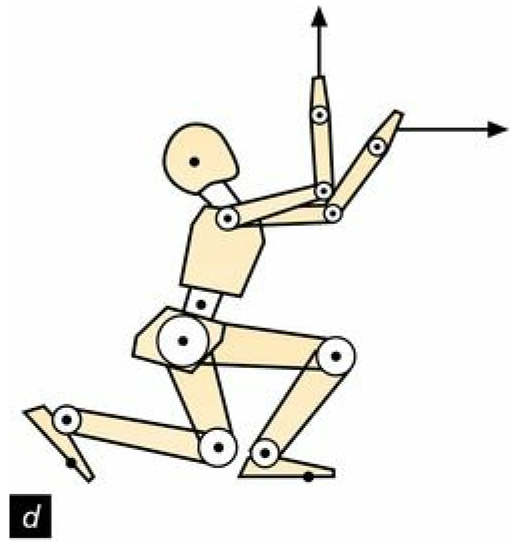


Figure 6.3 Ergowatch computes the joint-specific, three-dimensional orthopedic moments at each joint and uses several metrics to calculate risk. For example, the low back risk includes compression and shear load threshold values along with accumulated loads during repetitive work by comparing the load–time integrals with epidemiological data obtained from a large surveillance study. Two examples are shown: (a, b) a reaching lift and (c, d) an overhead assembly task.





The Ergowatch approach represents a compromise between simple models that are easy to implement but are not very robust in their risk assessment and more complex approaches. Ergowatch is still relatively easy to use, and it provides some degree of sensitivity to the way individual workers perform complex tasks. It uses many risk indexes to quantify the subsequent risk from a specific task performed over a work shift.

Euler Angles and Orthopedic Moments

Estimations of three-dimensional joint moments are typically performed using three orthogonal axes (XYZ). But as a person moves, the orthopedic axes of the joints (e.g., in the lumbar spine, axis 1—flexion–extension, axis 2—lateral bend, axis 3—axial twist) also move so that they no longer align with the inertial XYZ axes. The difference between the orthopedic joint axes and the XYZ axes is described with Euler angles. The orthopedic moments are then obtained from the E-vector approach described by Grood and Suntay (1983). This involves taking the cross-product of the long axis (the primary axis, usually twist) of two adjacent segments to form the secondary axis (usually flexion–extension). The cross-product of the primary and secondary axes forms the E-vector, which in turn becomes the third joint orthopedic axis (usually lateral bend). Moments computed about axes XYZ can now be transformed into joint-specific orthopedic axes. This technique ensures that the joint axes coordinate system "stay with the joint" as the person moves within the inertial coordinate system.

Biological Signal–Driven Model Approaches

The final approach for risk assessment is to measure biological signals from each subject to capture the unique ways people perform their jobs and then use sophisticated anatomical, biomechanical, and physiological relationships to assign forces to the tissues.

Marras Model and McGill Model

The Marras model (Marras and Sommerich, 1991a, 1991b) measures electromyograms (EMGs) from several muscles and, using known physiological relationships, assigns forces to the muscles during virtually any industrial task. As noted earlier, this approach revealed powerful evidence linking the physical demands of specific occupational tasks with the incidence of LBD.

The McGill model (see chapter 1) uses the same philosophical approach to assign forces to the muscles, but attempts to include the highest level of anatomical accuracy possible. For example, it also measures three-dimensional spine curvature to assess forces to the passive tissues, including the intervertebral discs and the ligaments. By assigning forces to muscles and passive tissues throughout the full range of spine motion, it captures the ways people perform their jobs and even how they change with repetitions of the same jobs. The obvious liability of the approach is its enormous computational requirements, postprocessing of data, and difficulty collecting such comprehensive data from a wide number of workers in the field.

The question of the validity of this type of model must be addressed along with other models based on the biological approach. Some have argued that because these models contain known biomechanical and physiological relationships, they contain a certain amount of content validity. Moreover, both the Marras model and the McGill model have been quite successful in estimating the passive tissue and muscle forces that sum together to produce flexion and extension, lateral bend, and axial twisting moments. (These moments have been well predicted, with the exception of the twisting moment.) In summary, if twisting is not a dominant moment of force in a particular job, these models appear to predict accurate distributions of forces among the support tissues.

EMG-Assisted Optimization Approach

Perhaps the current pinnacle of model evolution is a hybrid modeling approach known as EMG-assisted optimization (developed by Cholewicki and McGill, 1994) with stability analysis and tissue load prediction (Cholewicki and McGill, 1996, was an early example, and Ikeda and McGill, 2012, is a more recent example). This approach exploits the asset of the biological EMG approach to distribute loads among the tissues based on the biological behavior of the subject. It then uses optimization to fine-tune the balancing of joint torques about several low back joints. The optimization takes the biologically predicted forces and adjusts muscle forces the minimal amount possible to satisfy the three-dimensional moment axes at every joint over the length of the lumbar spine. Once the three-dimensional moments have been assigned and tissue forces determined, an analysis of spine stability is performed via comparison of the joules of work imposed on the spine through perturbation with the joules of potential energy existing in the stiffened column.

This most highly evolved of spine models provides the biomechanist or ergonomist with insight into injury mechanisms caused by instability (as witnessed by Cholewicki and McGill, 1992) such as occurs while picking up a very light load from the floor. Even though patients have reported back injury from incidents such as bending over to tie a shoe, previous modeling approaches were sensitive only to tissue damage and injury scenarios produced from large loads and moments. Now a biomechanical explanation is available to explain the subsequent tissue damage, and a method is available to detect the risk of its happening. Although the routine use of this type of sophisticated model by ergonomists is not feasible, it is useful (for trained scientists) for analyzing individual workers and identifying those who are at elevated risk of injury because of faulty personal motor patterns.

Simple or Complex Models?

In summary, the complex models provide a tool to investigate the mechanisms of injury and the effects of technique during material handling on the risk of injury. The most complex and most highly evolved models provide insight into how injury occurs with all types of heavy and light loads. On the other hand, the simpler models, although sacrificing accuracy, can be powerful tools for the routine surveillance of physical demands in the workplace, but they must be wisely interpreted in each case given their limitations and constraints. Biomechanics and ergonomics require the full continuum of models. The choice of which one to use depends on the issue in question.

A Final Note

This chapter summarized and critiqued several approaches for their assets and liabilities. The choice of the most appropriate tool depends on the objective at hand. Yet many ergonomists and industrial engineers believe that minimal tissue loading is best for all jobs, motivating them to direct their interventions toward making all jobs easier. This concept is faulty. Biological tissues require repeated loading and stress to be healthy. Virtually all risk assessment tools consider only the risk of too much load. Risk assessment tools that survey too little loading for optimal health are just starting to emerge. The challenge is to develop a wise rest break strategy to facilitate optimal tissue adaptation. More robust assessment tools will be developed in the future based on the healthiest combination of work and rest. This will be achieved first through an understanding of the biomechanical, physiological, and psychological parameters of injury and human performance and then through thoughtful applications of this wisdom.

Chapter 7
Reducing the Risk of Low Back Injury

As I have suggested in previous chapters, many of the classic instructions from experts about lifting (e.g., bend the knees and not the back) are in most cases neither feasible nor justifiable. In fact, very few occupational lifting tasks can be performed this way. The resulting tragedy is that clinicians or other experts lose credibility in the eyes of the worker; the worker knows she cannot do her job that way. She lifts objects from the floor, out of parts bins, over tables, and so forth. The science shows that squatting can have an increased physiological cost and that, depending on the characteristics of the load, it may not even reduce loads. Squatting is not always the best choice of position; it depends on the specifics of the task (e.g., the size, weight, and density of the object; the pick and place location; the number of repetitions). For example, the golfer's lift (see figure 7.6 later in this chapter) may be preferable for reducing spine loads to bending the knees and keeping the back straight for someone repeatedly lifting light objects from the floor.

Reducing pain and improving function for patients with low back pain involve two components:

- Removing the stressors that create or exacerbate damage
- Enhancing activities that build healthy supportive tissues

This chapter addresses the issue of prevention—specifically, reducing the overloading stressors that cause occupational low back disorders (LBDs). After reviewing lessons from the literature and presenting the scientific issues, I provide two sets of guidelines: one for workers and another for employers. Because back belts have also been a much-debated topic and the subject of legislation in some jurisdictions, a reasonably in-depth discussion is presented. Finally, I offer a few notes to enhance the effectiveness of consultants. The lists provided in this chapter will result in more successful injury prevention programs. Practitioners and consultants who employ them will stand out from their peers.

Upon completion of this chapter, you will be able to formulate scientifically based guidelines for any activity that reduce the risk of occupational low back disorders. Further, you will be aware of, and so be able to avoid, the pitfalls that cause ergonomic approaches to fail. Finally, you will be a more effective consultant by harnessing the real expertise—that of the worker.

Lessons From the Literature

Why does industry care about the backs of workers? Competitiveness in the new economy requires being profitable, and for many, the only way to enhance profit is to maximize loss control. A major source of losses to North American industry is worker injury, which results in direct costs for compensation and indirect costs of hiring and training replacement workers as well as reduced productivity because of decreased speed and increased errors. For this reason, preventing injury and promoting the rapid return to work of injured workers have become a major focus for industry. Because back injury represents an enormous cost in both real dollars and suffering, companies are realizing the benefits of supporting injury prevention and rehabilitation programs. Following are a number of studies that have addressed the issues that must be examined if such programs are to be successful.

Compensation Board Statistics—An Artifact?

When completing injury report forms, clinicians are often requested to name the event that caused the injury. These events are compiled by compensation boards to create their impressions of injury mechanisms —for example, the injured worker lifted and twisted. Although these named events may have been the culminating event, they were not the cause of the cumulative trauma that was present. As a result, the injurious event continues. Addressing the culminating event rather than the cause of the cumulative trauma will have little effect.

Ergonomic Studies

If LBDs are associated with loading, then changes in loading should change injury and absenteeism rates. But surprisingly, the literature does not provide clear guidance for reaching this objective; it needs interpretation. The traditional ergonomic approach is to establish a criterion that, if applied in the job design, would lead to a reduction in incidence rate. In a review in 1996, Frank and colleagues suggested that modified work with lower demands can reduce the number of injuries, but that other changes such as organizational parameters made it difficult to conclude that physical demand changes accounted for any reported differences. This is because very few studies have simply altered job demands. Furthermore, Winkel and Westgaard (1996) noted that the implementation of ergonomics can lead to what they called an ergonomic pitfall. That is, the new ergonomic consciousness causes many workers to report concerns that they did not previously realize were a result of their job situations. Thus, many newly implemented ergonomic intervention programs have resulted in a temporary rise in musculoskeletal disorders. This effect appears to have confounded any study of ergonomic efficacy of insufficient duration. The number of good investigations documenting the effects of job change, or the implementation of ergonomic principles, is also low because such research is very time-consuming and difficult to perform on-site.

Nonetheless, several nice studies qualify by documenting only the effect of ergonomic job redesign. For example, in a report on a series of studies of working women in Norway, Aaras (1994) noted a collectively documented reduction in sick leave due to musculoskeletal troubles as a result of job redesign (specifically, from 5.3 to 3.1%) and a reduction in employee turnover from 30.1 to 7.6%.

Rehab and Prevention Studies

Loisel and colleagues (1997) conducted a very interesting and important study in which they used a randomized control trial design with four groups. One group of people with back injuries received clinical interventions consisting of a visit to a back pain specialist, back school, and functional rehabilitation therapy. Another group received an occupational intervention consisting of a visit to an occupational physician and then an ergonomist to arrive at ergonomic solutions. A third group, the full intervention group, received both of these approaches, and the fourth group received usual care. The group receiving full intervention returned to regular work 2.41 times faster than the usual-care group, although the specific effect of occupational intervention (ergonomics) accounted for the largest proportion of this result with a rate ratio of return to regular work of 1.91.

Those paying for injury (government agencies, compensation boards, and the insurance industry) could reasonably argue from this evidence that, to reduce costs, care for those with injured backs should be removed from medical hands (once the medics have ruled out red flag conditions such as tumors) and given to ergonomists! Tongue-in-cheek as this statement may be, its point is worthy of deep consideration. Hopefully, the last section of this book will provide clues for more efficacious medical interventions. Krause, Dasinger, and Neuhauser (1998) reviewed the literature on the role of modified work in the return to work and rated as high quality the Loisel study, as well as the study of Baldwin, Johnson, and Butler (1996). This latter review concluded that modified work (involving the modification of musculoskeletal loading) is effective in facilitating the return to work of disabled workers.

In addition to workplace design modifications, workplace interventions may benefit from addressing the personal movement strategies of each worker. In a fascinating study, Snook and colleagues (1998) demonstrated that of 85 patients randomly assigned to a group that controlled the amount of early-morning lumbar flexion, the experimental group had a significant reduction in pain intensity compared to a control group. When the control group received the experimental treatment, they responded with similar reductions. This is yet another example of how personal spine motion patterns and loading posture can influence whether the person will become injured. The next section blends notions of job design with personal movement strategies to reduce loading and the risk of LBD.

Studies on the Connection Between Fitness and Injury Disability

It is fruitful to discuss briefly the role of fitness in the link between injury and disability. Although several studies have shown links between various fitness factors and the incidence of LBD (e.g., Suni et al., 1998, who showed that higher $\dot{V}O_2max$ scores were linked to LBDs), these cross-sectional studies cannot infer causation.

Probably the most widely cited longitudinal study was that of Cady and colleagues (1979), who assessed the fitness of Los Angeles firefighters and noted that those who were rated fitter had fewer subsequent back injuries. However, what is not widely quoted by those citing this study is that when the fitter firefighters did become injured, the injury was more severe. Perhaps those who were fitter were willing to experience higher physical loads. Several have suggested that a psychological profile is associated with being fit (e.g., Farmer et al., 1988; Hughes, 1984; Ross and Hayes, 1988; Young, 1979) and that the unfit may complain more about more minor aches. Along those lines, some athletes have demonstrated the ability to compete despite injury. Burnett and colleagues (1996) reported cricket bowlers with pars fractures who were still able to compete. Is this due to their supreme fitness and ability to achieve spine stability, or to their mental toughness? Perhaps it is both. The issue remains unresolved.

It is also interesting to note that personal fitness factors appear to play some role in first-time occurrence. Biering-Sorensen (1984) tested 449 men and 479 women for a variety of physical characteristics and showed that those with larger amounts of spine mobility and lower extensor muscle endurance (independent factors) had an increased occurrence of subsequent first-time back troubles. Luoto and colleagues (1995) reached similar conclusions. It would appear that muscle endurance, and not anthropometric variables, is protective.

Our most recent work with firefighters and elite task force police officers adds further insight into the role of fitness (Frost et al., 2011, 2012, 2014, 2015, in press; McGill et al., 2013a, 2013b). From this collective work, we observed that fitter people have more back pain. There are several possible explanations. In athletic groups, we have observed that the fitter ones get more play time and thus more exposure (McGill, Anderson, and Horne, 2012). It is quite possible that the fitter firefighters and police officers take on the challenging physical tasks in a crew, whereas the unfit beg off.

We also strongly suspect that many in these occupational groups have become enamored with the types of warrior training approaches characterized by excessive repetitions of substantial loads until exhaustion in the weight room. Another consideration is that in becoming fitter, their work capacity is expanded. Adding capacity without movement competency appears to lead to pain. We trained two groups of firefighters: one with warrior training approaches of adding more load without concern over movement quality, and another group with coaching to ensure movement competency. The coached group maintained movement quality in occupational exertions requiring pushing, pulling, lifting, chopping, and carrying. The warrior trained group did not. Thus, getting fitter, and by default adding more capacity, needs movement coaching to reduce injury rates (Frost et al., 2011, 2012, in press).

Beyond Ergonomics: Is It Time to Modify the Worker?

Ergonomics is often described as fitting the task to the person. Many efficacy studies now show that ergonomic job change is effective to varying degrees, but not as effective as might be thought. For example, a study of mechanized lifting aids in a patient care facility showed that the ergonomic aids reduced physical demands and decreased staff fatigue, yet injury rates remained unchanged (Yassi et al., 2001). Although training the worker has been shown to be necessary with virtually any physical ergonomic intervention (e.g., van der Molen et al., 2005), the way the worker moves also appears to be highly important. Reducing injury with ergonomic approaches appears to have some efficacy, but any further advances will probably require changing the person to fit the task. Gagnon (2003) observed expert lifters and concluded that their lifting strategies and personal body movements led to their health success. Our work for the past several years has converged on the notion that the way the exertion is performed determines the risk. Our quantification of martial arts tasks, weightlifting, strongman competitions, and so on, proves that technique determines the risk in very challenging physical tasks. The next section of this chapter incorporates these notions within the context of reducing the risk of back injury.

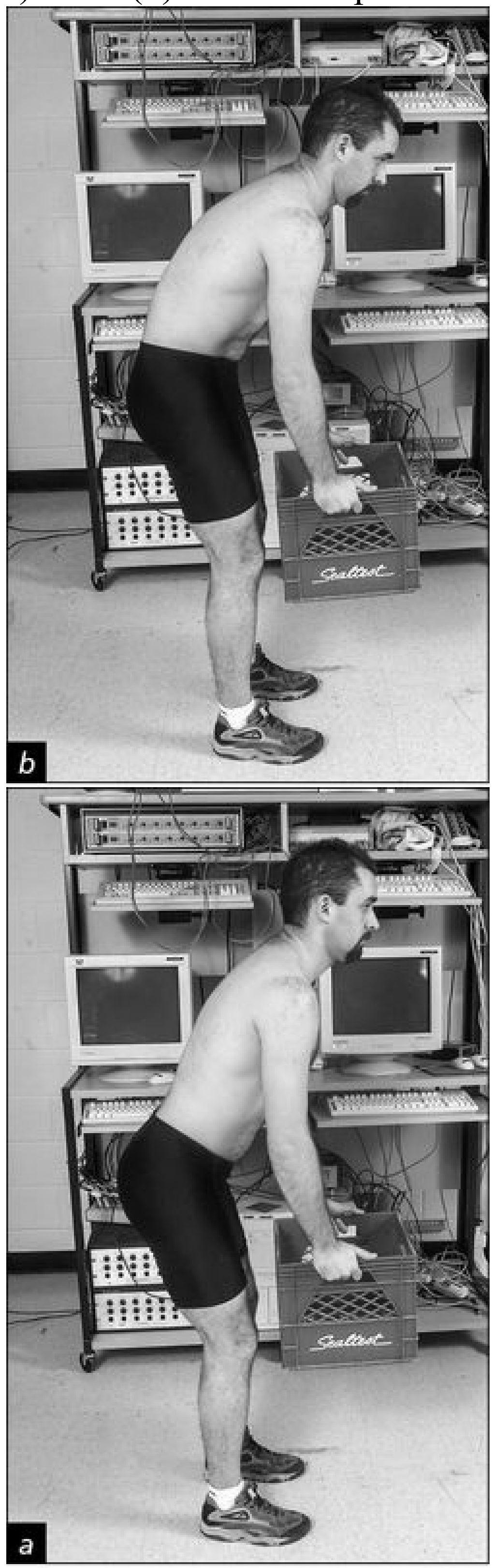
LBD Prevention for Workers

This section addresses a list of issues that are scientifically justifiable to reduce the risk of occupational LBDs.

Should Workers Avoid End Range of Spine Motion During Exertion?

Generally, the answer to whether end range of motion should be avoided is yes—for several reasons. First of all, the strategy works (Ikeda and McGill, 2012; Suni et al., 2013). Generally, if the spine is under load, it is best not to move it—keep it stiff. This principle is not contestable. The second best choice, if the spine is bent during a lift, is to hold it in an isometrically fixed posture with the motion focused about the hip joints. The worst choice is to move the spine over and over, because the combination of load with motion will slowly and cumulatively delaminate the disc collagen. Furthermore, maintaining a more neutrally lordotic spine maximizes shear support, ensures a high tolerance of the joint to withstand compressive forces, eliminates the risk of ligamentous damage because the ligaments remain unstrained, and qualitatively emulates the spine postures that Olympic lifters adopt to avoid injury (see figure 7.1, a and b, for an illustration of a flexed and a neutral spine). Unfortunately, this issue has become confused with issues such as whether it is better to stoop or squat. Another source of confusion has evolved from the common recommendation to perform a pelvic tilt when lifting; the scientific base for this clinically popular notion is nonexistent. Performing a pelvic tilt increases tissue stresses and the risk of injury!

Figure 7.1 (a) Flexing the torso involves either hip flexion or spine flexion, or (as in this case) both. (b) A neutral spine with hip flexion.



Photos from Stuart McGill

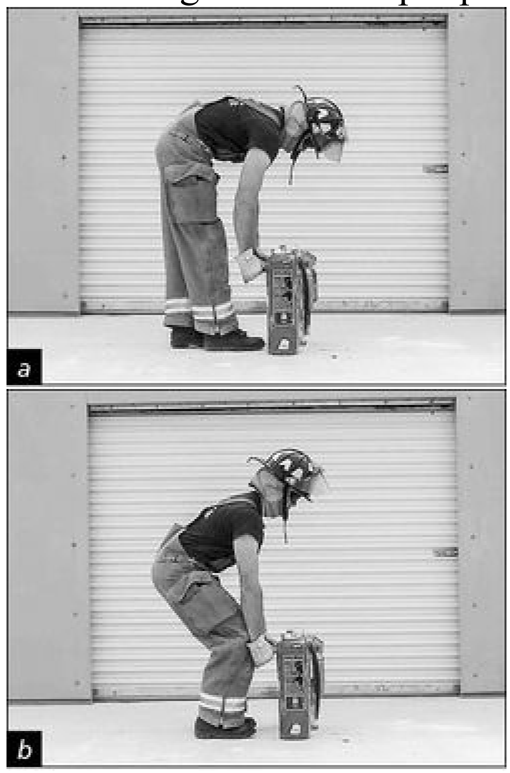
What spine and hip posture best minimizes the risk of injury? We do know that very few lifting tasks in industry can be accomplished by bending the knees and not the back. Furthermore, most workers rarely adhere to this technique when repetitive lifts are required—a fact that is quite probably due to the increased physiological cost of squatting compared with stooping (Garg and Herrin, 1979). However, a case can be made for preserving neutral lumbar spine curvature during lifting (specifically, avoiding the end range of spine motion about any of the three axes). This is a different concept from trunk angle, because the posture of the lumbar spine can be maintained independent of thigh and trunk angles.

The literature is confused between trunk angle or inclination and the amount of flexion in the lumbar spine. Bending over is accomplished by either hip flexion or spine flexion, or both. It is the issue of specific lumbar spine flexion that is of importance here. Normal lordosis can be considered to be the curvature of the lumbar spine associated with the upright standing posture. (To be precise, the lumbar spine is slightly extended from elastic equilibrium during standing; see Scannell and McGill, 2003.) In figure 7.2, a warehouse worker is sparing his spine by avoiding end range of spine motion even though he is not bending the knees; he has accomplished torso flexion by rotating about the hips. In figure 7.3, a firefighter is demonstrating a spine-damaging technique (figure 7.3a) versus a spine-sparing technique (figure 7.3b) for lifting extinguishers. Chapter 8 offers further occupation-specific examples (e.g., the construction workers demonstrating spine-sparing postures in figure 8.3).

Figure 7.2 This warehouse worker is not bending the knees, yet he is sparing the spine by electing to bend and rotate about the hips; the lumbar spine is not flexed.

Photos from Stuart McGill

Figure 7.3 (a) This firefighter, who is flexing his lumbar region, is loading the passive tissues and increasing his risk of back troubles. (b) Avoiding full lumbar flexion and rotating about the hips spare the spine.



Photos from Stuart McGill

Chapter 4 addresses the load distribution among the tissues of the low back. Of interest here are the dramatic effects on shear loading as a function of spine curvature. Recall the following facts:

- A spine that is not fully flexed ensures that the pars lumborum fibers of the longissimus thoracis and iliocostalis lumborum can provide a supporting posterior shear force on the superior vertebra, whereas full flexion causes the interspinous ligament complex to strain, imposing an anterior shear force on the superior vertebra. For these reasons, avoiding full flexion not only ensures a lower shear load but also eliminates ligament damage.
- A fully flexed spine is significantly compromised in its ability to withstand compressive load.
- Because herniation of the nucleus through the annulus is caused by repeated or prolonged full flexion, avoidance of the posture minimizes the risk of herniation (a cumulative trauma problem) and minimizes the

stresses on any developing annular bulges.

Olympic lifters provide a convincing example of the efficacy of avoiding lumbar flexion in lifting. They lock their spines in a neutral posture and emphasize rotation about the hips (see figure 7.4), lifting enormous weights without injury. The ambulance attendants in figure 7.5 are also sparing their backs by locking the lumbar regions to avoid flexion.

Figure 7.4 World-class performances in Olympic weightlifting are characterized by a lumbar spine locked in neutral and the motion taking place about the hips and knees. Spine motion would lead to either injury or an uncompetitive lift.

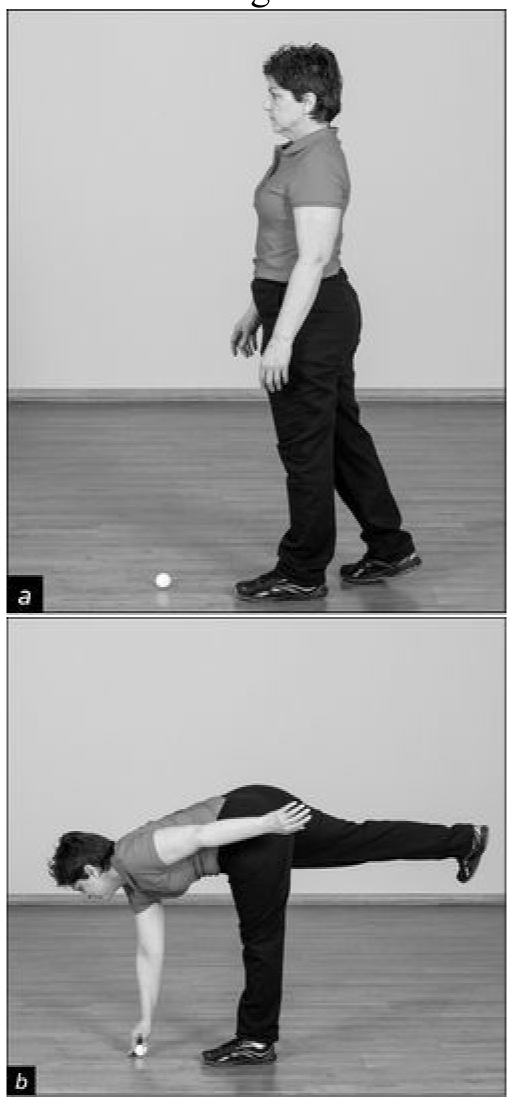
Figure 7.5 These ambulance attendants are attempting to spare their backs by avoiding lumbar flexion and lifting together to share the load. They have also been taught to lightly contract the stabilizing abdominal musculature.

Photos from Stuart McGill

Thus, the important issue is not whether it is better to stoop lift or to squat lift; rather, the emphasis should be to place the load close to the body to reduce the reaction moment (and the subsequent extensor forces and resultant compressive joint loading) and to avoid a fully flexed spine. Sometimes it may be better to squat to achieve this; if the object is too large to fit between the knees, however, it may be better to stoop, flexing at the hips but always avoiding full lumbar flexion to minimize posterior ligamentous involvement. (For a more comprehensive discussion, see McGill and Kippers, 1994; McGill and Norman, 1987; Potvin, Norman, and McGill, 1991.)

Yet another spine-sparing lifting posture is the golfer's lift, which reduces spine loads for repeated lifting of light objects (see figure 7.6, a and b) (Kinney, Callaghan, and McGill, 1996). The hips act as a fulcrum in which one leg is cantilevered behind with isometric muscle contraction, forming a counterweight to rotate the upper body back to upright.

Figure 7.6 (a, b) The golfer's lift has been documented to minimize low back motion and reduce the loads on the lumbar tissues by using the leg, which is cantilevered behind, as a counterweight; (c) the hips act as a fulcrum to raise the torso to upright. This is an effective technique for repeated lifting of light objects from floor level. Most still adhere to the general instruction to bend the knees and keep the back straight, not considering the spine-conserving benefits of the golfer's lift.



What Are the Ways to Reduce the Reaction Moment?

A popular instruction is to hold the load close to the torso when handling material. This is biomechanically wise; a reduced lever arm of the load requires lower internal tissue loads to support the reaction moment. But phrasing the principle in terms of holding the load close restricts the notion to lifting tasks. The real biomechanical principle is to reduce the reaction moment. When phrased this way, the principle is now applicable to any task involving the exertion of external force.

One technique is to expand the concept used by archers—termed the archer's bow by some of my Australian colleagues (figure 7.7). Here, one arm pushes to support the reaction moment needed to support the upper body while the other arm lifts the load.

Figure 7.7 (a) The archer's bow (b) is translated to a lifting situation to minimize back loading. Opposing hand forces are shown with arrows.

Photos from Stuart McGill

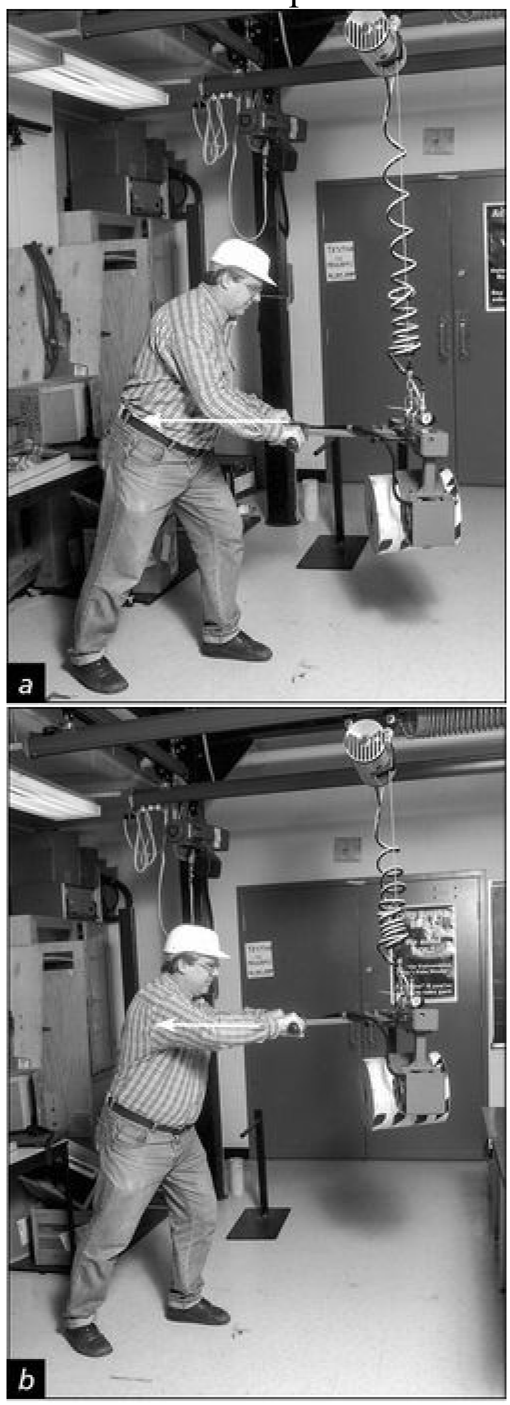
Directing the Pushing Force Vector Through the Spine

When pushing a cart handle, directing the pushing force vector through the lumbar spine reduces the reaction moment and therefore the tissue loads and spine load (see figure 7.8). In contrast, a pushing force directed through the shoulder would not be directed through the low back (see figure 7.9, a and b); this forms a transmissible vector. The large perpendicular distance from this force to the spine causes a high reaction torque that is balanced by muscular force; this imposes a corresponding compressive penalty on the spine. Redirecting the transmissible vector through the low back reduces this moment arm (and the moment), the muscle forces, and the spine compressive load. Another example from everyday activities is the technique used to open a door; directing the pulling force through the low back is sparing (see figure 7.10, a-c). Another example is vacuuming, which is often reported by our patients to exacerbate their symptoms. Holding the handle to the side creates a large moment arm for the pushing and pulling forces; this heavily loads the back (see figure 7.11a). Directing the pushing and pulling forces through the low back minimizes the moment arm (see figure 7.11b) and removes the loads that ensure that patients remain patients! These examples demonstrate how this powerful but rarely practiced technique of skilled control of the transmissible vector spares the back.

Figure 7.8 Pushing through the hands but directing the force vector to pass through the low back minimizes the low back moment (and minimizes the muscular loads).

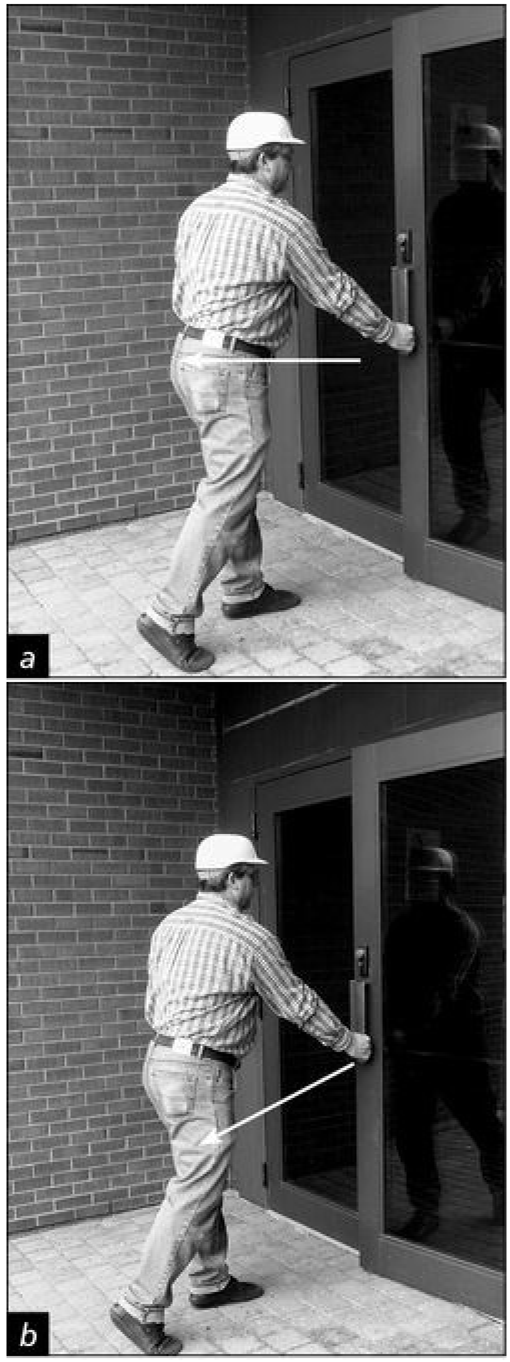
Photos from Stuart McGill

Figure 7.9 (a) Pushing or pulling forces that pass by the spine with a large moment arm ensure a high moment and corresponding high muscle forces and spine loads. (b) Pushing forces directed through the low back minimize the moment and spine load.



Photos from Stuart McGill

Figure 7.10 (a) Opening a door by directing hand forces through the low back spares the spine. Many people are not taught how to optimize this principle during the performance of daily tasks. (b) Instead, they sacrifice the spine by opening the door so that the force is lateral to the lumbar spine creating a twisting torque or (c) produce a pulling force that passes over the low back.



Photos from Stuart McGill

Figure 7.11 (a) Vacuuming is often reported as problematic by people with bad backs. The transmissible vector in this example has a large moment arm with respect to the back and requires large twisting torque forces (causing pain). (b) The transmissible vector is directed through the low back, minimizing the load and the pain and enabling vacuuming.

Photos from Stuart McGill

Diverting the Force Around the Lumbar Spine

Still other very effective ways exist to reduce the reaction moment. The next examples demonstrate how workers skillfully spare the low back by diverting force around the lumbar region when lifting. The workers lifting the shaft in a paper factory in figure 7.12 are minimizing the reaction moment by lifting the shaft with the thigh (by plantar flexing the foot). In this way, the weight of the shaft is directed down the thigh to the floor, bypassing the upper-body linkage and spine. Prior to redesigning the job, the workers had to lift the shaft with arm forces, causing low back problems and motivating our involvement. Figure 7.13, a through c, illustrates how a worker can perform the same maneuver to lift a patient. Observe that the lifter is pulling the patient's pelvis onto his thigh and standing along with the patient. (Note that patient lifts cannot be performed with universal technique because patients differ in many ways, including their ability to offer assistance.) Minimal forces are transmitted down the spine. Shoveling snow by resting the forearm on the thigh involves the same spine-sparing principle (see figure

7.14, a and b).

Figure 7.12 This task requires a worker to lift a heavy shaft while another worker slides on a core from the end. A high incidence of back troubles motivated our consulting recommendation to install a bar for the foot, which is plantar flexed to raise the shaft now supported on the thigh. The forces now transmit directly down the leg, completely bypassing the arm and spine linkage.

Photos from Stuart McGill

Figure 7.13 (a) Lifting the patient can be performed with minimal loading down the spine provided the height of the seat is close to the height of the lifter's thigh when in a squat position. (b) The patient's pelvis is pulled and slid onto the lifter's thigh, and (c) then the lifter stands up, hugging the patient's pelvis and minimizing the forces up the arms and down the spine. This spine-sparing technique is reserved for patients who are able to stand.

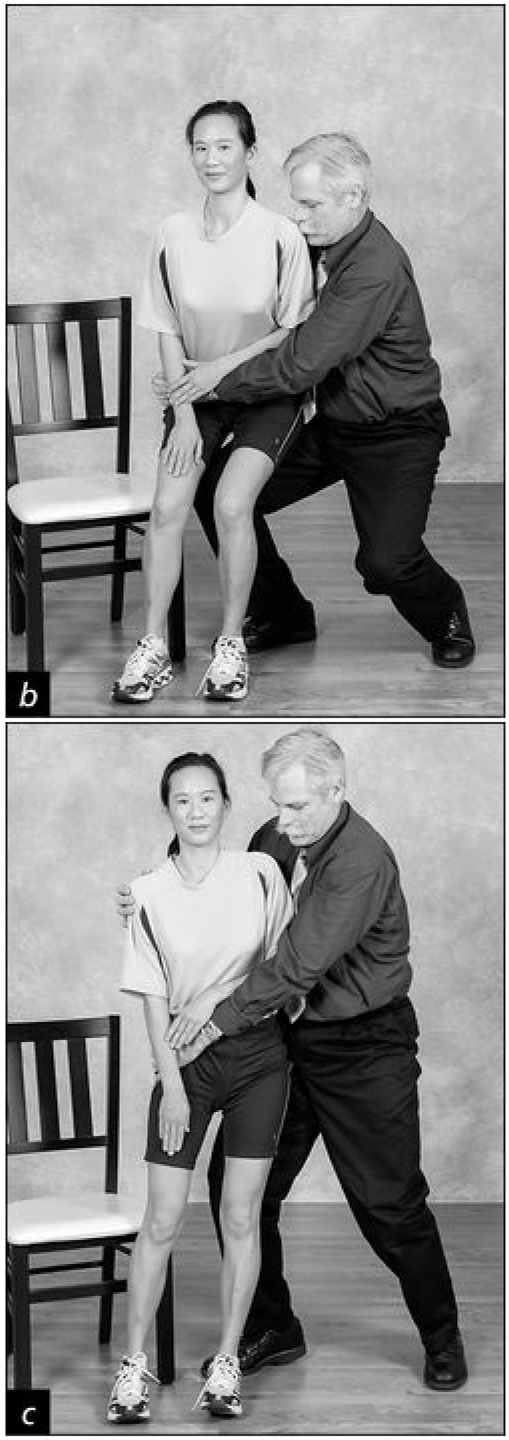

b
c

Figure 7.14 (a) Shoveling snow with a large moment arm for the load on the shovel loads the back. (b) Resting the arm on the thigh directs the forces to the ground, bypassing the arm and spine linkage.

Photos from Stuart McGill

A Note on Pushing and Pulling

Our studies comparing the expert techniques of firefighters to the techniques of graduate students were noted in chapter 4 (Lett and McGill, 2006). Specifically, for tasks such as hose pulling, the firefighters braced the body and pulled the hose with the force vector traveling through the low back to minimize lumbar loading (see figure 7.15). Some of our recent work documenting the techniques employed by hospital workers when they transfer patients highlighted once again this important technique (McGill and Kavcic, 2005). On occasion, friction-reducing devices are placed underneath patients that need to be transferred. Providers can then apply pushing and pulling forces to the patient. Even with the friction-reducing devices, the principle of directing the hand forces through the lumbar spine is a very important component in minimizing spine forces.

Figure 7.15 Directing pulling forces through the low back helps to minimize spine forces. Spine posture (neutral) and whole-body posture (to reduce the reaction moment) are also important variables for injury avoidance.

Photos from Stuart McGill

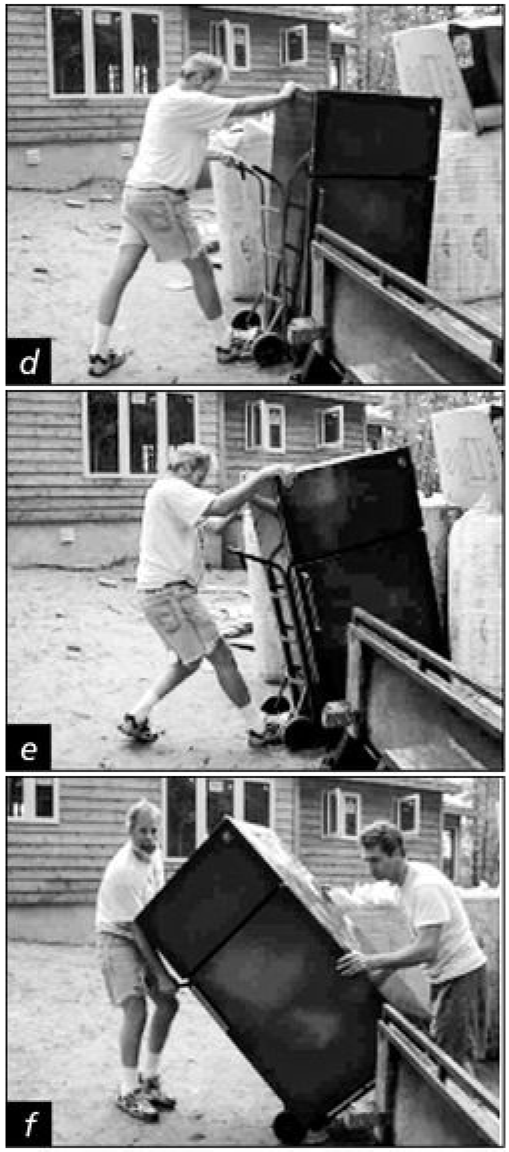
Reducing the Load

Finally, workers can use skill to reduce the actual load and hand forces when lifting large objects. Some tasks can be performed by lifting only half of the object at a time. For example, when loading long logs onto the back of a truck, a worker could lift just one end (effectively handling only half of the full weight of the log) and place it on the bed. The worker could then walk around to the other end of the log and lift while sliding the log onto the bed (see figure 7.16). Each lift is half of the total load. A sequence of unloading a refrigerator demonstrates the same technique; the full weight of the object is never lifted (see figure 7.17, a-f)! The worker lifting the mini-refrigerator (shown in figure 7.18, a and b) tilts it up onto an edge, raising its center of gravity together with the initial lifting height. Lifting from this higher starting position reduces the necessary moment. The concept of minimizing the reaction moment is much more robust than simply telling people to hold the load close to the body.

Figure 7.16 This girl loads the log into the truck by lifting only half its weight at a time. First she lifts one end onto the truck. Then she lifts the other end and slides the log onto the bed.

Photos from Stuart McGill

Figure 7.17 This worker unloads the refrigerator from the trailer without having to lift its full weight. (a) He “walks” the refrigerator on its corners to the edge of the trailer, where he balances it over the lip of the bed. (b) Next, he slides the refrigerator down the trailer’s tailgate. (c) He walks the refrigerator clear of the trailer and leaves it standing upright while he retrieves the dolly. (d, e) He pushes one edge of the refrigerator up just enough to slide the dolly under it. (f) Finally, he and a coworker wheel the appliance away.



Photos from Stuart McGill

Figure 7.18 (a) This man lifts the mini-refrigerator by tilting it up onto an edge, raising its center of gravity together with the initial lifting height. (b) Lifting from this higher starting position reduces the necessary moment so the man can reach the standing position without damaging his back.

Photos from Stuart McGill

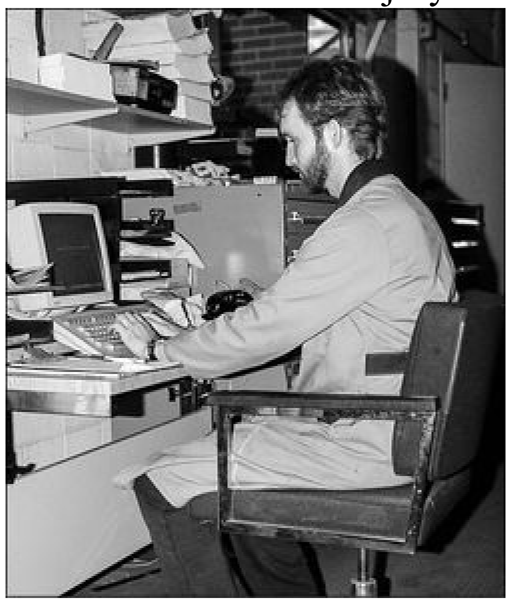
Should One Avoid Exertion Immediately After Prolonged Flexion?

Recall that prolonged flexion causes both ligamentous creep and a redistribution of the nucleus within the annulus (see chapter 4). In this way the spine tissues have a loading memory. Further, evidence from Jackson and colleagues (2001) and Le and colleagues (2009) suggests that prolonged flexion modifies the extensor neurological response and causes muscle spasming, at least until the ligamentous creep has been restored. In this case, it was 7 hours! Spine stability would be compromised during this period, and the risk of annulus damage remains temporarily high. If possible, after prolonged stooping or sitting activities, people should spend time standing upright before attempting more strenuous exertions. For example, it would be very unwise for the gardener in figure 7.19 to stand and lift a bag of peat moss or for the shipper/receiver in figure 7.20 to rise and immediately begin loading pallets. How long should one wait before performing an exertion? Data from Twomey and Taylor (1982) derived from cadaveric spines suggest that age delays the recovery of the spinal tissues. McGill and Brown (1992) noted that residual laxity remained in the passive tissues even after a half hour of standing following prolonged flexion—although the flexion was extreme in an occupational context. At least 50% of the joint stiffness returned after 2 minutes of standing following the session of prolonged flexion.

Figure 7.19 This gardener would be unwise to stand and immediately lift a heavy object after spending a long period of time in a stooped posture. Rather, standing for a brief period and even extending will prepare the disc and posterior passive tissues and reduce the risk of injury.

Photos from Stuart McGill

Figure 7.20 This shipper/receiver's work is characterized by periods of sitting followed immediately by lifting when a truck pulls up to the loading dock. Again, standing and waiting for a few minutes prior to the exertions will reduce the risk of injury.



Photos from Stuart McGill

As noted in chapter 4, because the mechanics of the joints are modulated by previous loading history, one should never move immediately to a lifting task from a stooped posture or after prolonged sitting. Rather, one should first stand, or even consciously extend the spine, for a short period. Obviously, people in certain occupations such as emergency ambulance personnel cannot follow this guideline. They arrive at an accident scene without the luxury of time to warm up their backs and may have to perform nasty lifts—such as a 150 kg (331 lb) heart attack victim out of a bathtub! For these people, the only strategy is to avoid a fully flexed spine posture while driving so that the spine is best prepared to withstand the imposed loads. This may be done with accentuated lumbar pads in ambulance seats and with worker training.

Should Intra-Abdominal Pressure (IAP) Be Increased During Lifting?

Generally, for occupational applications, the answer is no: at least IAP should not be increased consciously. Recall the discussion in chapter 5 concluding that IAP does not reduce spine loading but does act to stiffen it against buckling. By completing the rehabilitation training advocated in the final section of this book, people can train their breathing and IAP to be independent of exertion. In this way any specific instructions regarding breathing and exertion become moot points. In most cases IAP will rise naturally, and no further conscious effort is required.

A final caveat is required here. Very strenuous lifts, if they must be performed, require the buildup of IAP to increase torso stiffness and ensure stability (Cholewicki, Juluru, and McGill, 1999). On the other hand, we know that a substantially increased intrathoracic pressure (as occurs with lifting) compromises venous return (Mantysaari, Antila, and Peltonen, 1984). Further, breath holding during exertion raises both systolic and diastolic blood pressure (Haslam et al., 1988), which can be a concern for some. Blackout is not uncommon in strenuous lifting even though it is not clear which mechanism is responsible (MacDougall et al., 1985). Reitan (1941) proposed that blackout may be due to elevated central nervous system fluid pressure (IAP also raises central nervous system fluid pressure in the spine and up to the head), whereas Hamilton, Woodbury, and Harper (1944) proposed that an increase in cerebrospinal fluid pressure might actually serve a protective function (i.e., the consequent decrease in transmural pressure across the cerebral vessels could actually decrease the risk of vascular damage). At this point, given these issues and a lack of full understanding, moderate IAP may be warranted with the understanding of the negative side effects. Extreme lifting efforts involving conscious increases in IAP should not be performed at work. These situations are discussed later.

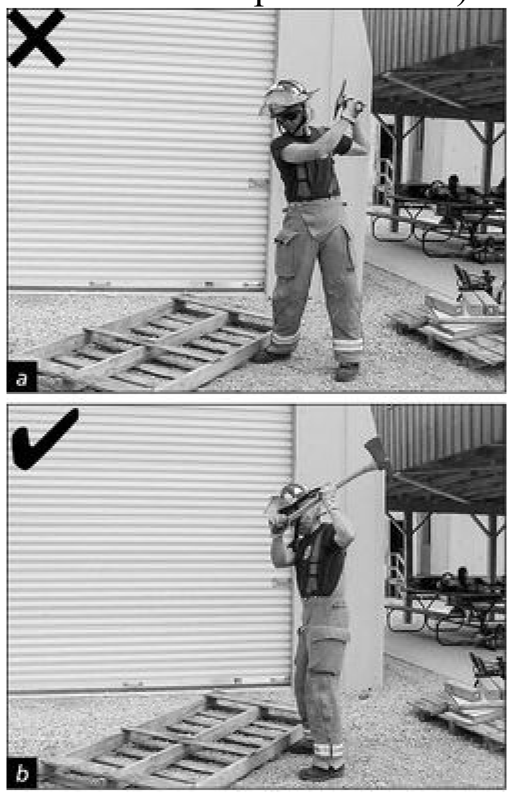
Are Twisting and Twisting Lifts Particularly Dangerous?

Although several researchers have identified twisting of the trunk as a factor in the incidence of occupational low back pain (Frymoyer et al., 1983; Troup, Martin, and Lloyd, 1981), the mechanisms of risk require some explanation. The kinematic act of twisting has been confused in the literature with the kinetic variable of generating twisting torque. Twisting torque in the torso can be accomplished whether the spine is twisted or not.

Generally, twisting to moderate degrees without high twisting torque is not dangerous. Some have hypothesized based on an inertia argument that twisting quickly imposed dangerous axial torques upon braking the axial rotation of the trunk at the end range of motion. Farfan and colleagues (1970) proposed that twisting of the disc is the only way to damage the collagenous fibers in the annulus leading to failure. They reported that distortions of the neural arch permitted such injurious rotations. Shirazi-Adl, Ahmed, and Shrivastava (1986) conducted more detailed analyses of the annulus under twist. They supported Farfan's contention that twisting indeed can damage the annulus at end range, but also noted that twisting is not the sole mechanism of annulus failure. In contrast, some research has suggested that twisting in vivo is not dangerous to the disc because the facet in compression forms a mechanical stop to rotation well before the elastic limit of the disc is reached; thus, the facet is the first structure to sustain torsional failure (Adams and Hutton, 1981). In a study of ligament involvement during twisting, Ueno and Liu (1987) concluded that the ligaments were under only negligible strain during a full physiological twist. However, an analysis of the L4-L5 joint by McGill and Hoodless (1990) suggested that posterior ligaments may become involved if the joint is fully flexed prior to twisting.

Generating twisting torque is a different issue (see figure 7.21, a and b). Because no muscle has a primary vector direction designed to create twisting torque, all muscles are activated in a state of great cocontraction. This results in a dramatic increase in compressive load on the spine when compared to an equivalent torque about another axis. For example, data from a combination of our previous studies indicate that supporting 50 Nm in the extension axis imposes about 800 N of spinal compression. The same 50 Nm in the lateral bend axis results in about 1,400 N of lumbar compression, but 50 Nm in the axial twist axis would impose over 3,000 N (McGill, 1997). It appears that the joint pays dearly to support even small axial torques when extending during the lifting of a load.

Figure 7.21 (a) Twisting the torso is occurring at the same time that twisting torque is required, which is a dangerous combination. (b) Generating the twisting torque but restricting the torso twist is a spine-sparing strategy (and can also enhance performance).



Photos from Stuart McGill

Although creating twisting torque increases the load on the spine, being twisted reduces the ability of the spine to bear load (Aultman et al., 2004, and Drake et al., 2005, observed the loss in load tolerance). Marshall and McGill (2010) documented how cyclic twisting causes circumferential delamination of the collagen, or what are known radiologically as disc tears. Thus twisting, when combined with twisting torque, becomes problematic.

To conclude, the generation of axial twisting torque when the spine is untwisted does not appear to be of particular concern. Nor is the act of twisting over a moderate range without accompanying twisting torque. But generating high torque while the spine is twisted appears to create a problematic combination and a high risk. This is of particular concern in several sports and is addressed in that context in chapter 11.

Is Lifting Smoothly and Not Jerking the Load Always

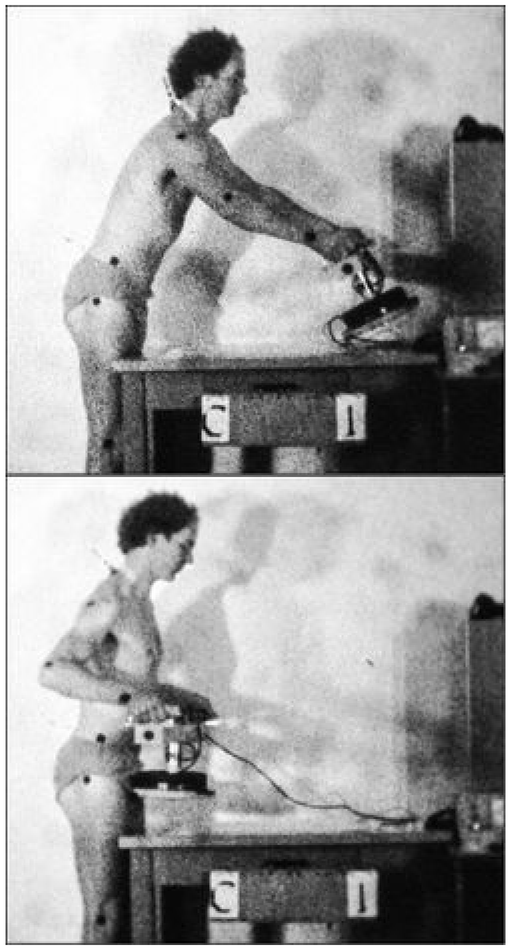
Best?

The answer to this question is no. We have all heard that a load should be lifted smoothly and not jerked. This recommendation was most likely made based on the belief that accelerating a load upward increases its effective mass by virtue of an additional inertial force acting downward together with the gravitational vector. However, this may not always be the case. It is possible to lift a load by transferring momentum from an already moving segment. Autier, Lortie, and Gagnon (1996) showed that, compared to novice lifters, expert materials handlers sometimes choose techniques that make more efficient use of momentum transfer. They do not always lift slowly and smoothly.

Troup and Chapman (1969) referred to the concept of momentum transfer during lifting, as did Grieve (1975), who coined the term kinetic lift. Later, McGill and Norman (1985) documented that smaller low back moments were possible in certain cases using a skillful transfer of momentum. For example, if a load is awkwardly placed, perhaps on a work table 75 cm (30 in.) from the worker, a slow, smooth lift would necessitate the generation of a large lumbar extensor torque for a lengthy duration of time—a situation that is most strenuous on the back. However, the worker could lift this load with a very low lumbar extensor moment or quite possibly no moment at all. If the worker leaned forward and placed his hands on the load, with bent elbows, the elbow extensors and shoulder musculature could thrust upward, initiating upward motion of the trunk to create both linear and angular momentum in the upper body (note that the load has not yet moved). As the worker straightens his arms, coupling takes place between the load and the large trunk mass (as the hands then start to apply upward force on the load), transferring some, or all, of the body momentum to the load and causing it to be lifted with a jerk.

This mechanical solution was proven to be effective in a very early experiment in my research career. The person shown in figure 7.22 is demonstrating a task in which the load can be lifted slowly, which would load the low back unduly, or with the kinetic lift technique, which, if correctly performed, will spare the back. (Obviously the markers on the model's body were to assist the measurement of body segment movement.) This highly skilled inertial technique is observed quite frequently throughout industry and in some athletic events such as competitive weightlifting, but it must be stressed that such lifts are conducted by highly practiced and skilled people. In most cases, acceleration of loads to decrease low back stress in the manner described is not suitable for the average person conducting the lifting chores of daily living. The momentum-transfer technique is a skilled movement that requires practice; it is feasible only for awkwardly placed lighter loads and cannot be justified for heavy lifts.

Figure 7.22 Lifting an awkwardly placed load slowly and smoothly, as common wisdom suggests, compromises the back. Rather, accelerating the torso first and then transferring the momentum to the load as the arms straighten can reduce the spine load. Accelerating the torso and the load at the same time is poor technique that violates this principle and causes higher spine loading. These photographs are from an early experiment in the author's research career.



Photos from Stuart McGill

Figure 7.23 shows another example of the skillful use of this principle by a worker performing the saddle toss lift.

Figure 7.23 This lift is termed the saddle toss by some; the load is swung with knee contact, minimizing muscular force in the back.

Photos from Stuart McGill

Another mechanical variable should be integrated into the analysis of a dynamic technique. The tissue property of viscoelasticity enables tissues to sustain higher loads when loaded quickly (Burstein and Frankel, 1968). Troup (1977) suggested that viscoelasticity may be used to increase the margin of safety for spine injury during a higher strain rate but cautioned that incorporating this principle into lifting technique should depend on the rate of increase in spinal stress, the magnitude of peak stress, and its duration. Tissue viscoelasticity means that under faster loading, the tissues do not have time to deform, even when the magnitude of the force is high. In this way the critical levels of tissue deformation required to cause damage are not reached. But given variability in response to load rate among the tissues, and among individuals, no specific guidance pertaining to actual load rate can be offered here.

The instruction to always lift a load smoothly may not invariably result in the least risk of injury. Indeed, it is possible to skillfully transfer momentum to an awkwardly placed object to position the load close to the body quickly and minimize the extensor torque required to support the load. In addition, tissue viscoelasticity can be protective during higher load rates. Clearly, reducing the extensor moment required to support the hand load is paramount in reducing the risk of injury; the best way to accomplish this is to keep the load as close to the body as possible.

Is There Any Way to Make Seated Work Less Demanding on the Back?

Prolonged sitting is problematic for the back. Unfortunately, this fact seems to be rather unknown in the occupational world. Those recovering from back injuries who return to modified work are often given light duties that involve prolonged sitting. Such duties are perceived as being easy on the back, but this can be far from the truth. Even though the returning worker states that she cannot tolerate sitting, that in fact she would be more comfortable walking and even lifting, she is accused of malingering. This is the result of a misunderstanding of sitting mechanics.

Sitting Studies

Epidemiological evidence presented by Videman, Nurminen, and Troup (1990) documented the increased risk of disc herniation in those who perform sedentary jobs characterized by sitting. Known mechanical changes associated with the seated posture include the following:

- Increase in intradiscal pressure when compared to standing postures (Nachemson, 1966)
- Increase in posterior annulus strain (Pope et al., 1977)
- Creep in posterior passive tissues (McGill and Brown, 1992), which decreases anteroposterior stiffness and increases shearing movement

(Schultz et al., 1979)
- Posterior migration of the mechanical fulcrum (Wilder, Pope, and Frymoyer, 1988), which reduces the mechanical advantage of the extensor musculature (resulting in increased compressive loading)

These changes caused by prolonged sitting have motivated occupational biomechanists attempting to reduce the risk of injury to consider the duration of sitting as a risk factor when designing seated work. A recently proposed guideline suggested a sitting limit of 50 minutes without a break, although this proposal will be tested and evaluated in the future.

Strategies to Reduce Back Troubles During Prolonged Sitting

We have developed a four-point approach for reducing back troubles associated with prolonged sitting:

1. Use an ergonomic chair, but use it properly (very few actually do). Many people believe that they should adjust their chair to create the ideal sitting posture. Typically, they adjust the chair so that the hips and knees are bent to 90° and the torso is upright (see figure 7.24). In fact, this is often shown as the ideal posture in many ergonomic texts. This may be the ideal sitting posture, but for no longer than 10 minutes! Tissue loads must be migrated from tissue to tissue to minimize the risk of any single tissue's accumulating microtrauma. This is accomplished by changing posture. Thus, an ergonomic chair is one that facilitates easy posture changes over a variety of joint angles (see figure 7.25). Callaghan and McGill (2001a) documented the range of spine postures that people typically adopt to avoid fatigue. Some have three or four preferred angles. The primary recommendation is to continually change the settings on the chair. Many workers continue to believe that there is a single best posture for sitting and are reluctant to try others. This is, of course, unfortunate, because the ideal sitting posture is a variable one. Many employees need to be educated about how to change their chairs and about the variety of postures that are possible.
2. Get out of the chair. There simply is no substitute for getting out of the chair. Some guidelines suggest performing exercise breaks while seated, and some even go as far as to suggest flexing the torso in a stretch. This

is both nonsense and disastrous! A rest break must consist of the opposite activity to reduce the imposed stressors. Extension relieves posterior annulus stress, but more flexion while seated increases it. The recommended break that we have developed involves standing from the chair and maintaining a relaxed standing posture for 10 to 20 seconds. At this stage, some may choose to perform neck rolls and arm windmills to relieve neck and shoulder discomfort from their desk work. The main objective is to buy some time to allow the redistribution of the nucleus and reduce annular stresses. The person then raises the arms over the head (see figure 7.26, a-c) and then pushes the hands upward to the ceiling. By inhaling deeply, the person finds that the low back is fully extended. In this way, the person has taken the back through gentle and progressive lumbar extension without having been taught lumbar position awareness or even understanding the concept. Some will argue that in their jobs they cannot stand and take a break; they must continue their seated work. These people generally will need to be shown the opportunities for standing. For example, they could choose to stand when the phone rings and speak standing. With these simple examples, they will soon see the opportunities to practice this part of good spine health.

3. Remove wallet from back pocket. Viggiani and colleagues (2012) documented the point load in the hip capsule and sciatic nerve when sitting on a wallet (see figure 7.27). Interestingly, some experiments have used this as an intentional inducer of back pain!
4. Perform an exercise routine at some time in the workday. Midday would be ideal, but first thing in the morning is unwise (see the previous guideline). A good general back routine is presented in the last section of this book.

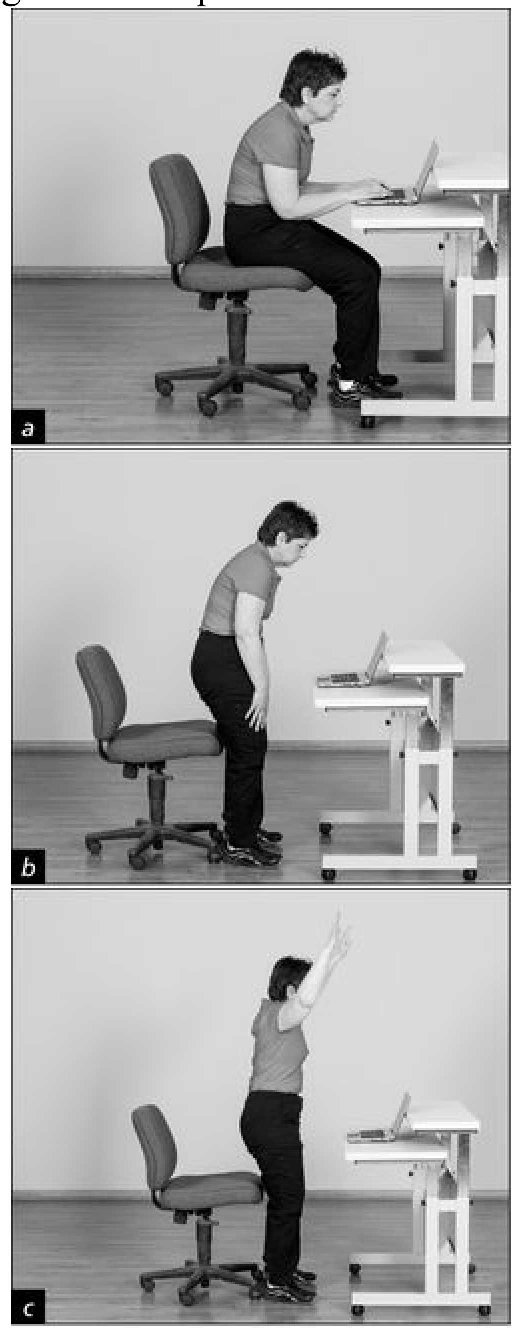
Figure 7.24 The so-called ideal sitting posture (90° angles at the hips, knees, and elbows) described in most ergonomic guides. This is erroneous; the ideal sitting posture is one that involves a variety of postures.

Photos from Stuart McGill

Figure 7.25 Good posture for prolonged sitting is a variable one that migrates the internal loads among the tissues. Possible short-term sitting posture options are shown.

Photos from Stuart McGill

Figure 7.26 A strategy to thwart the accumulation of disc stresses from (a) prolonged sitting is to (b) stand (note the forward antalgic posture often observed after sitting), (c) reach for the ceiling, stretch pushing the hands upward, inhale deeply and (d) then perform the final extension. This sequence, performed slowly, causes a gentle and progressive extension of the lumbar region and dispels the stresses of sitting.



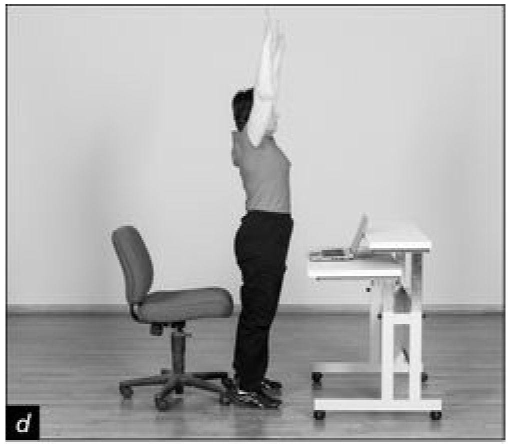

d

Figure 7.27 Map of the pressure distribution between the chair and a person's pelvis and legs showing the concentrations due to the wallet in the back pocket.

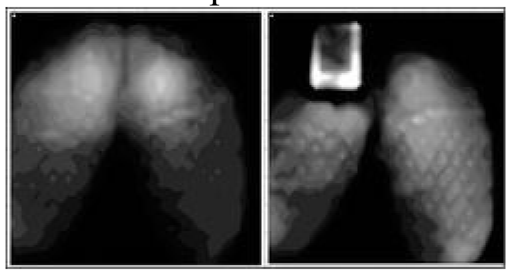
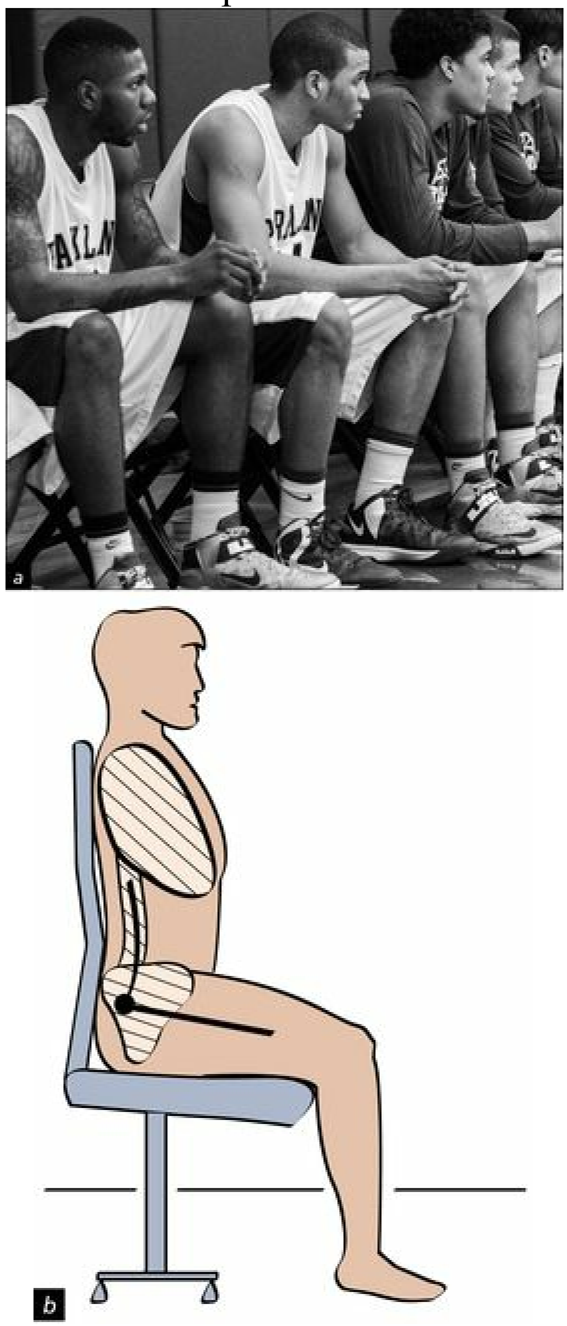


Photo from Daniel Viggiani

A couple of athletic examples may provide insight as well. Athletes who sit on the bench between plays (figure 7.28a) are often ill prepared for immediately resuming play (Green, Grevier, and McGill, 2002). To help combat this problem, they should sit in taller chairs with angulated seat pans to reduce lumbar flexion (figure 7.28b), and stand and sometimes pace approximately every 20 minutes. In addition, we question the many exercises performed in a seated position, which appears to be for convenience rather than related to any scientific rationale.

Figure 7.28 (a) Sitting on the bench with a flexed lumbar spine is problematic because it creates or exacerbates a posterior disc bulge (or both), and it causes loss of the benefits obtained from warming up the back. (b) Sitting in taller chairs with angled seat pans helps extend the hips and lumbar spine.



Some Short-Answer Questions

The following questions and their answers provide further guidance to reduce the risk of occupational LBD.

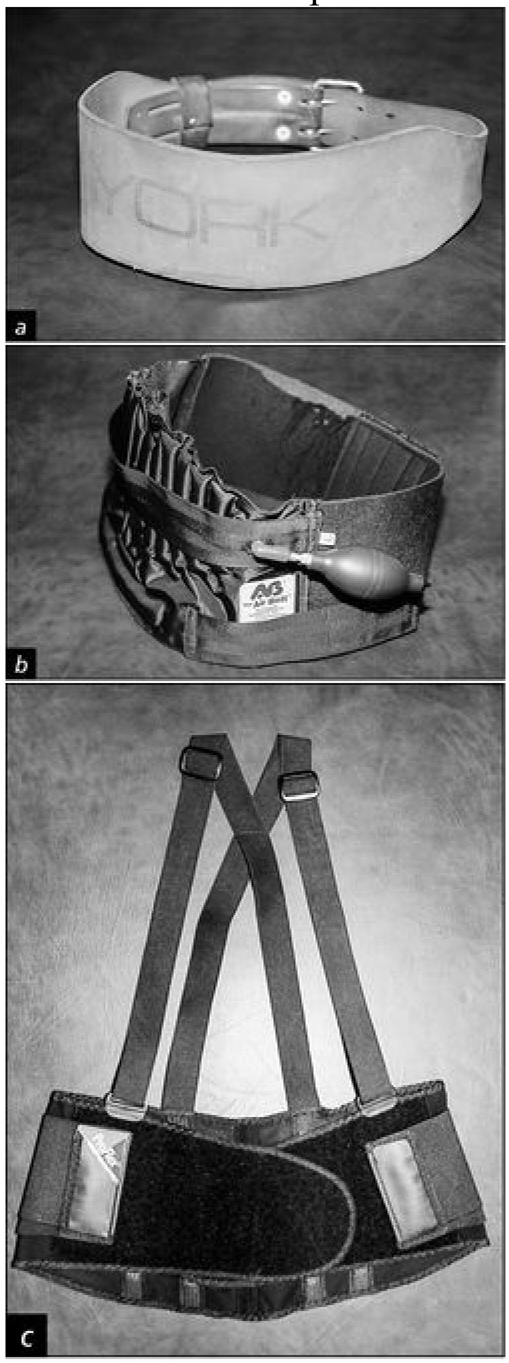
- Is it advisable to maintain a reasonable level of fitness? As much as we would all like to believe that higher levels of fitness are protective for low back disorders, it is argued by some that the literature is not strongly supportive. This is for several reasons. Many clinical trials in which the intervention was designed to enhance fitness actually chose exercises that inadvertently increased the risk of spine damage. For example, many assumed that enhancing abdominal strength was a good idea and addressed this goal by prescribing sit-up exercises. Sit-ups damage the backs of most people; they do not increase back health. Perhaps this has acted as an artifact biasing the literature. Interestingly, the most recent studies that have used biomechanical evidence to develop exercises—particularly stabilization exercises—have shown them to be efficacious. In fact, a review of preventive interventions by Linton and van Tulder (2001) suggests that well-chosen exercise is the most powerful strategy for preventing occupational back disorders. A stable spine maintained with healthy and wise motor patterns, along with higher muscle endurance, is protective.
- Move well. Employing the spine-sparing principles noted here such as hip hinging rather than spine bending has reduced the incidence of back pain in large test groups (e.g., the military; Suni et al., 2013). Choose the most appropriate movement tools (chapter 8) to accomplish the task (e.g., the lunge to move to the floor).
- Should people lift or perform extreme torso bending shortly after rising from bed? The answer is no. Recall the biomechanics of daily changes in the spine as discussed in chapter 4: the discs are hydrophilic and imbibe fluid overnight. This is why people are taller in the morning than when they retire at night. This is also why it is much more difficult to bend in the morning to put one's socks on compared to taking them off at night; the bending stresses are much higher, as is the risk of disc damage. This diurnal variation in spine length and the ability to flex forward have been well documented. As previously noted, Snook and colleagues (1998) found the strategy of restricting forward spine flexion in the morning to be very effective in reducing symptoms in a group of patients with back trouble.

- Should workers adopt a lifting strategy to recruit the lumbodorsal fascia or involve the hydraulic amplifier mechanism? As noted in chapter 4, these mechanisms have been shown to be untenable for sparing the back. Although some still attempt to train workers to invoke these strategies, they have little scientific support. In many cases such strategies are detrimental.
- Should the trunk musculature be cocontracted to stabilize the spine? As noted in chapter 5, the answer is generally yes. In chapter 4, we noted that although such coactivation imposes some penalty on the spine, it is best for the spine to pay this price to enhance stiffness and resist buckling and to reduce the risk of other unstable behavior. How much cocontraction is necessary? As noted in chapter 5, in most tasks, sufficient stability is ensured with very modest amounts of cocontraction —somewhere in the magnitude of 5 to 10% of maximal voluntary contraction for the abdominal wall and other antagonists. Achieving added stiffness in the spine through cocontraction will help prevent injuries that can otherwise result even while lifting a very light object, sneezing, or tying one's shoe. Of course, when the potential exists for surprise loading or handling nonrigid material or heavier loads, the coactivation magnitudes must increase. Damaged joint tissues also require higher levels of cocontraction to avoid unstable joint behavior. The necessary levels of coactivation would depend specifically on the task and the person's history. Thus, lightly cocontracting the stabilizing musculature upon exertion is a reasonable guideline; this should become automatic following stability training for those who do not naturally stiffen.

The Question of Back Belts

This section focuses on occupational belt use (see figure 7.29, a-c, for the typical types of belts used and tested). For information on the athletic use of belts, see Ultimate Back Fitness and Performance (McGill, 2014) or McGill (2008). After reading this section, you will be able to make decisions on who should wear a belt and to justify their prescription and use.

Figure 7.29 Several types of belts are worn and have been tested; (a) the leather belt, (b) the inflatable cell belt, and (c) the stretch belt with suspenders are a few examples.



Photos from Stuart McGill

Issues of the Back Belt Question

The average patient must be confused when observing both Olympic athletes and people with back injuries wearing abdominal belts. Years ago I conducted a review of the effects of belt wearing (McGill, 1993) and summarized my findings as follows:

- Those who have never had a back injury appear to receive no additional protective benefit from wearing a belt.
- It would appear that those injured while wearing a belt risk a more severe injury than if they had not worn a belt.
- Belts appear to instill in people the belief that they can lift more and may in fact enable them to lift more.
- Belts appear to increase intra-abdominal pressure and blood pressure.
- Belts appear to change the lifting styles of some people to either decrease or increase spine load.

As more studies became available, I updated the review (McGill, 1999a, 1999b). Even though there were more data, the conclusion did not change then and has not changed since. In summary, given the assets and liabilities of belt wearing, I do not recommend them for healthy people either in routine work or in exercise participation. However, the temporary prescription of belts may help some workers return to work.

Manufacturers of abdominal belts and lumbar supports continue to sell them to industry in the absence of a regulatory requirement to conduct controlled clinical trials similar to those required for drugs and other medical devices. Many claims have been made about how abdominal belts reduce injury. For example, some have suggested that belts do the following:

- Remind people to lift properly.
- Support shear loading on the spine that results from the effect of gravity acting on the handheld load and mass of the upper body when the trunk is flexed.
- Reduce compressive loading of the lumbar spine through the hydraulic action of increased intra-abdominal pressure associated with belt wearing.
- Act as a splint, reducing the range of motion and thereby decreasing the risk of injury.
- Provide warmth to the lumbar region.

- Enhance proprioception via pressure to increase the perception of stability.
- Reduce muscular fatigue.

These ideas, among others, are addressed in this chapter. The following section addresses the science regarding the occupational use of belts and concludes with evidence-based guidelines.

The 1994 U.S. National Institute for Occupational Safety and Health (NIOSH) report, "Workplace Use of Back Belts," contained critical reviews of a substantial number of scientific reports evaluating back belts. The report concluded that back belts do not prevent injuries in uninjured workers nor are they protective for those who have not been injured. Although this is generally consistent with our position stated in 1993, my personal position on belt prescription is somewhat more moderate.

Scientific Studies

In this section I divide the scientific studies into clinical trials and those that examined biomechanical, psychophysical, and physiological changes from belt wearing. Finally, based on the evidence, I recommend guidelines for the prescription and use of belts in industry.

Clinical Trials

Many clinical trials reported in the literature were fraught with methodological problems and suffered from the absence of a matched control group, no posttrial follow-up, limited trial duration, and insufficient sample size. As a result, I review only a few clinical trials in this chapter, while acknowledging the extreme difficulty in executing such trials.

- Walsh and Schwartz (1990) divided 81 male warehouse workers into three groups:
  - A control group (n = 27)
  - A group that received a half-hour training session on lifting mechanics (n = 27)
  - A group that received a 1-hour training session and wore low back

orthoses while at work for the subsequent six months (n = 27)

Instead of using more common types of abdominal belts, this research group used orthoses with hard plates that were heat molded to the low back region of each worker. Given the concern that belt wearing was hypothesized to cause the abdominal muscles to weaken, the researchers measured the abdominal flexion strength of the workers both before and after the clinical trial. The control group and the training-only group showed no changes in abdominal flexor strength or any change in lost time from work. The third group, which received training and wore the belts, showed no changes in abdominal flexor strength or accident rate but did show a decrease in lost time. However, the increased benefit appeared only to accrue to workers who had had a previous low back injury. Van Poppel and colleagues (1998) reached a similar conclusion in a study of 312 airline baggage handlers.

- Reddell and colleagues (1992) studied 642 baggage handlers who worked for a major airline. They divided the baggage handlers into four treatment groups:
    - A control group (n = 248)
    - A group that received only a belt (n = 57)
    - A group that received only a 1-hour back education session (n = 122)
    - A group that received both a belt and a 1-hour education session (n = 57)

  The trial lasted 8 months, and the belt used was a fabric weightlifting belt 15 cm (6 in.) wide posteriorly and approximately 10 cm (4 in.) wide anteriorly. The researchers noted no significant differences among treatment groups for total lumbar injury incident rate, lost workdays, or workers' compensation rates. Although the lack of compliance by a significant number of subjects in the experimental group was cause for consideration, those who began wearing belts but discontinued their use had a higher lost-day case injury incident rate. In fact, 58% of workers belonging to the belt-wearing groups discontinued wearing belts before the end of the 8-month trial. Further, an increase in the number and severity of lumbar injuries occurred following the trial in those who previously wore belts.
- Mitchell and colleagues (1994) conducted a retrospective study of 1,316 workers who performed lifting activities in the military. Although this

study relied on self-reported physical exposure and injury data over 6 years prior to the study, the authors did note that the costs of back injuries that occurred while wearing a belt were substantially higher than the costs of injuries sustained while not wearing a belt.

- Kraus and colleagues (1996), in a widely reported study, surveyed the low back injury rates of nearly 36,000 employees of the Home Depot stores in California from 1989 to 1994. During this study period, the company implemented a mandatory back belt use policy. Injury rates were recorded. Even though the authors claimed that belt wearing reduced the incidence of low back injury, an analysis of the data and methodology suggested that a much more cautious interpretation may be warranted. The data showed that whereas belt wearing reduced the risk in younger males and those older than 55, belt wearing appeared to increase the risk of low back injury in men working longer than 4 years by 27% (although the large confidence interval required an even larger increase for statistical significance) and in men working less than 1 year. However, of greatest concern is the lack of scientific control to ferret out the true belt-wearing effect: there was no comparable non-belt-wearing group, which is critical given that the belt-wearing policy was not the sole intervention at Home Depot. For example, over the period of the study, the company increased the use of pallets and forklifts, installed mats for cashiers, implemented postaccident drug testing, and enhanced worker training. In fact, a conscious attempt was made to enhance safety in the corporate culture. This was a large study, and the authors deserve credit for the massive data reduction and logistics work. However, despite the title and claims that back belts reduce low back injury, this uncontrolled study cannot answer the question about the effectiveness of belts.
- Wassell and colleagues (2000), in response to the huge promotion of the Kraus and colleagues (1996) paper by some interest groups and the study's methodological concerns, replicated Kraus and colleagues' work under the sponsorship of NIOSH. These researchers, however, used better scientific control to evaluate only the effect of the belts. Surveying over 13,873 employees at newly opened stores of a major retailer, in which 89 stores required employees to wear belts and 71 stores had only voluntary use, they discovered that the belts failed to reduce the incidence of back injury claims. This study has more power than the

Kraus study.

- Calmels and colleagues (2009) provided people with recent-onset back pain with belts with metal stays, which fall into the brace, or corset, category. Subjects showed a very modest reduction in pain scales, on average, over the subsequent 90 days. The standard deviation was larger than the main effect, suggesting that the belt really helped some and not others. This is consistent with past studies.

In summary, difficulties in executing a clinical trial are acknowledged. The Hawthorne effect is a concern, because it is difficult to present a true double-blind paradigm to workers (those who receive belts certainly know so). In addition, logistical constraints on duration, diversity in occupations, and sample size are problematic. However, the data reported in the better-executed clinical trials cannot support the notion of the universal prescription of belts to all workers involved in the manual handling of materials to reduce the risk of low back injury. Weak evidence suggests that those already injured may benefit from belts (or molded orthoses) with a reduced risk of injury recurrence. However, evidence does not appear to support uninjured workers' wearing belts to reduce the risk of injury; in fact, the risk of injury seems to increase following a trial of belt wearing. Finally, some evidence suggests that the cost of a back injury may be higher in workers who wear belts than in workers who do not.

Biomechanical Studies

Researchers who have studied the biomechanical issues of belt wearing have focused on spinal forces, intra-abdominal pressure (IAP), load, and range of motion. The most informative studies are reviewed in this section.

IAP and Low Back Compressive Load

Biomechanical studies have examined changes in low back kinematics and posture in addition to issues of specific tissue loading. Two studies in particular (Harman et al., 1989, and Lander, Hundley, and Simonton, 1992) suggested that wearing an abdominal belt can increase the margin of safety during repetitive lifting. Both of these papers reported ground reaction force and measured IAP while subjects repeatedly lifted barbells. Both reports observed an increase in IAP in subjects who wore abdominal belts. These researchers assumed that IAP is a good indicator of spinal forces, which is highly contentious. Nonetheless, they assumed that the higher recordings of IAP indicated an increase in low back support that in their view justified the use of belts. Neither study measured or calculated spinal loads.

Several studies have questioned the hypothesized link between elevated IAP and reduction in low back load. For example, using an analytical model and data collected from three subjects lifting various magnitudes of loads, McGill and Norman (1987) noted that a buildup of IAP required additional activation of the musculature in the abdominal wall, resulting in a net increase in low back compressive load and not a net reduction of load, as researchers had previously thought. In addition, Nachemson, Andersson, and Schultz (1986) published some experimental results of directly measured intradiscal pressure during the performance of Valsalva maneuvers, documenting that an increase in IAP increased, not decreased, the low back compressive load. Therefore, the conclusion that an increase in IAP due to belt wearing reduces compressive load on the spine seems erroneous. In fact, such an increase may have no effect or may even increase the load on the spine.

IAP and Low Back Muscles

Several studies have put to rest the belief that IAP affects low back extensor activity. McGill and colleagues (1990) examined IAP and myoelectric activity in trunk musculature while six male subjects performed various types of lifts either wearing or not wearing an abdominal belt (a stretch belt with lumbar support stays, Velcro tabs for cinching, and suspenders for when subjects were not lifting). Wearing the belt increased IAP by approximately 20%. Further, the authors hypothesized that if belts were able to support some of the low back extensor moment, one would expect to measure a reduction in extensor muscle activity. There was no change in activation levels of the low back extensors or in any of the abdominal muscles (rectus abdominis or obliques).

In a study of the effect of belts on muscle function, Reyna and colleagues (1995) examined 22 subjects for isometric low back extensor strength. They found that belts provided no enhancement of function (although this study was only a 4-day trial and did not examine the effects over a longer duration).

Over a 4-week period, Ciriello and Snook (1995) examined 13 men lifting 29 metric tons in 4 hours twice a week both with and without a belt. Median frequencies of the low back electromyographic signal (which is sensitive to local muscle fatigue) were not modified by the presence or absence of a back belt, strengthening the notion that belts do not significantly alleviate the loading of back extensor muscles. Once again, this trial was not conducted over a very long period of time.

Belts and Range of Motion Restriction

In 1986 Lantz and Schultz observed the kinematic range of lumbar motions in subjects wearing low back orthoses. Although they studied corsets and braces rather than abdominal belts, they did report restrictions in the range of motion, although the restricted motion was minimal in the flexion plane.

In another study McGill, Seguin, and Bennett (1994) tested the flexibility and stiffness of the lumbar torsos of 20 male and 15 female adult subjects, both while they wore and did not wear a 10 cm (4 in.) leather abdominal belt. The stiffness of the torso was significantly increased about the lateral bend and axial twist axes when subjects wore belts but not when subjects were rotated into full flexion. Thus, these studies seem to indicate that abdominal belts help restrict the range of motion about the lateral bend and axial twist axes, but do not have the same effect when the torso is forced in flexion, as in an industrial lifting situation.

Posture of the lumbar spine is an important issue in injury prevention for several reasons. In particular, Adams and Hutton (1988) showed that the compressive strength of the lumbar spine decreases when people approach the end range of motion in flexion. Therefore, if belts restrict the end range of motion, one would expect the risk of injury to be correspondingly decreased. Although the splinting and stiffening action of belts occurs about the lateral bend and axial twist axes, stiffening about the flexion–extension axis appears to be less.

A more recent data set presented by Granata, Marras, and Davis (1997) supports the notion that some belt styles are better at stiffening the torso—namely, the taller elastic belts that span the pelvis to the rib cage. Furthermore, these authors also documented that a rigid orthopedic belt generally increased the lifting moment, whereas the elastic belt generally reduced spinal load. Nevertheless, the authors noted a wide variety in subject response. (Some subjects experienced increased spinal loading with the elastic belt.) Even in well-controlled studies, belts appear to modulate lifting mechanics in some positive ways in some people and in negative ways in others.

Studies of Belts, Heart Rate, and Blood Pressure

Hunter and colleagues (1989) monitored the blood pressure and heart rate of five males and one female performing deadlifts and one-arm bench presses and riding bicycles while wearing and not wearing a 10 cm (4 in.) weight belt. Subjects were required to hold in a lifting posture a load of 40% of their maximal weight in the deadlift for 2 minutes. The subjects were required to breathe throughout the duration so that no Valsalva effect occurred. During the lifting exercise, blood pressure (up to 15 mmHg) and heart rate were both significantly higher in subjects wearing belts. Given the relationship between elevated systolic blood pressure and an increased risk of stroke, Hunter and colleagues (1989) concluded that people who may have cardiovascular system compromise are probably at greater risk when undertaking exercise while wearing back supports than when not wearing them.

Subsequent work conducted in our own laboratory (Rafacz and McGill, 1996) involved the investigation of the blood pressure of 20 young men performing sedentary and very mild activities both with and without a belt. (The belt was the elastic type with suspenders and Velcro tabs for cinching at the front.) Wearing this type of industrial back belt significantly increased diastolic blood pressure during quiet sitting and standing both with and without a handheld weight, during a trunk rotation task, and during a squat lifting task. Evidence increasingly suggests that belts increase blood pressure!

Over the past decade I have been asked to deliver lectures and participate in academic debates on the back belt issue. On several occasions occupational medicine personnel have approached me after hearing of the effects of belts on blood pressure and IAP and have expressed suspicions that long-term belt wearing at their particular workplace may possibly be linked with more incidents of varicose veins in the testicles, hemorrhoids, and hernias. As of this writing, there has been no scientific and systematic investigation of the validity of these suggestions. Rather than wait for strong scientific data to either lend support to these ideas or dismiss them, it may be prudent to simply state concern. This will motivate studies in the future to track the incidence and prevalence of these pressure-related disorders to assess whether they are indeed linked to belt wearing.

Additional studies have examined the effect of belt wearing on other physiological phenomena. Bobick and colleagues (2001) reported lower mean oxygen consumption, and Parker and colleagues (2000) reported reductions in lung ventilation tidal volumes in groups with higher abdominal fat, but increased tidal volumes in groups with lower fat. Collectively, these data would have implications regarding lower work rates during belt wearing, or modulated lung function that would affect performance, or both. Once again, the effect of belt wearing appears to depend on the characteristics of the worker, making it difficult to justify a single, universal belt-wearing policy or guideline.

Psychophysical Studies

Some have expressed concern that wearing belts fosters an increased sense of security that may or may not be warranted. Studies based on the psychophysical paradigm allow workers to select weights that they can lift repeatedly using their own subjective perceptions of physical exertion. McCoy and colleagues (1988) examined 12 male college students while they repetitively lifted loads from the floor to knuckle height at the rate of three lifts per minute for 45 minutes. They repeated this lifting bout three times, once without a belt and once each with two types of abdominal belts: a belt with a pump and air bladder posteriorly and the elastic stretch belt described in the McGill, Norman, and Sharratt (1990) study. After examining the magnitudes of loads that subjects had selected to lift in the three conditions, the researchers noted that wearing belts increased the load that subjects were willing to lift by approximately 19%. This evidence may lend some support to the theory that belts give people a false sense of security.

Summary of Back Belt Prescription Guidelines

My earliest report on back belts (McGill, 1993) presented data and evidence that neither completely supported nor condemned the wearing of abdominal belts for industrial workers. After more laboratory studies and field trials, my position (which has been implemented by several governments and corporations) has not changed.

Given the available literature, it would appear that the universal prescription of belts (i.e., providing belts to all workers in an industrial operation) is not in the best interest of globally reducing both the risk of injury and compensation costs. Uninjured workers do not appear to receive any additional benefit from belt wearing and, in fact, may be exposing themselves to the risk of a more severe injury if they were to become injured. Moreover, they may have to confront the problem of weaning themselves from the belt. However, if some workers perceive a benefit from belt wearing, they should be allowed to wear a belt conditionally but only on trial. The mandatory conditions for prescription (for which there should be no exception) are as follows:

- Given the concerns regarding increased blood pressure and heart rate and issues of liability, all candidates for belt wearing should be screened for cardiovascular risk by medical personnel.
- Given the concern that belt wearing may provide a false sense of security, belt wearers must receive education on lifting mechanics (back school). All too often, belts are promoted to industry as a quick fix to the injury problem. The promotion of belts, conducted in this way, is detrimental to the goal of reducing injury because it redirects the focus from the cause of the injury. Education programs should include information on how tissues become injured, techniques to minimize musculoskeletal loading, and what to do about feelings of discomfort to avoid disabling injury.
- Consultants and clinicians should not prescribe belts until they have conducted a full ergonomic assessment of the person’s job. The ergonomic approach should examine and attempt to correct the cause of the musculoskeletal overload and provide solutions to reduce the excessive loads. In this way, belts should only be used as a supplement for a few people, whereas a greater plantwide emphasis should be on the development of a comprehensive ergonomics program.
- Belts should not be considered for long-term use. The objective of any small-scale belt program should be to wean workers from the belts by insisting on mandatory participation in comprehensive fitness programs and education on lifting mechanics, combined with ergonomic

assessment. Furthermore, consultants would be wise to continue their vigilance in monitoring former belt wearers for a period of time following belt wearing, given that this period appears to be characterized by an elevated risk of injury.

LBD Prevention for Employers

Not all prevention strategies can be implemented solely by workers. Employers, too, play a role, which is outlined in the following guidelines.

- Provide protective clothing to facilitate the least stressful postures. Workers sometimes handle material that is too noxious or too hazardous to hold close to their bodies. For example, I have been involved in cases in which workers held dirty material away from the body to keep their shirts clean, unnecessarily loading their backs. The solution was to provide protective coveralls to spare workers' backs. Leather aprons are helpful if the material is sharp to foster holding the load against the abdomen, as for sheet metal workers, for example. Knee pads are good for prolonged work on the ground. Once the employer has provided the necessary protective clothing, workers will figure out the variety of working postures that can spare their backs.
- Should abdominal belts be prescribed to manual materials handlers? Employers are encouraged to follow the prescription guidelines in the previous section of this chapter.
- Optimize containers and packaging of raw material to spare workers' backs. Often in the design of the industrial process, employers don't consider sparing workers' joints. When considering the industrial process, employers should consider whether handled materials can be bulk containerized. Can raw material be handled in smaller bundles or in bundles of different dimensions? Sometimes the matter is as simple as contacting the suppliers to find alternate ways of packaging material that foster handling in a way that conserves the body joints. The purchasing department can play a role in reducing injury! Bins and containers with folding sides help if parts must be picked up out of bins.
- Encourage workers to practice lifting and work, or task, kinematic patterns. Some people simply do not move, bend, and work in ways that spare their spines. In a recent study (McGill et al., 2003), we noted that workers who had had a history of back troubles were more likely to adopt motion patterns that resulted in higher spine loads! Kinematic patterns need to be practiced and grooved into movement repertoires. (Remember the expression quoted earlier: Practice does not make

perfect; it makes permanent.) Some people must practice the spine-conserving motions every day—especially before attempting a particularly strenuous task. Even high-performance athletes must continually regroove motion patterns daily. Some worker groups have attempted to fabricate their own job-specific training and practice equipment. The dummy sitting in a wheelchair in figure 7.30 is an example of this type of worker professionalism. Nurses built the dummy from plumbing pipes and use it to practice one- and two-person patient lifts.

Figure 7.30 Nurses at a patient care facility fabricated this dummy to practice their patient lifts—an example of worker professionalism.

Photos from Stuart McGill

- Optimize worker rest breaks. A properly designed rest break consists of the opposite activity (and consequently the opposite loading) from that required by the job. For example, the sedentary secretary is best served by a dynamic rest break. The welder, on the other hand, is better served with rest and perhaps a stretch. The following example illustrates a violation of this principle that caused grief. Back in the 1960s, operators in a power plant monitored the process from a chair and had to respond to a vigilance buzzer on their desks that went off every 10 minutes (see figure 7.31, a and b). At each buzzer interval they would stand and walk around the control panel making adjustments. There was no history of back troubles. The room recently became obsolete and was replaced with an updated control room. The design team for the new control room believed that rising from the chair every 10 minutes was too strenuous.

Consequently, the job was redesigned so that the operators were able to stay seated to perform all operations. These operators worked 12-hour shifts. Having the workers sit for this period of time (and removing the need to get out of the chair regularly) resulted in an increase in low back problems. The power plant then hired a consulting group that recommended rest breaks that consisted of having the workers sit on an exercise bike. The workers' backs failed to improve because the consultants did not understand that the rest break must not exacerbate or replicate the forces of the job. In this case, sitting on a bike was not a break from sitting on the job.

Figure 7.31 (a) The first control room was built in the 1960s and required the operator to stand every 10 minutes to respond to the vigilance buzzer and make an adjustment to the analog instrumentation. There were no reported back troubles. (b) This room was replaced with a new layout based on the designers' assumption that standing every 10 minutes was too strenuous; the operators were able to sit for hours. A huge low back problem emerged.

Photos from Stuart McGill

- Involve workers in the ergonomic process. Design teams often neglect to consult with the expert on a job—the worker who has done it for years. Quite often, the worker knows of a good solution, and it is simply a matter of listening and facilitating the intervention. An added benefit, psychologists claim, is that workers are more likely to comply with the intervention if they perceive themselves to be a major part of the solution.
- Design work to be variable. As several previous examples have documented, accumulation of tissue stress is thwarted by a change in posture, loading, or activity. Human beings were not made to perform repetitive work that emphasizes only a few tissues. Nor were humans

designed not to be stressed. Research has established that tissues adapt and remodel in response to load (e.g., bone: Carter, 1985; ligament: Woo, Gomez, and Akeson, 1985; disc: Porter, 1992; vertebrae: Brinckmann, Biggemann, and Hilweg, 1989). Too little activity can be as problematic as too much. Krismer and colleagues' study (2001) strongly reinforces the idea so frequently stated in this text that the object of good work design is not to make every job easier; in fact, some jobs should be made more demanding for optimal health. In the Krismer study, students who reported low back pain were distinguished from those who did not by several risk factors. Two of those factors were as follows:

- Watching TV or playing computer games more than 2 hours per day (sedentary)
- Regularly going beyond personal limits in sport activities (too much loading)

Good occupational health from a musculoskeletal perspective is achieved when people perform a variety of tasks with well-designed rest activities, along with all of the traditional components such as proper nutrition, adequate sleep, and avoidance of smoking. Design work to be variable!

As illustrated in the previous examples and guidelines, management can play a role in reducing back troubles in workers. It is a mistake to think that management does not need to understand the science to justify specific injury prevention approaches. As we saw previously, injury prevention involves a thorough understanding of the industrial process, the way goods and materials are purchased, protective equipment, appropriate training, the costs and benefits of intervention, and so forth. Training of both workers and management ensures the best results.

Injury Prevention Primer

To use biomechanics to its full potential in injury prevention, workers and employers must have a reasonable understanding of the concepts as we understand them today. Workers must be educated in the biomechanically justifiable principles described earlier using examples with which they are familiar. Armed with the general principles, they can tackle any job and devise the best joint-conserving strategies. The intention is to enhance the industrial process and enable workers to retire in good health. The highlights of this chapter are summarized here:

- First and foremost, design work and tasks that facilitate variety. Perhaps the most important guideline should be this: Don't do too much of any one thing. Both too much and too little loading are detrimental.
    - Too much of any activity leads to trouble. Relief from cumulative tissue strains is accomplished with posture changes or, better yet, other tasks that have different musculoskeletal demands.
    - Although the tasks of many jobs cannot be changed, the working routines and arrangement of tasks within a job can be designed scientifically to incorporate this principle. Sometimes it is as easy as continually changing the sitting posture.
- Spine power: Keep it low. Power is the product of force and velocity. High power damages discs.
    - If the force on the spine is high, the movement velocity (bending velocity) must be low, ideally zero.
    - If the movement velocity is high, then force must be low. In contrast, the hip joints were designed to generate and sustain power.
- During all loading tasks, avoid a fully flexed or bent spine and rotate the trunk using the hips (preserving a neutral curve in the spine). Doing this has the following benefits:
    - Disc herniation cannot occur.
    - Ligaments cannot be damaged, because they are slack.
    - The anterior shearing effect from ligament involvement is minimized, and the posterior supporting shear of the musculature is maximized.

    - Compressive testing of lumbar motion units has shown increases in tolerance with partial flexion but a decreased ability to withstand compressive load at full flexion.
- During lifting, choose a posture to minimize the reaction torque on the low back (either stoop or squat or do something that is somewhere in between), but keep the external load close to the body.
    - A neutral spine is still maintained, but sometimes the load can be brought closer to the spine with bent knees (squat lift) or relatively straight knees (stoop lift). The key is to reduce the torque, which has been shown to be a dominant risk factor.
    - When exerting force with the hands or shoulders, try to direct the line of the force through the low back. This will reduce the reaction torque and the spine load from muscle contraction.
- Consider the transmissible vector. Attempt to direct external forces through the low back to minimize the moment arm, which causes high torques and crushing muscle forces. This principle should be applied when applying pushing and pulling forces while opening a door, when vacuuming, and when performing other household chores.
- Use techniques that minimize the weight of the load being handled. The log-lifting example given in this chapter demonstrates how to lift an entire log into the back of a truck by lifting no more than half of its weight at any point.
- Balance loads between both hands. Carrying a load in one hand creates more spine load than carrying the same load in both hands—and doubling the total weigh carried (McGill, Marshall, and Andersen, 2013). (See figure 4.17 in chapter 4.)
- Allow time for the disc nucleus to equilibrate, the ligaments to regain stiffness, and the stress on the annulus to equalize after prolonged flexion (e.g., sitting or stooping), and do not immediately perform strenuous exertions.
    - After prolonged sitting or stooping, spend time standing.
    - This principle can be adapted to many jobs, but some workers do not have the luxury of being able to take the time to allow the disc nucleus to equilibrate. For example, ambulance drivers are often called on to lift heavy loads immediately after significant periods of driving. A strategy for them is to use a lumbar support in the seat while driving to the incident so that the spine is not flexed. Thus, it

can be prepared for the load with minimal disc equilibration (part of the process of warm-up).

- Avoid lifting or spine bending shortly after rising from bed.
    - Forward-bending stresses on the disc and ligaments are higher after rising from bed compared with later in the day (at least 1 or 2 hours after rising), causing discs to become injured at lower levels of load and degree of bending.
    - This principle is problematic for some occupations such as firefighting, in which workers are often aroused from sleep to attend a fire. Such workers should not sit in a slouched posture with the spine flexed when traveling to the scene, but rather, sit upright. In this way the spine will be best prepared for strenuous work without a warm-up.
- Prestress and stabilize the spine even during light tasks.
    - Lightly cocontract the stabilizing musculature to remove the slack from the system and stiffen the spine even during light tasks such as picking up a pencil. The exercises shown in chapter 10 were chosen to groove these motor patterns.
    - Mild cocontraction and the corresponding increase in stability increase the margin of safety of material failure of the column under axial load.
- Avoid twisting and simultaneously generating high twisting torques.
    - Twisting reduces the intrinsic strength of the disc annulus by disabling some of its supporting fibers while increasing the stress in the remaining fibers under load.
    - Because no muscle is designed to produce only axial torque, the collective ability of the muscles to resist axial torque is limited, and they may not be able to protect the spine in certain postures. The additional compressive burden on the spine is substantial for even a low amount of axial torque production.
    - Generating twisting torque with the spine untwisted may not be as problematic, nor is twisting lightly without substantial torque.
- Use momentum when exerting force to reduce the spine load (rather than always lifting slowly and smoothly, which is an ill-founded recommendation for many skilled workers).
    - This is a skill that sometimes needs to be developed.
    - This strategy is dangerous for heavy loads and should not be used

for lifting them.
    - It is possible that a transfer of momentum from the upper trunk to the load can start moving an awkwardly placed load without undue low back involvement.
- Avoid prolonged sitting.
    - Prolonged sitting is associated with disc herniation.
    - When required to sit for long periods, adjust posture often, stand up at least every 50 minutes, extend the spine, and, if possible, walk for a few minutes.
    - Organize work to break up bouts of prolonged sitting into shorter periods that are better tolerated by the spine.
- Use good rest break strategies. Customize this principle for various job classifications and demands.
    - Workers engaged in sedentary work are best served by frequent, dynamic breaks to reduce tissue stress accumulation.
    - Workers engaged in dynamic work may be better served by longer and more restful breaks.
- Use protective clothing to foster joint-conserving postures. Use coveralls for dirty material handling, heavy aprons for sharp metals, knee pads for working at ground level, and so on.
- Practice joint-conserving kinematic movement patterns. Some workers need to constantly regroove motion patterns such as locking the lumbar spine when lifting and rotating about the hips.
- Maintain a reasonable level of fitness. While it is betterto be undertrained than overtrained, a moderate level of fitness is better than poor fitness.
- Combine these guidelines for special situations. For example, some people have difficulty rolling over in bed because of back pain. Nearly everyone with back pain can be taught to manage pain and still accomplish this task by combining a momentum transfer with the minimal twisting guidelines (see figure 7.32, a-d).

Figure 7.32 Rolling over in bed can be taught to those who maintain that doing so is too painful. The figure illustrates rolling from the left side to the right. (a) While lying on one side, the patient braces the torso so that the spine does not twist in the steps that follow. (b) Then the upper arm and leg are raised together with the lower arm and leg prying off the floor. (c) This is performed quickly enough to generate momentum that will carry the patient through the roll. (d) The patient should now be resting comfortably on the other side.

Photos from Stuart McGill

- In cases of morning stiffness, consider the bed. Some people with spine joints that generate pain when in a particular posture benefit from a mattress that fosters pain-free positioning. Zhang and colleagues (2009) reported that bed characteristics, together with spine loading, were the most predictive elements of those diagnosed with disc herniation. There are no rules to guide mattress selection, although Haex (2004) suggested that those with angular body types generally do better with firm mattresses covered by a soft pillow top. Haex also reported evidence that the amount of standing lumbar lordosis helped with bed selection. For example, they reported that those in Asian cultures, typified with less lordosis, find comfort with covered futons made of wood slats. Europeans, who tend to have more lordosis, found these less comfortable. Memory foam mattresses are very comfortable for some, particularly those who do not change position often. Rolling up and over the shaped foam, however, disturbs those who need to change position more often.
- Suggest the best positions for sexual activity. Primary contact physicians see a number of couples who have long periods of celibacy because of long-lasting back pain following coitus. Physicians have no guidelines to make recommendations. Our pioneering work ranked various positions for spine load (Sidorkewicz and McGill, 2014a, 2014b). In general, we recommend perfecting the hip hinge rather than spine bending, and avoiding a deviated static spine posture.

A Note for Consultants

Acting as a consultant, I have made many mistakes, some of which motivated the following tips.

- Don't fall into the trap of thinking that you are the expert and know what is best for a worker (unless you have done the job for years yourself). Always consult the worker. Successful workers have developed personal strategies for working that assist them in avoiding fatigue and injury. Their insights are the result of thousands of hours of performing the task, and they can be very perceptive. Try to accommodate them.
- Do not take the instructions for a specific worker verbatim from the preceding Injury Prevention Primer. Instead, explain the relevant biomechanical principle in language and terminology that are familiar to the worker.
- Do not focus only on the most demanding tasks. Given the links among different tasks from a tissue load perspective, you can often obtain better solutions by considering the full complement of exposures. In a similar vein, some consultants tend to focus on a single metric of risk (e.g., low back compression) or rely on only a few simple solutions. The average ergonomist probably does not have the training necessary for achieving the best solutions for low back problems. Perhaps I am biased because in recent years I have been asked to consult only when consultants' poorly conceived ergonomic approaches have failed. I am requested to become involved when the company faces lawsuits or other issues that have raised the stakes. Remember that many solutions are neither simple nor unidimensional, regardless of your training. Use the Injury Prevention Primer as a checklist to evaluate whether better and more comprehensive solutions are available or possible.
- Do not focus exclusively on the musculoskeletal issue. Rather, look for the opportunities that lie in enhancing the industrial process. Any management board will recognize the worth of a consultant who makes the process more efficient, produces a higher-quality product, or reduces injury compensation costs.
- Movement is taught more effectively when it is considered a complex

movement skill. Simply having an expert (i.e., yourself) demonstrate more effective work techniques will usually fail. Coaching the movement and motor patterns in an interactive session leads to performance enhancement and greater safety (see McGill, 2014, for a full overview of this process).

- Never make a recommendation that is not feasible to implement, for monetary or any other reason.
- Finally, consider becoming a job coach—a person with expertise in ergonomics and reducing the external load applied to the body. Job coaches also have expertise in personal body mechanics and movement patterns needed to further reduce tissue loads and enhance load tolerance. Our consulting experience shows that the payback, in terms of injury costs, far outweighs the expense of setting up the job coach approach.

Reducing the Risk in Athletes

Although this chapter has focused on occupational back injury prevention, these principles are also appropriate for athletic situations. Athletes and teams from a variety of sporting activities—from world-class professionals to amateurs—have sought my advice as a low back injury consultant. In many cases their painful and disabled backs were ending their careers. But as we have seen in preceding chapters, success in dealing with back disorders requires efforts to address both the cause of the troubles and the most appropriate rehabilitative therapy. In many cases, addressing the cause meant that athletes had to change their techniques. Without exception, they had to change the way they trained. Their backs were breaking down for a reason! As loads applied to the body reach world-class levels, technique must be impeccable. Techniques for athletes are well covered in my other textbook, Ultimate Back Fitness and Performance (McGill, 2014).

When I am asked to help a team reduce back injuries, I follow an approach I have developed over the years:

- Understand the challenges. Collect data on players and the team. Injury data together with performance data need to be mined for patterns and relationships. These reveal mechanisms that can be addressed. The more detail, the better. For injuries, we want to know details regarding diagnosis, time of the day or season of symptoms, training regimens, training/recovery schedules, peak and taper schedules, and so on. Performance statistics should include injury resilience (games without injury), points and assists scored, and minutes played, among other things.
- Devise a player assessment protocol relevant to the sport. Using the information obtained from the preceding procedure, we have a clear idea of suspected injury mechanisms and imbalances thwarting performance. We combine this information with what is already established about the science of the sport. This creates our baseline characterization of each player.
- Create a monitoring program. Each player is assessed on a schedule—one for in-season and another for out-of-season. The most successful clubs are on a 3-week schedule during the in-season. The testing is

minimally invasive and obtrusive but includes biomechanical, physiological, neurological and psychological elements. This reveals fatigue that leads to injury, player distress causing detriments in play, and so on. The intention is to keep the machine well tuned and injury-free.

- Follow up to assess program efficacy. Here team and player results are compared to those from previous years.

We have assessed the ability of commercial approaches to predict performance and injury resilience. None have lived up to their claims. Athletes have bodies suited to their sports. For example, basketball players are unique in that their height and different body segment proportions do not allow them to pass traditional movement screens. Their long legs simply do not allow a passing grade on a squat. In terms of physiology, explosive players, for example, can only retain their athleticism for a short period. Otherwise, they would rapidly degrade to ordinary performance and higher risk. These special athletes prosper with shorter playing time intervals.

For athletes in individual sports, I assess a game or two and chart their demands. For example, for a mixed martial arts athlete, I would catalog the amount of time spent in an upright posture, the number of fast, explosive strikes, the time spent in isometric control, and so forth. Then I would devise a training program to meet the demands in a spine- and joint-sparing way.

A Final Note

The practitioners most successful in reducing back injury and disability determine the cause and address it prior to prescribing therapeutic interventions. Use this chapter to guide you.

Part III
Low Back Rehabilitation

This part of the book presents better rehabilitation practices based on the evidence discussed in previous chapters. The chapters in part III discuss evaluating patients and developing beginner and advanced exercise and rehabilitation programs.

Chapter 8
Building Better Rehabilitation Programs for Low Back Injuries

All body systems depend on movement for health. For spine health, however, the traditional approaches for enhancing fitness must be reprioritized. For those with painful backs, attaining movement skill and competency must replace energy systems training that emphasizes cardiovascular and muscle components. Too many patients are taught that 45 minutes on the treadmill or stationary bike will rid them of pain. Also important is the notion that we are not born physical equals. A St. Bernard has little chance at the greyhound track except to become injured. But the greyhound attempting to carry a St. Bernard's load up a mountain will crumble. Both can enhance their pain-free athleticism, but they need different approaches.

Many people develop painful backs because of movement flaws. Lifting even extremely heavy loads can be accomplished safely by athletes with perfect form. But movement flaws cause repeated or prolonged loading that is abnormal for the tissue, so it slowly becomes painful. Part of the stabilization approach is to correct the aberrant patterns to metaphorically stop picking the scab. As a result, the tissues become less sensitized, the repertoire of pain-free tasks increases, and motion returns. This is why it is essential to perform therapeutic exercise pain free. The presence of pain also leads to substitution patterns as the spine literally learns to limp. These patterns must be corrected, and they require pain-free motion. So, don't worry about the concept of restoring function too soon. This retards progress. Address the painful tissues and then work on function. Choosing the correct movement tool is also necessary for back rehabilitation success.

Consider the action of rising from a chair. Clearly, a squat pattern is best. Now consider someone who must lie on the floor to perform an exercise. Should she choose a squat pattern to move to the floor? If she does, she will create large knee and low back forces, which, over time, will lead to the development of painful disorders. Instead, this person should choose a lunge, which spares the joints. For a person who has lost some of the ability to squat deeply as a result of a damaged knee or back, or advanced age, the lunge pattern makes moving to the floor possible, and pain free.

Movement competency is different for different people. For example, a construction worker needs different movements, strength exertions, postures, and endurance than a mail carrier or a bank teller. A high school student wanting to enhance her volleyball spike desires more explosive hip power and core stability, but her grandfather needs to maintain his ability to step quickly after stubbing his toe to prevent falling and breaking a hip. Both want to enhance their movement skill. However, the student's training program to maximize her movement ability would be very different from that of her grandfather.

The definition of good movement changes as we grow, mature, and age. Moving well reduces joint load, in turn reducing the risk of degenerative conditions. Moving well increases efficiency, which increases the number of repetitions that can be done before performance is compromised by fatigue or pain. Moving well also enhances performance by eliminating the weak links among all components of movement.

This book promotes the establishment of spine stability first, followed (sometimes) by spine mobility in some patients with back injuries. Although a small proportion of patients need mobilizing in a local spinal region first, most patients—from those looking for functional enhancement and pain relief to athletes seeking performance enhancement—benefit from stabilizing the spine first. In fact, once stability is established and the pain resolves, many patients find that their mobility returns with no further intervention (Moreside and McGill, 2012). Mobility elsewhere in the body linkage creates better back function. When testing back strength, remember that a good score requires a stable spine. In contrast, strong hips require good mobility. Poor hip function and control compromise back and torso function.

The evidence presented throughout this book is unanimous: A spine does not behave like a knee or shoulder, and approaches that work with these joints are often not effective for back therapy. Loading throughout the range of motion—which works well for joints in the extremities—is the nemesis of many backs, at least in the early stages of rehabilitation. During this period, training for strength is usually counterproductive. Unfortunately, the principles used in bodybuilding pervade rehabilitation clinical wisdom. This approach hypertrophies muscle at the expense of developing the functional motor and motion patterns needed for optimal health. This chapter provides several general recommendations to maximize the chance for successful rehabilitation, followed by considerations of the stages of patient progression and guidelines for developing the best exercise regimen for each patient.

Pain-Reducing Mechanisms of Exercise

How does exercise reduce back pain? The mechanism of pain reduction is different for different types of back pain. When spine joints are unstable, abnormal micromovements irritate tissues. The stabilization exercises presented here have been shown to enhance stiffness (Lee and McGill, 2015), which reduces this aberrant motion. In addition, creating proximal stability enables controlled, high-quality movement distally, reducing tissue stress.

For specific movement intolerance, the irritation is reduced simply by avoiding the exacerbating movement. Those with identified tissue entrapment can reduce the pressure and pain with specific exercises. For example, we have shown that certain exercises can return displaced nucleus-impinging nerve roots (Scannell and McGill, 2009).

As previously noted, patients do not get used to pain, and continual tissue irritation increases their pain trigger sensitivity. Here the key is to desensitize the pain trigger, which is known as winding down the sensitivity. For those with fibromyalgic overlays to their back pain, in whom virtually everything causes pain, this means finding pain-free movements and loading patterns. Their virtual body map, including pain perception, has been distorted with trauma or disease (Butler and Mosely, 2013, provide a wonderful review). Repeating these exercise patterns teaches the brain pain-free movement. This process sometimes starts with left side–right side discrimination and imagined and reflective movement followed by actual body movement. Slowly, this pain-free movement repertoire expands.

Neurophysiological mechanisms of pain are highly modulated by movement and exercise. Endorphin release has long been known to occur with exercise, but the molecular basis for these links is not well understood. However, many more exercise-induced analgesic pathways are coming to light. One example is that exercise increases neurotrophin-3 production, which appears to have an analgesic role in cutaneous and deep tissue pan (Sharma et al., 2010).

Psychological components are important for some patients and influence the approach used to introduce and program exercise. For example, some patients obsess over their pain and what is going on in their bodies. Patients with high fear avoidance do better with physical therapy, which de-emphasizes anatomical findings (George et al., 2003). For these types of patients, I suggest not using daily pain charts, but directing their focus to achieving good exercise form. Some patients have back pain because they overload themselves with daily regimens at the gym. For the type A personality, we ensure that exercise is not overexecuted (for people who believe that 25 repetitions of an exercise will do them more good than the 10 that were prescribed). For the type B personality who may bargain with the clinician about doing less exercise, performing the exercise dose as prescribed should be encouraged. These two personality types require different approaches to prescribing exercise.

Some pain is caused by what are referred to as sticky tissues. The concept that inflammation can cause sticky nerves was introduced for cases in which movement increased neural tissues' ability to slide with less pain-inducing friction. Fascial and muscular elements have been shown to get sticky and painful, and manual therapy approaches have been shown to help with this issue prior to movement exercise (Findley, 2011). Exercise has been shown to enhance pain-related ischemic conditions by promoting vascular sprouting (Wahl, Bloch, and Schmidt, 2007).

Some people have pain as a result of tissues that are inappropriately weak for the applied demands. In such cases the goal of exercise is to strengthen tissue, which requires a certain kind of programming. Athletes I have worked with who have recovered well from compression fractures have incorporated sufficient rest intervals to allow the injury to adapt between loading sessions. This appears to not work with collagenous disc injuries given the time required (years) for migrated nucleus material to gristle and form a plug (Adams and Dolan, 2005). Thus, the type of tissue that is the target for strengthening should determine the exercise approach.

Imbalances in joint and muscle function create odd stress patterns and more pain. For example, hip and back pain can cause gluteal muscle inhibition (Freeman, Mascia, and McGill, 2013), which makes the hamstring muscles dominant in creating hip extension moment. Because of the lines of action of these muscles, this causes more load on the anterior acetabulum and labrum of the hip (Lewis, Sahrmann, and Moran, 2009), resulting in associated hip pain.

There is also substantial support for the notion of the existence of a psychological profile associated with fitness—that is, that fitter people ignore small discomforts. As discussed in the section Why and How You Should Read This Book, fitness reduces the tendency to catastrophize pain.

How is the most appropriate exercise determined to reduce each of these pain mechanisms? Although the assessment will reveal the pain mechanism, many challenging patients must be considered experiments in progress. Tweaking the exercise form and the dosage helps you to hone in on the optimal.

Five-Stage Back Training Program

We have developed a five-stage approach to back training that begins with identifying faulty movement patterns and ends with pain-free performance readiness. Consider this the big picture, because only athletes and people who perform demanding tasks will complete all five stages. However, it is important to understand the components and objectives of each stage, as well as their order. For example, patients may be unknowingly following a strengthening regimen (stage 4) without having addressed perturbed motion patterns (stage 1). Doing so will delay their recovery, or make them worse. So when approaching the program, you must first ask, Is the objective pain reduction and rehabilitation or athletic performance? Health objectives demand a focus on motion and motor patterns, stability, and endurance to achieve low tissue loads and a low-risk environment. Performance requires more overload, which naturally results in an elevated risk. The trick in the case of performance is to stay within the lowest risk possible.

Although all five major stages are listed here, only the first three are appropriate for rehabilitation and addressed in this book. Rigorous strength, speed, and power training are only for those interested in enhancing these attributes. The average person does not need them to have good back health. I address more advanced athletic training in detail in my book Ultimate Back Fitness and Performance (McGill, 2014). Begin with an assessment to understand the pain mechanisms. Then use the appropriate approach to address the mechanisms and build the person's tolerance and work capacity. Here is a summary of all five training stages:

- Stage 1: Establish quality movement engrams (motion patterns, motor patterns, and corrective exercise).
    - Identify perturbed patterns and develop appropriate corrective exercise.
    - Address basic movement patterns through to complex activity patterns.
    - Address basic balance challenges through to complex and specific balance challenges.
- Stage 2: Build whole-body and joint stability (focus on spine stability

here).
    - Build stability while sparing the joints.
    - Ensure sufficient stability commensurate with the demands of the task.
    - Apply these patterns to the performance of daily activities.
- Stage 3: Increase endurance.
    - Address basic endurance training to ensure the capacity needed for stabilization.
    - Address activity-specific endurance (duration, intensity).
    - Increase the ability to repeat pristine movement patterns without fatigue-induced compromise.
    - Build the base for eventual performance training (only in those with this goal).
- Stage 4: Build strength.
    - Spare the joints while maximizing the neuromuscular compartment challenge.
    - Progress to skill movements.
- Stage 5: Develop speed, power, and agility.
    - Develop ultimate performance based on the foundation laid in stages 1 through 4.
    - Focus on optimizing elastic energy storage and recovery.
    - Employ the techniques of superstiffness.

If you master the first three stages, which are discussed in this chapter, you will understand how to get bad backs to respond and how to develop better rehabilitation programs.

Finding the Best Approach

Given the wide variety of patients with low back issues, we cannot expect to succeed in low back rehabilitation by treating everyone with the same cookbook program. The following strategies will help guide clinical decisions to individualize—and thus optimize—the rehabilitation program.

- Encourage patients to train for health versus performance. The notion that athletes are healthy is generally a myth, at least from a musculoskeletal point of view. Training for superior athletic performance demands substantial overload of the muscles and tissues of the joints. An elevated risk of injury is associated with athletic training and performance. Unfortunately, many patients observe the routines used by athletes to enhance performance and wrongly conclude that copying them will help their own backs. Training for health requires quite a different philosophy; it emphasizes muscle endurance, motor control perfection, and the maintenance of sufficient spine stability in all expected tasks. Although strength is not a goal, strength gains do result. If a patient with back pain states that his objective is to play tennis or golf, then he has the wrong short-term objective. First and foremost, the objective is to eliminate pain. Then the objective may shift toward a performance objective such as participation in a sporting activity.
- Integrate prevention and rehabilitation approaches. The best therapy rigorously followed will not produce results if the cause of the back troubles is not addressed. Part II provided guidelines for reducing the risk of back troubles: the importance of removing the cause of tissue overload cannot be overstressed. Linton and van Tulder (2001) demonstrated the efficacy of exercise for prevention; exercise satisfies the objective for both better prevention and better rehabilitation outcomes. First, teach patients what is causing their troubles; then work with them to eliminate the cause.
- Work toward a slow, continuous improvement in function and pain reduction. The return of function and the reduction of pain, particularly for those with chronic bad backs, is a slow process. The typical pattern of recovery is akin to that of the stock market. Daily, and even weekly, price fluctuations eventually result in higher prices. Patients have good

days and bad days. Many times lawyers have hired private investigators to make clandestine videos of people with back troubles performing tasks that appear inconsistent with those troubles. I am hired to provide comment. Some of these people are true malingerers and get caught. Others are simply having a good day when they are video-recorded. In such cases, I see all sorts of movement pathology consistent with their chronic history, and they are exonerated.

- Have the patient keep a journal of daily activities. Sometimes it is difficult to hone in on the pain mechanism and the correct dosage and exercise form. Examining daily pain and activity patterns can help identify the link with mechanical scenarios that exacerbate the pain. Two critical components should be recorded in a daily journal: how the back feels and what tasks and activities were performed. When patients encounter repeated setbacks, they should try to identify a common task or activity that preceded the pain episode. Likewise, even when progress is slow, patients should be encouraged to see some progress nonetheless. Without referring to the diary, patients sometimes do not realize that they are improving. Linking pain with a dose of activity is different from recording pain on a 10-point scale, which is typical of behavior modification programs. I have seen too many patients from these programs obsessing over their pain levels; for these people, we suggest stopping pain recording.
- Ensure a positive slope in progress. Chapters 10 and 11 introduce the big three exercises in different forms. We designed these exercises to spare the spine from large loads and to groove stabilizing motor patterns. Use the three to establish a positive slope in patient improvement. Once the slope is established, you may choose to add new exercises one at a time. The patient may tolerate some exercises well and others not so well. If the improvement slope is lost after adding a new activity, remove it, go back to the big three, and reestablish the positive slope. If the patient requires advanced exercises for athletic performance, perhaps to increase spine mobility, you may add exercises to achieve such objectives after establishing the positive slope. How long should each stage be? There is no single answer for everyone. Some progress quickly, whereas others require great patience. Your job is to determine the initial challenge, to gauge progress and enhance the challenge accordingly, and to keep the patient motivated, even during periods of

no apparent progress. The great clinicians blend keen clinical skills and experience with scientifically founded guidelines and knowledge.

- Determine whether the patient is willing to make a change. Obviously, the patient must change the current patterns that caused her to become a back patient. This requires motivation, which is not always easy to establish. Some have listed the importance of, and steps for developing, a change in motivation and attitude (e.g., Ranney, 1997). Briefly, such a program begins with the setting of goals—for example, returning to a specific job or partaking in a leisure activity. The employer's role in enhancing motivation is to ensure that modified work is available together with the opportunity for a graduated return to duty. Employers can also enhance motivation by fostering a culture in which worker success equates to company success, which in turn helps the worker. The second step in a motivation program is to formulate a realistic plan for reaching the goal established in the first step. It is beyond the mandate of this book to develop the components of maintaining and enhancing motivational opportunities at each stage of recovery.
- Determine whether the patient needs initial mobilization. Although everyone should incorporate spine stabilization exercises into daily activity, a small group of people will benefit from some directed soft tissue work (e.g., manipulation, trigger point therapy, Active Release Techniques, the use of foam rollers). These techniques are not the focus of this book. A word of caution is required here. Too many make the mistake of trying to mobilize a painful spine region that already has mobility. Nonetheless, there is good evidence that those with documented hypomobility may benefit from some initial manipulation or mobilization with a transition into stabilization training (Fritz, Whitman, and Childs, 2005).
- Consider other soft tissue treatments. A good manual medicine clinician may perceive local muscle spasms and odd-feeling local muscle texture. Further, these spasms and local neurocompartment disorders are associated with larger dysfunctions of the agonist and synergist muscles involved in a movement. In many cases these dysfunctions delay recovery or prevent complete recovery. Clinicians use a variety of soft tissue treatments to reduce spasm and release tissues that can impede attaining more normal muscular and joint function. Documenting them is beyond the scope of this book. We simply alert you to their potential

significance and role in rehabilitation.

- Avoid spine power. Spine power is the product of velocity and force (power = force × velocity). This means that the spine is bending quickly and there is velocity in the muscles' lengthening and shortening. Techniques that involve high velocity in the spine have been shown to lead to back troubles, because they usually indicate high power (Marras et al., 1993; Stevenson et al., 2001). To minimize power and maximize safety, the forces transmitted through the trunk must be low if the spine is moving. If the forces transmitted through the trunk are high, then the velocity must be low. The power must be generated at the hips and shoulders and transmitted through an isometrically stabilized torso. Fortunately, this fundamental tenet for safety also helps to maximize performance.

Stages of Patient Progression

Before we can undertake to remove the activities that exacerbate low back troubles, we must determine what they are. This is a crucial part of the rehabilitation process. Uncovering the activities that cause back disorders begins with a patient interview. Some clinicians perform provocative testing at this time as well. Chapter 9 thoroughly discusses how to interview and test the patient. Once this has been done, rehabilitation can proceed. You may choose to overlap the stages involved in this process or conduct them in parallel. At all times, however, the objective is to establish and maintain the positive slope of continual improvement.

Most patients with chronic conditions do well with the universal principles of spine health: Move in a spine-sparing way throughout the day; develop appropriate spine and torso stiffness to eliminate painful spine micromovements; mobilize the hips; enhance endurance to maintain these pristine patterns throughout the day; know postures of comfort when you have overdone it; and know how and when to employ the movement techniques for a push, pull, lift, carry, squat, and lunge.

People in acute pain learn to settle their pain with pristine movements and by adopting appropriate pain-free postures (e.g., spine extension postures for posterior disc bulges). Finally, some conditions require special consideration, such as scoliosis and stenosis.

Stage 1: Establish Quality Movement Engrams

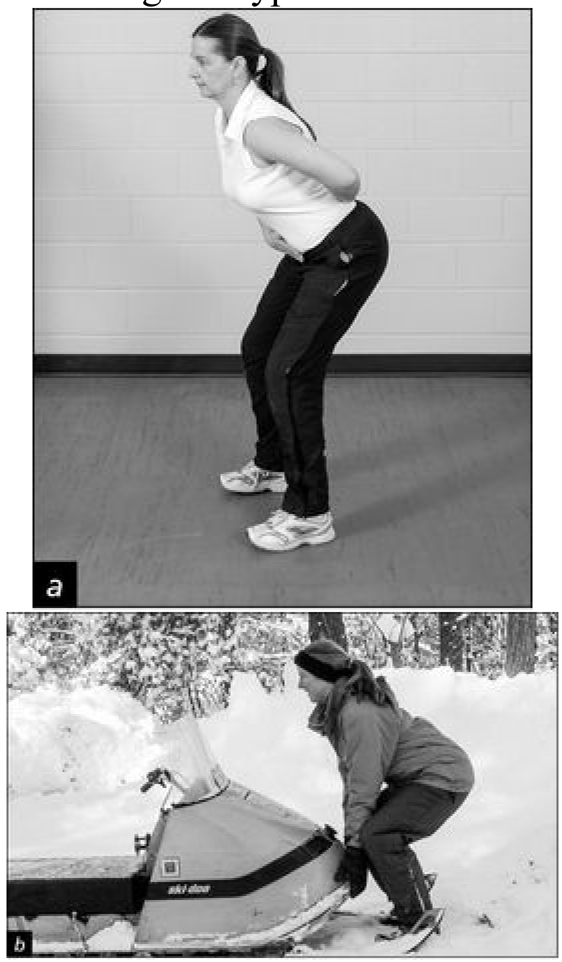
Some people are very body aware and can adopt a neutral spine or a flexed spine on command. Others can be frustratingly clueless. The objective of this stage is to detect and then correct perturbed movement patterns or engrams.

Distinguish Hip Flexion From Lumbar Flexion

The initial objective of stage 1 is to have the patient consciously separate hip rotation from lumbar motion when flexing the torso. For the more difficult cases, we typically begin by demonstrating on ourselves lumbar flexion versus rotation about the hips. Other techniques that we have found particularly helpful are as follows:

- Have the patient place one hand on the abdomen while placing the other over the lumbar surface. This way the patient can feel whether the spine is locked and motion is occurring about the hips (see figure 8.1a).

Figure 8.1 For people who are not body aware and are unable to adopt a neutral or a flexed spine on command, we suggest (a) rehearsing the spine neutral position and hip (not lumbar) flexion while doing knee bends before (b) exertion, such as lifting this typical Canadian household item.



Photos from Stuart McGill

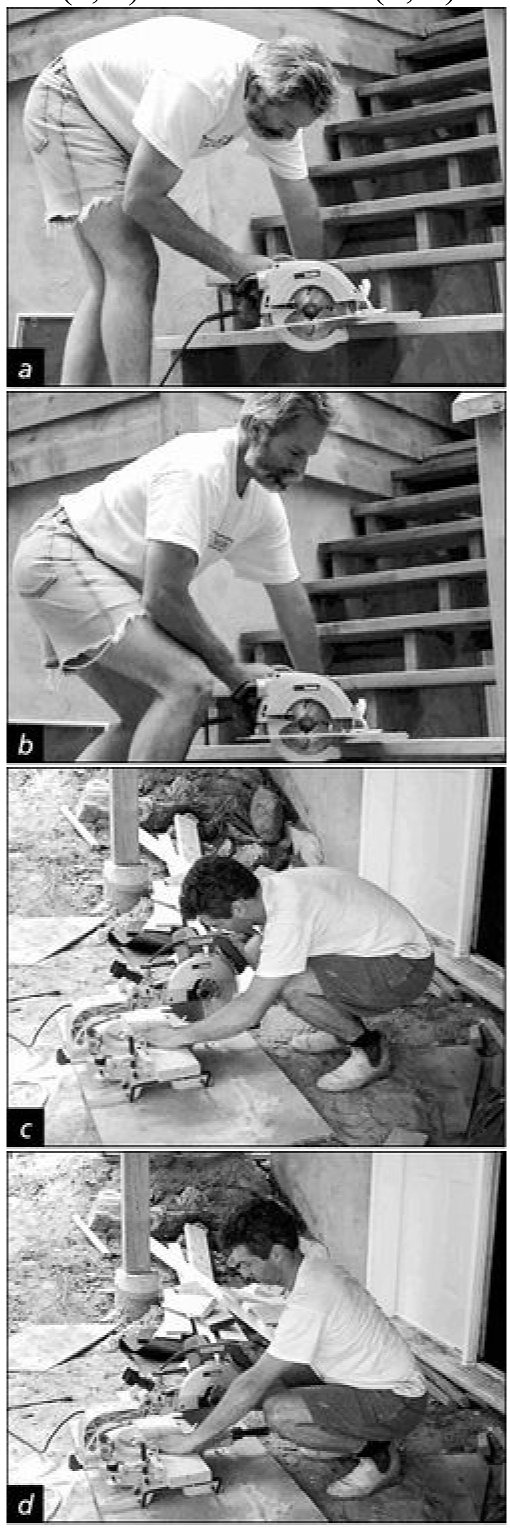
- Sometimes patients are receptive to being coached while using a practice load. The dummy constructed by nurses to help them rehearse proper patient lifting (shown in figure 7.30 in chapter 7) is an example of such a practice load.
- Other patients respond well to photos of people correctly doing tasks that they will also be called on to do in the course of their jobs or their everyday activities. Figure 8.2, a and b, shows examples of such photos.

Figure 8.2 Sometimes patients are receptive to task-specific illustrations showing spine and hip postures. (a) A rescue worker is applying a pulling force to the victim with the spine flexed. Discussion with the patient of this flexed spine posture together with one that would better spare the spine is very helpful. (b) Also discussed are exercise postures such as the neutral lumbar posture adopted by this patient performing cable pulls.

Photos from Stuart McGill

- Before (incorrect) and after (correct) photos, such as those in figure 8.3, a through d, can be especially helpful.

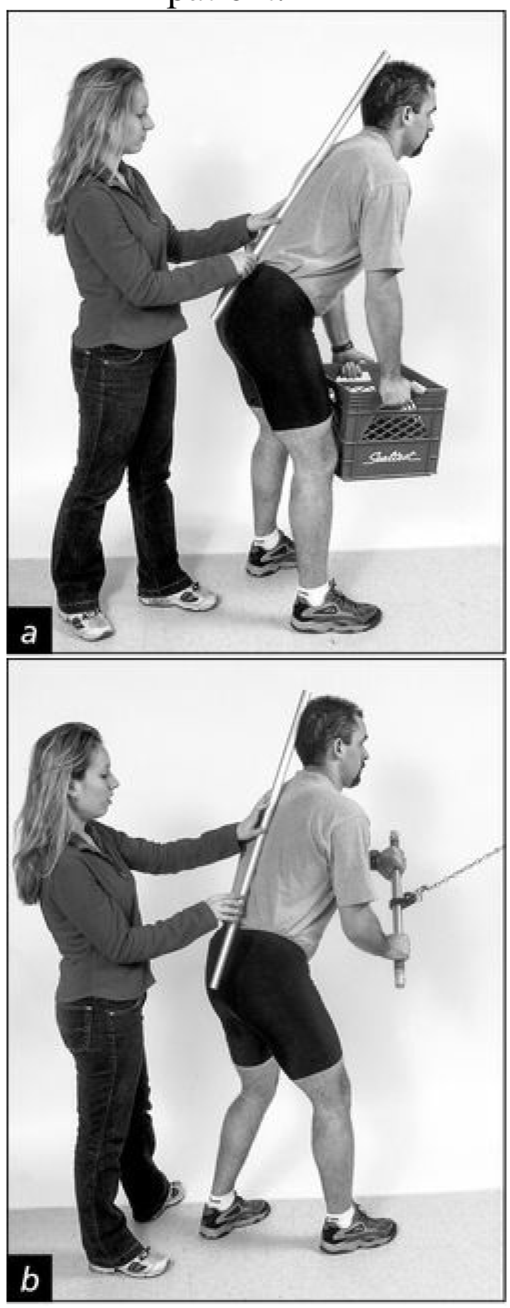
Figure 8.3 Workers relate to tasks with which they are familiar. These photos are helpful for construction workers, especially because they can readily see the difference between the (a, c) incorrect and (b, d) correct spine postures.



Photos from Stuart McGill

- Yet another technique is to place a stick along the spine with the instruction to flex the torso forward using the hips but maintain contact with the stick over the length of the spine (see figure 8.4, a-c). Although this begins in the clinic, other, more complex bending tasks can also help groove these patterns into the general motion patterns used in work and other daily activities.

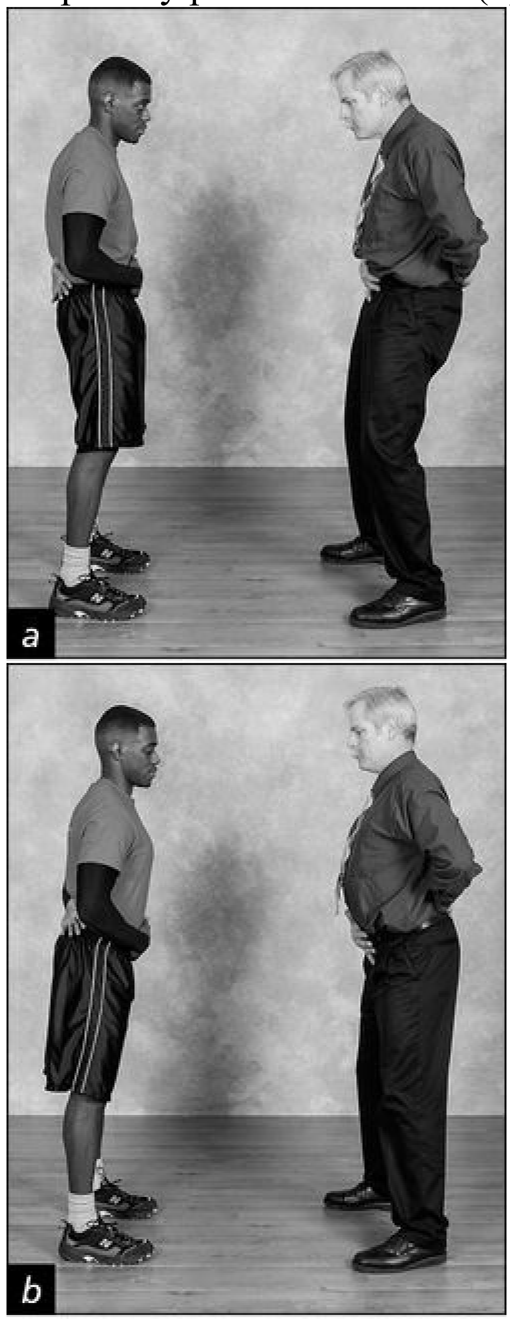
Figure 8.4 Placing a stick along the spine with the instruction to flex forward but maintain contact with the stick over the length of the spine helps patients separate hip rotation from lumbar motion when flexing the torso. Examples of motions that can be used are (a) sagittal plane symmetrical lifts, (b) three-dimensional cable pull exercises, and (c) work tasks that are familiar to the patient.

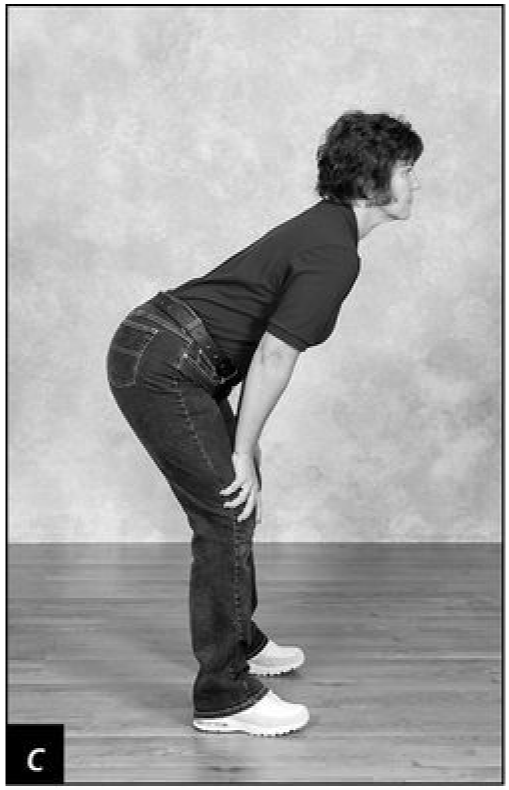


Photos from Stuart McGill

- When all of these attempts fail, we resort to the final technique: having the patient perform the "midnight movement" (rolling the pelvis)—this is lumbar motion. Interestingly, some patients who found sex painful never associated pelvis tilting with lumbar flexion (see figure 8.5, a-d). Pointing this out to them often facilitates their making the next leap in spine position awareness and in being able to avoid these painful motions and postures.

Figure 8.5 Allowing the patient to feel spine (a) flexion and (b) extension helps them to eliminate these from the squatting motion. Others get it by (c) adopting the shortstop ready position and then (d) standing up.






d

Once you begin to see that any or several of the techniques just listed are helping these "difficult" patients learn how to achieve and maintain a neutral spine position during their daily activities, you may have them attempt some tasks to see whether the concept of the neutral spine is becoming ingrained. One such task is to take the spine stick and ask the patient to knock down an imaginary spiderweb in an overhead corner of the room. If he loses the lumbar neutral posture, point this out to him so he can correct it. Obviously, you should continue spine position awareness training with such a patient.

Some people have a very difficult time remembering the protective neutral spine pattern. We tell these types of patients to do the following:

1. Stop before an exertion (perhaps prior to lifting a household item).
2. Place the hands on the abdomen and lumbar region.
3. Practice a few knee bends with the motion about the hips and not the lumbar spine.
4. Then perform the lift.

This practice is effective for many people.

Three Movement Tools

For many people, learning to lock the rib cage on the pelvis is essential for injury prevention and for performance—though of course not for all. We have developed a teaching progression that is effective for most patients and is fully explained in chapter 8. However, we begin with three movement patterns that allow the patient to perform many daily activities without triggering pain (hip-hinge squat, lunge, and stop twist). Once these patterns are perfected, the patient learns when to use each tool (e.g., the lunge, not the squat, for moving to the floor).

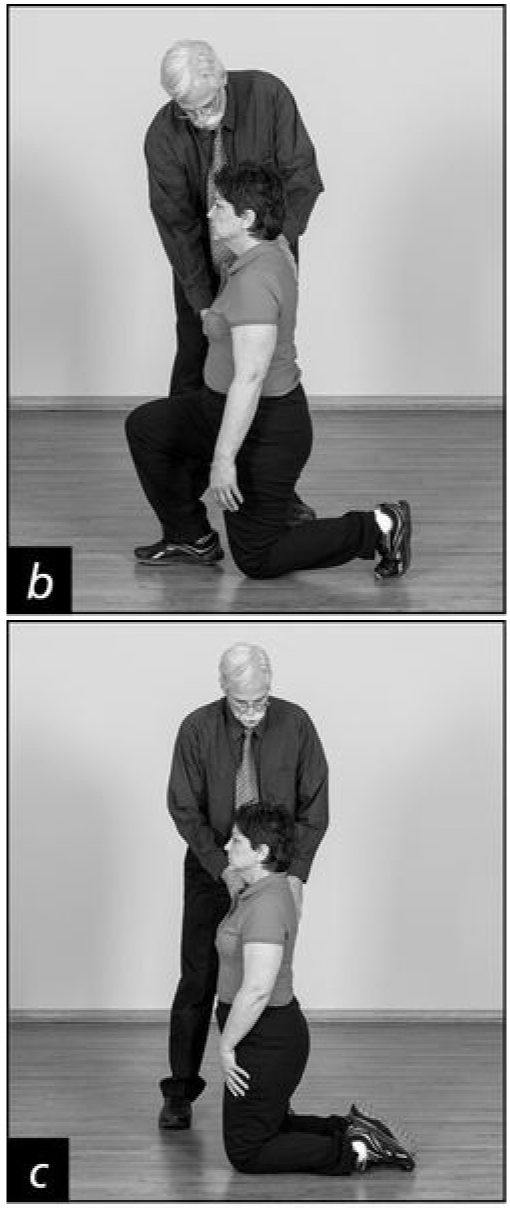
- Hip-hinge squat. This pattern translates to rising from a chair or getting off the toilet, brushing teeth, and lifting. The motion is taken from the spine and transferred to the hips. The spine is stiffened into its pain-free posture (see figure 8.6).
- Lunge. This pattern assists in moving to and from the floor. No motion occurs in the painful spine (see figure 8.7).
- Stop twist. This pattern translates to people pushing and pulling on doors and performing other tasks in which twisting torque must be controlled so that the spine does not twist. Instead, the motion occurs at the ball and socket joints: the shoulders and hips (see figure 8.8).

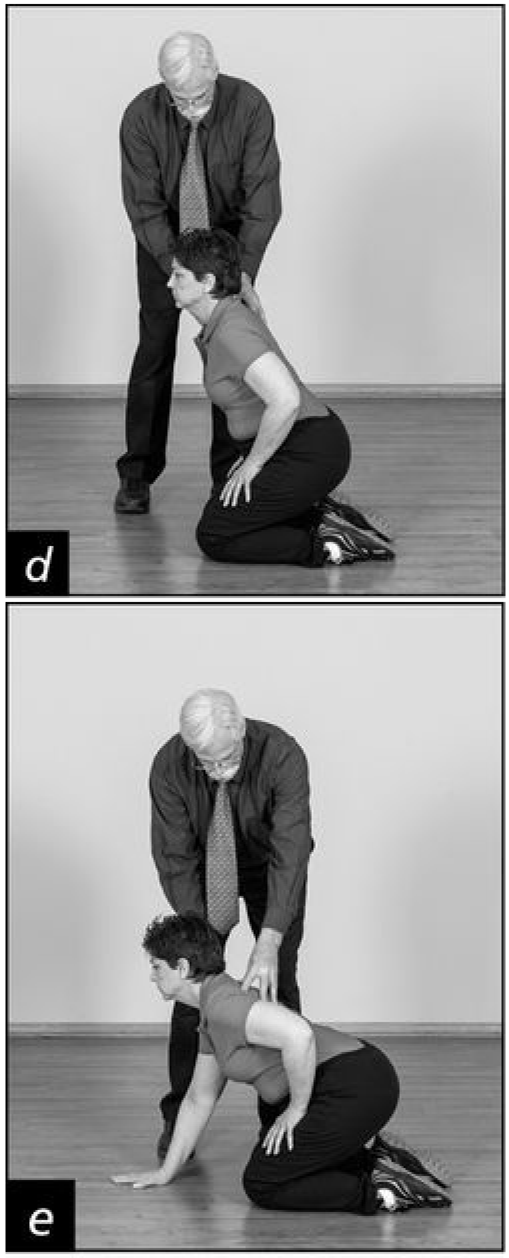
The motion pattern should be accompanied by the abdominal brace motor pattern (see figure 8.6). More athletic versions of the squat progression are detailed in McGill (2014).

Figure 8.6 (a) The hip-hinge squat begins with placing the palms on the thighs. (b) The hands slide down the thighs and grab around the knees. Note that a vertical line through the knees falls between the heels and the balls of the feet. Now the spine is positioned into a neutral posture with cues such as "Lift your tail." (c) Then the torso weight is projected down the arms, which adds stiffness to the torso with latissimus and pectoral coactivation. (d) To cue the rise, the instruction is “Don’t lift with the back, but pull the hips forward as you slide your hands up your thighs." This eliminates pain from bending, lifting, and so on. Test the person’s competency by asking her to brush her teeth at the bathroom sink. Have her replicate the hip hinge and brush.


c
d

Figure 8.7 (a, b) The lunge begins by taking a step and descending until one knee is on the floor (note that no spine motion occurs at any stage during this movement). The torso is stiffened sufficiently to remove any pain-causing aberrant movement. (c) Then the person moves to both knees. The hands slide down the thighs by (d) hip hinging (no spine motion), and (e) the hands walk forward into a (f) hands-and-knees support posture. The person then rolls to the floor by sliding one hand forward and the same-side knee back. The pelvis and rib cage drop to the floor in unison. (g, h) The roll is completed by lying on the back by stiffening the rib cage to the pelvis with an abdominal brace. (i, j, k) To rise, one leg remains straight while the other knee is raised together with the same-side arm. (l) As the roll continues, just as the knee is planted in the crawl posture, the shoulder is stiffened as the person rises on the support elbow. (m) The other knee is now pulled through with no spine motion. (n, o) The hip hinge is used to obtain an upright kneeling position from which the (p, q) lunge is used to get to the feet. The test is to ask the person to tie her shoe. From here she should lunge to a chair and take the hips to the target. Then she can tie her shoe with no spine motion or pain.


b
c


d
e


f
g
h


i
j
k
l


m
n
o


p


q

Figure 8.8 (a) The stop twist is a wall roll that begins with the person in the plank position with both elbows planted on the wall. (b) Many initiate the movement with the pelvis twisting and stressing the spine. (c) This is corrected when the focus is on spine and hip motion, generally adjusting the spine and hip posture to shift the pelvis toward the wall to find the sweet spot in spine position with minimal pain. (d , e) The abdominal muscles are braced and the rib cage is locked on the pelvis. The person pivots on the balls of the feet, pulling one elbow off the wall. No spine motion is permitted. These corrections are repeated until the person can control the spine and eliminate pain. The test is to then pull on a door, locking the rib cage to the pelvis; there should be no pain.


c
d
e

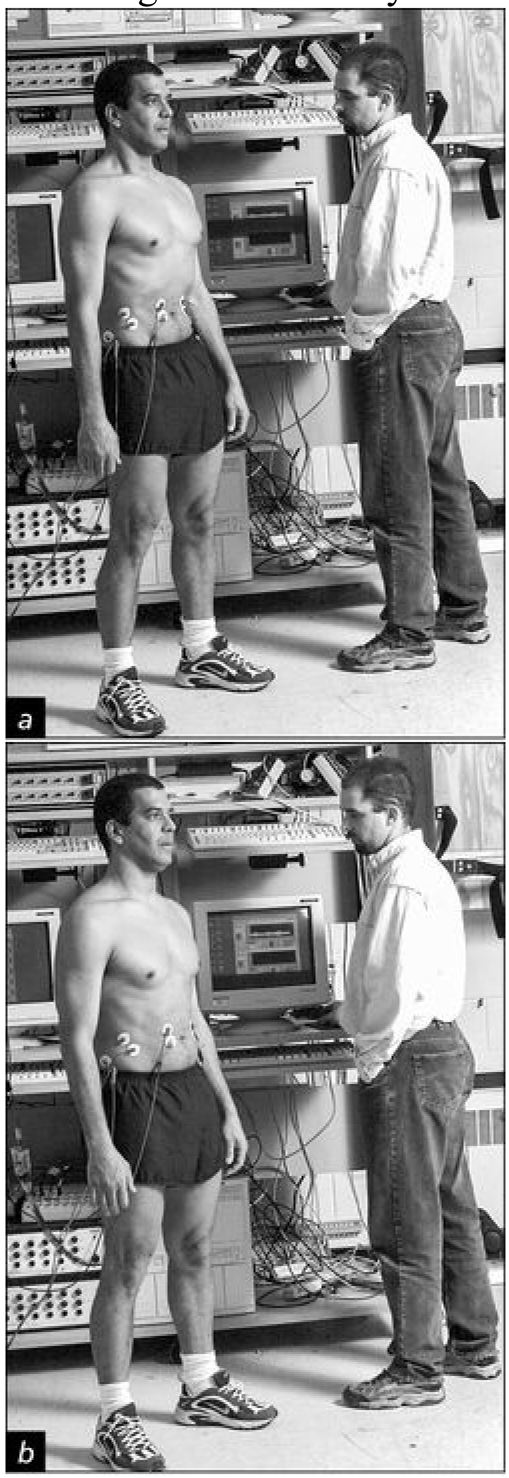
Distinguish Abdominal Bracing From Abdominal Hollowing

Maintaining a mild contraction of the abdominal wall can also help ensure sufficient spine stability. This maneuver is called abdominal hollowing in many back care circles; however, we prefer to avoid that terminology, because it suggests to most people either pulling in or puffing out the abdominal wall. When the contraction is performed correctly, no geometric change at all occurs in the abdominal wall. In other words, rather than hollowing or drawing in the abdominal wall, the patient simply activates the muscles to make them firm and stiff. We call this contraction abdominal bracing.

There is confusion in the interpretation of the literature regarding abdominal hollowing and abdominal bracing is discussed in chapter 5. Abdominal bracing, which activates the three layers of the abdominal wall (external oblique, internal oblique, transverse abdominis) with no drawing in, is much more effective than abdominal hollowing at enhancing spine stability

(Brown and McGill, 2008, 2010; Brown, Vera-Garcia, and McGill, 2007; Grenier and McGill, 2007; McGill, 2001) (see figure 8.9). An important issue is the intensity. Bracing effort runs the full continuum, from very mild for those intolerant of compressive load but need some control (roughly 2 to 5% of maximal contraction), to lifting and carrying a load that must be controlled (5 to 20% of maximal contraction), to those maximizing storage and recovery of elastic energy (about 25% of maximal contraction), to those performing a maximal-effort lift (well over 50% of maximal contraction).

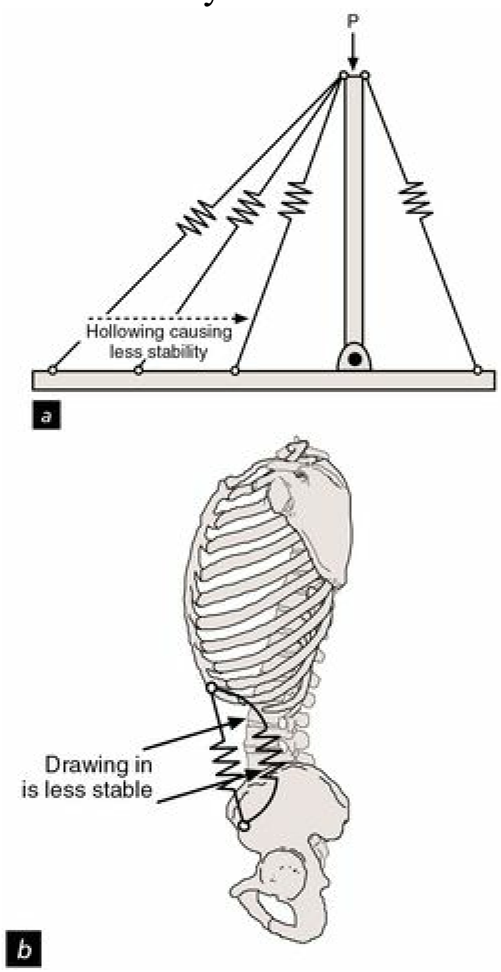
Figure 8.9 (a) Hollowing involves the sucking in of the abdomen to activate transverse abdominis. (b) On the other hand, the abdominal brace activates the three layers of the abdominal wall (external oblique, internal oblique, transverse abdominis), with no drawing in. The cue to the patient is to push the fingers out laterally.

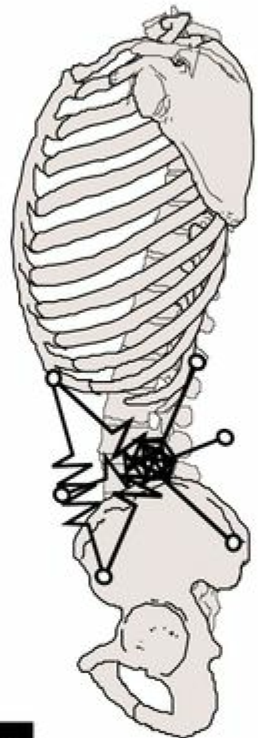


Photos from Stuart McGill

Two mechanisms can shed some light on this issue of hollowing versus bracing. First, the supporting “guy wires” are more effective when they have a wider base (see figure 8.10, a-c); that is, when the abdomen is not hollowed. Second, the obliques must be active to provide stiffness with crisscrossing struts, which measurably enhances stability. Bracing enhances oblique activity, whereas hollowing inhibits it. The lumbar torso must prepare to withstand all manner of possible loads, including steady-state loading (which may be a complex combination of flexion–extension, lateral bend, and axial twisting moments); sudden, unexpected complex loads; and loads that develop from planned ballistic motion. Stiffness is required in every rotation and translation axis to eliminate the possibility of unstable behavior. The abdominal brace ensures sufficient stability using the oblique cross-bracing, although high levels of cocontraction are rarely required—usually about 5 to 10% of maximal voluntary contraction (MVC) levels of the abdominal wall during daily activities are sufficient. The patient must match the level of contraction to the needed stability—there is no need to crush the spine with overcontraction.

Figure 8.10 (a) Hollowing the muscles reduces the size of the base of the guy wires, as well as the incidence angle at which they attach to the spine. (b) This inherently reduces their contribution to spine stiffness in various modes, which compromises spine stability. (c) Bracing assists in keeping a wide base to the guy wires and recruits the oblique muscle to supply cross-bracing struts for stability in all axes.

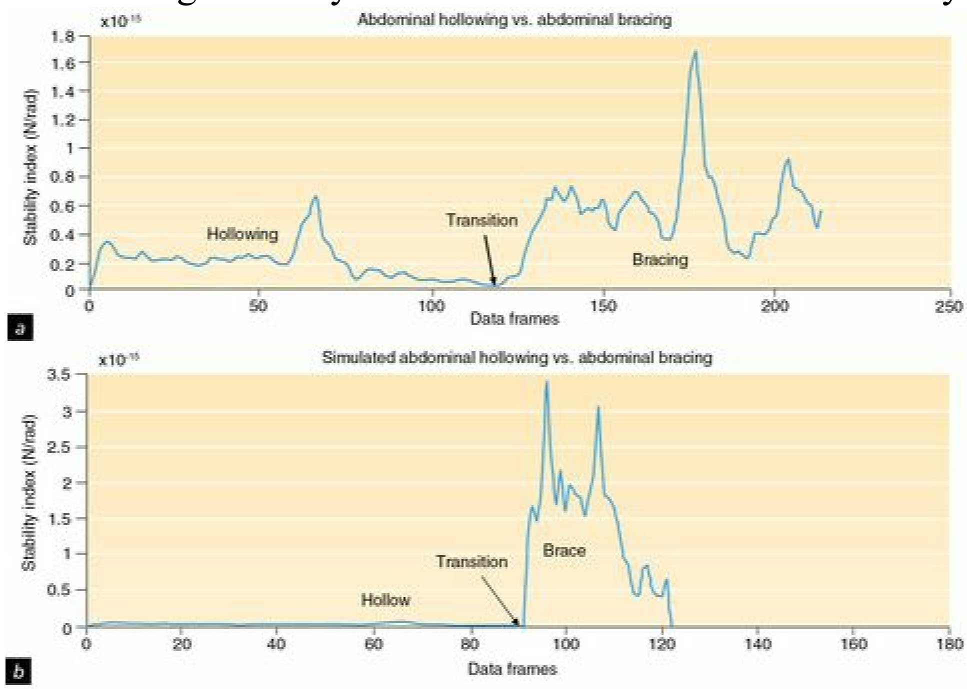




c

A quantitative comparison of the hollow and the brace is clearly seen in a person standing upright with loads in the hands. Simply hollowing can cause the stability index to drop to low levels or even negative levels, which indicates the possibility of instability (see figure 8.11). Bracing increases the positive stability index value. The subject in these figures is typical in that even though the hollow was taught to target the transverse abdominis, all abdominal muscles were activated when measured. Thus, stability was created while attempting hollowing, although true bracing is superior to create lumbar stability. If a true hollow is accomplished with just the transverse abdominis, as simulated in figure 8.10b, stability is low compared to that with a brace in which the three layers of the abdominal wall are activated (figure 8.10c). Simply hollowing causes the stability index to drop to negative levels when the load is placed in the hands, compressing the spine. A negative value indicates that instability is possible. In contrast, bracing maintains a positive stability index value, eliminating the possibility of buckling.

Figure 8.11 (a) An example comparing the hollow with the brace (higher stability) in a person standing upright, arms at the sides, with loads placed in the hands. The problem for patients is that isolating transverse abdominis is virtually impossible. (b) A "perfect" hollow was created with simulation and shown to be significantly inferior to the brace to create stability.



Teach Abdominal Bracing

Generally, to demonstrate abdominal bracing to the patient, we stiffen one of our own joints, such as an elbow, by simultaneously activating the flexors and extensors. The patient then palpates the joint both before and after we stiffen it. Then we ask the patient to attempt to stiffen her own joint through simultaneous activation of flexors and extensors. Once she can stiffen various peripheral joints, we demonstrate (again on ourselves, with patient palpation) the same technique in the torso, achieving abdominal bracing. Finally, we again ask her to replicate the technique in her own torso (see figure 8.12). Occasionally, we use a portable electromyographic (EMG) monitor so the patient can learn through biofeedback what, for example, 5%, 10%, or 80% of maximal contraction feels like (see figure 8.13). We use similar devices to teach patients how to maintain the contraction while on a wobble board and in functional situations such as when picking up a child, getting on and off the toilet, and getting in and out of cars.

Figure 8.12 (a) A good cue is for the clinician to place the fingers into the lateral obliques, or have the patient do this themselves. (b) The instruction is to stiffen and push the fingers out laterally.

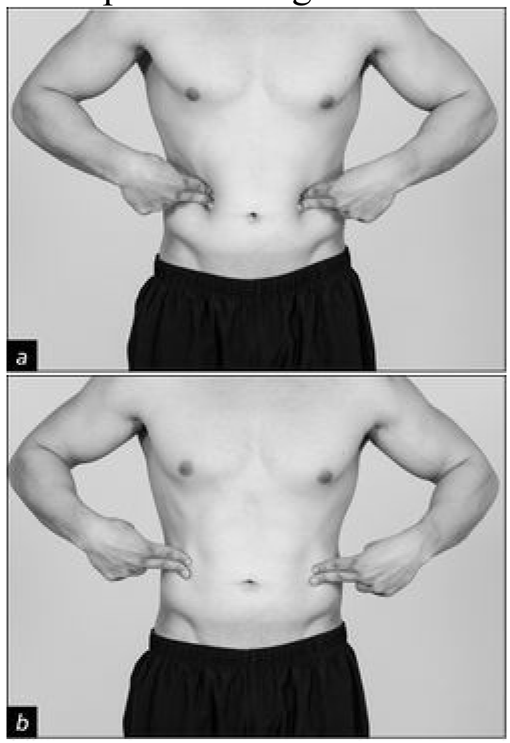


Figure 8.13 EMG biofeedback devices are an economical way to provide feedback to the patient regarding the level of abdominal activation during any type of functional task, from standing on a wobble board to getting into a car.

Photos from Stuart McGill

Given our research on the importance of spine position awareness to spare the spine and our experience in teaching positioning, we became interested in proprioceptive training for the back. The fact that very little evidence was available to validate the use of proprioceptive rehabilitation for the lumbar spine motivated our recent work on spine proprioception (Preuss, Grenier, and McGill, 2005). The purpose of this work was to quantify the effects of a 6-week rehabilitation program to improve lumbar spine position sense and sitting balance. Twelve subjects with a previous history of low back pain were evenly split into a training group and a control group. The subjects in the control group received no intervention, whereas the subjects in the training group received a 20-minute rehabilitation session three times per week emphasizing spine stabilization exercises with a neutral spine. The ability to correct lumbar spine position during four-point kneeling and while sitting substantially improved in the training group over the study. This small but initial study demonstrated that proprioception and position awareness in the lumbar spine can improve through active rehabilitation.

Mental Imagery

The use of mental imagery is helpful for both spine position and muscle activation awareness. Following is a general protocol that we have adapted from the imagery literature for use with spine training.

1. Focus on feeling the surface under the feet or buttocks. Whatever body part is touching a surface, be aware of the sensation.
2. Practice simple motions such as tightening and then relaxing specific muscles in different areas of the body. Then graduate to performing the abdominal brace.
3. Palpate, and have the patient self-palpate, the muscle involved while he is attempting to tighten and relax it. Sometimes a full-body mirror is helpful. The focus for the patient is on the specific muscle(s) involved.
4. Perform motions slowly, chunking them into segments and sequences; then visualize the total motion. For example, beginning with a simple task such as a forward reach, visualize the neutral spine, then activating the extensors and the bracing abdominal muscles, and finally the motion about the hips.
5. Practice the imagery independent of physical action. Of course, the patient will have already been successful in learning spine position awareness, proper muscle control, and desirable motion patterns.

Source: Kathryn McGill, sports psychology consultant

Build Squat Patterns

To have a healthy back, a person needs good gluteal muscle function, and function demands balanced hip power about each axis. This section describes some hip motor patterns that inhibit spine-sparing patterns, and documents several training progressions to address them.

The crossed-pelvis syndrome is a term given to the condition in which the gluteal complex appears to be inhibited during squatting patterns; this syndrome is very common in those with a history of back troubles (and in those who sit for extended periods during work). Interestingly, we still do not know whether the crossed-pelvis syndrome exists prior to back troubles or is a consequence of having them. Nonetheless, the syndrome is noticeable in many patients referred to our research clinic. This results in two concerns. First, those with aberrant gluteal patterns cannot spare their backs during squatting patterns because they use the hamstrings and erector spinae to drive the extension motion. Subsequently, the erector spinae imposes unnecessary loads on the lumbar spine. In this way, healthy gluteal patterns are needed to spare the back. Second, it is impossible to rebuild optimal squat performance, either for strength or hip extensor power, without well-integrated hip extensor patterns. In fact, the failure of many people to properly rehabilitate is due to neglect of the ability to squat and rise off the toilet, or egress from a car, or walk up stairs, as a result of aberrant gluteal patterns that have not been addressed.

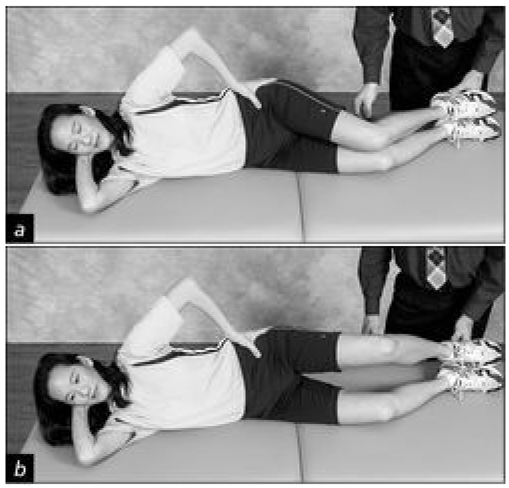
Retraining the gluteals cannot be performed with traditional squat exercises that use a machine. Performing a traditional squat requires little hip abduction. Consequently, there is little gluteus medius activation, and the gluteus maximus activation is delayed during the squat until lower squat angles are reached. This is well documented in McGill (2014). In contrast to the traditional squat, a one-legged squat activates the gluteus medius immediately to assist in the frontal plane hip drive necessary for spine-sparing function together with sooner integration of gluteus maximus during the squat descent motion.

Learning to Activate the Gluteals

The following maneuvers will enable most patients to learn how to isolate and activate both gluteus medius and gluteus maximus.

Gluteus Medius

The first stage involves isolating gluteus medius. Once again, the patient needs to feel the muscle and perceive its activation. The patient lies on her side, places her thumb on the anterior superior iliac spine (ASIS), and reaches with the fingers posteriorly—the tips will be over the gluteus medius (a). With the hips and knees flexed, the patient spreads the knees apart like a clamshell, with the feet remaining together and acting as a hinge (b). The patient feels with the fingers the activation of gluteus medius. This maneuver is to simply activate the gluteus medius and should not be considered a strengthening exercise. There is no need to offer resistance at this stage (resistance is imposed later during strength training). True isolation of the gluteus medius is not possible; other muscles are active. In this posture, the external hip rotators are recruited. Recent work of Sidorkewicz, Cambridge, and McGill (2013) has shown that although muscles such as the tensor fascia latae are activated, the gluteus medius dominates this exercise, and the changing hip flexion does not substantially influence the relative activation of these two muscle groups.



Gluteus Maximus

Lying on the back with the knees flexed and the feet on the floor, the patient places the fingers on gluteus maximus to feel its activity. Have the patient imagine a coin placed in the gluteal fold that must not be dropped. The patient activates gluteus maximus by squeezing the buttocks—not by creating hip extension. The focus is on the pelvis at this stage to ensure that no pelvic tilting occurs. The lumbar spine remains in neutral posture (a). Then, once the activation has been mastered, the patient begins bridging the torso off the floor. The clinician at this stage palpates the hamstrings (b). Those who are hamstring dominant and gluteal deficient will immediately activate the hamstrings just prior to the occurrence of motion. This pattern is very dominant in those who have the aberrant crossed-pelvic syndrome, but is also seen in some sport-specific athletes such as cyclists. The patient must repeatedly try to begin the bridging action without hamstring activity (or at least only mild activity).

For some patients, overriding the hamstring-dominant tendency requires coaching and cuing from the clinician. For these challenging cases, we place our foot against the patient's toes and instruct the patient to continue with the preparatory gluteal activation but then also very slightly activate the quadriceps by very mildly attempting to extend the knees (c). Buttressing the patient's feet with the clinician's foot assists this. A gentle stroke on the quads to assist the patient's imagining and perception of mild knee extension also facilitates this pattern to enhance gluteal dominance. Then the patient repeats the attempt to bridge with gluteal dominance. Once this is mastered, squat performance will improve.

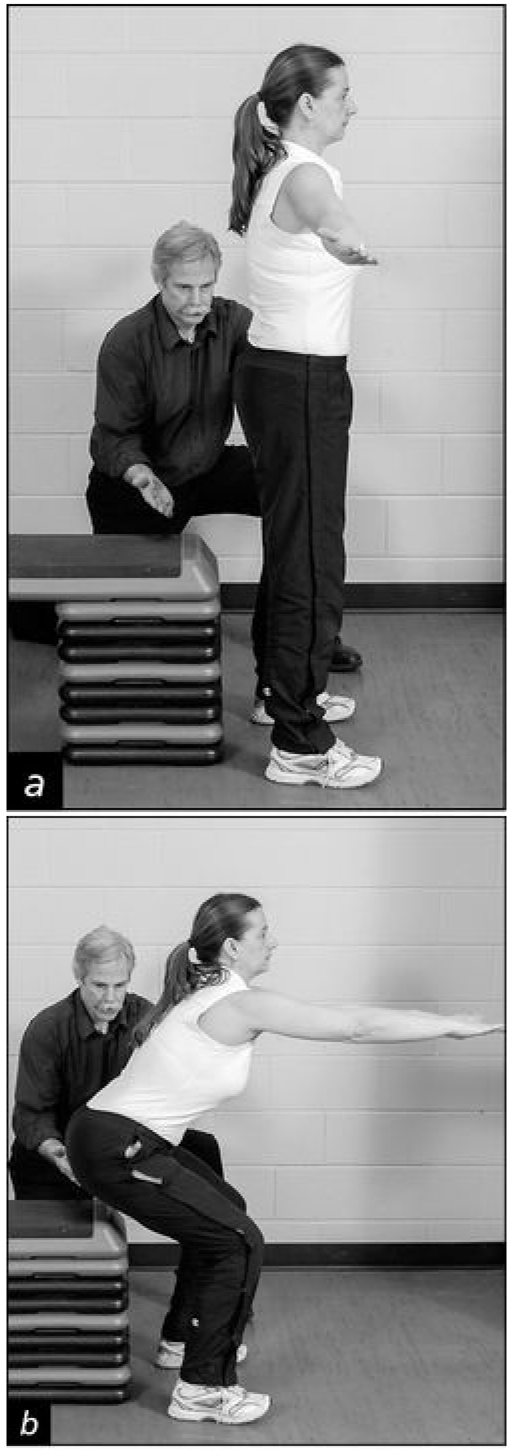
Imagining squeezing the gluteus maximus muscles prior to performing the back bridge will assist in grooving gluteal-dominant hip extension patterns. Then the patient performs the bridge with a focus on gluteal contraction throughout the full range. For those having difficulty, quadriceps stroking can assist with mild knee extension to further reduce hamstring contribution.

Beginning Basic Squat Patterns

First a word of caution: Do not start this too early with patients who either are disabled or have very painful backs.

Potty Squat

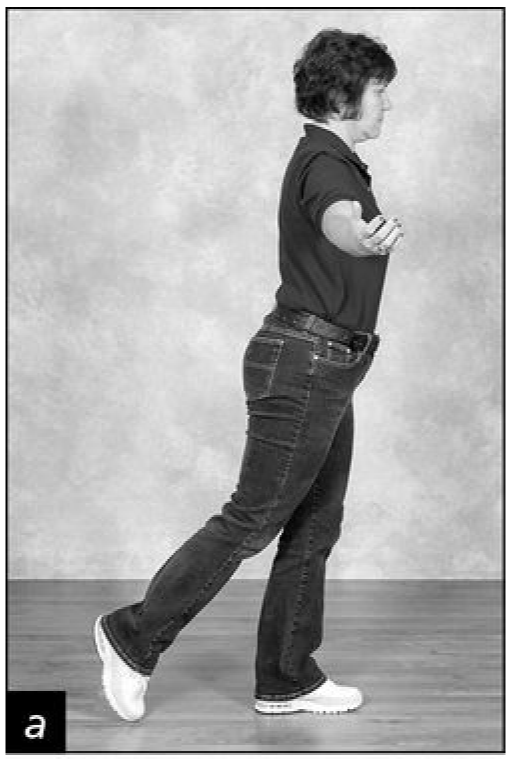
When appropriate, we begin a basic squat progression with a potty squat. Sitting on the corner of a chair or a stool, the patient positions the feet under the body to rise off the chair without using any momentum shifts. The lumbar spine is neutral and braced. This begins to groove a good two-legged squat position. Then, with progression to a standing position, the arms are held out laterally (a) and moved in front of the body as the patient squats (b). Of course, emphasis is on maintaining a neutral lumbar spine and abdominal bracing. The hips follow a trajectory along a line about 45° from the vertical. “Squat back” is a better instruction than “Squat down.” The motion is predominantly at the hips, which is known as the hip hinge.

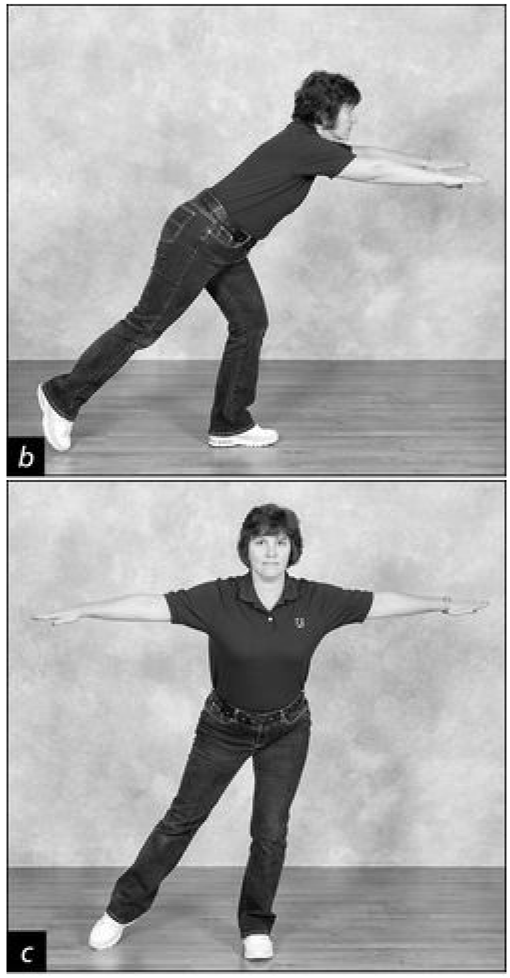


Photos from Stuart McGill

Single-Leg Squat

A single-leg squat involves the same arm motion as the potty squat to assist balance. As the single-leg squat is performed, the free leg is held behind and the knee is touched to the floor, or the toe is reached with an outstretched leg to a distant object behind. (a , b) Then, the free leg is reached out to a distant object placed laterally during the squat (c). Variations include working the free leg to various positions around the clock. This challenges the full hip extensor, flexor, and abduction torque generators together with keen motor control. Full integration with the pelvis and lumbar spine is achieved with emphasis on the appropriate motor and motion patterns. Specific focus is on maintaining a neutral lumbar spine. Focus on hip motion, the gluteal muscles producing the hip extension torque, and a stiff torso. Caution with anterior foot placement is necessary with some patients who are unable to maintain a neutral spine.

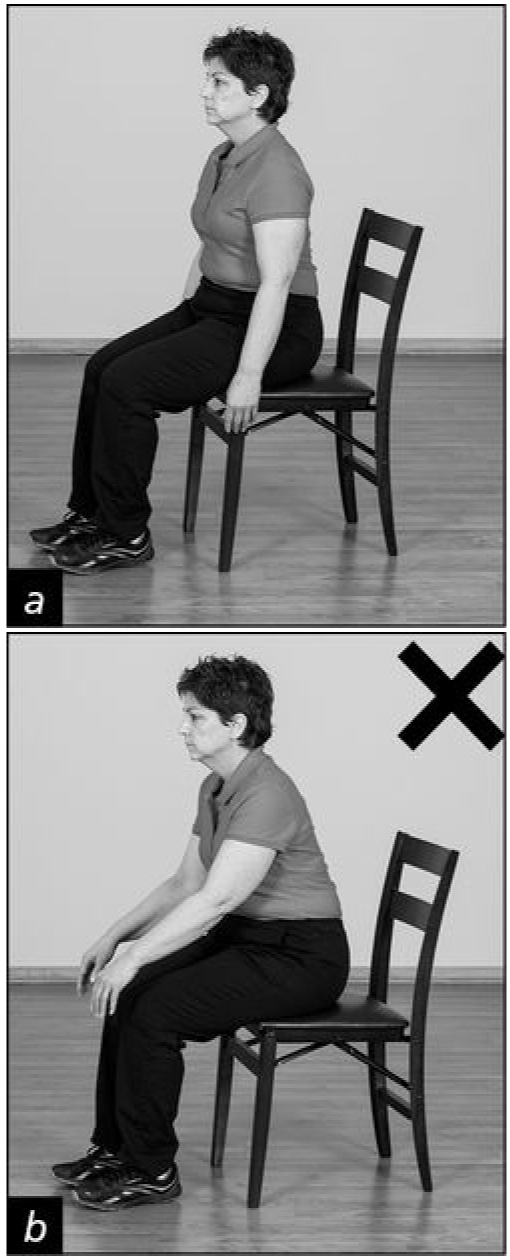




The single-leg squat follows the same hip-hinging motion as the potty squat. The nonsupport leg is projected to the rear and to the side. Be cautious with forward projection of the foot to the front, because it often causes spine flexion. The abdominal muscles are braced, the lumbar spine is in neutral, and the mental focus of the patient is on hip extension torque. Notice that the hips are drifted posteriorly during the descent to place more emphasis on the gluteals for hip extension, unloading both the knees and the back.

Rising From a Chair

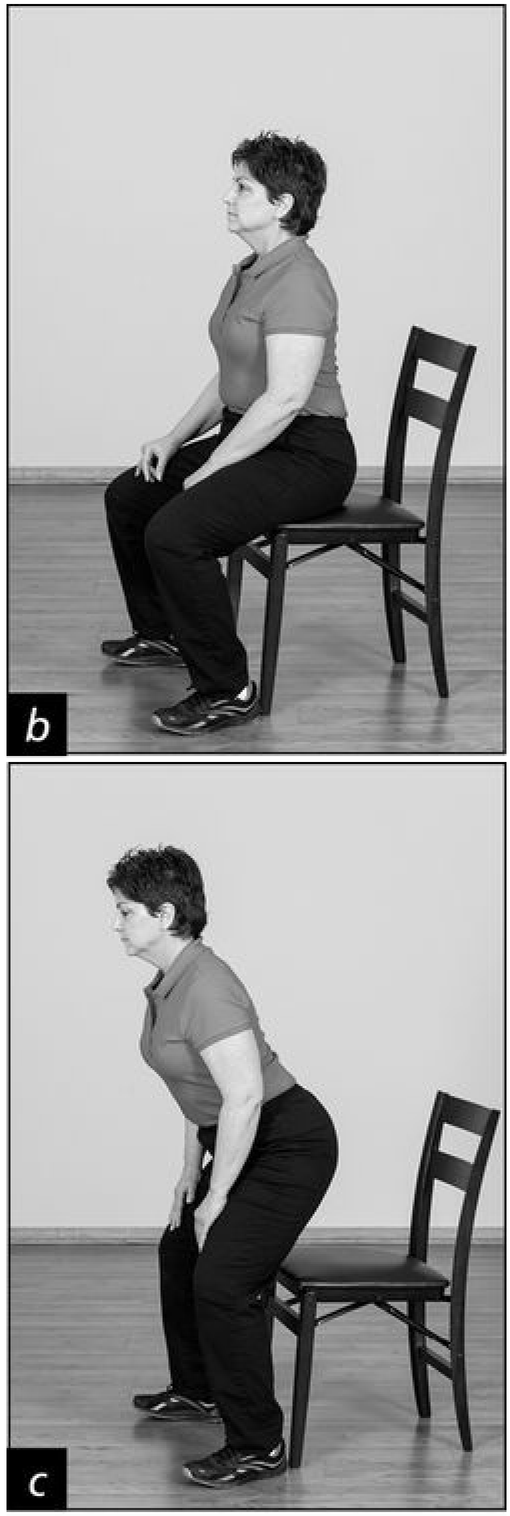
Rising from a chair follows the potty squat mechanics. Faulty motion patterns include initiating the motion with spine flexion. Instead, the spine should be extended with the rib cage rising and the hips flexed. The feet are drawn underneath the patient, the knees and feet are spread, and the hips externally rotate to integrate the gluteal muscles for hip extension.


a
b


c


d

(a, b) The first faulty motion is initiated with spine flexion. (c) Then the hips are extended with the hamstrings, and (d) the spine is extended with the back extensors. Poor technique is characterized by spinal flexion and lifting from the back. (e) The stooped standing posture results from a failure to pull the hips through with extension.


b
c

Corrected rising from the chair begins by spreading the knees and placing the feet under the client's center of mass (a). Then the torso leans forward with hip flexion, not spine flexion (b). Sniffing the air first helps with form. Then the hips are pulled through as the hands slide up the thighs (c), until an upright posture is achieved (d).

Stage 2: Establish Stability Through Exercises and Education

For patients to achieve stability not just during rehab, but also in all aspects of their lives both during and after rehab, both exercise and education are crucial. The fundamental tenet of human motion is that a proximal joint must be stiffened and stabilized before a distal joint can produce force, torque, and movement. Skilled stiffness and stability techniques must be established.

- Establish stabilizing motor patterns through stabilization exercises. Generally, stage 2 should begin with stabilization exercises. The trick is to find an appropriate starting level. For those who arrive at our university clinic having failed traditional therapy, we generally undershoot what many would consider an appropriate loading level. The typical progressive improvement philosophy of work hardening with weekly improvement goals does not work for these people; they require more patience. They will have good and bad days, but the general positive slope in improvement must be established—however gradual. Once we can document a positive slope in improvement, then we can increase the rehabilitation challenge. Specific exercises are discussed and illustrated in chapter 10.

- Ensure stabilizing motion patterns and muscle activation patterns during all activities. You must clarify the range of activities (e.g., daily living, occupational, athletic) for which the patient must be prepared. This is obtained through interview. Although there is no standard form, we begin by documenting the patient's daily routine, which includes occupational demands and those of daily living. Previous chapters offered many examples of sparing the spine with an appropriate posture and muscle activation pattern. The guidelines in the next section will help you determine what exercises to prescribe for patients in this stage. In addition, we spend time rehearsing daily activities to be sure the patient is learning and using spine-sparing motion and motor (muscle activation) patterns.

Stage 3: Develop Endurance

Endurance is necessary for maintaining stabilizing patterns of muscle activity. There is a progression to building endurance, and it starts with building endurance without becoming tired! Here is what you should keep in mind as you progress patients toward greater endurance:

An Important Reminder

Remember, because half the battle is to remove the pain triggers, make sure to follow the recommendations of the previous chapters.

- Typically, initial endurance is built with repeated sets of relatively short holds. Holds should be no longer than 7 or 8 seconds. The duration is based on recent evidence from near-infrared spectroscopy that indicates a rapid loss of available oxygen in torso muscles contracting at these levels. A short relaxation of the muscle restores oxygen (McGill, Hughson, and Parks, 2000). The endurance objectives are achieved by increasing the repetitions of the exertions rather than the duration of each hold.
- Identify endurance deficits. Motivated by the evidence of the superiority of extensor endurance over strength as a benchmark for good back health, we recently documented normal ratios of endurance times for the torso flexors relative to the extensors and lateral musculature (see chapter 9). Use these values to identify endurance deficits—both absolute values and values for one muscle group relative to another.
- Use the reverse pyramid for endurance training. This approach to designing endurance sets is founded in the Russian tradition of maintaining excellent technique and form. The idea is to train endurance without becoming tired. For example, a training session of sets for the side bridge exercise using five repetitions would look like this:
    1. Five repetitions on the right side
    2. Five repetitions on the left side
    3. Rest a few seconds
    4. Four repetitions on the right side
    5. Four repetitions on the left side
    6. Rest a few seconds
    7. Three repetitions on the right side
    8. Three repetitions on the left side
    9. Finished

Good technique is facilitated as the repetitions are reduced with each fatiguing set. This is generally done to build the endurance base, because the objective of maintaining sufficient oxygen levels is met so that failure is not a result of oxygen starvation or acidic metabolite buildup. This is the approach for those with back pain. Endurance training progressions involve much longer durations for more athletic patients and are described in McGill (2014).

Summary: Checklist for Patient Progression

Clinical Relevance

As we have noted, the first three stages of our five-stage program focus on rehabilitation. Following is an expanded checklist for rehabilitation that adds some critical elements.

1. Identify and remove the exacerbating activities.
2. Have the patient record in a journal daily how the back feels as well as the tasks and activities performed.
3. Identify and correct perturbed motion and motor patterns using corrective exercise. Develop spine position awareness (hips vs. lumbar motion) and the ability to maintain appropriate levels of abdominal bracing. Establish spine-sparing motion and motor patterns.
4. Begin appropriate spine exercise and appropriate stabilization and mobilization tasks.
5. Develop muscular endurance.
6. Transfer to daily activities.

Note that training athletes in our program involves the same stages with the addition of two—training strength and power (see chapter 11 and McGill, 2014).

Guidelines for Developing the Best Exercise Regimen

Reports of the effectiveness of training and rehabilitation programs for the low back are quite variable: some claim great success, whereas others report no success or even negative results (Faas, 1996; Koes et al., 1991). The discrepancy regarding the effectiveness and safety of exercise programs is probably due to clinicians prescribing inappropriate exercises because they do not understand the tissue loading that results during various tasks. The approach described in this book has been used with great success for preventing back disorders (e.g., the Finnish military; Suni et al., 2006). Resist the urge to enhance mobility just to adhere with the disability rating system. That system is for legislative convenience only. Rather, judge your success by how well you are able to reduce patients' pain and restore their ability to complete tasks. Also, there must be a reason to perform an exercise, and to perform it in a specific way. Chapter 9 will help you identify deficits in patients so that you can address them in a specific program. If there is no reason to prescribe an exercise, then don't—work only within the restricted capacity of the patient to achieve specific goals.

Developing a Sound Basis for Exercise Prescription

I have quantified and selected the exercises in the following chapters based on tissue-loading evidence and the knowledge of how injury occurs to specific tissues (described in previous chapters). Choosing exercises, however, still involves making the best educated guess, which is developed through clinical experience. The following example illustrates the need for quantitative analysis to evaluate the safety of certain exercises.

We have all been told to perform sit-ups and other flexion exercises with the knees flexed—but on what evidence? Several hypotheses have suggested that this disables the psoas or changes the line of action of the psoas, or both. Magnetic resonance imaging (MRI)–based data (Santaguida and McGill, 1995) demonstrated that the psoas line of action does not change as a result of lumbar or hip posture (except at L5-S1) because the psoas laminae attach to each vertebra and follow the changing orientation of the spine. However, there is no doubt that the psoas is shortened with the flexed hip, modulating force production. But the question of whether there is a reduction in spine load with the legs bent remains. In 1995 McGill examined 12 young men using the laboratory technique described previously and observed no major difference in lumbar load as the result of bending the knees (average moment of 65 Nm in both straight and bent knees; compression of 3,230 N with straight legs and 3,410 N with bent knees; shear of 260 N with straight legs and 300 N with bent knees). Compressive loads in excess of 3,000 N certainly raise questions of safety. This type of quantitative analysis is necessary to demonstrate that the question of whether to perform sit-ups using bent knees or straight legs is probably not as important as the question of whether to prescribe sit-ups at all! There are better ways to challenge the abdominal muscles.

Basic Issues in Low Back Exercise Prescription

Several exercises are required to train all the muscles of the lumbar torso, but which exercises are best for a given person? This determination depends on a number of variables, such as the person's fitness level, training goals, history of previous spinal injury, and other factors. However, depending on the purpose of the exercise program, several principles apply. For example, someone beginning a postinjury program would be advised to avoid loading the spine throughout the range of motion, whereas a trained athlete may indeed achieve higher performance levels by doing so. Another general rule of thumb is to preserve the normal low back curve (similar to that of upright standing) or some variation of this posture that minimizes pain. Although in the past many clinicians have recommended performing a pelvic tilt when exercising, this is not justified; we now know that the pelvic tilt increases spine tissue loading, because the spine is no longer in static–elastic equilibrium. Thus, the pelvic tilt appears to be contraindicated when challenging the spine. Basic issues you should consider when prescribing exercises for low back rehabilitation are discussed in the following sections.

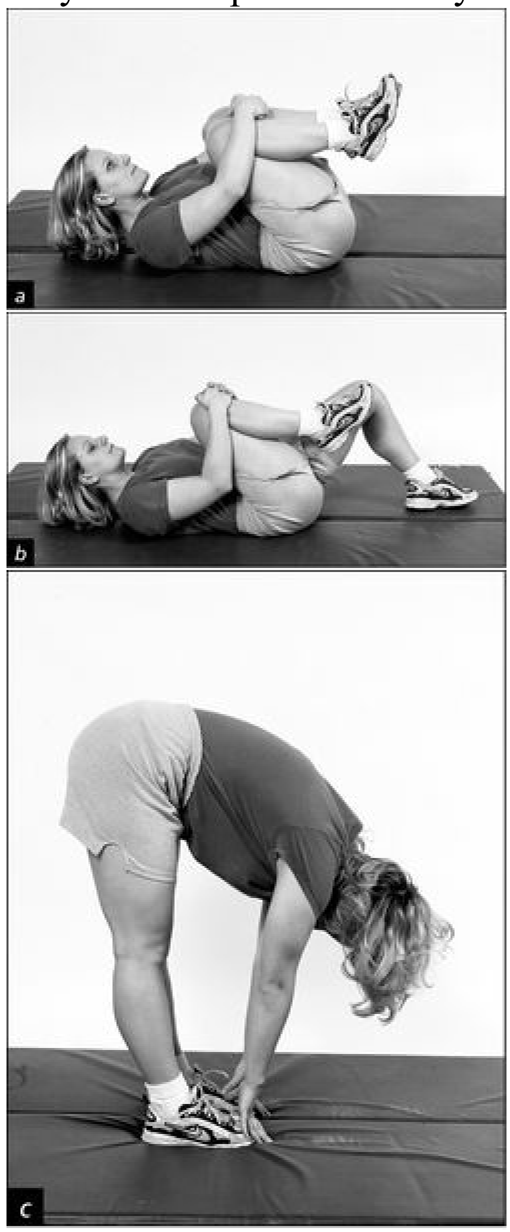
Flexibility

The decision of whether to train for optimization of spine flexibility depends on the person's injury history and exercise goal. Generally, for the injured back, spine flexibility should not be emphasized until the spine has stabilized and has undergone endurance and strength conditioning—and some may never reach this stage! Despite the notion held by some, few quantitative data support the idea that a major emphasis on trunk flexibility improves back health and lessens the risk of injury. In fact, some exercise programs that have included loading of the torso throughout the range of motion (in flexion–extension, lateral bend, or axial twist) have had negative results (e.g., Nachemson, 1992), and greater spine mobility has been associated with low back trouble in some cases (e.g., Biering-Sorensen, 1984). Further, research has shown that spine flexibility has little predictive value for low back trouble (e.g., Sullivan, Shaof, and Riddle, 2000).

In the context of trying to stretch the back and train flexibility, the insightful work of Solomonow and colleagues (2002) has shown that the stretch reflex is diminished and muscle spasms can result. The most successful programs appear to emphasize trunk stabilization through exercise with a neutral spine (e.g., Hides, Jull, and Richardson, 2001; Koumantakis, Watson, and Oldham, 2005; Saal and Saal, 1989) while stressing mobility at the hips and knees. (Bridger, Orkin, and Henneberg, 1992, demonstrated advantages for sitting and standing, and McGill and Norman, 1992, outlined advantages for lifting.) Note that stretching programs that have enhanced hip range of motion used several static and dynamic approaches and considered technique directed to muscle, nerve, fascia, and the joint connective tissues in all directions (Moreside and McGill, 2012).

Finally, removing lumbar flexion from morning activities substantially improves patients, on average (Snook et al., 1998). Despite this evidence, many patients are still instructed to pull their knees to their chest in the morning and perform toe touches (see figure 8.14, a-c). The destabilizing consequences of full flexion were described in chapters 3 and 4.

Figure 8.14 “Silly stretches,” such as (a, b) pulling the knees to the chest and (c) toe touches, are often prescribed to patients to do in the morning. These can cause instability! Paradoxically, the stretch receptors in the back are stimulated, providing a false sense of relief that may last about 20 minutes. In reality, they increase pain sensitivity later.



Photos from Stuart McGill

On occasion, a patient will want to mobilize the spine. There are also therapeutic reasons for mobilizing the spine through a range—for example, gentle movement postsurgery to prevent adhesions and short scarring of neural and connective tissues. We have quantified that the most spine- and disc-sparing method is to adopt the hands and knees position and perform the cat–camel exercise (see chapter 10). This unloaded range of motion cannot be achieved in a standing posture and is suited to those concerned with safety. Those interested in specific athletic activities may sometimes be an exception to this rule. (Of course, spine flexibility may be more desirable in athletes who have never suffered back injury.)

Strength

In general, strength seems to have little to do with back health even though increasing torso muscle strength is a popular objective of low back rehabilitation protocols (note that the issue is rehabilitation and not athletic performance). This is not to imply that strength is not important. Rather, it is to emphasize that the way the spine moves is relatively more important in terms of spine health.

Back muscle strength in particular has not been found to be a significant predictor of first-time injury. (In the context of cause and effect, predicting first-time injury offers special insight.) Only Troup, Martin, and Lloyd (1981) found, while testing torso muscles, that reduced dynamic strength was a predictor of recurring back pain. However, in a prospective study, Leino, Aro, and Hasan (1987) found that neither isometric nor dynamic trunk strength predicted the development of low back troubles over a 10-year follow-up period. The Biering-Sorensen (1984) study, previously noted, found that isometric back strength did not predict the appearance of low back trouble in previously healthy subjects over a 1-year follow-up. Holmstrom and Moritz (1992) recorded reduced isometric trunk extensor endurance times in male workers with low back disorders (LBDs) compared to those without, but found no differences in isometric flexion or extension strength.

Strength appears to have little, or a very weak, relationship with low back health. Strength is for athletic performance objectives. In contrast, muscle endurance, when separated from strength, appears to be linked with better back health.

Our data have suggested that although having a history of low back troubles is not related to reduced strength, it is related to a perturbed flexion-to-extension strength ratio (McGill et al., 2003). This difference in the ratio appeared to be mainly influenced by greater extensor strength relative to flexor strength in those with disorders. However, the most influential ratio was strength to endurance (see the next section for an explanation).

Endurance

Two cross-sectional studies, those of Nicolaisen and Jorgensen (1985) and Alaranta and colleagues (1994), found reduced extensor endurance in workers who reported disabling low back pain. Both Biering-Sorensen (1984) and Luoto and colleagues (1995) suggested that although isometric strength was not associated with the onset of back troubles, poor static back endurance scores were. Some have expressed concern that patients with poor muscle endurance scores have poor scores from a lack of effort (a psychological variable) rather than any physiological limitation. A study by Mannion and colleagues (2001) suggested that of the total variance measured in the endurance scores of their back patients, 40% was explained by physiological fatigue (quantified as declines in the EMG power spectrum), and only 10% was explained by psychological variables (quantified as motivation and fear of pain variables, from a questionnaire). Another study (McGill et al., 2003) suggested that having a history of low back troubles appears to be associated with a different flexion-to-extension endurance ratio—the extensors have less endurance and the flexors have more endurance. This imbalance in endurance also appeared between the right and left lateral musculature as evidenced by the asymmetry in right and left endurance holding times (e.g., right side bridge/left side bridge (RSB/LSB) ratio of 0.93 for those with an LBD history vs. 1.05 for those without). The next issue addressed the question of whether strength and endurance are related. Interestingly, the flexor strength (Nm)-to-endurance (sec) ratio was between 3 and 3.5 for the flexors in both those with normal backs and those with a history of repeated episodes of disabling pain, and for the extensors of those with normal backs. The ratio was much larger for the extensors (5.3, $p = .033$) in those with a history of LBD.

Our most recent work with firefighter and police populations simply enhanced our historical impressions. It appears that many get hurt in the training room when focusing on strength training. The stronger men were not protected from back injury. We now believe that the key is to develop skills to control movement with the available strength. People get injured when they break form. More strength will not prevent this. Rather, enhanced endurance coupled with control skills enables people to maintain good form during repeated strength exertions. This is why endurance must be established before strength is enhanced in those recovering from repeated and recurrent back pain episodes.

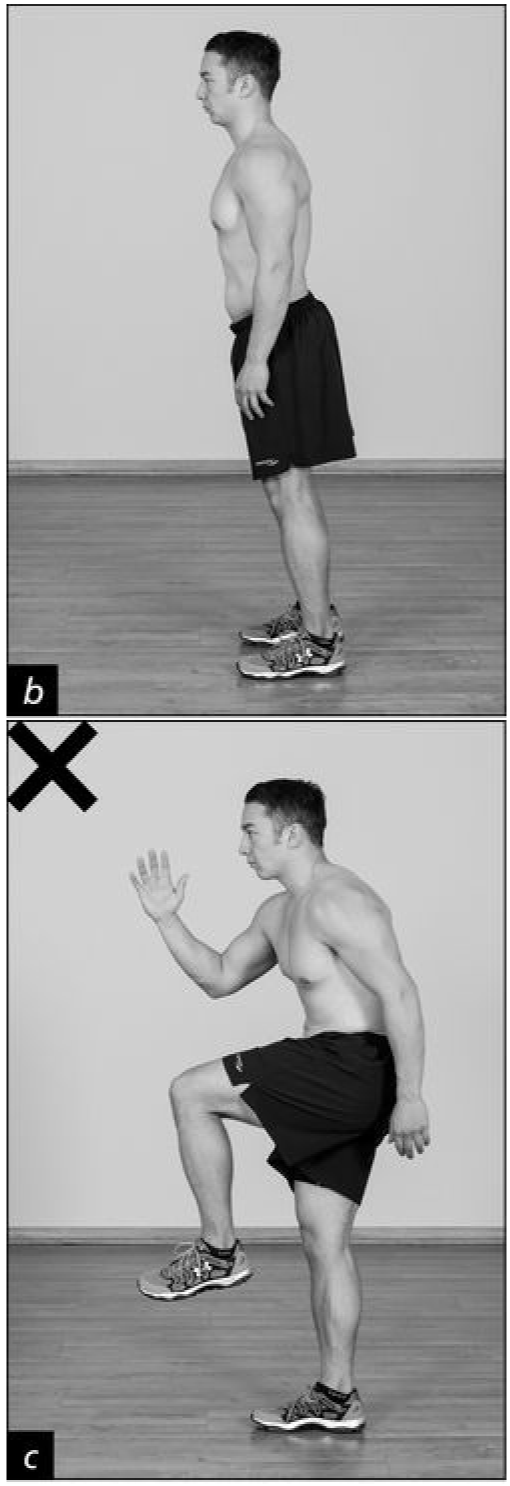
In summary, several studies have suggested that diminished trunk extensor endurance and not strength is linked to low back troubles. Recent data confirm this notion and enhance it further by suggesting that the balance of endurance between flexor and extensor muscles and the balance between the right and left sides of the torso appear to be linked to a history of back troubles severe enough to result in work loss. Note that these were losses that lingered long after the disabling episode. In those with a history of disabling LBD, the average length of time since the last work loss episode was 261 weeks (standard deviation = 275), whereas the average length of time lost from work in that episode was 7 days (standard deviation = 10). The lesson for exercise prescription is that graduated, progressive exercise programs (i.e., of longer duration and lower effort) that emphasize endurance before strength development are preferable.

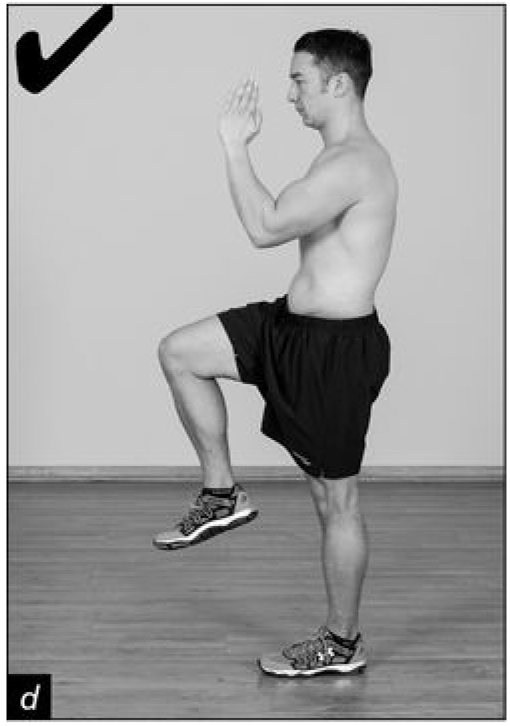
Aerobic Exercise

The role of aerobic exercise in both reducing the incidence of low back injury (Cady et al., 1979) and treating low back patients (Juker et al., 1998) is compelling. Much new knowledge has already been introduced regarding the analgesic effects of aerobic exercise at the molecular level. An investigation into loads sustained by the low back tissues during walking (Nutter, 1988) confirms very low levels of supporting passive tissue load coupled with mild, but prolonged, activation of the supporting musculature. Callaghan, Patla, and McGill (1999) documented that fast walking with the arms swinging results in lower oscillating spine loads. When tolerable, aerobic exercise, particularly fast walking, appears to enhance the effects of back-specific exercise.

Many patients reveal torso and pelvis frontal plane strength and endurance deficits. Our recent work quantifying walking and carrying loads with one and two hands showed this to be an excellent progression for addressing this deficit. The approach is tolerable to most pained patients. Fast-paced walking creates the aerobic stimulus and the frontal plane challenge necessary to train the musculature to support the pelvis in a stiffened position. This facilitates stance and leg swing with no pain. We start some patients with simple weight transfer from side to side, then progress to marching in place, then walking (see figure 8.15). Carrying continues the progression, recognizing that load in just one hand results in more compressive force on the spine than the same load in each hand (McGill, Marshall, and Andersen, 2012).

Figure 8.15 (a) Walking progressions begin with weight transfer from one leg to the other, followed by (b) learning to control forward lean using the ankles, called the leaning tower. Move on to marching and then walking. The focus should be on spine and pelvic stiffness and control, while freeing the hips. (c) Poor form; (d) good form.


b
c



Order of Exercises Within a Session

Because the spine has a loading memory, a prior activity can modulate the biomechanics of the spine in a subsequent activity. For example, if a person sat in a slouched posture for a period of time sufficient to cause ligamentous and disc creep, she would have residual ligament laxity for a period of time. (We have measured laxity of over a half hour in some cases; McGill and Brown, 1992.) The nucleus volume appears to redistribute upon adoption of a standing posture (Krag et al., 1987). This redistribution takes time. If the spine is flexed in one maneuver, then it probably should return to neutral or extension for the next.

Viscosity is another property of biological tissues—in this case, a frictional resistance to motion within the spine and torso tissues. This is why motion exercises (such as the cat–camel) are usually performed first as part of a warm-up; once the viscous friction has been reduced, subsequent motion can be accomplished with less stress.

Establishing Movement Patterns

We have found that patients are best served when we establish the preferred movement patterns for spine stability at the beginning of the session. These exercises produce a residual stiffness in the spine for both pain control and improvement of function.

A final consideration is the need to continually reinforce healthy, joint-conserving, and stabilizing motor patterns. Depending on the exercise objectives, we most often begin an exercise or training session with some spine stabilization exercises to reinforce the patterns that will continue over to other exercises in the program. Our recent work has shown that protective stiffness is retained in the torso tissues following the performance of the big three exercises (Lee and McGill, 2015). Understanding spine biomechanics, function, and pain mechanisms guides the optimal ordering of tasks in a training session.

Breathing

Debate continues regarding training for breathing during exertion. Should one exhale or inhale during a particular phase of movement or exertion?

In the rare cases of very heavy lifting or maximal exertions (which would not be part of a rehabilitation program), high levels of intra-abdominal pressure (IAP) are produced by breath holding using the Valsalva maneuver. This elevated IAP, when combined with high levels of abdominal wall cocontraction (bracing), ensures spine stiffness and stability during these extraordinary demands.

Another motivation given for striving to achieve higher IAP is the need to reduce the transmural gradient in the cranium to lessen the risk of blackout or stroke (McGill, Sharratt, and Seguin, 1995). The explanation for this risk reduction is as follows:

- Building IAP is associated with a rise in the central nervous system (CNS) fluid pressure in the spine, which forms an open vessel to the CNS and brain.
- Upon exertion, enormous elevation in blood pressure occurs (documented in weightlifters to be well over 400 mmHg).
- Pressure in the cranial vessels creates a large transmural pressure gradient that is reduced if the CNS fluid pressure is likewise elevated,

reducing the load on the vascular vessels.

Although this explanation is valid, the mechanism should be considered only for extreme weightlifting challenges—not for rehabilitation exercise.

In the design of rehabilitation exercise, a major objective is to establish spine stabilization patterns. An important feature of stable and functional backs is the ability to cocontract the abdominal wall (abdominal brace) independently of any lung ventilation patterns. Good spine stabilizers maintain the critical symmetrical muscle stiffness during any combination of torque demands and breathing patterns (such as when playing a basketball game). Poor stabilizers allow abdominal contraction levels to cycle with breathing at critical moments when stability is needed. Entraining a particular direction in lung air flow to a particular part of an exertion is not helpful for these patients. This would be of little carryover value to other activities; in fact, it would be counterproductive. As the patient progresses to performance objectives and higher loads with speed, the relationship changes. Consider the tennis players serving the ball with an audible grunt. This overdrives the abdominal wall contraction, creating a superstiffness in the core and enabling higher shoulder velocity and a faster serve. This mechanism and technique is explained in McGill (2014).

In summary, patients susceptible to pain should train to breathe freely while maintaining the stabilizing isometric abdominal wall contractions. Entrained breathing comes later as the progression shifts from eliminating pain to enhancing performance.

Time of Day for Exercise

As pointed out in part II, the intervertebral discs are highly hydrated upon rising from bed; the annulus is subjected to much higher stresses during bending under these conditions, and the end plates fail at lower compressive loads as well. Thus, performing spine-bending maneuvers at this time of day is unwise. Yet many manual medicine physicians continue to suggest that patients perform their therapeutic routines first thing in the morning. This appears to be due to convenience and ignorance. Because the discs generally lose 90% of the fluid that they will lose over the course of a day within the first hour after rising from bed, we suggest simply avoiding this period for exercise (that is, bending exercise) for either rehabilitation or performance training. Although there hasn't been a study on the enhancements obtained during exercise routines as a function of time of day, Snook and colleagues (1998) did discover that the conscious avoidance of forward spine flexion in the morning improved their patients' backs.

Notes for Rehabilitation Exercise Prescription

Clinical Relevance

Exercise professionals face the challenge of designing exercise programs that consider a wide variety of objectives. Consider these guidelines:

- Although some experts believe that exercise sessions should be performed at least three times per week, low back exercises appear to be most beneficial when performed daily (e.g., Mayer et al., 1985).
- The no pain, no gain axiom does not apply when exercising the low back, particularly when applied to weight training. Scientific and clinical wisdom suggests that the opposite is true.
- Research has shown that general exercise programs that combine cardiorespiratory components (such as walking) are more effective in both rehabilitation and injury prevention (e.g., Nutter, 1988).
- Diurnal variation in the fluid level of the intervertebral discs (discs are more hydrated early in the morning after rising from bed) changes the stresses on the disc throughout the day. People should not perform full-range spine motion under load for 1 to 2 hours after rising from bed (e.g., Adams and Dolan, 1995).
- Low back exercises performed for health maintenance need not emphasize strength with high-load, low-repetition tasks. Rather, more repetitions of less demanding exercises enhance endurance and strength. There is no doubt that back injury can occur during seemingly low-level demands (such as picking up a pencil) and that injury from motor control error can occur. Although the chance of motor control errors that result in inappropriate muscle forces appears to increase with fatigue, evidence also indicates that passive tissue loading changes with fatiguing lifting (e.g., Potvin and Norman, 1992). Given that endurance has more protective value than strength (Luoto et al., 1995), strength gains should not get ahead of endurance.

- No set of exercises is ideal for everyone. An appropriate exercise regimen should consider the person's training objectives, be they rehabilitation, reducing the risk of injury, optimizing general health and fitness, or maximizing athletic performance. Although science cannot be used to evaluate the optimal exercises for each situation, the combination of science and clinical experience will result in enhanced low back health.
- Both patients and clinicians should be patient and stick with the program. Increased function and reduced pain may not occur for 3 months (e.g., Manniche et al., 1988).

A Final Note

The five stages described in this chapter have a rationale and a progression based on scientific evidence of mechanisms and efficacy. Not every patient will need each exercise. However, following the stages greatly enhances the chances for success.

Chapter 9
Evaluating the Patient

The typical orthopedic exam determines the range of spine motion, some neurological measures such as the strength of reflexes, and perhaps some qualitative measures of muscle strength. These measures provide little guidance for designing prevention and rehabilitation programs. Further, we published a study (Parks et al., 2003) in which we tracked back pain patients in a pain clinic. Scores obtained from patients' assessment had very little relation to who recovered and returned to work. Quite often, these numbers are more for the legal determination of disability than for aiding in the clinical decision process. For example, some erroneously believe that all patients should have normal to average range of motion (ROM) values even though they probably were not average prior to becoming a patient. The tests discussed in this chapter offer more useful indicators of pathology to assist you in making treatment decisions.

Of the several deficits in low back variables identified in earlier chapters, many are the direct result of injury. They include aberrant lumbar motion patterns, perturbed motor patterns of muscle recruitment, and aberrant joint motion with concomitant pain and loss of muscle endurance. Unfortunately, testing for these deficits is not easy. The challenge is to find the tests that can best identify the deficits and that are reasonably safe and do not require expensive or specialized equipment. The tests that come closest to meeting these criteria are described in this chapter, along with guidelines for tests to quantify patient deficits. The results form the rehabilitation objectives for the patient and provide clues for designing exercise. This chapter also discusses how patients can help to define their own rehabilitation targets.

There is no such thing as nonspecific back pain—there are only people who have not had thorough assessments. Provocative testing, when combined with movement screens for joint symmetry of motion, strength, and endurance, underpins a powerful classification for people with back pain. Classification enhances the therapy plan and identifies what to avoid. The process continues throughout the recovery process to define tolerable levels of load in specific postures and movements so that the dosage of therapeutic exercise can be tuned to the person.

The general approach begins with an interview followed by an assessment of movements, postures, and loads that cause pain. Then the patient is shown the cause of the pain. This is crucial for learning to avoid the cause through movement technique. Then, and only then, is corrective and therapeutic exercise considered. Yet too many clinicians unwittingly engage the patient in strengthening routines without this process, and fail to achieve a pain-free state.

Most Crucial Element in Evaluation

Before learning about any tests or techniques, it is vital that you understand the central element in all diagnoses: your brain! If your own observation and reasoning skills are not well developed, the best tests and the most advanced technology will be of little use, as the following text will show.

Geoff Maitland, the well-known Australian physical therapist, promoted the hypothesis formulation approach to diagnosis—which is very similar to our own. Like a crime detective, a clinician must consider evidence from all sources. As each piece is considered, the hypothesis is either strengthened or weakened. However, unlike the detective, the expert clinician can obtain a definite history, even though a patient may present conflicting signs and disguised characteristics. This complexity means no more than that the patient has a complex presentation. Such patients may be farther down the degenerative cascade of tissue and nervous system change, or they may have some biological processes under way that complicate the search. Biomechanical knowledge is critical for success in hypothesis formation. Usually, the overused tissues are the ones involved in symptom creation. The solution often lies in changing the biomechanics to avoid loading painful tissues.

A keen clinical eye is a feature of all great clinicians. I often tell the story of when I was invited to teach at a renowned spine center and part of my course was to conduct three examinations in front of the center's 18 clinicians. The clinicians included orthopedists, neurologists, physical therapists, and so on. We went to the waiting room and were introduced to the first patient. The patient rose from the chair and walked to the large training room that served as our exam room. Then I asked the clinicians to turn away from the patient and face the wall. I asked them to tell me about the patient—and not one of them could. I told them that they had all failed.

Interpreting Patient Presentation

Clinical Relevance

Form a working hypothesis and continually reassess the hypothesis, doing the following:

1. Observe everything, starting with the person's sitting posture, how she rises from the waiting room chair, how she stands, and how she walks.
2. Elicit and record the history: link injury mechanisms and pain mechanisms.
3. Perform provocative tests to determine the loads, postures, and motions that exacerbate the pain and those that relieve it.
4. Perform functional screens and tests to determine whether perturbed postural, motion, and motor patterns exist.

I then described all that I had observed, beginning with the seated posture of the patient, which was a full-flexion, slumped posture (this usually suggests flexion intolerance). Rising from the chair was initiated with more spine flexion and less hip extension. Then the hips rose, indicating typical hamstring dominance for hip extension and gluteal deficiency in generating the hip extensor torque. The spine extended last with spine extensor muscle activation. This strengthened the working diagnostic hypothesis that was forming in my head. The classic antalgic walking posture was shown with arms swinging and bending at the elbows instead of the shoulders. A keen ear could hear the asymmetry in footfall with the right-sided deficit in tibialis anterior. Then I observed the classic standing posture with the weight shifted predominantly onto one leg.

After pointing out these features, I stated that my working diagnostic hypothesis was a disc bulge on the right posterolateral side of L4-L5. Further testing showed this to be correct. The other two patients included a stenotic person and one with a highly unstable spine lumbar hinge with overstiffness in the thoracic region. After conducting the same qualitative observations on these two, the clinicians understood what I meant when I said that their eyes, ears, and hands were their best scanners. Interestingly, a few older clinicians stated that they had forgotten their observational skills because of access to high-tech medical imaging—this was a poignant reminder to rehone their observation and assessment skills.

What Every Clinician Must Know

Assessing a patient usually reveals several dysfunctions. I have seen patients with a litany of dysfunctions in the referral, all with prescribed treatment plans. Yet none of the dysfunctions listed caused their back pain. As clinicians, we need to focus on reducing the pain first, meaning address the big issues and show the patient quickly how to reduce the pain. Teaching him how to activate the transverse abdominis rather than showing him how to get out of a bed pain free is a disservice. The painless dysfunctions can be addressed later.

The implication of the previous paragraph is that the clinician must have expert competence in, skill in, and knowledge of the following:

- Injury and pain mechanisms as well as mechanisms of pain desensitization and tissue repair. Those without this knowledge make statements such as, We do not know the source of pain. Those with this knowledge and skill use the precise application of stress to a targeted structure, or tissue within a structure, while looking for the onset of painful symptoms. They can replicate the patient's familiar pain or create new pain.
- The information content of each test and an understanding of what each test reveals. Some simple tests are reliable but limited in what they reveal. The more involved tests are not reliable and require clinical skill, but have great potential to reveal the pain mechanism. Both are necessary to understand patients' pain.
- How to adjust each provocation test posture, load, range, trajectory, and neural and other tissue tensions to deeply probe the mechanism of the pain.
- Corrective and therapeutic exercise selection, dose, technique modification, and progression.
- Comorbidities that cause or mask back pain. Sembrano and Polly (2008) diagnosed 289 patients presenting at a spine surgeon's practice. Of these, 65% had spine-only pathology, and 17.5% had additional hip or sacroiliac (SI) pain sources. Eight percent had hip SI joint pathology (or both) with no spine pathology, and 10% had an undefined pain source. This is consistent with our clinical experience.
- Subgroups of patients. Average clinicians may have three or four subgroups that are assigned specific treatments or approaches. Highly skilled clinicians have finer criteria to the point that each patient

becomes a unique case study.

- Variables that influence the assessment. A patient who is flexion posture intolerant will experience exacerbated pain symptoms from the drive to the assessment appointment. The keen clinician understands that her observations are modulated by the prior loading history of the patient.
- The influence of perception and personality on the presentation.

What Every Patient or Client Needs to Know

The medical system does not always give all involved parties the necessary information to optimize recovery. All workers with back pain need to know the following to facilitate their recovery:

- Exam results. Their current scores give context to their goals.
- Natural history and prognosis. There is no evidence that back disorders last into retirement; in fact, they are often addressed with appropriate classification and treatment plans.
- Causes of pain. Patients are often amazed to learn that the way they move and activate muscles can eliminate pain.
- What to avoid. Removing the cause of the disorders is an obvious strategy; this also improves the efficacy of the therapy.
- Recovery plan. A progression should begin by addressing the movement disorders with corrective and therapeutic exercise, and then move on to stabilizing body areas needing stabilization and mobilizing those that need mobilization. Next in the progression is enhancing endurance so that joint-sparing movement patterns can be repeated even when fatigued, followed by building some strength and possibly some power-generating ability at the hips and shoulders if appropriate.

First Clinician–Patient Meeting

Every person with back pain has limited capacity for physical work. Every aspect of therapeutic exercise must be justified; otherwise, capacity is wasted. The objective is to determine the capacity, determine what is tolerable, determine the deficits, and design the best therapy to rectify the shortcomings. Having a road map to follow will allow you to plan for your patient the fastest and most efficient journey to optimal back health. Finally, you should proceed only after you have screened your patient for all red flag conditions. The following checklist will help you determine appropriate rehabilitation exercise.

1. Identify the rehabilitation objectives (specific health or performance objectives). The specific rehabilitation objective determines the acceptable risk-to-benefit ratio. A performance objective carries higher risk. Because the principles of bodybuilding and athletic training are so pervasive, you need to be sure that all patients understand the difference between athletic performance objectives and those for pain reduction and improved daily function. This implies that the recovery plan should first direct all efforts toward pain elimination and then when successful, address enhancing athleticism.
2. Ask key questions. Ask the patient, Do your symptoms change in intensity? Do you have better and worse periods? If the answer is yes, you are guaranteed success. Identify what makes the pain worse and what alleviates it. Ask, Do you get a sharp pain rolling over in bed? If the answer is yes, the patient has instability to the point that when the spine is not controlled with muscle stiffness, joint micromovements cause sharp pain. He will most likely do well learning to replicate these relieving patterns. Another key question is Do you have morning stiffness or pain that diminishes throughout the morning, or does the pain ramp up throughout the day? Pain that ramps up usually indicates that loading causes cumulative symptoms. Teaching the person “pristine” spine-sparing movement strategies will delay the symptom onset. Morning stiffness may indicate an inappropriate bed or mattress. Follow up this suspicion by asking whether the person has a different pain pattern after sleeping, for example, in a hotel bed. Other questions

and responses hone the focus for the remainder of the interview stage.

3. Consider patient's age and general condition. Younger patients (from the teens to the fifth decade) tend to have more discogenic troubles, whereas arthritic spines tend to begin developing after the age of 45, and stenotic conditions develop after that. Note how patients walk and sit. Are they in noticeably poor condition, either emaciated with little muscle mass or heavy with fat rather than muscle? It is also assumed that patients have been medically screened and cleared for cardiovascular concerns, or any red flag conditions such as tumors.
4. Identify occupation and lifestyle details. Generally, you should begin by documenting patients' daily routines: when and how they rise from and retire to bed, meal routines, and exercise and recreational habits. Then direct specific focus toward areas of concern. For example, if the patient reports watching TV for 2 hours in the evening, ask for details on the type of chair, range of postures used, and so on. After gathering information about the patient's daily routines, inquire about occupational demands. All of this information, when added to the clinical presentation, will help you evaluate common links. Discogenic troubles are linked with prolonged sitting (particularly prolonged driving) and repeated torso flexion. A passive or inactive lifestyle is also associated with disc troubles. Arthritic conditions, facet troubles, and the like, are more linked with jobs and activities that involve large ranges of motion and higher loading. Former athletes such as soccer players also fall into this category, although long-distance runners do not because they do not, presumably, take the spine to the end ROM.
5. Consider the mechanism of injury. Attempts to re-create injury mechanisms are fruitful only when the real mechanisms are understood. These were detailed in chapters 3 and 4. Once identified, the mechanisms can be linked with specific tissue damage (much of which is otherwise not diagnosable). Not only will this assist in designing the therapeutic exercise, but it will also help in teaching patients to avoid loading scenarios that could exacerbate the damage and symptoms. Note that some of these injury mechanisms will have acute onset, whereas others progress slowly. Slow onset may result in some patients' being unable to identify the mechanism of injury. Nevertheless, a culminating event is usually involved. Careful questioning about events leading up to that event will provide clues as to the mechanisms of injury.

6. Have the patient describe the perceived exacerbators of pain and symptoms. Prompt the patient to describe the tasks, postures, and movements that exacerbate the pain. Examine these tasks from a biomechanical perspective to determine which tissues are loaded or irritated. These tissues should be spared in the exercise therapy, and the exacerbating movements minimized with movement pattern coaching.
7. Have the patient describe the type of pain, its location, whether it is radiating, and specific dermatomes and myotomes. Descriptions of the type of pain are usually helpful; patients may describe their pain as deep and boring, scratchy, sizzling, at a particular point, general over the back region, continually changing, and so on. You may need to help some people describe their pain by offering adjectives to choose from. In chapters 3 and 4 I described the link between pain types or patterns and specific tissues and syndromes. Keep in mind that changing symptoms over the short time of an examination generally suggest more fibromyalgic syndromes, which can sometimes be resistive to exercise therapies—particularly ones that cause pain. The issue here is that pain-free motion must be found and repeated, so that people can slowly expand their repertoires of pain-free motion.
8. Consider patient personality and perception. Patients perceive similar symptoms differently. For example, the patient who exhibits self-manipulating behavior during the interview (e.g., twisting the spine to initiate a crack) usually give high priority to the stretch reflex. The same patient will likely report enjoying yoga and daily stretching. In contrast, another patient perceives stretching as discomfort—she interprets the stretch reflex differently. These two patients require different clinical approaches. In a similar fashion, some perceive muscle work as pain, whereas others perceive the difference between muscle work and back pain. Distinguishing these two presentations, and coaching patients in interpreting their perceptions, will influence their compliance. The influence of personality on compliance is poignant when we work with experimental groups of diabetics and breast cancer survivors at our university clinic. The buzz in the room with each group is the polar opposite of that of the other. When the cancer survivors are asked to perform 10 repetitions of a therapeutic exercise, typically they perform more when the clinician is not looking. They are typically type A people, a group of overachievers who take control to regain their fitness.

The diabetic group is typically the opposite, being made up of more type B personalities. When those with diabetes are asked to do the same 10 reps, they bargain for why they need to do only 3. They are generally, as a group, movement averse. Personality influences their presentation and response to advice. I consciously note whether the patient will need encouraging to engage in therapeutic exercise or will need holding back so he does not work until pain erupts.

9. Take dermatomes and myotomes into account. With radiating symptoms, the dermatomes and myotomes can assist in understanding the involved segmental levels and whether the pain originates from a specific nerve root. For example, direct pressure on the root could indicate a unilateral disc bulge or end-plate fracture that would cause a loss in disc height together with a loss of root outlet foramen size. In this way the spinal level can be linked with the dermatome or myotome but not the actual tissue damage. Further, nerve root pressure can occur at a specific spinal level on the outlet nerve, consistent with a dermatome or myotome, or on the traversing nerve from above if there is pressure on the cauda equina centrally. Thus, dermatomes and myotomes are another consideration when forming an opinion from the results obtained from several tests that can include medical imaging and provocative tests.
10. Perform provocative tests. Having observed the patient sit, rise from a chair, stand, and walk, you have developed a working diagnostic hypothesis. Once you suspect that specific tissues are damaged or sensitive, you can load them to see whether loading produces pain. This is provocative testing. Many patients have more complex presentations, with several tissues involved. Nonetheless, the provocative procedure still indicates which postures, motions, and loads cause pain and specific motions, postures, and loads that should be avoided when designing the therapeutic exercise. Generally, patients' descriptions of the activities they find exacerbating of their pain (item 6 on this list) will guide your decision as to which tissues to load and stress. For example, lumbar extension with a twist can provoke the facets, whereas the anterior shear test may be warranted for suspected instability (shown in the Testing for Lumbar Joint Shear Stability).
11. Perform functional screens. To determine whether a patient is moving in a spine-conserving and efficient way, use functional screens. In addition, functional screens indicate the suitability of a specific exercise or serve

as qualifying tests prior to exercise prescription.

Interpreting Pain Reports Obtained During the Interview

Clinical Relevance

A skilled clinician can often create a very robust hypothesis simply by listening to the patient's story and asking strategic questions. This sidebar presents observations from my own clinical experience given the fact that we follow up with every patient who comes to our research clinic. This allowed us to assess how effectively we interpreted patients' pain reports and how well they responded to our specific recommendations.

- Of course, recognizing red flag conditions is essential. These include a history of trauma, bowel or bladder incontinence, a history of cancer, unexplained weight loss, intravenous drug use, systemic illness, low back pain with fever, progressive and constant night pain, abdominal pain (between the navel and pubis), saddle anesthesia, and no response to motions, postures, and loads.
- Disc symptoms that are radiating generally centralize, or are relieved with specific postures and movements. Onset can be sudden. A feeling of giving way and other similar descriptions of instability in neutral joint ranges are common. Interestingly, reported dermatome distributions are more accurate than neurological signs. Although radicular patterns (pain, dermatomes, myotomes) demonstrate anatomic variability, the following are most typical:
    - L1, L2: Pain in the lower abdomen, groin, and medial thigh with hip flexor myotomes
    - L3: Pain in the anterior thigh and knee, with knee extensor myotomes
    - L4: Pain in the medial malleolus and small toes, with an ankle dorsiflexion myotome and a patellar reflex (L2, L3, L4)
    - L5: Diminished pain in the great toe and lateral calf, with ankle eversion myotomes and a diminished Achilles reflex (L5, S1)
- Sacroiliac (SI) joint symptoms are rarely above the L5 level; rather, they are along the spine midline or a few centimeters lateral. They are often

exacerbated by standing up and performing split lunges, and may be associated with a history of trauma. Typical wisdom is that generally three or more traditional SI joint provocation tests should be positive to conclude that SI joints are the pain source (but these do not isolate SI joint loading). We put more weight in the fist pulse test and the walking compression test described in the section Test for Sacroiliac Joint Pain. Interestingly, pain during provocation is on the unstable, hypermobile side, yet symptoms are more often on the hypomobile side. Clinicians often treat the wrong side!

- Facet joint pain can be radiating, and is usually slower in onset and in resolution. Pain is usually isolated with extension and rotation. Facet disorders are rarely the primary injury since they are usually secondary to discogenic disorders.
- Muscle pain is often mistaken as the only source given that it usually indicates underlying pathology. The pain usually peaks 24 to 48 hours following trigger exposure and responds to techniques such as active release therapy (ART) and contrast heat and cold. It is sometimes confused with neural tightness, particularly in the hamstrings.
- Spondylolisthesis is usually confirmed with radiological imaging, but symptom confirmation can be done with the spondylolisthesis test described later in this chapter. Evidence from MRI T2 images of fluid in facet interspaces is another strong indicator. People typically report having a continually "grumpy back" that is exacerbated by motion, prolonged postures, and sleeping postures. Standing extension pain is often lessened during the one-legged stork test.
- Stenosis may be confused with claudication (motor losses after stair climbing or walking). Symptoms have a slow onset and include pain (although sometimes not in the back) and radiating numbness. This is one of the few conditions that respond to traction and traction with extension. As with many of these syndromes, a key for recovery is to train in short, tolerable intervals.
- Compression fractures are most common in people who are osteoporotic, although people who participate in outdoor sports (hunting and fishing) also experienced them. Radiological confirmation (e.g., vertebral collapse, split vertebral end plates and sacrum, Schmorl's nodes) is needed. The heel drop test conducted with care assists in confirming symptoms, which can range from local periosteum pain to

radiating symptoms.

- Ankylosing spondylitis has an insidious onset usually of morning stiffness and a progressing flexion antalgic posture. Lewit extension exercises performed daily may assist with posture and pain control.
- Arachnoiditis cases are some of the saddest I have seen. They are often the result of postsurgical nerve root scarring. This very debilitating condition may also be instigated by complications from epidurals, myelograms, and so on. We have had a few successes with nerve flossing and movement control.
- Psoas abscess is a rare condition usually associated with a history of fever, infection, and prior surgery. MRI and blood work are needed for confirmation.
- Fibromyalgia is usually characterized as swirling pain around the shoulders, hips, and back, with accompanying headache. Because more activity increases symptoms, exercise therapy can be challenging. I am convinced that people with fibromyalgia have usually been associated with a history of trauma sufficient to rewrite and rewire movement engrams. Thus, the brain perceives these movements as pain generators. The key, I believe, to the few successes that we have had is for people to find pain-free movement patterns and practice them. They should then expand their pain-free repertoires as their potential grows.
- Neurological engram disruption occasionally follows trauma. These patients have forgotten basic movement and motor patterns. They are unable to cross their legs when sitting, or patterns of rising from a chair are missing. This may occur as a result of motor cortex rewiring from a traumatic event or of traumatic spinal cord shock when the engrams that reside in the cord are lost. The therapeutic approach is to help people relearn movements with repeated, short-duration practice. Insist on impeccable form.
- Scheuermann’s disease requires radiological confirmation, which shows wrinkled vertebral end plates. Patients report constant pain exacerbated by activity and workload. We have observed this to appear more frequently in taller and lankier males. If you coach the person in spine-sparing movements, he will likely grow out of these adolescent pains.
- Hip pain from the joint may radiate into the groin, scrotum, medial thigh, femur, and deep buttock. The hip is confirmed as the source with provocative testing isolating the hip capsule, labrum, and acetabulum.

Hip pain results in what is called gluteal amnesia and often psoas tension. This may be confused with piriformis syndrome, which is usually associated with blunt trauma to the buttock posterior hip joint region, or the sciatic nerve piercing the muscle (confirmed radiographically). Interestingly, hip and back pain often occur simultaneously because of the mechanical linkage. When hip extension or stiffness is different unilaterally, the spine often acquires a twisted rotation, which changes both static and dynamic mechanics.

- Complex symptoms may simply reflect a constellation of disorders. People with complex symptoms are sometimes labeled bizarre or, worse, accused of malingering or told that the pain is all in their heads. Proper interpretation is required to unravel these comorbid syndromes.

Assessing Posture and Movement Quality

Posture and movement patterns influence the loading of specific tissues. These habitual patterns result from engrams, which are encoded motor control patterns. Apart from acute trauma, these motor control patterns are probably the most important determinant of back injury. This opinion has been gained from both working as a consultant to athletic teams and programs and performing longitudinal studies. For example, I have consulted with a number of Olympic programs and sport organizations, both professional and amateur. Watching the movement patterns of individual athletes early in the season motivated me to write down who I thought would become injured and in what way. For one team, I predicted that two players would sustain back complaints that would affect their play (they tended to initiate movement with the spine rather than the hips, as their teammates did). I also predicted that two athletes would sustain ankle injuries because of the heavy pounding of poorly directed forces down their legs, and that one would sustain a knee injury because he used the knees to brake his motion rather than the hips. I correctly predicted four out of five injuries (the knee case did not materialize that season). Movement patterns predicted who would become injured. The corollary is that this knowledge could be used to intervene and reduce the risk.

Using a more scientifically rigorous example, we performed a study on 76 workers who performed the same physical job (McGill et al., 2003). A total of 26 had recurrent back episodes lasting on average a couple of weeks per year, but all were at work and functioning pain free on the testing day. Those who had recurrent painful back attacks differed from their healthy colleagues. When bending to pick up a coin from the floor, they bent their spines more and their hips less than their healthy colleagues did. They had more back strength when tested. We assumed that this was because they overused their backs when working. They also had less muscle endurance. It became clear that although they were stronger, their lack of endurance caused them to break form when lifting, imposing stresses to their back that led to constant tissue irritation. They had stiffer hips, particularly in flexion and internal rotation. Interestingly, psychosocial markers were present in the recurrent pain workers, on average, but they were less important than those considered biomechanical and motor control in nature.

In a study of adolescents, Smith, O'Sullivan, and Straker (2008) showed higher odds for having back pain with nonneutral sagittal spine postures when standing. It's interesting that skilled clinicians can often pinpoint the location of the pain index simply by seeing the region of highest deviation or the location of a spinal hinge observed in bending or during the cat–camel exercise.

The combination of biomechanics and motor control variables in provocative tests and movement assessment screens is perhaps the most powerful component of an examination of a person with back pain. However, just because a person can move in a certain way during testing does not mean that she will move in a joint-sparing way when performing tasks of daily living. This means that a simple movement screen, one that is performed quasi-statically and without substantial external load, does not predict how a person will move in other tasks. We studied the movement patterns of firefighters in simple tests and then quantified their movements while they performed tasks such as carrying ladders, chopping holes in roofs, and pulling hoses. The simple tests did not predict how they loaded their bodies when loads and speeds were increased to real-life conditions (Frost et al., in press). In another study, we screened 180 students to quantify their squat and lunge mechanics together with their hip and back range of motion. Interestingly, there appeared to be little relationship between whether they could move in a joint-sparing way versus whether they chose to move in a joint-sparing way. In other words, just because a person can move in a certain way in a controlled test doesn't mean he will move that way in real life. Motor control patterns, or patterns of movement, need to be trained in certain people. Similarly, people should be assessed while performing real-life tasks.

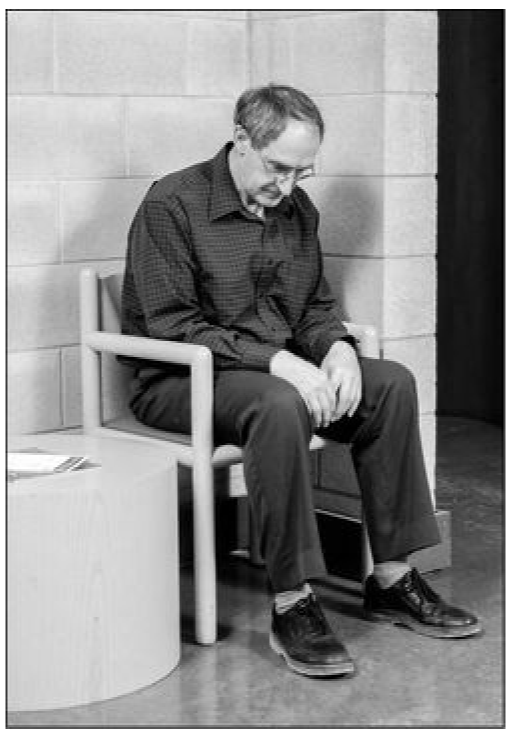
Our movement assessment begins during the interview by observing the person sit. Then we observe sitting-to-standing, walking, and pulling to open doors while walking from the interview office to the assessment clinic. We look for hip-hinge patterns versus spine bending, walking with stiff hips and shoulders, pulling with spine motion rather than hip movement, and a stiffened spine. These patterns were introduced in chapter 8.

Formal Assessment of Posture

A formal assessment of posture includes an assessment of sitting, standing, and lying. Pain is present because the back is currently a weak link. Functional tests help to sort out why the back is a weak link and help indicate what is required to bolster the deficit. Some tests are posturally based, whereas others assess control of motion. The following postural tests are very helpful. Recall that screening need not involve manipulation or special positions. Some of the most useful "tests" are simply intelligent observation.

Sitting Posture

Remarkably, patients with troubled backs often sit in a way that causes more back troubles. For example, patients who show flexion intolerance during provocative tests often have a typical sitting posture that involves a lot of spine flexion. In a similar way, those who show extension intolerance often sit with the spine locked into extension. The sitting posture is the first clue in hypothesis formation because this is often the posture that is first observed in the waiting area. Sitting in the waiting room in a flexed posture often suggests flexion intolerance.



Photos from Stuart McGill

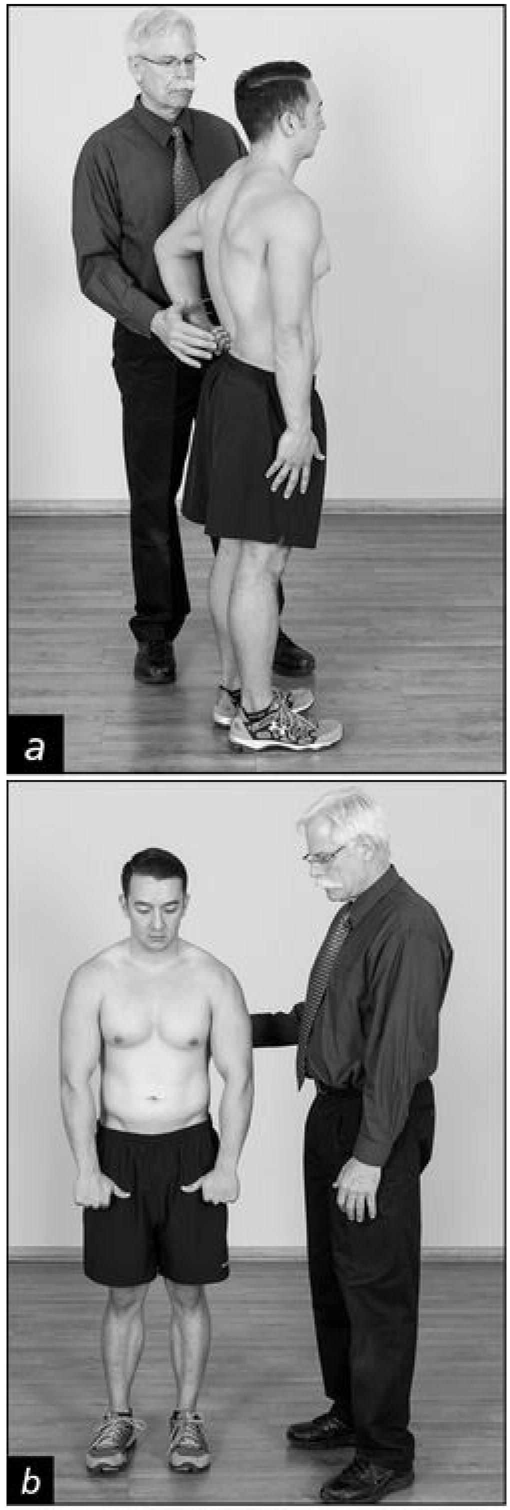
After noting the sitting posture, observe how the patient shifts from a slouched sitting posture to an upright one. Did she lift the rib cage, stressing the thoracolumbar junction (a and b). Or did she flex the hips, rolling the pelvis forward to align the spine (c). Our recent work has shown that the best choice for most patients is to use a combination of the two (Castanhero et al., 2014) to minimize stress and avoid pain.

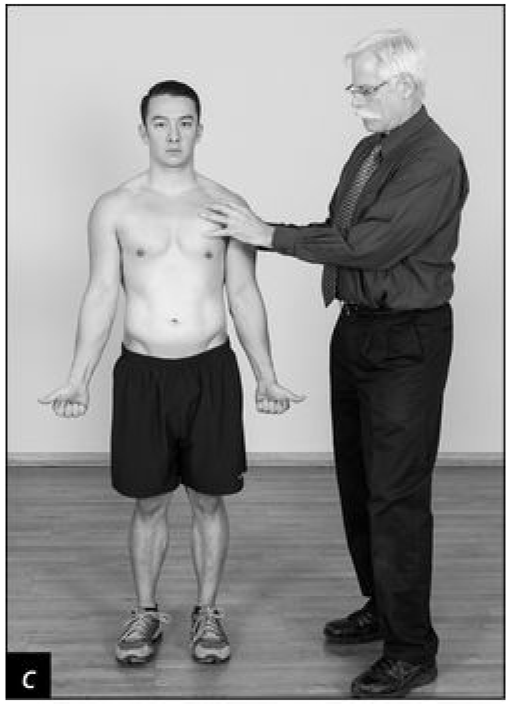
Does the patient sit with a neutral spine (a) or a flexed spine (b)? When shifting from a slouch to a more upright posture, is the movement concentrated about the thoracolumbar junction or hips (c)?

Standing Pattern

All sorts of clues are revealed in the standing posture. Simple palpation of the standing patient’s lumbar extensor muscles will reveal whether the patient is chronically crushing the back with extensor contraction. Simple postural corrections such as extending the hip, retracting the shoulders posteriorly, and pulling in the chin can shut these muscles off.

Teach patients to palpate the erectors at the L3 level, and learn where the switch point is between muscle activation and muscle relaxation. They do this by standing upright, head erect and shoulders back, and then leaning forward slightly (a). Then they perfect this strategy to relax the muscle. Teach them that chin poking (b) and shoulder slouching (c) activate the muscles that cause fatigue cramps. Have them try the hitchhiking thumb position as a correction strategy, externally rotating the arms about the shoulders and then letting the shoulders relax into this new position (d).Having patients palpate the lumbar erectors helps them learn self-correcting strategies—are the erectors silent or active (a)? If they are active, show the patient how to shut them off by unloading the spine, which often provides immediate relief of muscular pain. Cues include shifting the head and neck from a chin poke to chin retraction position, adopting a hitchhiking posture with external rotation of the arms and shoulders, and pulling the hips forward (b, c).



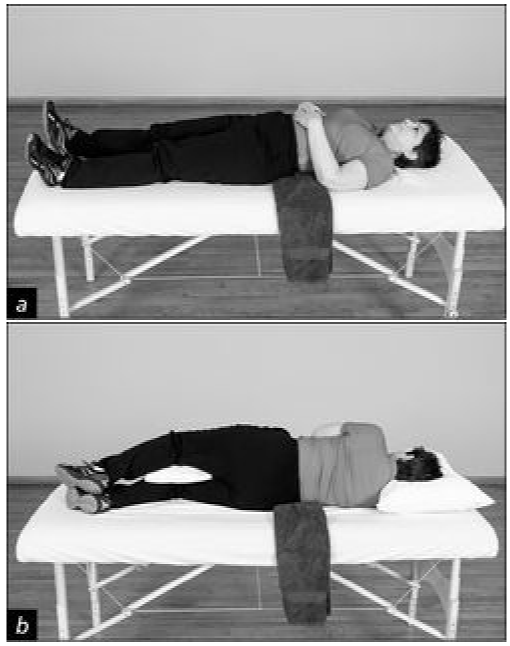


No amount of muscle relaxants will shut off the painful back muscles if the patient stands in a slouched posture. Standing to spare the spine requires a finely tuned blend of postural adjustments and muscle activation patterns. One correction is to steer the thumbs in external rotation, retracting the shoulders (see b and c) to a spine-sparing, corrected posture.

Some patients stand with no measurable extensor or abdominal muscle activation—they simply balance on their passive elastic spine tissues. These people tend to have thick torsos with flaccid abdominal walls. For these patients, pain reduction can be achieved with mild abdominal contraction; 2 or 3% of maximal voluntary contraction is considered normal abdominal wall muscle tone during standing (Juker et al., 1998).

Lying Pattern

Stress during lying is a function of body shape, the lying surface, and the pain mechanism. For back sleepers with protruding buttocks, lying on a hard surface causes a bridge under the lumbar spine. This causes spine flexion as the back flattens to the mattress and pain in the flexion-intolerant back. This may be addressed with support such as a folded towel (a, b). In a similar fashion, wide hips cause full, and painful, spine lateral bend in side-lying. In this case, a pillow-top surface can provide relief.



(a-b) Painful backs with lying and sleeping postures are usually relieved with support to foster a neutral posture. This support may be in the form of mattress selection (adjusting firmness and the thickness of the pillow-top) or buttressing devices such as a folded towel in a person whose spine is bridged with protruding buttocks (a). A pillow to support the neck and knees may assist with side lying (b).

Formal Assessment of Movement

Qualitative observations begin the instant the patient is greeted. At our clinic the assessment room is downstairs from the interview room, which provides an opportunity for observing sitting-to-standing, walking, descending stairs, and pulling and pushing heavy doors. A more formal assessment of basic movement patterns is conducted following the interview. A poor and painful pattern (a-d) and a better spine conserving pattern (e-h) is shown.


a
b


c
d


e
f

Rising From a Chair

Rising from a chair can be very revealing of numerous motion and motor patterns. For example, the flexion-intolerant patient initiates rising from a chair first by lumbar flexion and even sometimes hip extension**.** Such patients' movements exacerbate the painful tissues, preventing recovery. A conserving approach for the flexion-intolerant person would be to initiate the movement with a lifting of the chest, causing lumbar extension first with corresponding hip flexion motion. The feet are underneath the center of mass of the body, and hip extension drive is dominated by gluteal muscle activation. Hip power in this case is often helped by shifting the pelvis forward in the chair and planting the feet wider apart on the floor.

00:00 / 00:00

Also, rising from a chair often reveals spine hinges. These are regions, or motion segments, in which a disproportionate amount of the motion occurs. These overused regions are often the site of local symptoms, and until they are addressed, the back often remains chronic.

Assessing Basic Patterns of Movement

Motion restrictions other than in the spine may also be observed when a patient rises from a chair—for example in the hip, knee, or ankle. These restrictions may be complicating the diagnostic hypothesis and may or may not become a component of the back therapy.

Squat: Observe where movement is initiated—is it in the spine, hips, knees? Teach the shortstop squat position, with appropriate corrections. Then drop a coin on the floor and ask the person to pick it up. Observe the choice of movement strategy. If the person squats, point out that this is the wrong movement tool. Demonstrate the golfer's lift.

Lift: Have the person lift a heavy object (appropriately heavy for them). Teach appropriate torso stiffness, the antishrug, the hip hinge, and appropriate bracing for movement and pain control.

Lunge: Observe the basic lunge pattern and correct to help the person maintain an upright torso. Then have him lie on the floor. Did he choose the lunge with a crawl pattern as the preferred movement tool? (See chapter 8.)

Pull: Have the patient open a door (a). Did he lean forward with the torso, and did he stiffen the torso? (b)

Here the patient flexes the spine to reach for a door (a). Because the spine has little stiffness, the shear forces create pain. Preparatory muscle bracing is absent (b). Correct these flaws by teaching the patient to prepare to apply load with bracing and to maintain the brace until the door is released and the body is upright.

Push: Assess the strategy in the same way as with the push.

Gait Pattern

Is walking a provoking task for the patient? If so, can it be turned into therapy? Many clues can be obtained during the observation of walking patterns. For example, a slouched posture, small steps, swinging arms from the elbows, duck feet, and a low-cadence shuffle are all suggestions that walking is an exacerbating activity (a). Correcting these will turn walking from an exacerbating activity into therapy. Lifting the chest, swinging the arms from the shoulders, taking longer and faster steps, or correcting duck feet often relieves the pain (b). The biomechanical changes show more efficient storage and recovery of elastic energy, actually reducing spine loads with this approach. Further, walking is changed from a static spine activity into a reciprocating motion and load activity.

Observing the dynamics of the rib cage relative to the pelvis is also revealing. A lumbar spine may be out of control with large motion; pain in these patients is often relieved with abdominal bracing patterns. Yet others with lumbar symptoms may be overly stiff in the thoracic and shoulder regions, with motion occurring only in the lumbar region. Too many young males who are bodybuilding fall into this category. The correction is to have them walk with little stiffness above the lumbar region and with some stiffness in the lumbar region. Developing an attitude during walking often helps. Using cues such as "Walk like you own the world" or "Walk with no worries" often lightens the upper regions to unload the painful lumbar spine.

Some Provocation Tests

Provocative testing is intended to identify the postures, motions, and loads that cause discomfort or pain. They are used to guide the design of pain-free therapeutic exercise and avoidance strategies to remove the cause. The following list comprises some that we have designed or quantified (or both). Because they may mimic the mechanism of injury, it is up to you, the clinician, to set the intensity so that it is sufficient only to provoke discomfort and not to risk further injury.

Some video clips are provided in the online video that accompanies this text, I have also made a DVD of the tests and patients' reactions to them. The many nuances for diagnosis and treatment decisions are demonstrated in the DVD Assessment and Therapeutic Exercise. If you are seeking to become an expert diagnostician, I encourage you to view this unique footage, which is available at www.backfitpro.com.

Compression Tests

Each compression test is well suited to establish compressive tolerance in specific postures and to provide clues for the working diagnostic hypothesis. In compression tests, patients generate their own loads. Caution them not to crush themselves, but simply to perceive whether the pain is worsened.

Heel Drop Test

Schmorl’s nodes and fractured end plates result from compressive overload. Annulus damage typically does not compromise compression tolerance until the disc is flexed. The heel drop test can cause pain or feel uncomfortable in those with pain-provoked compression in an upright and neutral spine. True periosteum pain is often described as local and boring. The dynamic load peak is usually around 2.5 times body weight as seen on a force plate (registering total body weight). A positive pain response would strengthen a hypothesis of recent end-plate damage, or perhaps compromise to the vertebral body (e.g., cancellous bone damage, osteoporotic wedge fracture). If the patient adopts a more extended posture, and the compression is repeated, provoked pain may suggest more posterior bony damage such as to the facet joints or neural arch.

The standing heel drop test is performed with the patient standing relaxed (a), then rising onto the balls of the feet. The patient then drops down to a flat foot, causing a rapid compressive loading of the spine (b). Although the test is conducted cautiously, repeated trials when the patient is pain free may be more aggressive until a rigid drop produces a lumbar compressive magnitude up to about 2.5 times the body weight. Then bracing is performed to evaluate whether it is an effective pain-reducing strategy (cued here with the instruction to harden the abdominals out laterally) (c).

Two patients come to mind as examples of responses to the heel drop test. The first had a T9 wedge fracture, and upon heel drop compression, local spine pain (lower thoracic) was coupled with rectus abdominis sensations of numbness (rectus is innervated from this spinal level). The second patient had prosthetic discs at L5 and L4 and a flattened disc at T10. Once again, the heel drop produced local lumbar pain together with rectus abdominis perceptions, which is innervated from the T10 level, revealing that multiple levels were causing the multiple symptoms in the patient.

Other techniques that provide insight during the heel drop test are tuning the brace and changing cervical posture. If pain is provoked during the uncoached heel drop, instruct the patient to brace the abdominals and repeat. Adjust the intensity of the brace to see whether pain changes. If the pain worsens, this indicates that the spine cannot tolerate the extra compression resulting from the brace. Instead, try a shoulder-bracing strategy in which the pectorals and latissimus dorsi muscles cocontract, pulling the shoulders down into the antishrug. Test whether this reduces the pain.

If radiating symptoms are exacerbated with the heel drop, follow up by adjusting the cervical posture to influence neural tension. For example, if the right great toe goes numb with the heel drop, have the patient bend the cervical spine to the right and repeat. Then bend the cervical spine to the left. Changing pain patterns define the neural tensions and spine postures that relieve and exacerbate pain.

Seated Compression Tests

Seated compression tests reveal whether compression intolerance is modulated by posture. For example, posterior disc herniations generally tolerate compression better when the spine is in a neutral posture, yet these patients feel very uneasy when the spine is flexed. This makes perfect sense considering that flexion creates hydraulic pressure on a posterior bulge. The seated compression test is helpful in such cases. Have the patient sit upright, grab the stool seat pan and pull up; then repeat with the spine (not the neck) flexed in a slouched sitting posture.


00:00 / 00:00


a
b


c
d

The photos show (a) seated compression with the spine in an upright posture; (b) then compression is repeated in a slouched posture. The patient pulls upward on the stool seat and repeats for each variation of the test. Have the patient adjust the spine posture, lift the rib cage, and repeat. This may create pain at the thoracolumbar junction. Alternatively, have her flex the hips to align the spine vertically and repeat the test. Then have her flex the neck to see whether neural tensions modify the pain sensitivity (c). Then laterally flex the neck to see if radiating pain occurs (d). If pain occurs, rest whether it is relieved by extending the neck but maintaining a slouched torso (e). This will reveal the link between pain and neural tension. Find the posture that affords the highest tolerance, and teach that posture and strategy to the patient.

Also be observant during the standing up motion. Pain may be from the discs and SI joints, but rarely from the facet joints (Young, Aprill, and Laslett, 2003).

Extension Tests

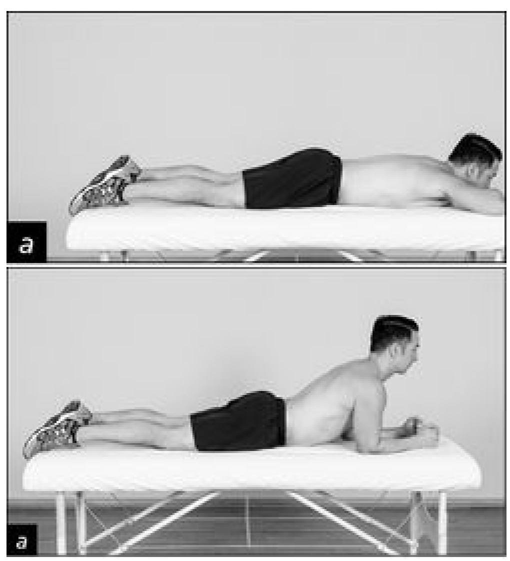
Spine extension as a provocative test can be interesting, because the mechanics involve several structures. For example, a prone, relaxed extension (some McKenzie postures) may relieve pain, suggesting that the pain was discogenic. Although for years the mechanism was thought to be actual migration of the nucleus anteriorly, recent work has shown a more interesting phenomenon. As the annulus slowly and progressively breaches, layer by layer, nuclear material infiltrates the annulus. Prone extension has been shown to wring out the annulus and squeeze the nucleus fluids back into the annulus in some patients (Scannell and McGill, 2009). But this also depends on the type of annulus damage, its location, disc shape, and the disc height remaining. Obviously, a more posteriorly located partial herniation should respond to this hydraulic mechanism. Sometimes the delaminations of the layers in the annulus travel around to the lateral, and even anterior, portions of the annulus. In these cases, extension may drive more nuclear material anteriorly following the path of delamination. Clinically, a side bend (known as the side glide to McKenzie-based clinicians) is effective for some patients. We speculate that these may be the ones with lateral delaminations. The side glide is then followed by extension, which, once again, is effective for some. The nuclear material may be then directed back to the central nucleus or may be driven back to the lateral rent.

Prone Extension Test

The patient begins in a relaxed standing posture, and you ask about current pain and the general feeling about the back. Then the patient lies prone, adopting one of the following three levels of the extension posture progression. (Decide which is suitable based on the patient's flexibility and symptoms. More extension is not better; simply find what is most tolerable).

- (a) Prone with the arms relaxed, hands are flat and stacked under the chin
- (b) Prone and supported on the elbows
- (c) Prone with elbow support but with cervical flexion (this reduces pain in a patient with an underhooked nerve root)


00:00 / 00:00


a
a

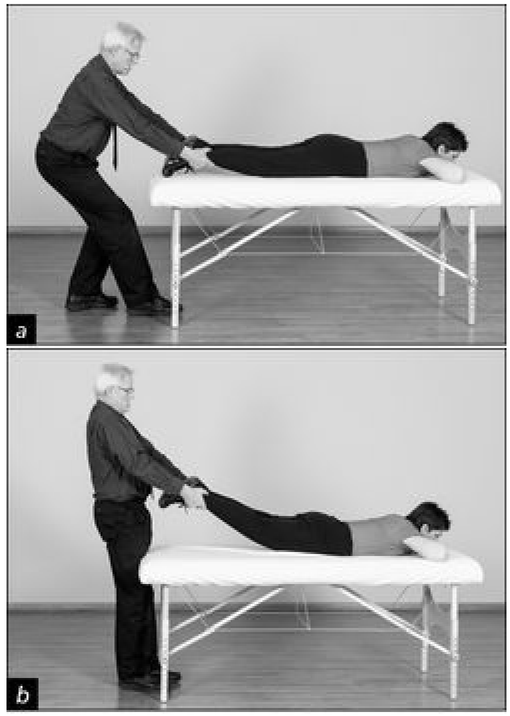
While in position a during the prone extension test, the pain mechanism can be probed. Begin with the forehead on the hands (d). If the patient experiences discomfort, push the eyebrows down to see if this stabilization maneuver helps relieve the discomfort. Then try the chin on the hands (e), then chin on one fist (f), then the chin on two stacked fists (g).

If the patient cannot tolerate one of these postures, you can assume that simple disc disturbances are not the source of troubles, or at least not the sole source. Many with uncomplicated posterior disc lesions, or herniations, find relief in these postures (Young, Aprill, and Laslett, 2003). The patient then returns to the standing posture. A patient who feels more stable or has less pain compared to the moments prior to lying prone is classified as posterior discogenic.

A therapy that greatly assists these patients is learning to lie prone in their relieving posture and then inhaling. They then exhale while consciously feeling the lumbar region fall toward the table (a).

Patients who are posterior discogenic should avoid flexion stretches and a flexed spine under load when performing exercise therapy. Those who feel worse upon standing could have a host of other possible conditions but will probably experience better progress by adopting a neutral to slightly flexed lumbar posture for exercise therapy.

Traction is then applied in the prone position first as a test, then subsequently as a treatment if it relieves pain. This provides greater relief for some patients, but exacerbates the symptoms of others. Traction applied to the ankles may help or hinder pain relief. The more unstable the joint is, the more likely traction will be to exacerbate the pain. Clinical tricks can be added throughout to help you understand the pain trigger and the associated avoidance strategy. For example, if pain is triggered during prone traction, cue the patient to push one eyebrow into the table and see whether the pain is relieved. Then have the patient turn the head and try the other eyebrow. This added muscular stiffness reduces symptoms in many patients, thus identifying a pain-reducing strategy once a specific trigger has been identified.



Postures to relieve pain for the posterior discogenic patient: Lying prone, as in panel a of the prone extension test, the patient inhales and then exhales focusing on allowing the lumbar spine to fall to the table. This gentle lumbar extension accelerates the stress relief for discs that respond well to prone extension. This does not cause the irritation of the facet joints that eventually occurs in those performing the floppy push-up, which our work has shown should be avoided. Static prone postures are sufficient. Traction applied to the ankles may help or hinder pain relief. The more unstable the joint is, the more likely traction will be to exacerbate the pain. (b) Sometimes it is more effective to lift the knees from the table. Then a small wiggle of the legs side-to-side at a rate of two cycles per second is about the most effective vacuuming technique to reduce a posterior disc bulge. However, as this first is a test, pain with the wiggle suggests instability (see the test for spondylolisthesis in the next section of this chapter).

Tests for Determining Suitability for ROM Training and Stretching

The McKenzie extension approach is well known for treating acute discogenic troubles. But the extension postures can also be used as a test to identify those with posterior disc troubles. In most cases, these posterior discogenic patients should never flex the lumbar spine when it is under substantial loading. See the preceding section prone extension test.

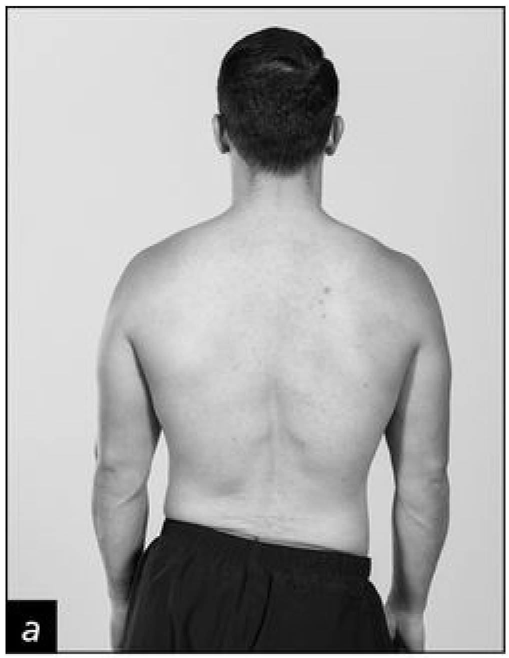
Spondylolisthesis Test

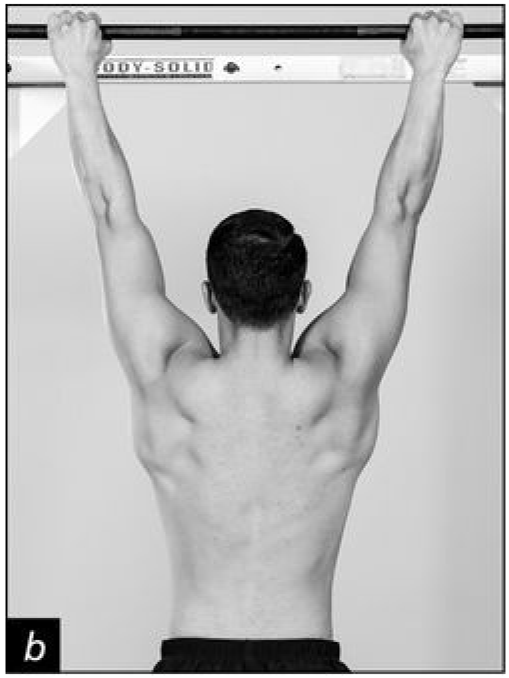
Most spondylolisthesis patients' pain is exacerbated by extension postures and anterior shear loads. In this test the patient lies prone. Pure traction (about 20 lb or 90 N) is applied via the ankles. Then the clinician raises the legs until the knees just clear the table. Back pain and sometimes radiating symptoms occur if the joint is unstable and a pain generator. Then a slow (1 cycle every 2 seconds) side-to-side shake may be applied. Pain provocation for spondylolisthesis begins with the patient prone. Traction loads of approximately 20 lb are applied (10 lb per leg); then the ankles are raised until the knees clear the table. Pain indicates a positive test. Conversely, this may provide relief suggesting that the disc, not spondylolisthesis, is the pain source. A slow side-to-side shake helps quantify the stability of the joint: pain is linked to instability, and relief is linked to stability. The more unstable the joint and listhesis is, the greater the pain is. Conversely, a more stable joint will enjoy the accelerated return of a posterior disc bulge with pain reduction that this technique creates.

Scoliosis Test

The scoliosis test reveals whether scoliosis (a) is correctable. The patient grabs an overhead bar. As the patient hangs supporting his full body weight with the arms, observe the spine curves. If the curves straighten under the traction load from the added body weight, the spine has the potential to respond to exercise therapy (b). Schroth therapeutic exercise is then administered (c). (Special condition exercises are beyond the scope of this book, although one is shown as an example.)

True scoliosis is sometimes confused with temporary conditions. False signs suggesting a temporary scoliosis include unilateral psoas contraction, which gives an appearance of an aberrant curve. Some clinicians falsely interpret this as a short leg, perform an adjustment, and declare that they have corrected the short leg—in fact, they released the psoas. Obviously, a psoas contraction that disturbs normal hip and spine mechanics requires targeted treatment to the psoas, of which there are several appropriate soft tissue release techniques.

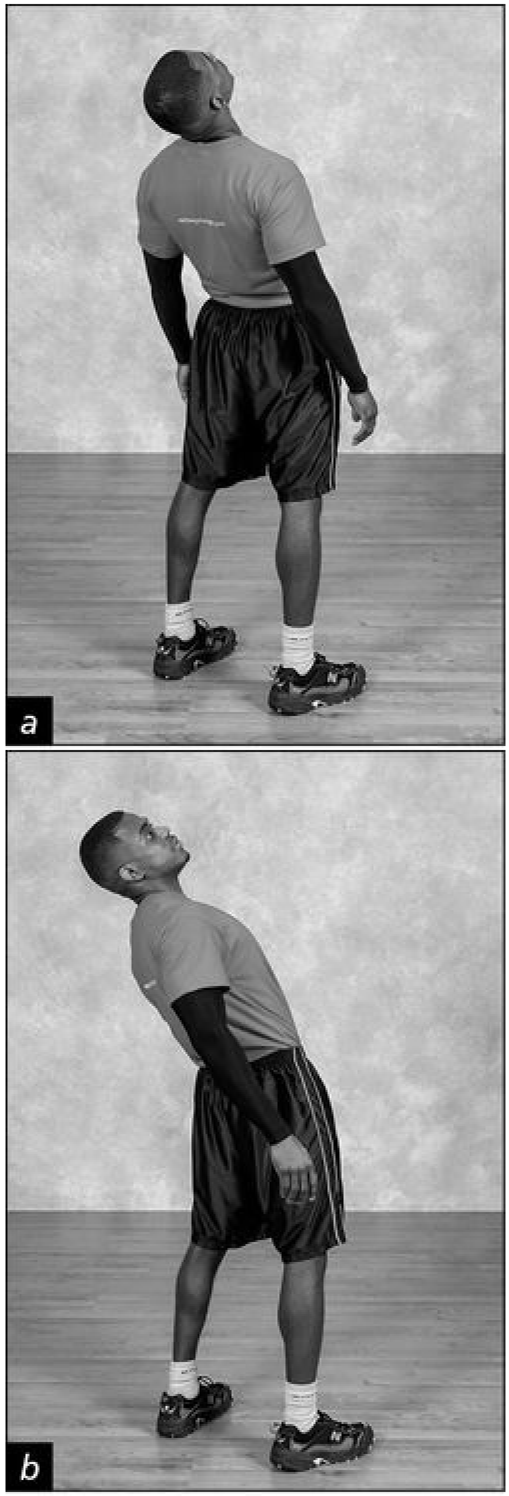


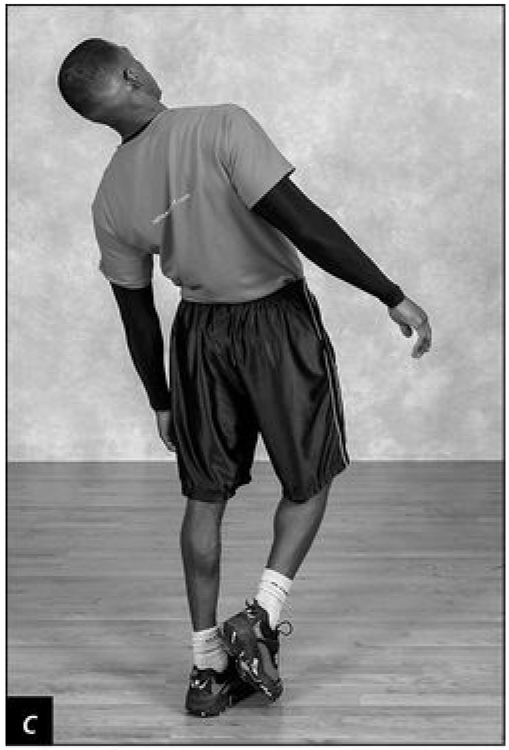

b

An example of a Schroth exercise for a contained lumbothoracic curve with a level pelvis and no substantially misshapen vertebrae is as follows. The hand contacts the rib cage on the convex side of the curve and applies pressure. The patient raises the arm on the concave side and pushes vertically. During the effort, the patient takes a deep breath, imagining the lungs filling with air on the concave side. The lung on the concave side is usually smaller.

Standing Extension Tests

Classically, extension postures have been thought to indicate posterior element or neural arch damage. However, as with disc pathology, this exacerbation provides only circumstantial evidence. The standing extension provocation tests include the extension quadrant and the one-legged stork test. Both postures load the facet capsules, compress the facet articular interface, and bend the pars and lamina. They also close down the size of the lateral nerve root foramen and tension the anterior longitudinal ligament and anterior annulus. Understanding the interplay between these tissues helps in understanding the signs during the standing extension postures. For example, sagittal plane extension may cause central pain that is further exacerbated by lateral bending to the right, then twisting to drop the right shoulder back. Shown in the photos are (a) standing extension, (b) the extension quadrant test, (c) the one-legged stork test.





The main implication, as with many provocation tests, is that the reaction to extension postures provides circumstantial evidence. When combined with other evidence, the impression and working diagnostic hypothesis are strengthened or weakened. Relief with prone extension appears to be a strong indicator of discogenic pain from posterior annulus damage, but exacerbation of pain requires more follow-up testing of facets, the neural arch, and so on. If the extension pain is reduced or eliminated during the stork test, this adds more weight to a diagnosis of joint instability because the stork test adds stiffness and stability to the joints in the frontal plane. Nonetheless, in terms of designing therapy, the pain exacerbator (an extended spine in this case) has been identified and should probably be avoided.

Neural Tests

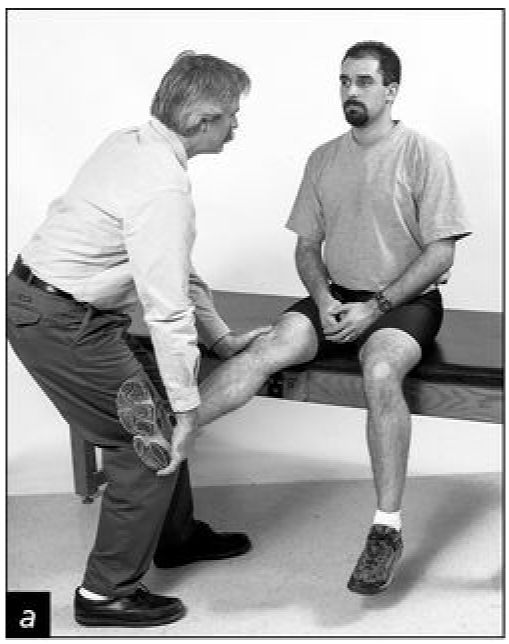
Pain is sometimes wrongly attributed to tight muscles when the genesis is actually an irritated nerve. Proper diagnosis is an important issue because the treatment of tight muscles usually makes an irritated nerve worse. The clinical techniques discussed here will help you enhance patient outcomes.

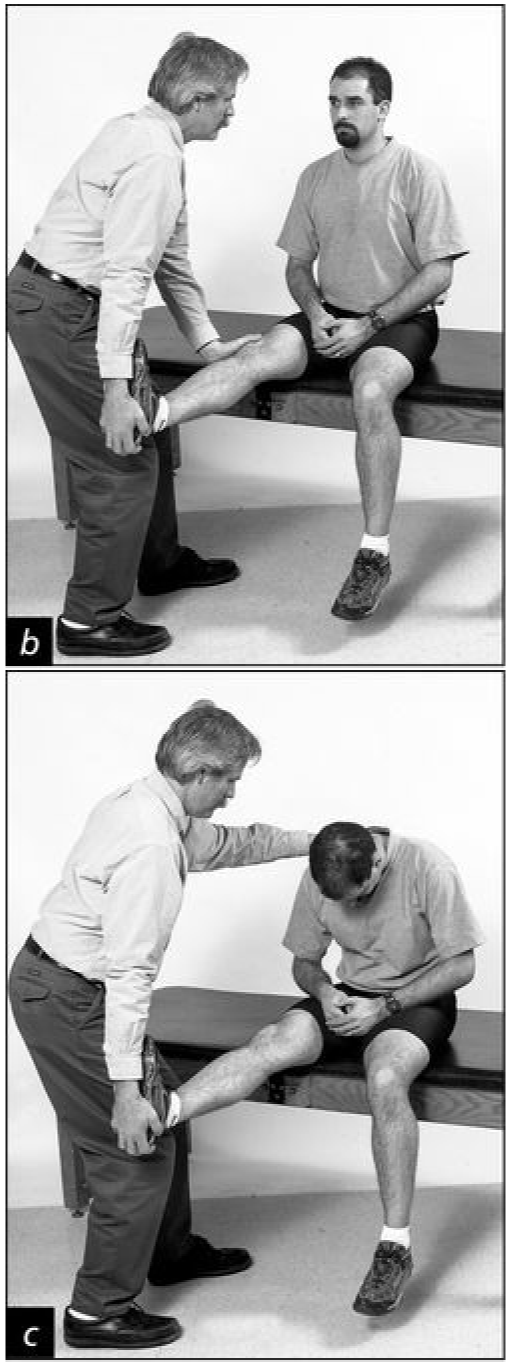
Sitting Slump Test

Several forms of the sitting slump test can be used to elicit radiating sciatic symptoms. These are designed to tense the sciatic nerve and irritate the lumbar nerve roots. Typically, the patient sits on the table or chair and then slumps or slouches. This test may be performed at the same time as the seated compression tests, because the load may work with the various postures to elicit pain. The intention is to progressively increase the nerve tension and define the trigger levels and conditions.

The test is as follows:

- (a) The leg is extended at the knee, tensing the nerve from below the lumbar spine.
- (b) Foot flexion can be added to further tense the nerve.
- (c) The nerve can be further tensed from above the lumbar region with the addition of cervical spine flexion.





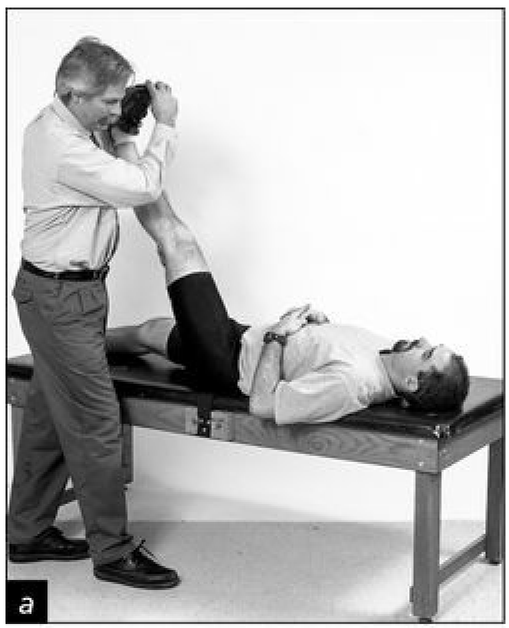
Photos from Stuart McGill

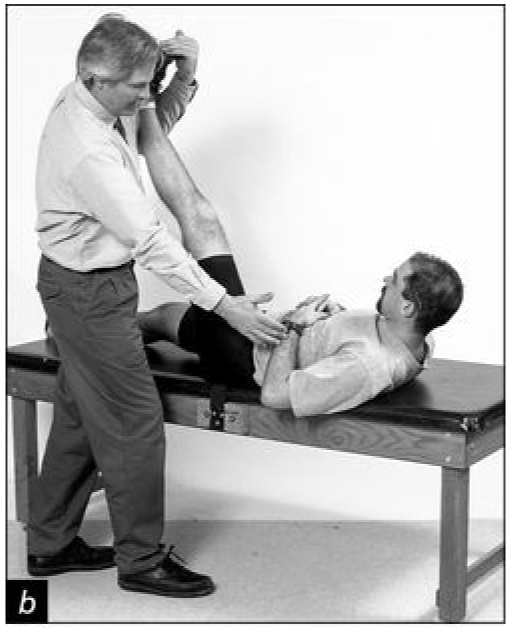
If the tension at any of these stages causes symptoms, progressive testing should be stopped. The patient may be a candidate for nerve flossing (described in chapter 10). Certainly, forms of flexion are contraindicated for these patients during stretching and exercise.

If flexing the neck does not increase the pain, then the sciatic nerve is not the tissue of interest and you can begin assessing the sacroiliac (SI) joints, the hamstrings, piriformis, and other muscle-based syndromes, ending with the hip. However, the slump test, in this form, is limited—a more insightful nerve tensioning test (the supine passive leg raise test) is described following the description of the Fajersztajn test and the neural underhook.

Fajersztajn Test

The Fajersztajn test should always be performed on both legs even when the sciatica is unilateral. Raising the well leg is needed to perform the well leg–raising test of Fajersztajn (DePalma and Rothman, 1970). Raising the well leg tensions the nerve root on the well side while causing tensions centrally along the midline of the cauda equina and to the nerve roots on the opposite side (a). Sometimes pain is provoked with simultaneous cervical flexion (b). Pain in the symptomatic side (side not raised) is an organic sign of disc lesion, usually a more central lesion. It is not a sign of malingering, as some have suggested.

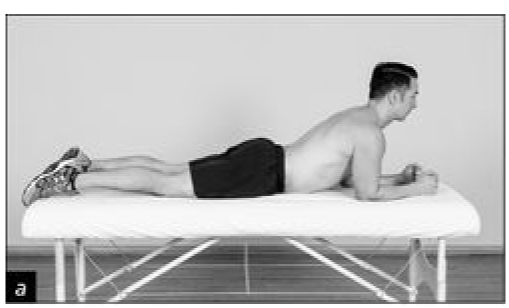




Photos from Stuart McGill

Neural Underhook

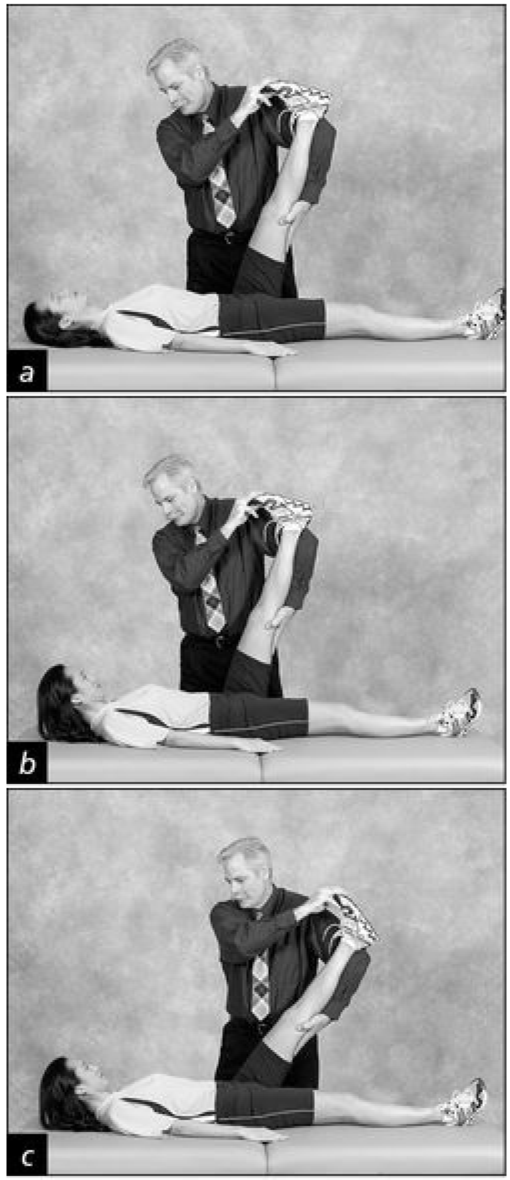
During neural tensioning tests, cervical flexion usually increases radiating lower limb symptoms—but not always. Sometimes cervical flexion relieves symptoms and extension increases symptoms. This sign usually indicates an underhook on the nerve root as the whole spinal cord slides caudally into the disc bulge. It also accounts for some patients who walk with their heads down in chronic cervical flexion, vexing clinicians who cannot understand why they do not stand upright. With this condition, the approach is usually to address the disc bulge with prone static McKenzie postures, and by avoiding lumbar flexion during daily activity. Over time, the ability to obtain an aligned, upright, pain-free posture should improve. The underhook also explains why some nerve flossing procedures require modified cervical postures and movements. Begin prone (a) with cervical extension, then only flex the lumbar spine (b).



Supine Passive Leg Raise Test (Sciatic Roots)

Technique while performing the leg raise test is very important. Both neural tension and muscular tension are changed with raising the leg, as in the sitting slump test. Thus, during the leg raise, some patients report pain that originates from muscle tension. To sort out the difference, always place one hand behind the knee so that the fingers can palpate the tension in the hamstring tendons. The patient's description of pain also assists in the impression of whether the pain is from neural or muscular tension—neural pain travels beyond the borders of the muscle (perhaps follow the sciatic tract in the buttock and below the knee). Internal rotation of the leg adds more tension to the sciatic tract.

If neural tension is present, follow up by determining whether the nerve root is free to floss. With the patient remaining in the supine posture, raise the pain-generating leg to the point of pain. The patient then raises the head with cervical flexion, and if that exacerbates the leg pain, this indicates that tension of the neural tract from above exacerbates the pain. Then lower the leg slightly. Alleviation of the pain with this maneuver establishes that releasing tension in the neural tract from below (while the tension from above remains) allows the nerve root to relax. Then returning the leg to the former position that generated pain and returning the head to the rested position on the table should reduce the pain. If this occurs, you will have established that the nerve root is flossing and not adhered. An adhered or pinched nerve root will not show different pain patterns from the coordinated cervical motion described here. Although we have been able to break nerve root adhesions (postsurgical) in the past with flossing regimens, the risk of creating exacerbated sciatica is large and requires strict guidance, which is discussed in chapter 10.


a
b
c

Shown in the photos is (a) the leg raised to the point of pain provocation and (b) cervical spine flexion added. Does this make the pain worse? Then (c) lower the leg. Does this reduce symptoms? If so, the nerve is flossing. Note the technique of cradling the leg in the crook of your arm so that your fingers can palpate the hamstring tendons.

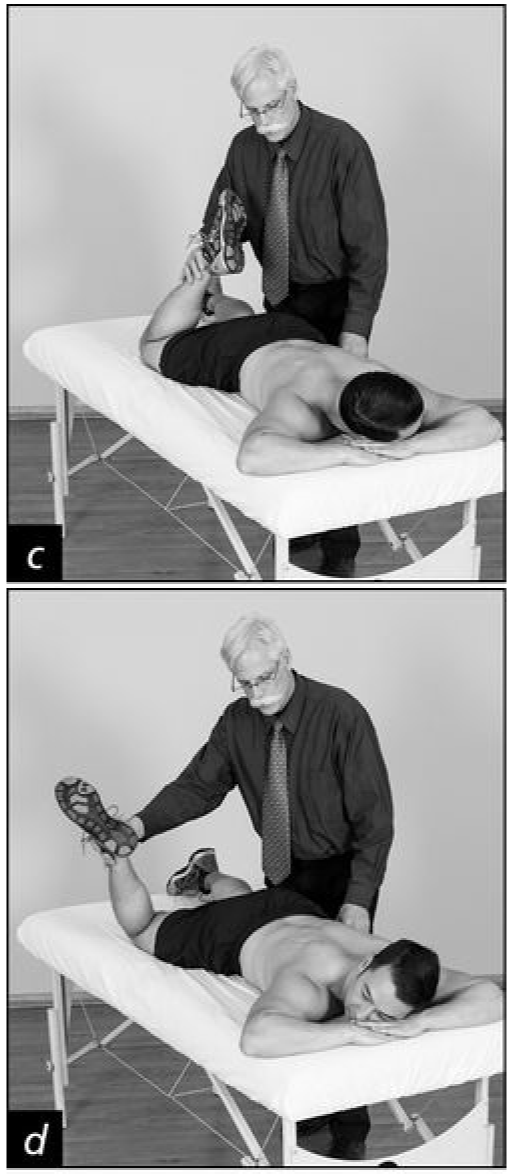
While performing the supine passive leg raise, monitor muscle tension by using the fingers to palpate the hamstrings. (a) Raise the leg to the point of pain provocation. (b) Then have the patient flex the cervical spine to further tension the spinal cord from above the lumbar roots. If the pain is exacerbated either in the back or along the sciatic tract in the lower extremity, neural tension is considered the culprit. (c) Then lower the leg. If the pain is reduced, this is a clear indication that the nerve root is not adhered.

Tests for Femoral Nerve Roots

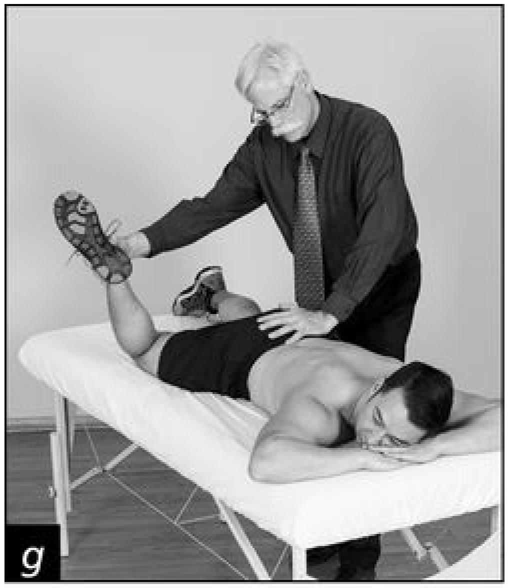
The upper lumbar spine (L1, L2, and L3) roots form the femoral nerve tract that is tensioned with hip extension and knee flexion. Pain-causing tension is tested in a prone posture. The clinician raises the lower leg into full knee flexion; then internally and externally rotate it. Femoral nerve roots are tensioned as the knee is flexed and the thigh is externally rotated. The pelvis rolling away from the knee being flexed is known as Ely's sign, which indicates either muscle or nerve tension. Follow up with a standing test to thrust the pelvis forward to tension the anterior hip capsule, while changing neural tract tensions to differentiate between muscle and nerve tension. Neural tensions are adjusted with cervical posture; frontal plane spine bending is probed with shoulder elevation.

Occasionally, this test may provoke back pain or radiating symptoms down the anterior thigh. If this occurs, manually stabilize the pelvis to see whether lumbar or SI joint instability is part of the pain trigger. Use cues for stiffening selected muscle groups to sort out pain-reducing potential.


a
b


c
d


e
f



The patient lies prone (a) as the clinician flexes the knee and asks the patient about pain or nerve crawling in the anterior thigh or back (b). The patient's head is rotated to the other side to see if perceptions change (c). The hip is internally (d) and externally rotated (e), then the clinician manually stabilizes the pelvis to probe whether this added control and stiffness changes the pain pattern (f, g).

Nerve tensions can be caused by pulls from either end. Here is an example where cervical posture affects femoral nerve tension and corresponding symptoms. The femoral nerve is tensioned by the clinician (a) and as an assistant flexes and extends (b) the cervical spine to reveal the mechanism of posture-driven pain.

Differential Neural Tests and Implications

Cervical nerve roots are different from lumbar nerve roots in that they have ligamentous connections to vertebrae making them susceptible to whiplash, unlike lumbar roots. In addition, cervical roots have separate pain and sensory roots, whereas these are not separate in the lumbar roots (LaBan, 2008). This means that pain and weakness are more correlated with function associated with the lumbar roots.

It is possible for higher neural pathology to influence lumbar symptoms. We have seen posttrauma patients who experienced spinal cord shock and the subsequent death of cells within the cord. They were labeled as having bizarre behavior by the compensation board clinicians until we found the damage in the thoracic cord. This damage leaves deficits in motor engrams normally encoded in this region of the spine. For example, one person forgot how to cross his legs. Many weeks were required to recode this movement into the movement pattern repertoire. Another person forgot how to coordinate movement patterns to rise from a chair. This needed to be patiently relearned. Many neurologists seem unaware of this aspect of cord shock and movement behavior. Another thing to keep in mind is that EMG test results are not always truthful during the few weeks following trauma or the onset of acute back pain.

Foot drop is another condition suggesting pathology in the L4 nerve root. Jeon at al. (2013) introduced a test to differentiate a lumbar genesis from a peroneal nerve genesis. Weakness when testing hip abduction suggested a lumbar pathology. These researchers then followed up with Trendelenburg and gluteal muscle tests (shown as the exercises of the side-lying and clamshell tests) to determine whether an L5 root pathology existed.

The typical tests of myotomes are also helpful for determining pathological levels (e.g., great toe dorsiflexion, ankle plantar and dorsal function).

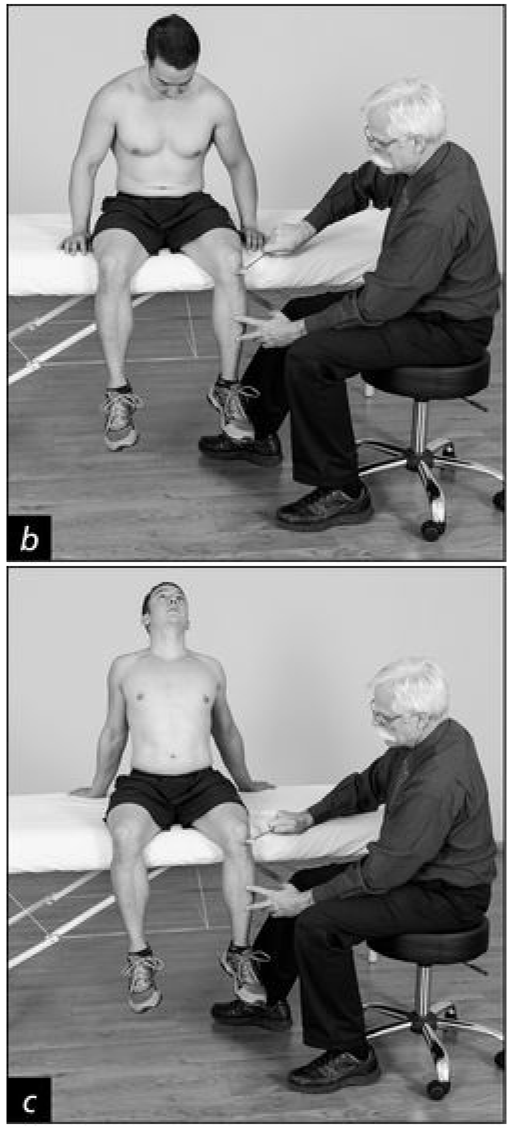
Reflex Testing to Find the Best Postures for Nerve

Mobilization

Peripheral reflexes such as the patellar reflex or the Achilles reflex are modulated by lumbar root tension or compromise. However, the actual nerve root tension that is modulated by posture is rarely considered. For example, patients with a diminished patellar reflex during the typical upright sitting posture are recorded as compromised. Yet in some of these patients, the reflex is restored when the posture is changed to reduce the neural tension. In this case, a logical posture is to have the patient lie back. A stronger reflex measured in this new posture establishes that the reflex is postural, and therefore biomechanically modulated.

Test for Posturally Modulated Reflex Diminution

Reflex intensity is evaluated as a function of neural tension. Adjust the seated posture of the patient to modulate the neural tension to determine whether the reflexes are modulated—for example, (a) with a neutral spine; (b) in a flexed posture, causing neural tension; or (c) in an extended posture, slackening the neural tract (or other postures between the two). If a stronger reflex response is generated in posture b, then this is adopted during flossing routines.



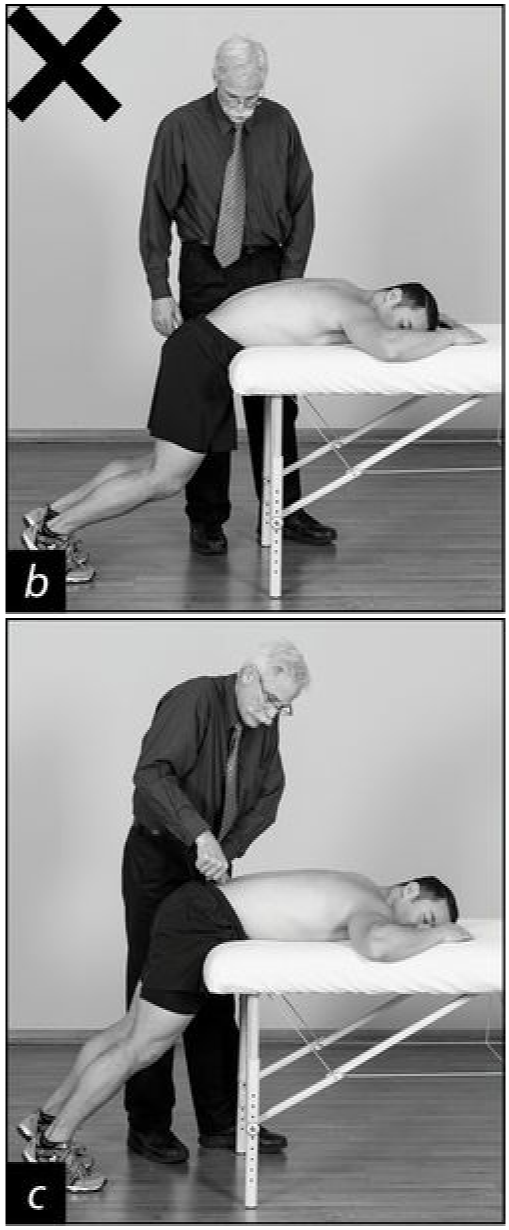
Testing for Lumbar Joint Shear Stability

Chapters 3 and 4 offer a well-developed discussion of the role of the lumbar extensors (longissimus thoracis and iliocostalis lumborum) in supporting anteroposterior shear of the vertebral motion segments. These muscles stiffen against unstable motion in the shear plane. In fact, good stabilization exercises must involve these muscles. A simple manual test for lumbar joint shear stability is presented here. If the result is positive (i.e., shear instability provokes pain), the patient could benefit from reducing the aberrant shear motion and will do well with stabilization exercises that groove cocontracting motor patterns of the shear musculature (Hicks et al., 2005).

Manual Testing for Lumbar Joint Shear Stability

The patient lies prone on a table with the legs over the edge and the feet on the floor (a). The person must relax the torso musculature. Correct positioning is essential for true results. Too much spine flexion causes pain immediately in some patients (b). You then apply direct force downward onto each spinous process in turn (starting at the sacrum, then L5, L4, L3, etc.). The force applied should be no more than 1 kg (2 lb), and is applied in an oscillatory way to rattle the segment. Unstable segments are identified when either the person reports pain or you feel actual shear displacement. The patient's reporting of segment-specific pain, in this case, is given more consideration. Then ask the patient to slightly raise the legs off the floor to contract the back extensors. You once again apply force on each spinous process, using the same technique as before. By virtue of their lines of action, the lumbar extensors will reduce shearing instability, if present. If pain is present in the resting position but then disappears or subsides with the active cocontraction, the test is positive. Patients who indicate more pain during active contraction may be compression intolerant. Or they may be trying to extend their legs with lumbar extension rather than hip extension. Correction of this aberrant pattern may make the test more precise. More technique adjustments enhance insight into the pain exacerbator or reliever. Try more hip extension with a conscious gluteal squeeze and find the sweet spot (e).

This test proves conclusively that activating the extensors stabilizes the shear instability and eliminates the pain. Now the trick is to incorporate these extensor motor patterns into exercise prescriptions to carry over to daily activities.

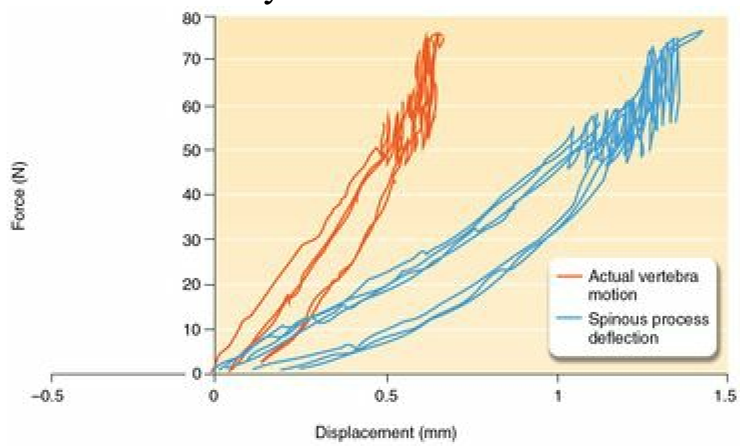

b
c

Locating where to apply force: The thumbs palpate the posterior–superior iliac spines (PSISs) (f) on a skeleton model. As in real life (g), the thumbs slide to the midline. This will either be the posterior spine of L5 or the first sacral tubrical (h).

A Note on Motion Palpation

Some clinicians use their hands to feel specific vertebral motion during a whole-trunk motion such as flexion or twisting. When they detect a blocked or stiff-feeling segment, they often declare it pathological and make it the target of mobilizing treatment. Clinicians rarely, however, consider the flexibility in the spinous process and bony neural arch noted in chapter 3. Two of my PhD students questioned whether the asymmetrical feel to a specific joint was true pathology or simply asymmetrical anatomy—perhaps asymmetrical facet joints (Ross, Bereznick, and McGill, 1999). By carefully documenting the motion occurring at the disc and in the spinous process in cadaveric specimens, as well as the applied loads (similar to clinical loads), they were able to quantify the effect of anatomy. The results demonstrated that over half of the motion felt by the clinician was actually the spinous process flexing and bending (see figure 9.1) and not vertebral body motion. Those joints that were anatomically asymmetrical had asymmetrical motion under an applied load from the clinician. Treating such a joint with mobilization therapy would be fruitless.

Figure 9.1 These results were measured as clinicians applied lateral forces to the tip of the spinous process of cadaveric motion segments to mimic motion-palpation testing. Most of the motion measured in the spinous process, which is what the clinician feels, was the result of spinous process bending rather than disc motion. Moreover, asymmetrical anatomy resulted in asymmetrical motion when loaded to mimic the clinician's hand forces. This suggests that this test should not be the only test to determine the site of treatment.



Distinguishing Between Lumbar and Hip Problems

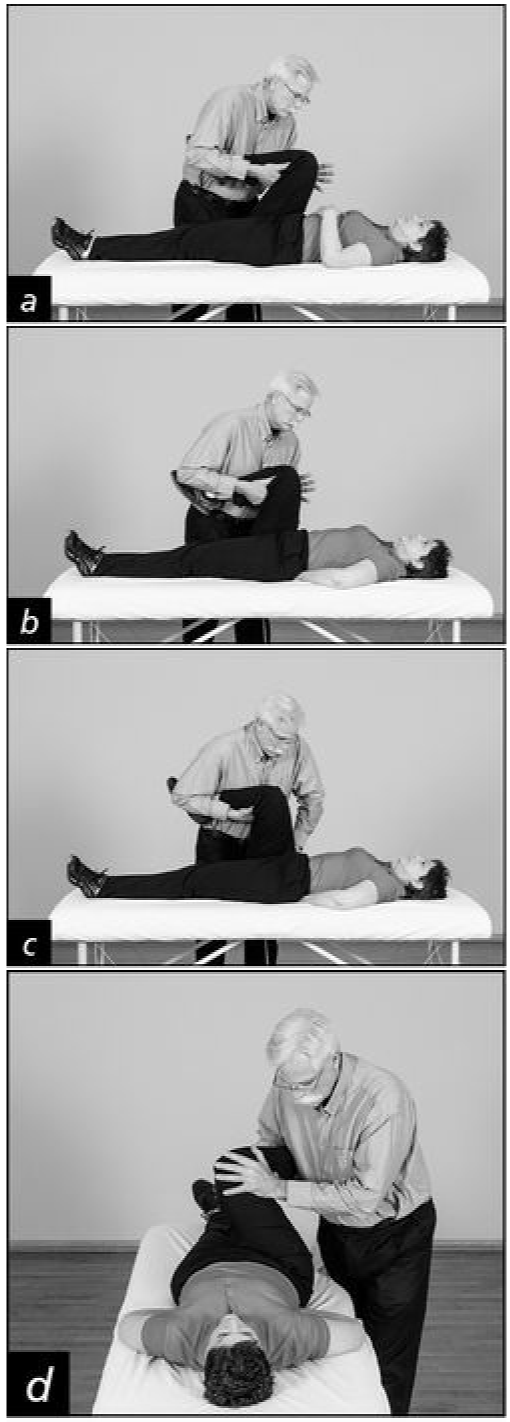
Pain in the buttock or radiating down the leg (or both) can have more than one source. For effective treatment, you must discover whether that source is in the lumbar spine or the SI joints, in various muscles, in tissues around the hip, or even in the hip joint itself.

I am continually surprised at the number of people with back troubles who also have hip troubles (McGill and colleagues, 2003, noted a correlation between back troubles and hip pathology in factory workers). It is sometimes difficult to separate hip pain from lumbar trouble because deep buttock pain can be from a sciatic root (lumbar) or originate in the hip joint. Typically, medial and anterior thigh pain are indicators of hip pathology. If the nerve tension tests are negative and more hip investigation is indicated, then hip flexion and rotation tests should be performed.

Hip Flexion and Rotation Tests

Have the patient lie supine with the hip flexed. Rotate the patient’s thigh segment into hip internal rotation (a). Stiffness or pain is a positive indicator of hip concerns. Hip joint pain is somewhat distinctive with radiation into the groin, or along the inguinal crease or down the anterior–medial thigh (or some combination of these). When more pressure is applied to the thigh challenging the hip joint in full flexion, the spine will also flex. When back pain occurs during hip flexion, stabilize the lumbar spine with the hand (c). Also have the patient place his hands under the lumbar spine to prevent flexion. If this causes back pain, stabilize the pelvis with your other hand so that no spine motion occurs and rotation is confined to the hip. If the back pain is gone, the spine was the source.

Then flex the patient’s hip (d) while moving the knee in circular motions, scouring the acetabulum, but with the knee bent so as not to confuse any pain with nerve tension related to the back. Now externally rotate the thigh, testing the joint capsule (e) and stabilizing the pelvis to isolate the hip (f-h). Finally, the hip distracted (i) and compressed (j).

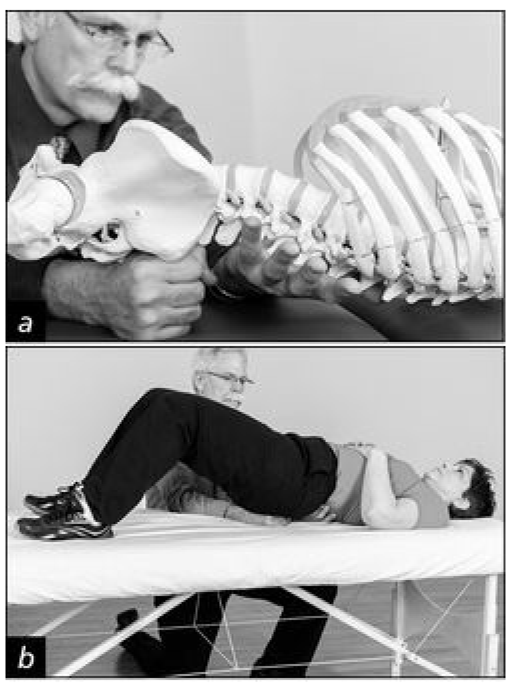



e
f
g
h

Tests for Sacroiliac Joint Pain

Young, Aprill, and Laslett (2003) noted that SI joint pain is rarely experienced along the midline, above the L5 level, and bilaterally. Rising from a chair tends to exacerbate the pain. Our clinical observations suggest that pain is provoked with lunges. Several tests challenge the SI joints and pelvic ring. The series begins with force applied down into the hip joint, followed by an uplifting force similar to that shown in photos i and j of the hip flexion and rotation test just described. Then the supine patient bridges the pelvis up and lowers the sacrum down onto your fist (see a and b). This may cause pain. Or you can perform rapid fist clenches to transfer sharp pulses into the pelvis via the SI joints. The clinician's other hand supports the lumbar spine, changing the amount of lordosis to probe whether this influences pain (c). This loads the SI joints in pulsating shear loads. Then the patient walks while you manually apply pressure to the iliac crests cracking the low half of the SI open (d, e). The patient then repeats the walk with manual pressure applied to the greater trochanters, cracking open the lower half of the SI joints (f, g). These tests help define the nature of the SI pain if it present. If no pain is provoked, the SI joints can be ruled out.

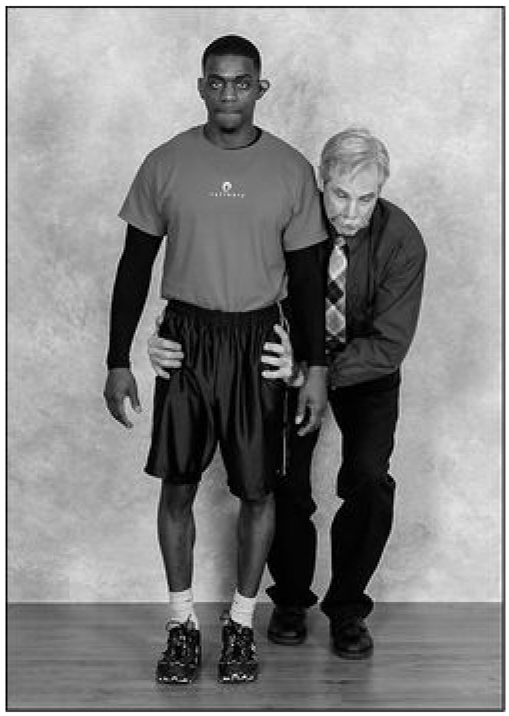
00:00 / 00:00




c
d
e

Manual Iliac Crest Compression Test

Continue probing the pelvic ring to learn more about the cause of the pain and what may help. If manual compression relieves the pain, try cuing abdominal wall stiffness while the patient walks to see whether this reduces pain. If any of these strategies relieves pain, it is a strong indicator of pelvic ring instability. The exercises prescribed for initial lumbar stabilization (chapter 10) also stiffen and stabilize the pelvis. Also, probe the patient to find the cause. Many report recent changes in their training such as adding many more loaded split squats. Too many of these exercises can loosen the SI joints. Postpartum women, particularly those who have returned to vigorous activity too soon, or those who have sustained a high-energy trauma such as a fall or motor vehicle accident, may present with unstable pelvic rings and associated back or pelvic pain.



Some Functional Screens

Pain is present because the back is currently a weak link. Functional tests help to sort out why the back is a weak link and to indicate what is required to bolster the deficit. Some tests are posturally based; others assess motion control.

Manual Tests for Motion Control

Motion control requires all of the attributes of physical performance—motor control, strength, endurance, specific flexibility, and so on. The following screens will help you identify some of these deficits while also serving as qualifying tests prior to engagement in more progressive therapeutic exercise.

Proprioception Acuity

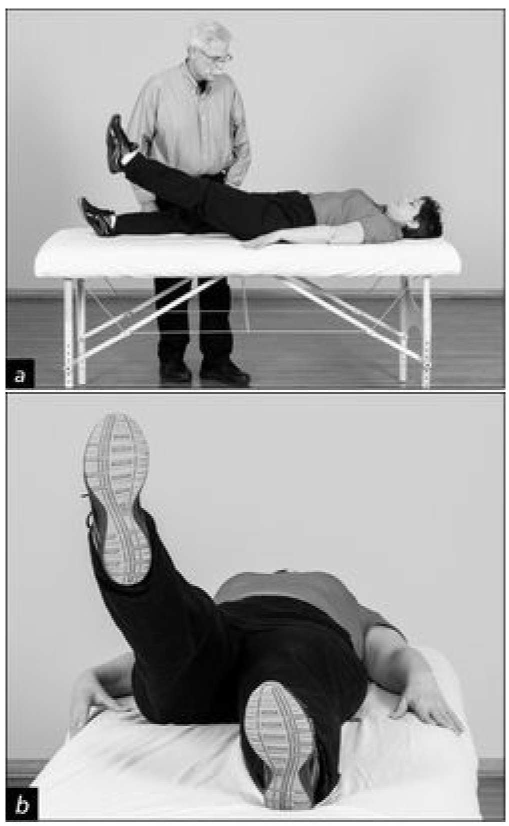
The ability to find a pain-free position for the spine appears to be compromised in those with chronic backs. Clearly, this ability can be trained (Preuss, Grenier, and McGill, 2005). Recall the wall plank that was part of the stop twist movement tool described in chapter 8. This drill can also be used first to identify those who are unable to accurately reposition the spine and second as a training exercise.


a
b

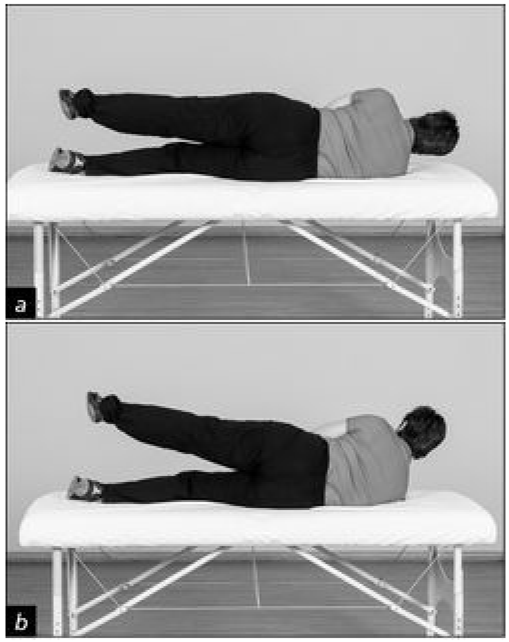
Wall planks begin with the patient’s finding and grooving the sweet spot for minimal or no pain. You then move the pelvis toward the wall using a combination of hip and spine flexion–extension (a). Once the no-pain position is found, (b) rake the patient’s abdominal wall with your fingers to stimulate oblique muscle activation and lock this pain-free posture into muscle memory. (c) The shoulder and latissimus may be brushed to cue the locking initiation of the torso rotation with lastissimus dorsi. (d) Finally, groove the locked torso into the movement engram of the patient.

Once the pain-free posture is found, the patient spins on the balls of the feet with the rib cage locked to the pelvis so that no lumbar motion occurs (c, d). The plank position is then taken once again. If the sweet spot for pain-free lumbar posture has been lost, the exercise is repeated to groove the pain-free position into the movement and posture repertoire.

Other tests provide indicators of movement control and, by default, pain control For example, the straight-leg raise test (SLRT) has been shown to test the ability to control the lumbar spine during a single-leg raise with the associated hip flexion (Liebenson et al., 2009) and to determine whether this is a pain trigger. Here, the patient raises a straight leg while the clinician looks for any aberrant spine twist or flexion. If the spine twists, the patient is coached to eliminate it (a, b). This allows the clinician to determine if the problem is "software" or poor understanding of the movement (or "hardware") where sufficient strength is lacking.



In a similar way, Nelson-Wong, Flynn, and Callaghan (2009) tested the side-lying hip abduction test (a). They noted that aberrant spine movement during the test predicted which asymptomatic people would develop back pain earlier during a prolonged standing task (b).



The evidence supports the notion that the ability to control spine movement during the production of hip moment reduces pain onset. Part a shows good control while part b shows poor control.

Looking for Spinal Hinges

Hinges are motion segments that bear more of their share of the motion. In the sagittal plane they are seen during sitting down or standing flexion. A common location is the thoracolumbar junction. In athletic populations, a successful strategy is often to buttress this lack of stiffness with latissimus dorsi activation through shoulder depression with the rib cage lifted (see McGill, 2006). Lumbar hinges are usually better buttressed with an abdominal brace (within the compression tolerance of the spine and at the sufficient level). Sometimes torsional hinges occur. These can be addressed by learning to avoid painful twisting or by buttressing with an appropriate muscular brace (or both). Over time, the natural degenerative cascade will work in favor of the patient to stiffen the painful segment. In this way the arthritic or degenerative process acts to stabilize the painful segment (or hinge). However, the clinical objective is to restore pain-free function in the meantime.

Photos from Stuart McGill

The test begins with the hips over the knees and the shoulders over the hands (a). The patient rocks the buttocks back to the heels, trying to maintain a neutral spine. A flexion hinge (arrow) at L2-L3 is the site of pain in this flexion-intolerant former athlete. General abdominal bracing removed his pain together with spine–pelvis position awareness training.

The Stiff Spine

Not all spines are painful because of instability. Rather, some are stiffened with muscular contraction and locked into painful postures. The chronic muscular lock, however, is often secondary to tissue damage and local instability. However, the choice of coping strategy is what causes the chronic symptoms. The muscular reaction is simply inappropriate: It is probably far too intense and is more than is needed to be sufficient. Thus the brace is tuned to the task—just enough to control pain. As noted earlier, stiffness is often regional, and various clinical approaches can be employed, ranging from passive approaches to active learned motion patterns that involve less muscular stiffening. Techniques for spine mobility, when appropriate, are shown later.

Control of Torsional Motion

Other forms of aberrant motion may indicate poor lumbar control, which is in contrast with the local segmental instabilities shown previously. For example, a rotational disconnect of the rib cage and pelvis during the push-up test described here can indicate that more work is required for enhancing lumbar control.

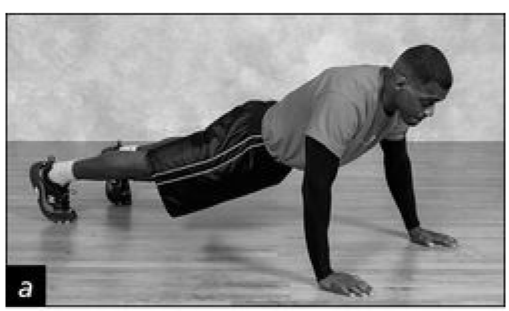
Torsional Control Tests

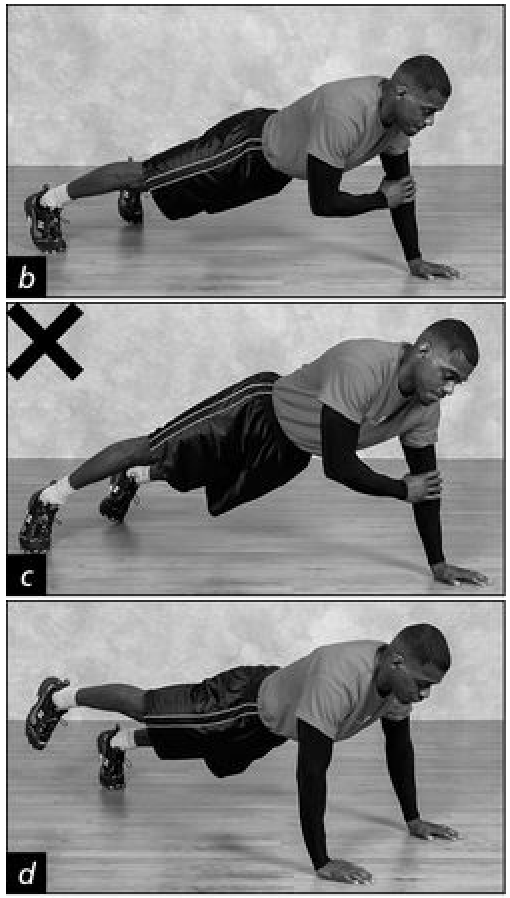
The stop twist movement tool can be considered to provide an initial impression of torsional control. **Begin with the wall plank test and assess standing twist competency.** The more advanced torsional tests described here indicate the presence or absence of the control necessary before engaging in more demanding torsional training.

Push-Up Tests

After the patient has assumed a correct push-up position (a), have him lift one hand in a controlled motion and place it over the opposite elbow (b). This is good form and shows torsional control. Poor control is shown in (c), contraindicating further torsional training. The pelvis and rib cage should remain level if torsional control is achieved.

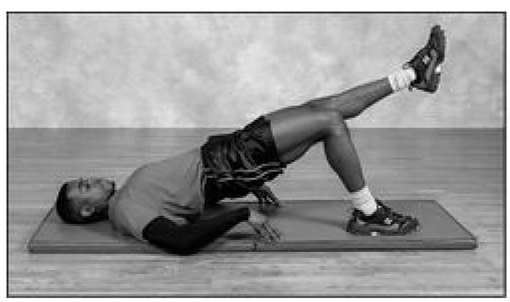
The one-legged hip extension from the push-up position provides a similar indicator of control (or lack thereof). A pelvis locked to the rib cage while raising one leg with hip extension also indicates lumbar torsional control (d). Hiking the pelvis indicates poor lumbar torsional control.





Back Bridge Test

The back bridge with a leg extended also indicates torsional control. Although this was originally proposed as a test for pelvic stability (Mens et al., 1999), we have found that it reveals lumbar torsional control, stiffness, and stability (Liebenson et al., 2009).



Have the patient get into the back bridge position; then ask the patient to extend one leg slowly. The patient should be able to control this motion with no twisting or flexion in the lumbar region.

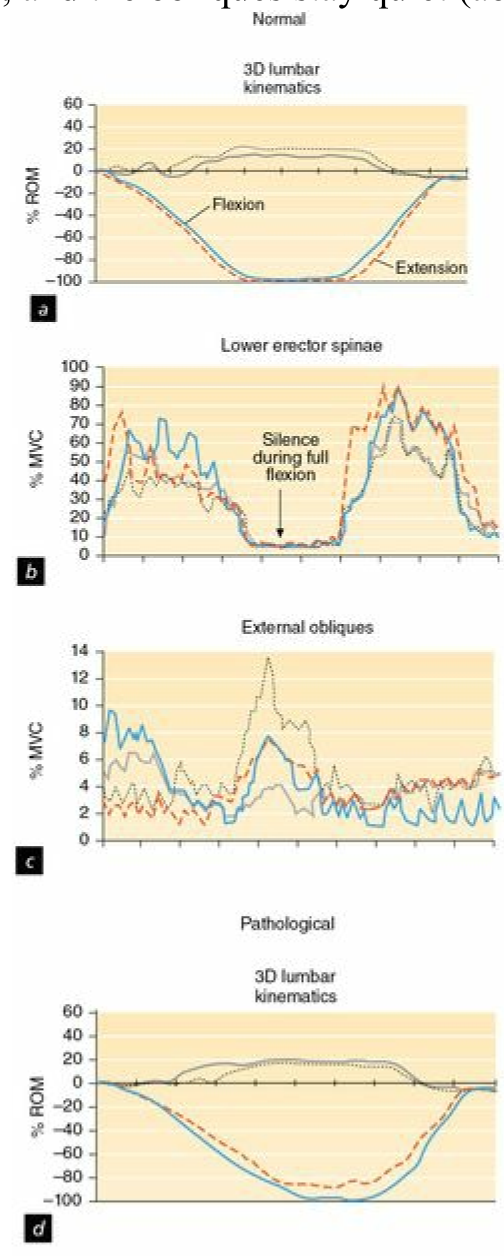
Testing for Aberrant Gross Lumbar Motion

Local spine instabilities are detected in several ways. The more skilled clinicians simply notice them. They appear as small deviations in the three-dimensional motion profiles. For example, the spine may clunk during standing flexion when the lumbar spine bends through 14°. This is repeatable once the refractory period has passed. The refractory period is not a set time but a condition in which the unstable joint is able to shift back to a neutral position so that it is able to clunk again. The clunks are almost always manifested in a shear mode. Sometimes they are audible with the naked ear or with a stethoscope. Other times they are a simple and very subtle shift in motion as the spine is moving in a specific way, but they cause pain at that instant. Correction exercises involve avoiding the motion that causes the pain, buttressing the instability with an appropriate bracing pattern, or both. A skilled clinician will see these instabilities through unaided observation; however, I have also found technology helpful, particularly when I have to document these cases for medical and legal reports. The following are a few examples.

Aberrant Gross Lumbar Motion and Electromyographic Evidence

The usual practice of assessment is to test for ROM about the three primary axes of motion by determining the degrees of motion possible from neutral. For example, a flexion ROM may be determined to be 45° from neutral (usually using standing posture as a reference base). As previously pointed out, this number is of little use as an indicator of pathology or for guiding therapeutic exercise decisions. Other well-known pathology markers include the inability to achieve myoelectric silence in the extensors at full flexion (and that full flexion is not reached—see figure 9.2). However, pathology may manifest within moderate ranges of motion, and simultaneously in axes other than the primary plane of motion. The following example illustrates this point. As one bends forward in the sagittal plane, a small amount of motion may occur in the lateral bend or axial twist axis, and it may be repeatable every time the spine passes a certain flexion position. This indicates an instability catch, or clunk. These are seen in the examples shown in figures 9.3 and 9.4.

Figure 9.2 Flexion response test. The normal person flexes forward to full flexion (a), shows silence in the erector spinae muscles (b) and actually pulls with the obliques (c). The person with pathological pain does not reach full flexion to transfer load from muscle to passive tissues (d), does not shut off the erector spinae (e), and the obliques stay quiet (about 3% MVC) (f).



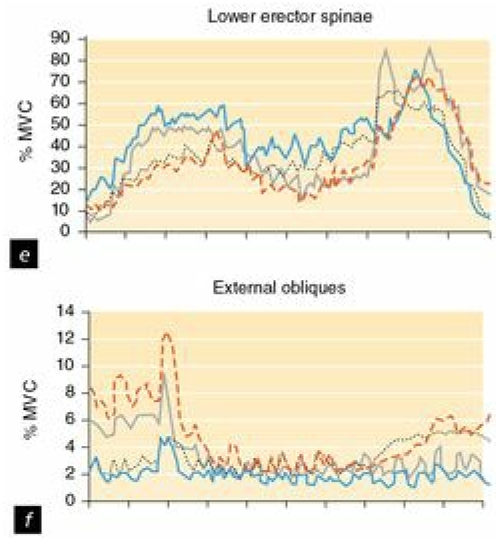

Lower erector spinae
90
80
70
60
50
40
30
20
10
0
% MVC
e
External obliques
14
12
10
8
6
4
2
0
% MVC
f

Figure 9.3 This patient had a normal ROM but demonstrated an instability catch. During a lateral bend test (from neutral 0 to 23°), every time the spine bent laterally passing 17° (event A) (b), a small flexion hitch of a degree and a half occurred in the flexion axis (event B) (a). There was no deviation in the twist axis (c). The bottom curve represents the first derivative of the flexion curve, which we use to better locate instability. The spike (event C) (d) clearly indicates the point at which the instability occurred in the flexion axis. In this patient, a muscle activation pattern was identified that removed the painful clunk, or catch.

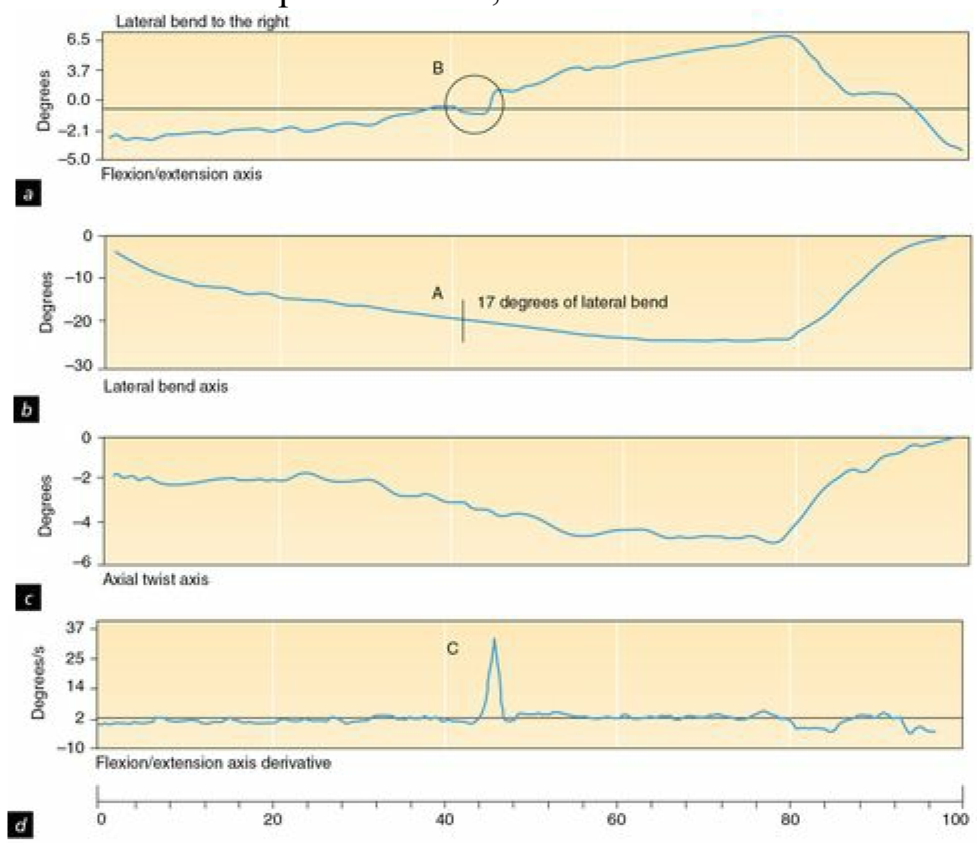
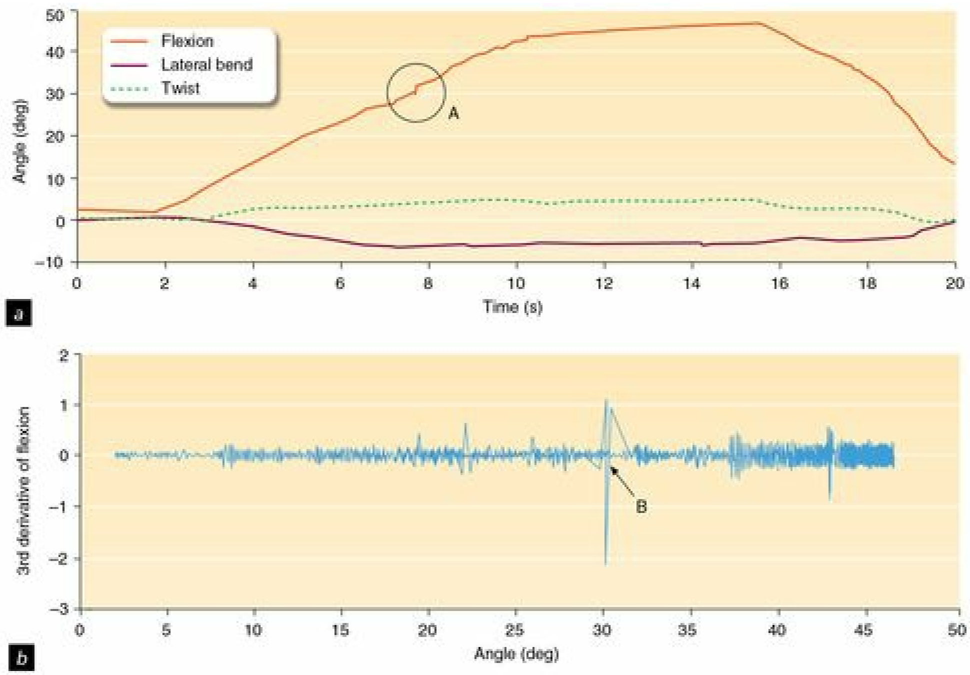


Figure 9.4 In this patient an instability catch, or clunk, occurred in the primary axis of motion (flexion). As he flexed, the clunk was seen in the flexion axis at 30° (event A) (a). This occurred in every trial of forward flexion. The instability is evident (event B) in the third derivative of the flexion axis motion (b). The rehabilitation objective was to stabilize the spine to eliminate the clunk. This was not fully successful over the short term. Symptoms resolved only when the patient developed spine position awareness and was able to avoid flexion to 30°. Specifically, in this case, the instruction was a mild abdominal brace.



The first patient (figure 9.3) had a normal ROM and by traditional tests would have been classified as normal. Yet during a lateral bend test (from neutral 0 to 23°), every time the spine bent laterally passing 17°, a small flexion hitch occurred in the flexion axis of a degree and a half. This was repeatable. The point is that the motion pathology was not in the lateral bend axis but was seen in the flexion axis. These very subtle aberrant catches in the "off" axes are often missed during examination. The more skilled clinicians see these or feel them with their hands.

In the second example (figure 9.4), a clunk, or catch, was observed in the flexion axis as the patient flexed forward and passed 30°—this was repeatable and observed in every flexion attempt. Interestingly, this instability catch, as well as the associated pain, was cured with some muscle patterning and stiffening in a very short period of time. Specifically, in this case, the instruction was a mild abdominal brace. Sometimes patients report pain as the catch occurs, and sometimes there is evidence of muscle activation bursts; but many patients simply move through the region. We use an electromagnetic instrument that tracks three-dimensional spine motion and graphically displays the results. Sometimes the catches are so subtle that it is far more illuminating to take the first derivative of the axis with the catch. Patients have a much higher incidence of these catches (Peach and McGill, 1998) than people without back issues, which indicates instability. People without symptoms who demonstrate these catches may be at elevated risk for becoming patients according to the aberrant motion model of Richardson and colleagues (1999).

A final example is very striking. One patient arrived after seeing many specialists who told him that his problems were in his head. He had intransigent sciatica and could not stand the weight of a bedsheet on his foot. The specialists had treated his foot! I asked him what caused his pain, and he started to wind his upper torso around. As he approached upright standing, I heard a grind and pop from his low back and he yelled out in pain. Clearly, he had a massive spinal instability with a trapped lumbar nerve root (see figures 9.5 and 9.6). Upon looking at his motion and muscle electromyogram (EMG) data, I could see that his spine became unstable when the muscles shut down as he returned to an upright posture. His nerves were highly sensitized, accounting for his seemingly bizarre (to others—not to us) behavior. We showed him some muscle-bracing patterns, which removed the audible clunk within about 4 days. After rapid progress, he was pain free in about 3 months. Bracing took away the daily irritation to the sensitized tissues. The bracing then also allowed his tissues to desensitize. We have done this time and time again with patients who have had no success with other approaches—the approach can be very successful with these unstable patients. We have seen many patients who were suicidal with their pain and who were told the problem was in their heads. It is tragic.

Figure 9.5 (a) The three-dimensional motion of the lumbar spine is shown with the instability catch occurring as the patient returned to an upright posture. (b) Observing the EMG records revealed that this was the instance in time when muscle activation (and stiffness and stability) was at a minimum.

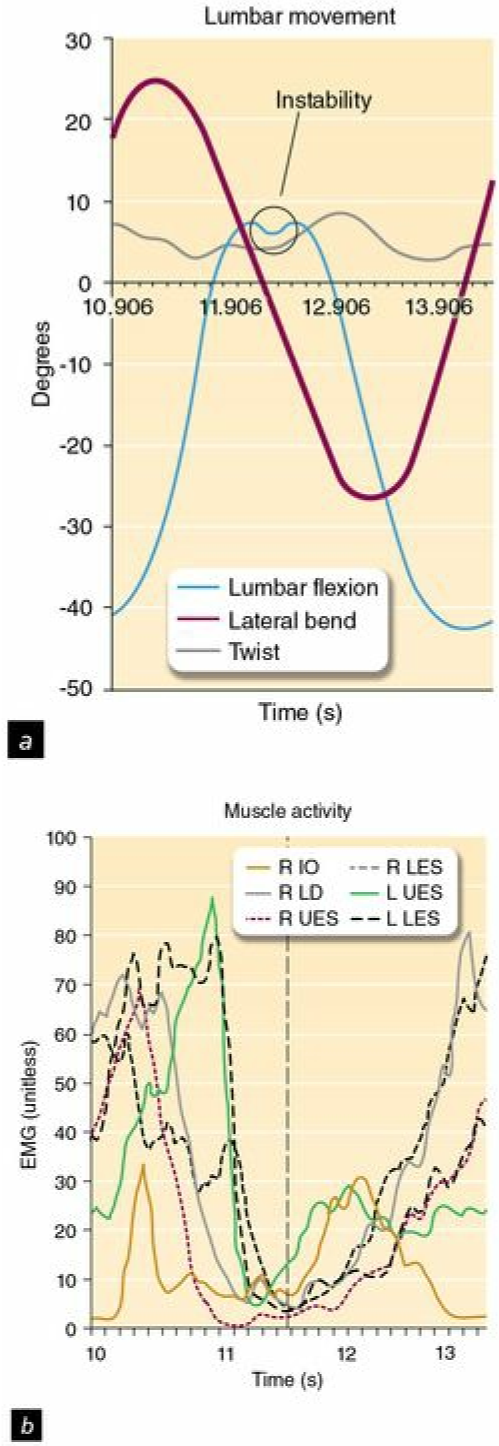
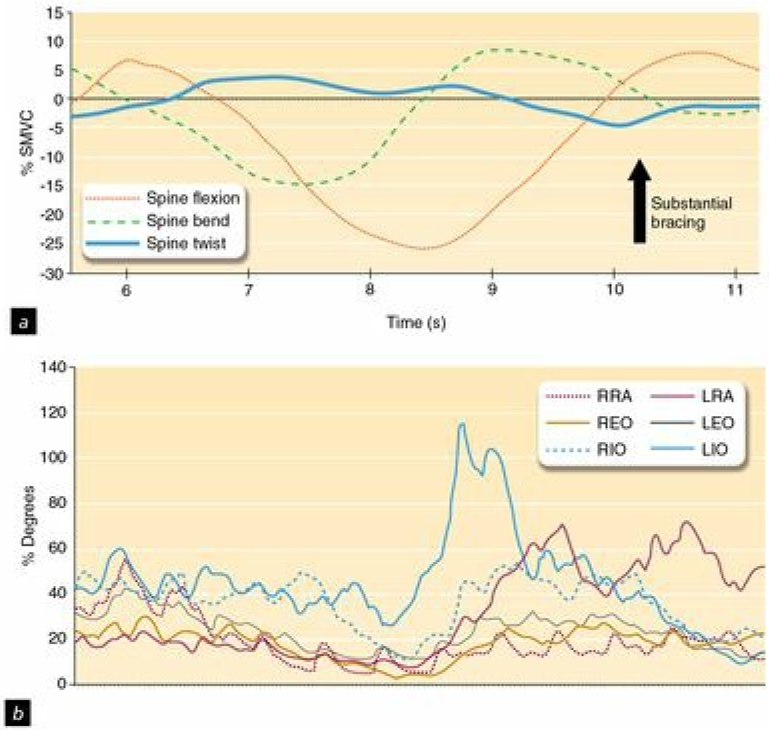


Figure 9.6 The same patient as figure 9.5 was tested three months later. Here, they performed the same movement and (a) no clunk is evident as they (b) learned to brace, causing stiffness sufficient to eliminate potential instability.



Testing for Aberrant Motor Patterns During Challenged Breathing

The test for aberrant motor patterns during heavy breathing is rather sophisticated, but it is probably the only way to identify a person’s ability to maintain spine stability during tasks that require higher physiological work rates. Candidates for this type of test include athletes and occupational workers such as construction workers and warehouse employees. We explored the link between breathing and spine stability in chapter 8. Challenged breathing requires heavy involvement of the abdominal muscles. Because we monitor the muscles with EMG electrodes and because muscle fatigue changes the EMG signal, we avoid creating heavy lung ventilation with exercise for this test. (Note that this consideration is only for testing; we intentionally increase the physiological work rate for exercises in chapter 5.)

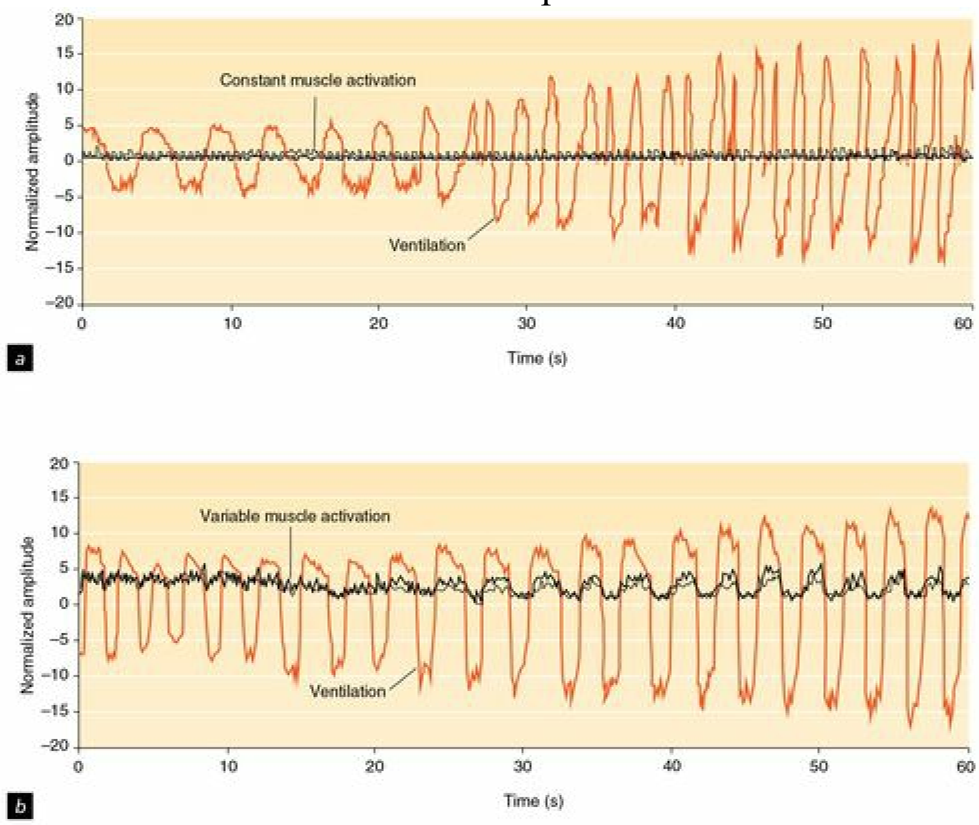
Challenged Breathing Test

During this test the person breathes a 10% CO_2 gas mixture to elevate the lung ventilation with only a few breaths, but the O_2 is adjusted so that the light-headedness that accompanies hyperventilation does not occur. Then a weight, usually about 15 kg (33 lb) for the average man, is placed in the hands while the torso is flexed forward about the hips to approximately 30°. This test reveals those who have aberrant motor patterns in the abdominal muscles during challenged breathing that compromise lumbar stability.

Photos from Stuart McGill

Good spine stabilization patterns observed in the muscle EMG signal obtained during the challenged breathing test include a constant muscle cocontraction ensuring spine stability. Figure 9.7, a and b, shows stable and unstable patterns. Our computerized analysis package measures spine stability using the methods explained in chapter 6. Many clinicians will not have this capability but should still look for constant levels of abdominal wall activation. Patients who are poor at stabilization in these situations will demonstrate short, temporary reductions in activation or even loss of activation. These mark the critical instances at which spine instability can occur, and classify the person as a candidate for spine stabilization during high physiological work rates. These patients must learn to contract the spine stabilization muscles independently of challenged breathing to ensure spine stability in conditions of heavy work.

Figure 9.7 Good spine stabilization patterns during the challenged breathing test include a constant muscle cocontraction, which ensures spine stability. Both (a) stable with constant muscle activation and (b) unstable variable abdominal muscle activation patterns are shown.



Many other provocation tests and functional screens are suited for more athletic populations. Some can determine the starting load for serious resistance training, whereas others indicate basic movement flaws that must be corrected prior to rigorous training such as with plyometrics. A few of these are shown in Ultimate Back Fitness and Performance (McGill, 2014).

Testing Muscle Endurance

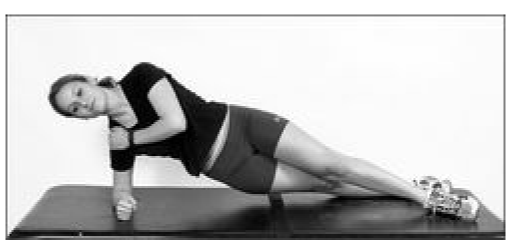
Several tests that can help you assess your patients and plan the most appropriate exercise are described in this section. You are the final judge as to which tests are relevant for specific patients.

Muscular Endurance Tests

Although Biering-Sorensen (1984) showed that decreased torso extensor endurance predicts those who are at greater risk of back troubles, recent work has suggested that the balance of endurance among the torso flexors, extensors, and lateral musculature better discriminates those who have had back troubles from those who have not. Because these three muscle groups are involved in spine stability during virtually any task, the endurance should be measured in all three. Simple tests that isolate these groups of muscles are difficult to find; I chose the following tests because each was shown to have high reliability coefficients (at least .98 or higher) when repeated over 5 consecutive days (McGill, Childs, and Liebenson, 1999).

Lateral Musculature Test

The lateral musculature is tested with the person lying in the full side bridge position (the test is also known as the side bridge or side plank). Legs are extended, and the top foot is placed in front of the lower foot for support. Subjects support themselves on one elbow and on their feet while lifting their hips off the floor to create a straight line over their body length. The uninvolved arm is held across the chest with the hand placed on the opposite shoulder. Failure occurs when the person loses the straight-back posture and the hip returns to the floor.



Photos from Stuart McGill

Flexor Endurance Test

Testing endurance of the flexors (rectus) begins with the person in a sit-up posture with the back resting against a jig angled at 55° from the floor (a). Both knees and hips are flexed to 90°; the arms are folded across the chest with the hands placed on the opposite shoulder; and the toes are secured under toe straps. To begin, the jig is pulled back 10 cm (4 in.) and the person holds the isometric posture as long as possible (b). Failure is determined to occur when any part of the person's back touches the jig.

Photos from Stuart McGill

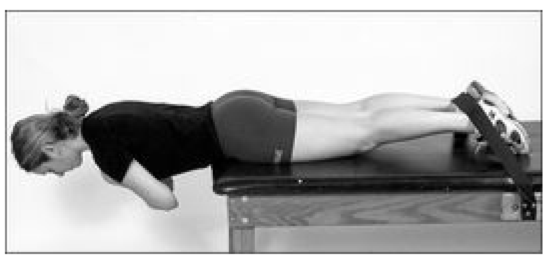
What Is the Best Test?

Concerns about using speed sit-ups as a test of core fitness in the mandatory annual fitness test in the U.S. military have been discussed previously. Recognizing the link between repeated sit-ups and the incidence of low back injury, Peterson (2013) evaluated alternate measures and suggested the front plank. The plank has the benefit of not needing any equipment, which is important for those deployed on ships and aircraft. The downside is that shoulder disorders may disguise true core endurance.

McGill and colleagues (2010) compared endurance data with the V-sit flexor endurance test and the front plank in a population of 620 firefighters for the first data set and 181 university students for the second. Although flexor endurance in firefighters peaked in their 40s, extensor endurance peaked in their 20s. In the study of university students, the plank scores were relatively higher than the V-sit scores in males but relatively lower than the V-sit scores in the females. A Pearson correlation test between the paired plank and V-sit scores of each subject rendered a coefficient of r = .34. This means that the performance on one flexor test predicted only 11% of the score in the other. An ANOVA comparison of scores based on subjects' history of having had shoulder or back troubles showed no significant link between V-sit and plank scores. The plank scores are not well correlated with the V-sit scores, suggesting that the two measure different variables. Because more data exist for the V-sit, measurement of flexor endurance in occupational settings using this test probably forms a stronger link to back injury.

Back Extensor Test

The back extensors are tested in the Biering-Sorensen position, with the upper body cantilevered out over the end of a test bench and with the pelvis, knees, and hips secured. The upper limbs are held across the chest with the hands resting on the opposite shoulders. Failure occurs when the upper body drops from the horizontal position.



Photos from Stuart McGill

Normative Data

Normative absolute endurance times collected from young healthy people are listed in table 9.1. Note that women have greater endurance than men in the extensors. Further, the flexor endurance time (as a proportion of the extensor endurance time) for healthy young men is about .84, whereas other tests have yielded lower ratios for healthy older men. McGill and colleagues

(2003) showed that the relationship of endurance among the anterior, lateral, and posterior musculature is upset once back troubles begin (see table 9.2); that upset in the relationship remains long after the symptoms have resolved. Typically, extensor endurance is diminished relative to both the flexors and the lateral musculature in those with lingering troubles. A final note regarding patient endurance testing: There is no need to exacerbate the pain in patients with endurance testing, because their daily variation in pain will invalidate their results. Wait until they are pain free. In addition, resist the urge to "train the tests." Rather, build endurance with the reverse pyramid approach introduced in chapter 8 and with repeated sets of shorter-duration holds.

Table 9.1 Mean Endurance Times (sec) and Ratios Normalized to the Extensor Endurance Test Score

	MEN			WOMEN			ALL		
Task	Mean	SD	Ratio	Mean	SD	Ratio	Mean	SD	Ratio
Extension	161	61	1.0	185	60	1.0	173	62	1.0
Flexion	136	66	0.84	134	81	0.72	134	76	0.77
RSB	95	32	0.59	75	32	0.40	83	33	0.48
LSB	99	37	0.61	78	32	0.42	86	36	0.50
Flexion/extension ratio	0.84			0.72			0.77		
RSB/LSB ratio	0.96			0.96			0.96		
RSB/extension ratio	0.58			0.40			0.48		
LSB/extension ratio	0.61			0.42			0.50		

Mean age of university students = 21 (men: n = 92; women: n = 137).

The ratios are determined for each person and then averaged. They are not derived simply by dividing the average scores of the group.

RSB = right side bridge; LSB = left side bridge.

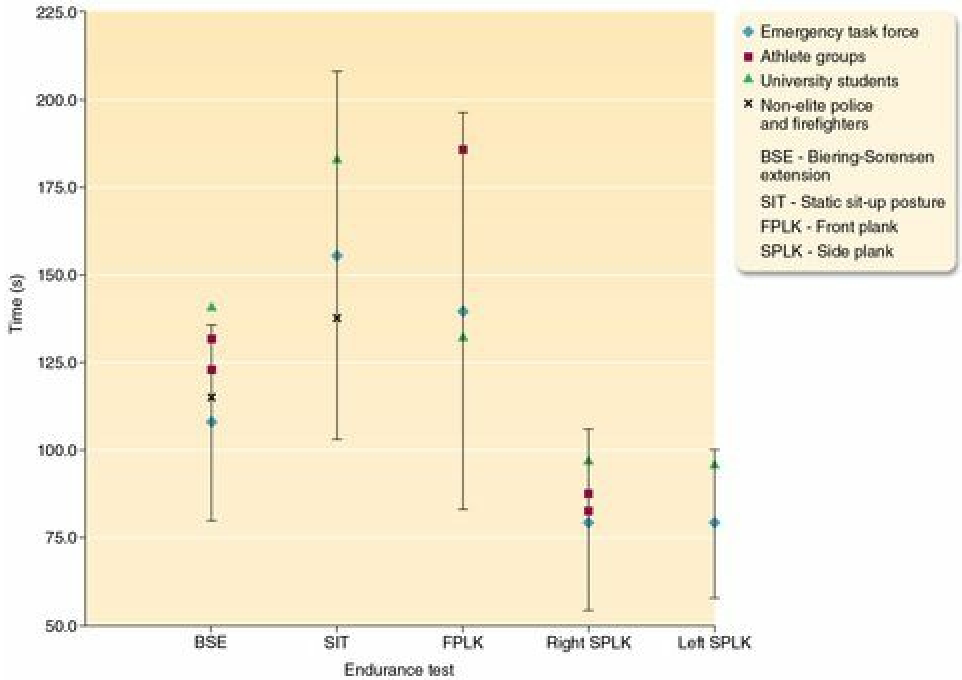
Table 9.2 Mean Endurance Times Comparing Normal Workers With Those Who Have Had Back Disorders

	NO BACK TROUBLES			HISTORY OF DISABLING BACK TROUBLES		
Task	Mean	SD	Ratio	Mean	SD	Ratio
Extension	103	35	1.0	90	49	1.0
Flexion*	66	23	0.64	84	45	0.93
RSB	54	21	0.52	58	23	0.64
LSB	54	22	0.52	65	27	0.72
Flexion/extension ratio*	0.71	0.26		1.15	0.66	
RSB/LSB ratio*	1.05	0.32		0.93	0.22	
RSB/extension ratio*	0.57	0.29		0.97	1.20	
LSB/extension ratio*	0.58	0.28		1.03	1.16	

Men, mean age 34 (never had back troubles: $n = 24$; lost work due to low back disorders: $n = 26$) from the same workplace. Variables that are significantly different between those with a history and those who have never had troubles have an asterisk. Note that all men were asymptomatic at the time of testing; these are long-lingering deficits subsequent to back troubles.

Endurance data from other populations give context to the links to back injury (see figure 9.8). Firefighters and police officers were followed over a period of 3 and 5 years, respectively, with particular focus on the links between certain fitness variables and the risk of developing back pain. Interestingly, in these professions, the fitter members had a higher risk. It appears that the injuries were more likely to occur in the station fitness training rooms (Frost et al., in press). This speaks volumes about the inappropriate training methods currently popular in these populations. Thus, the complexity of the interactions among exposure, movement competency, training, fitness, and injury in these worker groups may occlude their true relationships.

Figure 9.8 Endurance data from various populations.



Interpreting Endurance Scores

Interpreting absolute endurance is probably secondary to interpreting the relationship among the three muscle groups (flexors, lateral, and extensors). Normally, the following discrepancies suggest unbalanced endurance (note that the last ratio is a strength-to-endurance ratio).

Right-side bridge/left-side bridge endurance > 0.05

Flexion–extension endurance > 1.0

Side bridge (either side)–extension endurance > 0.75

Extensor strength (Nm)–extensor endurance (sec) > 4.0

Probably the most important ratio is the strength-to-endurance score.

Many strong men and women suffer repeated acute episodes of back pain. Most report the pain-inducing episode as resulting from a break in form. We interpret these observations to mean that endurance helps to preserve good spine-sparing form when performing tasks requiring some strength. Strength should not get ahead of the ability to control strength and retain good posture and form.

Other observations linking perturbed endurance with back disorders include those of Nelson-Wong and Callaghan (2014), who noted that those with poorer side bridge endurance scores were the first to develop pain when standing. Observing a series of case studies of professional hockey ice players, of the 5 who suffered sportsman's hernias, all had side bridge scores below 70 seconds at the beginning of the season. We are now using 70 seconds as a minimum for all players.

Several scientists have applied these scores to special populations such as elite golfers. It appears that golfers in this group have higher lateral endurance than normal. Such special groups do not conform to the average scores and rules for interpretation.

Integrating Evidence From Medical Images With Assessment Findings

There appears to be an increasing trend for physicians to order medical images for those presenting with back pain. Although this may be warranted in the presence of a red flag condition, it is rarely necessary. Some clinics advertise that if a patient sends images, they will determine whether their surgery will help them. In my view this is simply a business venture for profit. The best clinicians determine the pain causes and triggers from provocation testing. They also create the advantage of immediately determining what can take the patient's pain away. Further, the picture often bears little reflection of the amount of pain for reasons covered in chapters 1 and 2.

The next issue has to do with the use of the information contained in a radiologist's report. Offhand comments such as No tumor is present or The conus terminates at a normal level reveal that the radiologist has not been directed on what she should be looking for. The requisition should include details about the alleged or candidate injury or pain mechanism. This would help the radiologist optimize the patient setup (e.g., changing the load or posture to enhance the chance of revealing the pathology). Remember that the radiologist without such direction has no impression of the patient, the severity of the symptoms, or the postures that decrease or increase the pain. I see case after case in which the radiology was simply bogus. For example, a patient with a dynamic disc bulge that grows with flexion postures should sit flexed for a few minutes prior to the image acquisition. Failure to do this results in a bona fide patient being told he has no problem. We see patients with massive bony fracture only to receive a report of normal findings because the radiologist did not know how to position the patient appropriately (McGill and Yingling, 1999). The same is true for spine instability. So much more could be reported to the referring clinician in terms of the nature of the instability if the radiologists had some direction about acquiring the most appropriate image coupled with training to interpret the finding in a way to guide clinical treatment. The optimal situation is the clinician and the radiologist working together to discuss the case.

Obviously, images are very helpful in certain situations. I have found a few tumors over my career that were missed by the radiologist. Syndromes such as Scheuermann's disease, for example, can be confirmed radiologically. Yet even with Scheuermann's, there appears to be a constellation of features that would alert an astute clinician. For example, all of the patients with Scheuermann's whom I have seen have been lanky males with slouched postures (I am not sure what is the cause or consequence here).

The radiological diagnosis of degenerative disc disease has been discussed at several junctures in this book. This is not a disease. A thinned disc at one level should be followed up to examine the integrity of the end plate; most often this reveals damage, allowing the radiologist to report a useful directive rather than a red herring.

I also believe that it is well overdue for radiologists to become better schooled in the causes of the features they report. For example, Modic changes along the margins of vertebrae, in the presence of radiological instability, indicate a bone bruise. Clinically, this results in load intolerance for substantial periods of time. The most insightful monograph linking radiological findings with clinical features is Morgan (2013). A report of the muscle density and neurologically dead zones observed within muscle would also be helpful, yet these are not considered standard components of a report. Sadly, I never look at the radiology report anymore; I insist on seeing the original pictures.

A Final Note

The process of patient assessment is never ending. I find that my assessments take longer as I become better at probing the pain mechanism and the mechanisms responsible for aberrant movement patterns. But the effort is worth it. At the end of my assessments, the pain mechanism is almost always known, and the exact pain triggers are uncovered. (If you are unable to converge on a mechanical diagnosis, there is a good chance there is a more sinister cause of the pain.) Then I create the plan to help the patient avoid the pain triggers followed by progressions of exercise that address the precise clinical targets.

Chapter 10
Developing the Exercise Program

The exercises described in this chapter constitute a program founded on the principles discussed in previous chapters. This program is sufficient for many patients and for those interested in optimal back health. Those with performance ambitions are directed to my textbook Ultimate Back Fitness and Performance (McGill, 2014); also, chapter 11 offers some insight into this different world. Although I claim no expertise beyond the low back, I will broach other issues that have implications for the back. For example, improper technique when performing hip and knee mobilization and stretching can compromise the back; for this reason, I address these issues here. Upon completion of this chapter, you will be able to design exercise that challenges muscle and establishes stabilizing motor patterns, but spares the spine. The focus here is clearly on the person with back pain and on low back health for everyday activity.

Before presenting the exercises, I review some of the basic ideas discussed in this book thus far, such as stretching without stressing the back and abdominal hollowing and bracing. I also introduce the technique of flossing the nerve roots for those with sciatica.

Philosophy of Low Back Exercise Design

Many traditional notions that exercise professionals consider to be principles for exercise design, particularly when dealing with the low back, are not as well supported with data as generally thought. As previously noted, many clinicians still prescribe sit-ups—usually with the proviso that the knees remain bent. The resultant spinal loading of well over 3,000 N of compression to a fully flexed lumbar spine clearly shows the folly of such a recommendation. Other experts still recommend that people adopt a posterior pelvic tilt when performing many types of low back exercise. This actually increases the risk of injury by flexing the lumbar joints and loading passive tissues. The recommendation of flattening the lumbar region to the floor when performing abdominal exercise is another version of this ill-founded philosophy. Many continue to believe that having stronger back and abdominal muscles is protective and reduces bad back episodes, even though Luoto and colleagues (1995), among others, showed that muscle endurance, not strength, is more protective. This should not be misinterpreted to mean that having stronger muscles is not a good objective; rather, it reflects on many of the strength training approaches currently used that create back patients. Myths remain regarding the need for greater lumbar mobility, which the evidence suggests leads to more back troubles—not fewer (e.g., Biering-Sorensen, 1984)! Finally, we are disturbed by the fact that replicating the motions and spine loads that occur while using many low back extensor machines, intended as training and therapy, produced disc herniations when applied to spines in our laboratory!

Clinical Wisdom

Clearly, some current clinical wisdom needs to be reexamined in the light of the scientific evidence, much of which has been presented in this book. Removing the cause of pain is essential. Spine and torso stiffness enhances performance and reduces the risk of injury—this is unequivocal. The evidence indicates that the safest and most mechanically justifiable approach to enhancing lumbar stability through exercise is to emphasize endurance over strength. Clinicians using such an approach should encourage patients to maintain a neutral spine posture when under load and use abdominal cocontraction and bracing in a functional way. Studies of military personnel show this to be effective (e.g., Suni et al., 2013), as do large studies of people with back pain versus matched controls without pain. People with back pain move in ways that stress the back, ensuring pain (Luomajoki et al., 2008)! The bodybuilding approach often used in rehabilitation is not synonymous with health objectives and results in additional risk from joint loading through a range of motion. Further, the simple isolationist motor patterns do not promote pain-reducing stability.

Thoughts on Mobility

This book has introduced issues regarding spine mobility, whether stretching is appropriate or effective, and the consequences of pain on mobility. The next few sections guide the application of scientific findings on mobility and moving with competence.

Sparing the Back While Stretching the Hips and Knees

Given that tonic contractions in the psoas, which produce a shortening and hip extension restriction, are common in those with back troubles (McGill et al., 2003), people with this condition would probably benefit from hip stretching and other range of motion exercises. Sometimes, however, poor technique during the stretching of hip and knee tissues leads to unnecessary loading of the lumbar spine. This can easily be avoided by changing technique. A general guideline for sparing the back is to maintain an upright torso posture while performing hip and knee work. Preservation of a neutral spine ensures minimal loading from passive tissues. An upright torso minimizes the reaction torques, the associated muscle contraction, and spine load.

Abdominal bracing to an appropriate level is sometimes required for pain control. For example, lunges are a good exercise for challenging strength, endurance, balance, and mobility in the lower extremities. Lower-extremity capability is needed to facilitate spine-sparing postures when lifting and when performing a host of other tasks.

The technique for sparing the back is to maintain an upright torso while performing the lunge (see figure 10.1). Some clinicians recommend keeping the back leg straight, which causes patients to flex the torso forward. This is poor form for sparing the back (see figure 10.2). A general principle is to keep the torso upright with a neutral lumbar spine while stretching joints other than the back. Figure 10.3, a and b, for example, shows spine-sparing postures for stretching the quads and the hip adductors, respectively. Finally, the lunge with a neutral spine stretches the iliacus more than the psoas (the iliacus is a uniarticular muscle that crosses only the hip). Fully stretching the psoas requires a lateral bend of the torso away from the extended hip. The anatomy train is then engaged by raising the same-side arm and pushing the hand to the ceiling, but this is reserved for those with more robust backs (see figure 10.4, a-d).

Figure 10.1 The spine is spared performing the beginner's lunge by maintaining an upright torso and neutral spine curvature.

Figure 10.2 Some clinicians recommend keeping the back leg straight while performing lunges, which causes patients to flex the torso forward. This is poor form for sparing the back.

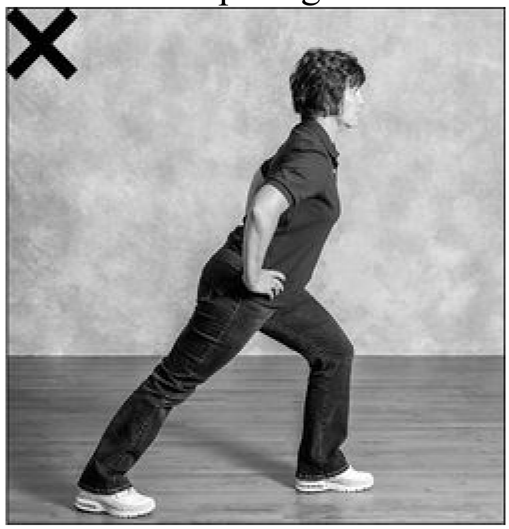
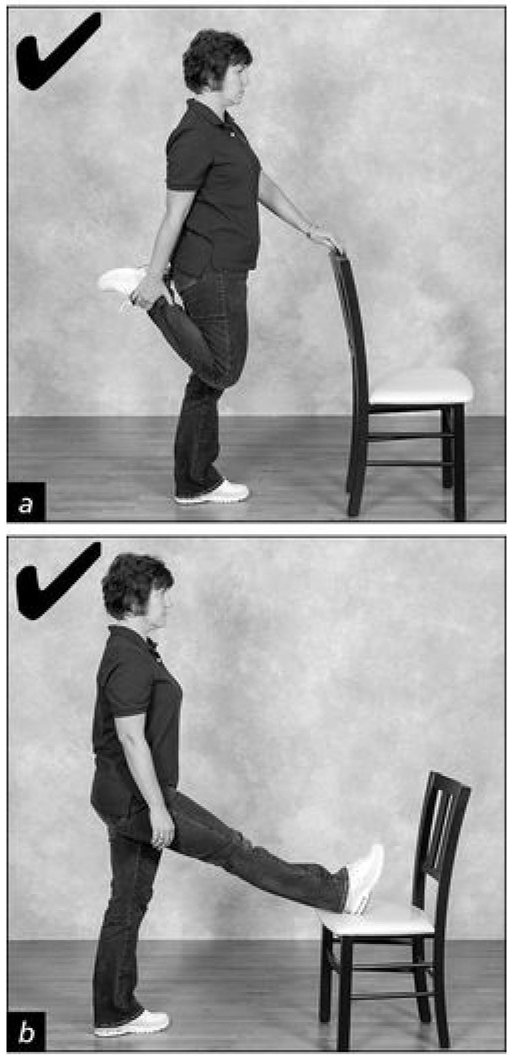


Figure 10.3 During stretching at joints other than the back, the spine is spared by maintaining an upright torso and neutral spine curve. (a) When stretching the quads, holding a chair for balance is a good way to ensure a straight back. (b , c) This person has sufficient hip flexion mobility to ensure an upright torso and a neutral spine. Those who are unable to maintain this spine posture should forgo this exercise until they can achieve the required hip flexion mobility. (d) This shows poor technique with spine motion.



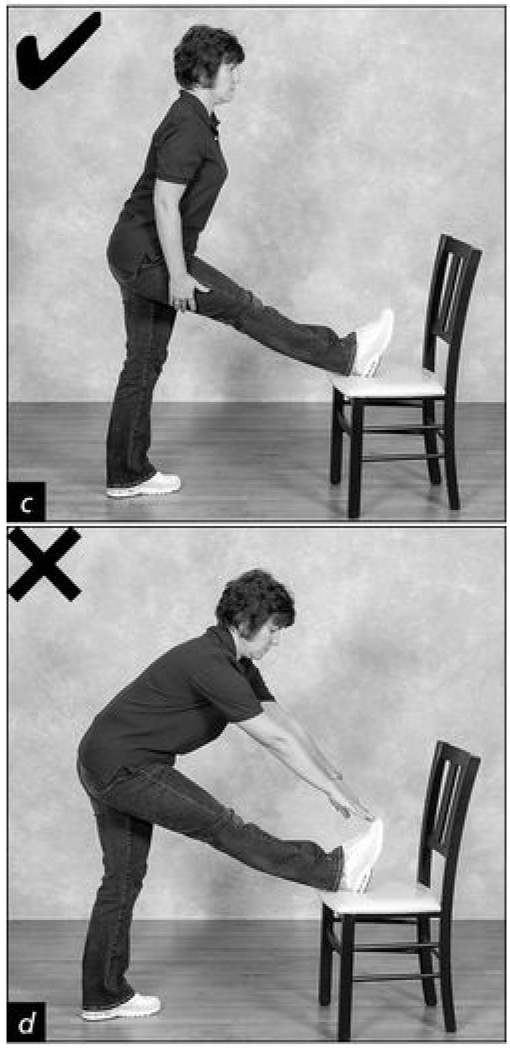

c
d

Figure 10.4 Unlike the uniarticular iliacus, the multiarticular psoas crosses the hip and entire lumbar spine, and needs specific techniques to stretch it. (a) The patient will feel the psoas tendon just medial to the rectus femoris, below the inguinal ligament. (b) Then they descend into a lunge, (c) noting the absence of tension in the psoas tendon. The ipsilateral arm is raised and pushed straight up. (d) The psoas should now be noticeable. Leaning the torso away from the arm pushing up will increase the psoas tension.

Flossing the Nerve Roots for Those With Accompanying Sciatica and Neural Tensions

Before presenting the flossing technique for sciatica, some background is required regarding the biomechanics and biochemistry of the spinal cord and nerves.

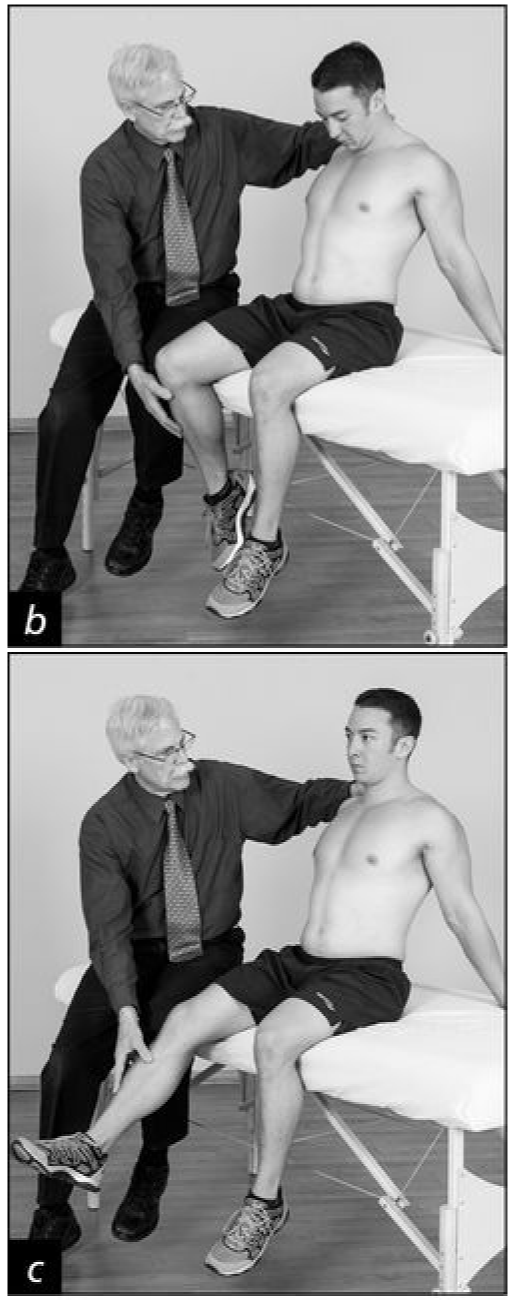
Excessive tension in the nerve is the cause of sciatica. Normally, the cord nerve roots travel through the foramens of the neural arch of each vertebra, and their sliding excursions are substantial with spine flexion, hip flexion, and knee extension. According to Louis (1981), the nerve can travel well over a centimeter (over 1/2 in.) during some of these movements. Thus, if a nerve root is impinged and cannot slide, any of the postures that would normally pull that nerve root through the foramen will increase nerve tension. Instead of moving, the nerve is stretched. Tension on such a nerve can be increased from the cranial end with simultaneous cervical flexion because the entire spinal cord moves slightly with cervical flexion and thus pulls at the nerve roots all along their length. This is the basis for the tests for nerve tension presented in chapter 9.

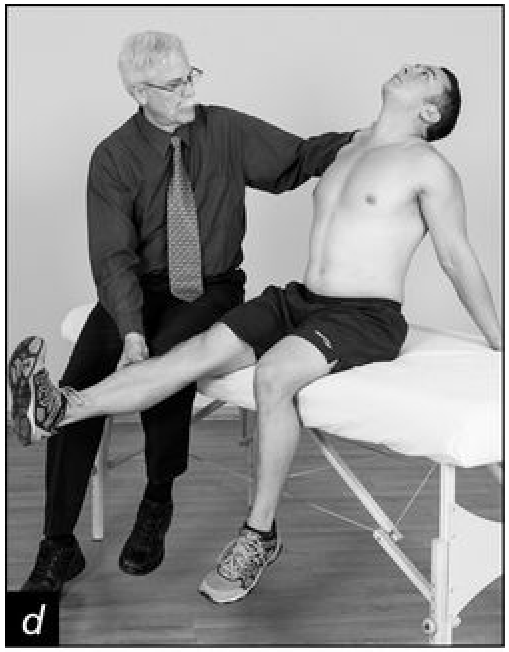
Recently, scientists have suggested that nerves have the ability to create their own pathways as long as they can move. They seem to have some chemically based ability to dissolve—over time—tissues impinging them, although the exact molecular mechanism remains elusive. The idea of flossing is to pull the cord and nerves from one end only, while releasing at the other, and then to switch the pull-and-release direction. In this way the nerve roots are flossed through the lateral foramina and in fact along their entire length. Thus, by working the nerves back and forth in whatever limited range they can manage in spite of impingement, we facilitate the dissolving of the impingement and the gradual release of the nerve to once again move freely (this approach was proposed by Butler, 1999). This flossing action is accomplished with coordinated hip, knee, and cervical motion.

A note of caution is needed here: Although this can be wonderful in helping chronic sciatica resolution, it can also cause an acute onset. Be very conservative in the first session. If the nerve is adhered so that it can't slide, the sciatic symptoms will be exacerbated. However, if the patient reports no change or even relief the next day, then increase the flossing. Monitor all patients and remove the procedure from the programs of any whose symptoms worsen.

Flossing

The patient, seated and with the legs able to swing freely (a), flexes the cervical spine while at the same time flexes the knee and plantar flexes the ankle (b). This creates a pull on the spinal cord from the cranial end and a release from the caudal end. This should not produce sciatic symptoms (see the discussion of the supine passive leg raise test in chapter 9). Then, the patient extends the cervical spine with simultaneous knee extension on the side with sciatica (c, d). This pulls the nerve from the caudal end with a corresponding release at the cranial end. The cycle is completed as the knee is flexed with coordinated motion of the cervical spine flexing. This causes the nerves to floss through the vertebral tissues. If the patient experiences minor sciatic symptoms when the cervical spine is flexed or the knee is extended, then the patient should reduce the range of motion at these joints until no pain is provoked. Some patients who have suffered sciatica pain for years report reductions in their sciatic symptoms within a few days to a couple of weeks; others report increasing symptoms.


b
c



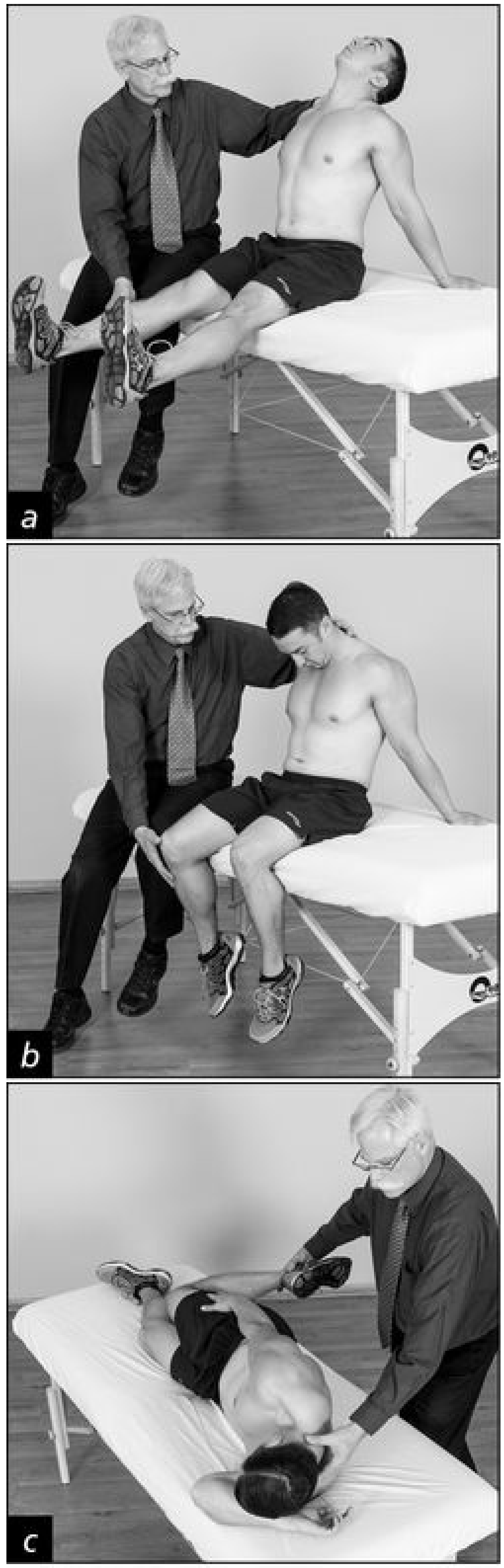
00:00 / 00:00

Technique Tips

- A tip to enhance efficacy is to first adopt a posture that relieves impingement. For example, the patient with a disc bulge who can reduce the bulge by adopting a prone extension posture (such as shown in the McKenzie posture test in chapter 9) should do so for a few minutes prior to flossing. The patient should then adopt the posture with the longest reflex response.
- The motion should be slow (with a flexion–extension cycle lasting about 5 seconds), but most important, coordinated, to create floss rather than length change in the neural tract.
- The objective is to create motion rather than a static stretch. The patient should not push into the end range, and never travel into a painful range.
- The patient should begin by performing 10 repetitions with each leg. Then, if symptoms are not exacerbated, he can perform these repetitions several times per day. The patient should not perform flossing within 2 hours after rising from bed.

Advanced Flossing

Recent work of Professor Olavc Airaksimen and colleagues (personal communication, June 2013) showed that double-leg flossing increased the central neural tract excursion distance and may be considered as an exercise to add to the routine (a, b). The progression continues in side-lying in which the hip joint becomes involved in creating large nerve excursions. The neck is flexed while the hip and knee are extended, and the ankle is plantar flexed (c). The cycle is completed with the opposite movement (d, e).




d
e

Identifying Safe and Effective Exercises

After a brief review of the muscles that stabilize the spine, we will have a look at exercises for training those muscles. Although many muscles have been regarded as primary spine stabilizers, the confirmation of their roles requires two levels of analysis. The first is engineering—a stability analysis must be conducted on anatomically robust spine models to document the ability of each component to stiffen and stabilize. Second, electromyographic recordings of all muscles (even deep muscles requiring intramuscular electrodes) are necessary to confirm the extent to which the motor control system involves each muscle to ensure sufficient stability.

Identifying safe and effective exercises for low back stabilization is a several-step process: incorporating and patterning the muscles involved in spine stability; eliminating unsafe exercises; and selecting or devising exercises that appropriately challenge the selected muscles at the various stages of rehabilitation and conditioning.

Incorporating and Patterning the Muscles

Our intramuscular and surface electromyography (EMG) and modeling studies to quantify spine stability demonstrated that virtually all torso muscles play a role in stabilization. (Some data are presented in chapter 4.) Although the back extensors and the abdominal wall all appear to play significant roles, the quadratus lumborum also appears to play an important role in many tasks. The fibers of quadratus lumborum cross-link the vertebrae; they have a large lateral moment arm via the transverse process attachments; and they traverse between the rib cage and iliac crests. In this way, the quadratus is an effective stiffener and appears to be effective in all loading modes, as a result of its architectural design. Typically, the first mode of lumbar buckling is lateral; the quadratus appears to play a significant role in local lateral buttressing. The three layers of the abdominal wall are also important for stability together with the muscles that attach directly to vertebrae—the multisegmented longissimus and iliocostalis and the unisegmental multifidii. Cholewicki and McGill (1996) also presented an argument for the role of the small intertransversarii in producing small but critical stabilizing forces. As well, latissimus dorsi plays a major stabilizing role in many loading tasks (Kavcic, Grenier, and McGill, 2004). On the other hand, psoas activation appears to have little relationship with low back demands; the motor control system activates it when hip flexor moment is required (see Andersson et al., 1996; Juker et al., 1998), limiting its role in stabilizing the spine when hip flexion is not involved. But when corresponding hip flexion is demanded, the psoas becomes an important spine stabilizer via shear stiffness, compression, and locking the pelvis to the lumbar spine.

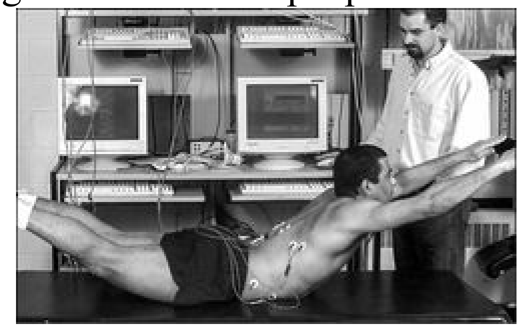
Eliminating Unsafe Exercises

Quantitative data have confirmed that no single abdominal exercise challenges all of the abdominal musculature while sparing the back (Axler and McGill, 1997). For this reason, more than one exercise is required. Unfortunately, many inappropriate exercises have been prescribed for people with low back difficulties, including the following:

- Sit-ups (both straight-leg and bent-knee) are characterized by higher psoas activation, with consequent high low back compressive loads that exceed the occupational guidelines of the National Institute for Occupational Safety and Health (NIOSH) in the United States.
- Leg raises cause even higher psoas activation and spine compression (actual values are presented in chapter 4).
- Most traditional extensor exercises are characterized by very high spine loads, which result from externally applied compressive and shear forces (from either free weights or resistance machines). A commonly prescribed spine extensor muscle challenge, the superman, involves lying prone while extending the arms and legs (see figure 10.5). This results in over 6,000 N of compression to a hyperextended spine, transfers load to the facets, and crushes the interspinous ligament. Needless to say, this exercise is contraindicated for anyone at risk of low back injury—or reinjury! Although some may believe that putting the hands on either side of the head rather than extending them may make this exercise safe, this is not true. This exercise should not be done in any form. Further, recall that the mechanism for disc herniation is reproduced by back machines that take the lumbar spine from full

flexion and through the range of motion under load from muscle contraction.

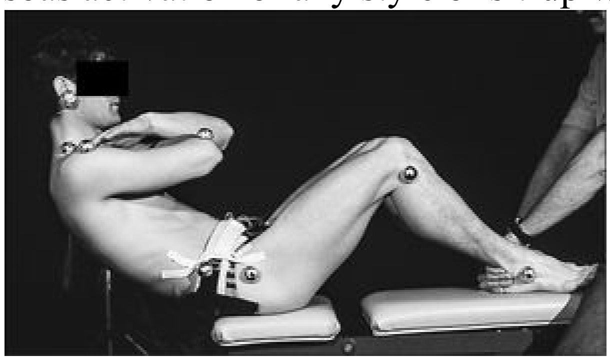
Figure 10.5 A commonly prescribed spine extensor muscle challenge (superman) involves lying prone while extending the arms and legs. This results in over 6,000 N of compression to a hyperextended spine. It is a poorly designed exercise for people with back pain.



Photos from Stuart McGill

In our investigations we made several relevant observations regarding psoas activation during abdominal exercises. The challenge to the psoas is lowest during curl-ups, followed by higher levels during the horizontal side bridge. Bent-knee sit-ups were characterized by larger psoas activation than straight-leg sit-ups, and the highest psoas activity was observed during leg raises and hand-on-knee flexor isometric exertions. The often-recommended press-heels sit-up, which has been hypothesized to activate the hamstrings and neurally inhibit the psoas, was actually confirmed to increase psoas activation! (See figure 10.6.) (Original data can be found in Juker et al., 1998; clinicians and coaches who intentionally wish to train the psoas will find these data informative.) Once again, the horizontal side bridge appears to have merit because it challenges the lateral obliques and transverse abdominis without high lumbar compressive loading and ensures a high stability index (a loss of contraction of the involved muscles would cause the patient to fall out of the bridge).

Figure 10.6 The press-heels sit-up was proposed by several clinical groups, on theoretical grounds, to inhibit the psoas by activating the hamstrings. In fact, EMG assessment (Juker et al., 1998) proved this to be mythical. This original photo from the intramuscular experiments shows the clinician's hands behind the heels of the subject as the hamstrings are being activated during the sit-up. Activating the hamstrings creates a hip extensor moment, and sit-ups require hip flexion. During this type of sit-up, the psoas is activated to even higher levels to overcome the extensor moment from the hamstrings and produce a net flexor moment. This type of sit-up produced the highest level of psoas activation of any style of sit-up we quantified!



Photos from Stuart McGill

Upper and Lower Rectus Abdominis?

Myoelectric evidence, normalized and calibrated, suggests that there is no functional distinction between an upper and lower rectus abdominis in most people; in contrast, the obliques are regionally activated with upper and lower neuromuscular compartments as well as medial and lateral components. There are, however, some highly trained people who can create small differences in activation. However, these differences are only at very low levels of activation and occur during what would be considered nonfunctional tasks, such as belly dancing (Moreside, Vera-Garcia, and McGill, 2008).

Selecting Safe and Effective Exercises

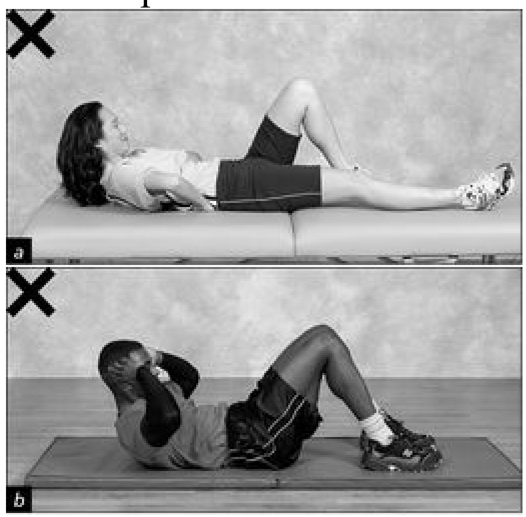
A wise choice for stabilization exercises in the early stages of training or rehabilitation, and for simple low back health objectives, is the big three that we have quantified to sufficiently challenge muscle, spare the spine of high load, and ensure sufficient stability:

- Curl-ups for rectus abdominis
- Several variations of the side bridge for the obliques, transverse abdominis, and quadratus
- Leg and arm extensions leading to progressions of the bird dog for the many back extensors

The variation of each of these exercises must be chosen with the patient's or athlete's status and goals in mind.

- Curl-up: training rectus abdominis. Calibrated intramuscular and surface EMG evidence suggests that the various types of curl-ups challenge mainly the rectus abdominis because psoas and abdominal wall (internal and external oblique and transverse abdominis) activity is relatively low (see the tables in chapter 4 for relative activation levels in a variety of exercise tasks). Curl-ups performed with poor technique, however, can be counterproductive, either failing to activate the rectus abdominis sufficiently or overstressing the spine (see figure 10.7, a and b). Curl-ups with a twisting motion are expensive in terms of lumbar compression because of the additional oblique challenge. Higher oblique activation with lower spine load is accomplished with the side bridge, which is therefore preferred over twisting curl-ups for training the obliques. The side bridge is presented next. The highest-level curl-up is presented in chapter 11.

Figure 10.7 (a) Poor form during the curl-up is to flex the cervical spine, loading the neck and not the rectus. (b) Another common type of poor form is to elevate the head and shoulders a large distance off the floor. This patient is elevating far too much, which is closer to replicating the much higher stresses of the sit-up. The intention is to activate rectus and not to produce lumbar spine motion.



- Side bridge : training the quadratus lumborum , lateral obliques , and transverse abdominis. Given the architectural and EMG evidence for the quadratus lumborum, transverse abdominis, and abdominal obliques as spine stabilizers, the optimal technique to maximize activation but minimize spine load appears to be the side bridge. Abdominal bracing is emphasized in all forms of this exercise. Maintaining the bridge ensures constant muscle activation, and the brace introduces new combinations of muscle recruitment to ensure stability. It is almost impossible for the spine to become unstable during the performance of a side bridge with a neutral spine.
- Bird dog: training the back extensors. In our search for methods to activate the extensors (including longissimus, iliocostalis, and multifidii) with minimal spine loading, we have found that the single-leg extension creates tolerable spine loading (<2,500 N) for many and activates one side of the lumbar extensors to approximately 18% of maximal voluntary contraction (MVC). Simultaneous leg extension with contralateral arm raise (the bird dog) increases the unilateral extensor muscle challenge (approximately 27% of MVC in one side of the lumbar

extensors and 45% of MVC in the other side of the thoracic extensors) but also increases lumbar compression to well over 3,000 N (Callaghan, Gunning, and McGill, 1998). This exercise can be enhanced with abdominal bracing and deliberate mental imaging of activation of each level of the local extensors. Once again, the technique for challenging the extensors should be guided by the patient's status and goals. This is an individual clinical decision.

Beginner's Program for Pain Control and Stabilization

Now that you are familiar with some specific recommended low back exercises, how do you put these together to create the best possible program for your patient? Assuming that there were no particular indicators obtained during provocative testing that would contraindicate beginning with a typical progression, we recommend considering the following sequence:

1. Throughout the day incorporate the movement tools to eliminate tissue irritation. Irritation sensitizes the tissues to trigger pain at lower levels of stimulus. (Recall chapter 8.)
2. Begin the training session with the flexion–extension cycles, also called the cat–camel exercise (see figure 10.8, a and b), to reduce spine viscosity. Note that the cat–camel is intended as a motion exercise, not a stretch, so the emphasis is on motion rather than pushing at the end ranges of flexion and extension. We have found that five or six cycles are often sufficient to reduce most viscous stresses—additional cycles rarely reduce viscous friction further. Those with sciatica may find increased symptoms during the flexion phase. Use pain to guide the choice of a suitable pain-free range of motion.
3. Perform slow partial squats in which the pelvis is directed downward and posterior along a 45° line. The spine has no motion (see the description of the potty squat in chapter 8).
4. Sciatic pain sufferers may try the nerve-flossing technique following a posture that relieves neural tension.
5. These motions are followed by anterior abdominal exercises—namely, appropriate curl-ups.
6. Lateral musculature exercises follow the preceding—namely, the side bridge for quadratus lumborum and the muscles of the abdominal wall, for optimal stability.
7. The extensor program begins in the quadruped position with single thigh extensions progressing to the full bird dog.
8. The person begins a walking program based on tolerable intervals. If she can walk only 10 meters (or yards) without pain, then she should walk 7 meters every hour. As she gains tolerance, increase the distance and the time between intervals. Always design the distance to be less than the

distance at which pain would occur.

Figure 10.8 Cat–camel exercise. Note that this is a motion exercise and not a stretch; the person should not push at the end range of motion. Viscosity is measurably reduced after just a few cycles.

Photos from Stuart McGill

In general, we recommend that the isometric holds performed in the curl-up, bridge, and bird dog be no longer than 7 or 8 seconds, given recent evidence from near-infrared spectroscopy indicating rapid loss of available oxygen in torso muscles contracting at these levels. Short relaxation of the muscle restores oxygen (McGill, Hughson, and Parks, 2000). The endurance objectives are achieved by building up repetitions of the exertions rather than by increasing the duration of each hold.

Motivated by evidence of the superiority of extensor endurance over strength as a benchmark for good back health, we documented normal ratios of endurance times for the torso flexors relative to the extensors and lateral musculature (see chapter 9). Use these values to identify endurance deficits—both absolute values and for one muscle group relative to another—and to establish reasonable endurance goals for your patients.

A Crucial Note to Clinicians

Clinical Relevance

Insistence on perfect technique will allow the patient to reach much higher levels of challenge without pain (Ikeda and McGill, 2012; McGill and Karpowicz, 2009). Do not allow spine deviation such as lumbar flattening to the floor or loss of neutral during holding of any stabilizing posture. Constant postural correction is necessary in some patients. Finding the optimal dosage of load is also critical. Keep correcting to eliminate any pain.

Exercises That May Be Used in a Stabilization Program

The basic big three exercises (an appropriate form of the modified curl-up, side plank, and bird dog) have been found to be the most effective in working with patients with low back troubles.

Beginner Through Advanced Curl-Ups

This series of curl-ups, along with neck-strengthening isometric exercises for the neck (as necessary), provides a good foundation for a strong rectus abdominis.

Curl-Up, Beginner

The curl-up technique is critical to spare the spine. The basic starting posture is supine with the hands supporting the lumbar region. The person should not flatten the back to the floor, which takes the spine out of elastic equilibrium and raises the stresses in the passive tissues. Although the position of elastic equilibrium is desired in the lumbar region, the hands can be adjusted to minimize pain if needed. One leg is bent with the knee flexed to 90° and the other leg remains relaxed on the floor. This adds further torque to the pelvis to prevent the lumbar spine from flattening to the floor. The focus of the rotation is in the thoracic spine; many tend to flex the cervical spine, which is poor technique. Rather, picture the head and neck as a rigid block on the thoracic spine. No cervical motion should occur—either chin poking or chin tucking. The intention is to activate rectus and the obliques and not to produce spine motion. People who report neck discomfort may try the isometric exercises for the neck that follow. In addition, particularly for patients experiencing neck discomfort, the tongue should be placed on the roof of the mouth behind the front teeth and pushed upward, which helps to promote stabilizing neck muscle patterns. Patients should leave the elbows on the floor while elevating the head and shoulders a short distance off the floor. The rotation is focused in the midthoracic region. The head and neck unit is locked onto the rib cage.

Photos from Stuart McGill

Shown in the photos are (a) a very mild form of the curl-up to just take the weight off the head and shoulders with almost no motion. (b) More challenge is obtained with raising the head and shoulders but focusing the motion on the thoracic spine with no cervical or lumbar flexion.

Isometric Exercises for the Neck

Those who experience neck symptoms with curl-ups may find relief by building the neck by developing better control (software) than muscular strength (hardware). Begin these isometric exercises where the head and neck unit does not move, and the tongue is on the roof of the mouth behind the front teeth and pushed upward to activate the deep flexor muscles (a). The fists are under the chin where they push up (b). The neck maintains isometric contraction to ensure that no motion occurs. The patient holds for several seconds, then relaxes, building up endurance and grooving stabilizing motor patterns by increasing the repetitions of the hold–relax cycles. (Believe it or not, some patients try this while chewing gum, which makes the establishing of stabilizing patterns impossible.) More traditional isometric exercises (c-e) may or may not help.

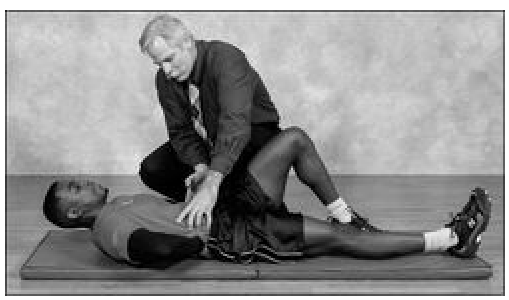

c
d
e

Curl-Up, Intermediate

The intermediate progression of the curl-up involves raising the elbows a couple of centimeters (a little less than an inch) so that the arms do not pry the shoulders up, thus shifting more load to the rectus. The person should not raise the head and neck any higher than in the beginner curl-up.

Curl-Up, Advanced

An advanced curl-up requires prebracing of the abdominal wall. The resistance is provided by the brace, and no additional motion takes place. This can be facilitated and learned with fascial raking by a clinician. Deep breathing is also added to train the diaphragm to function independently of the stabilizing abdominal musculature. The head and neck must move as a unit, maintaining their rigid-block position on the thoracic spine.



The side bridge is an excellent exercise to challenge the important stabilizers of quadratus lumborum, latissimus dorsi, and the abdominal obliques, while sparing the spine from high loads. Although there are several forms that comprise a logical progression, each form is characterized by a neutral spine with the rib cage locked to the pelvis.

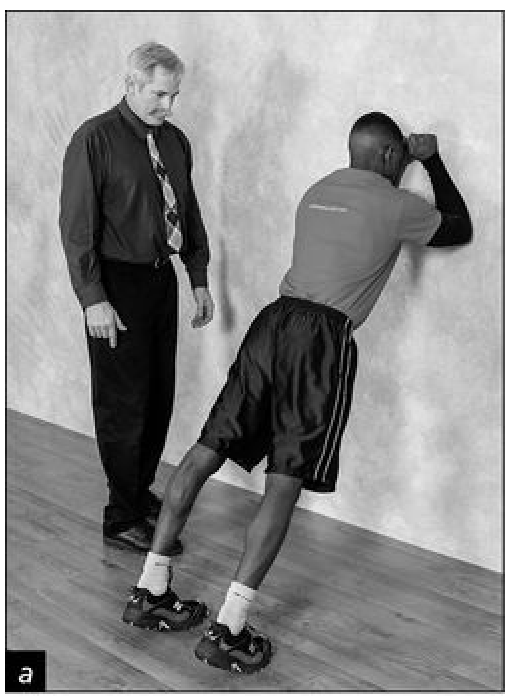
Side Bridge, Remedial

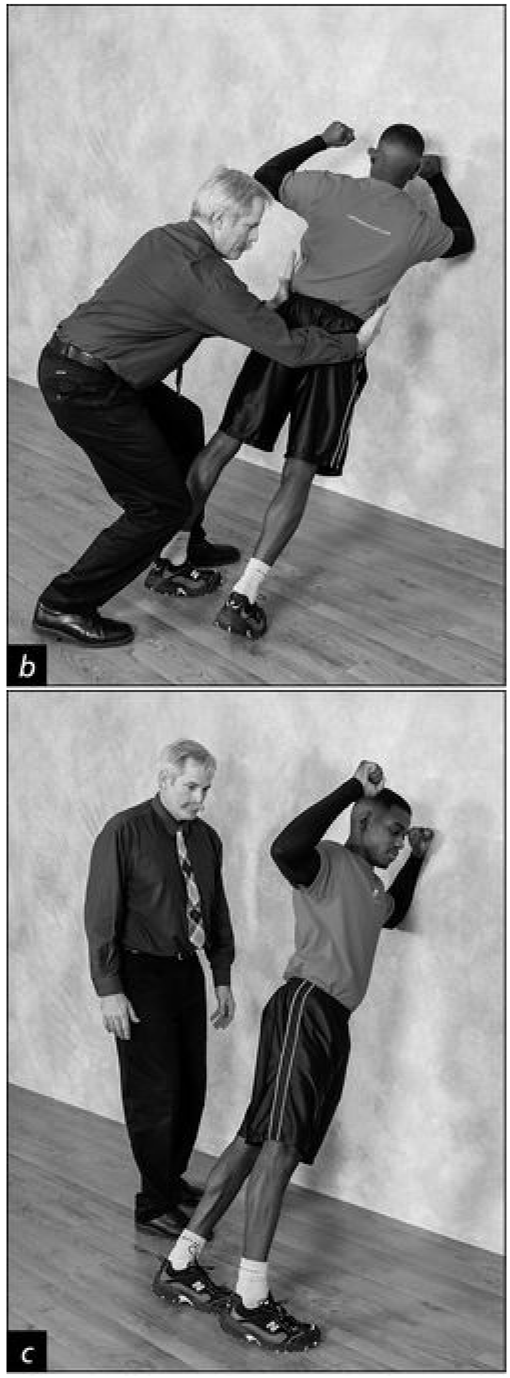
Many patients deserve special consideration—for example, the chronic patient who is quite deconditioned. These patients are sometimes unable to perform the side bridge even from the knees. Start these people with a side bridge while standing against a wall. Instruct them to move smoothly from the beginning position (a) through the intermediate position (b) to the final position (c), in one flowing motion. The patient should pivot over the toes, as if performing a fluid dance move. Clinical cues may include feedback such as keeping the heels of the hand on the pelvis and the fingertips on the rig cage to eliminate lumbar motion.

Once patients graduate to the floor, they can spare the spine by beginning (d) with the knees and hips quite flexed and move into the side bridge posture with an accordion-like unfolding of the legs (or a hip-hinge squat; d, e), all the time keeping a braced neutral spine. Never allow the patient to (f) slump into a laterally bent, deviated spine posture.

Another type of patient is the football player whose shoulders are so painful he cannot tolerate the shoulder load (some elderly women also fall into this patient group). These patients can perform a modified side bridge by lying on the floor and attempting to raise the legs laterally (g, h) or simply attempting to take the weight off the legs.

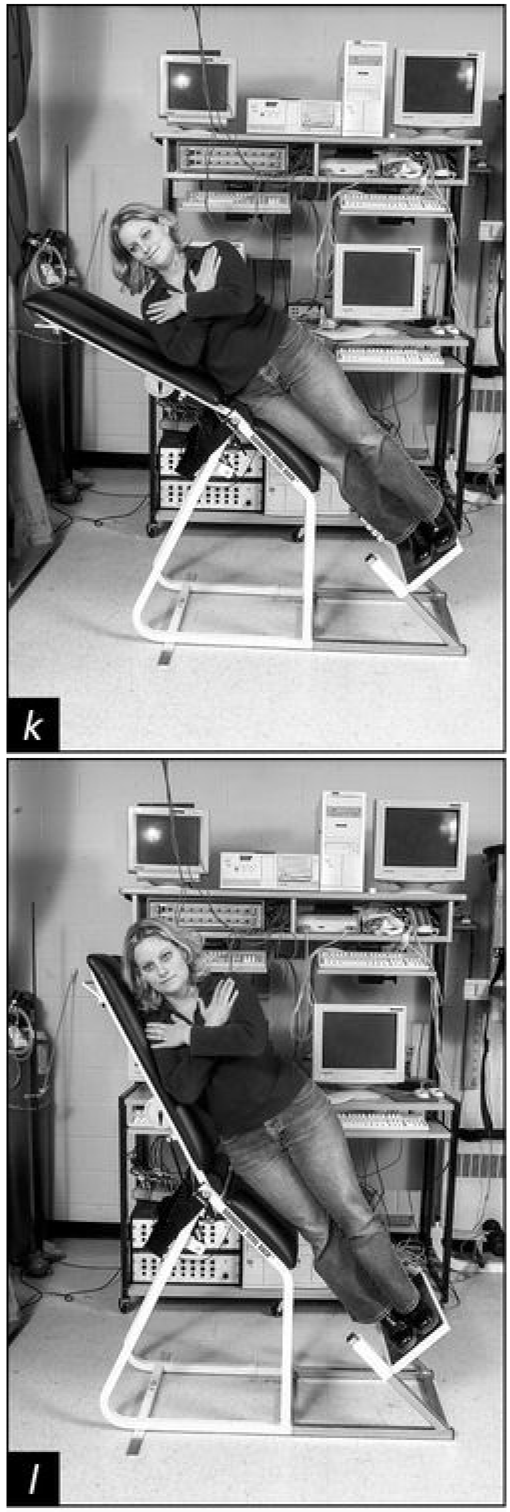
Another option for patients with shoulders that cannot tolerate load is to stand on a 45° bench with the feet anchored, which spares the shoulders (j-l).




b
c


i


j



Photos from Stuart McGill (photos d, e, g-l)

Side Bridge, Beginner Through Advanced

Once the client is comfortable performing the final remedial side bridge, she may move on to the beginner through advanced side bridge progression.

Side Bridge, Beginner

Beginners bridge from the knees. In the beginning position, the exerciser is on the side, supported by the elbow and hip. The knees are bent to 90°. Placing the free hand (the hand and fingers are spread and cap over the deltoid) on the opposite shoulder and pulling down robustly on it will help stabilize the shoulder. The torso is straightened until the body is supported on the elbow and the knee, with some input from the lower leg. The beginner side bridge can be slightly advanced by placing the free arm along the side of the torso—effectively placing more load on the bridge.

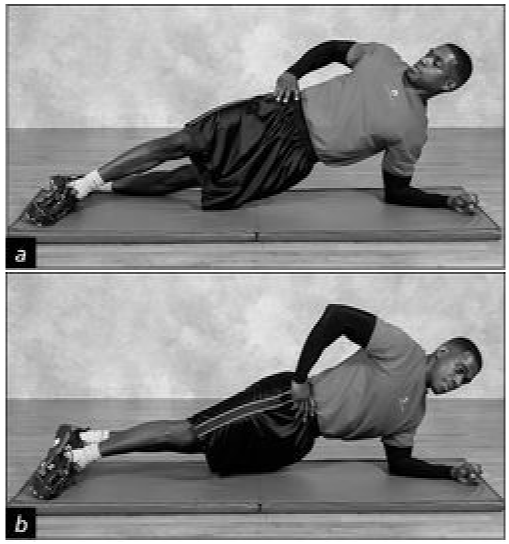
Photos from Stuart McGill

Side Bridge, Intermediate

The beginning position for the intermediate side bridge is like that for the beginner, except that the legs are straight. The torso is straightened until the body is supported on the elbow and feet (see photo). When supported in this way, the lumbar compression is a modest 2,500 N, but the quadratus closer to the floor appears to be active up to 50% of MVC (this is a preferred exercise for the obliques because they experience similar levels of activation).

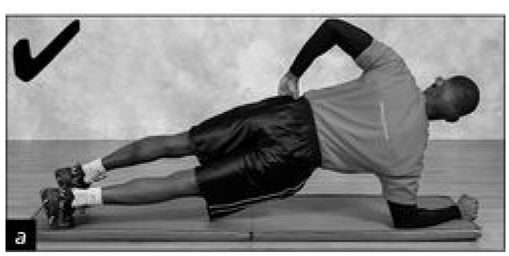
Side Bridge, Intermediate Variation

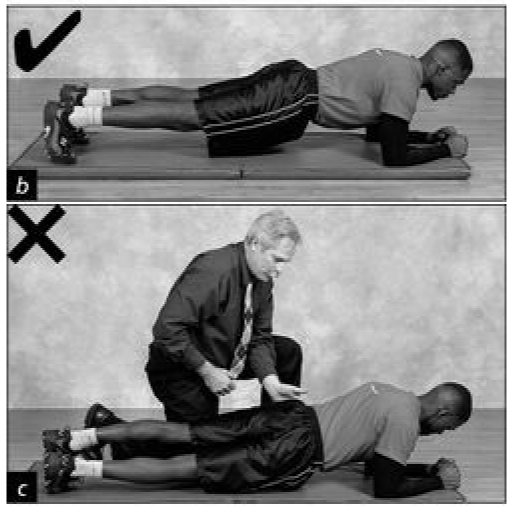
Placing the upper leg and foot in front of the lower leg and foot enables a longitudinal rolling of the torso to challenge both anterior (a) and posterior (b) portions of the abdominal wall.



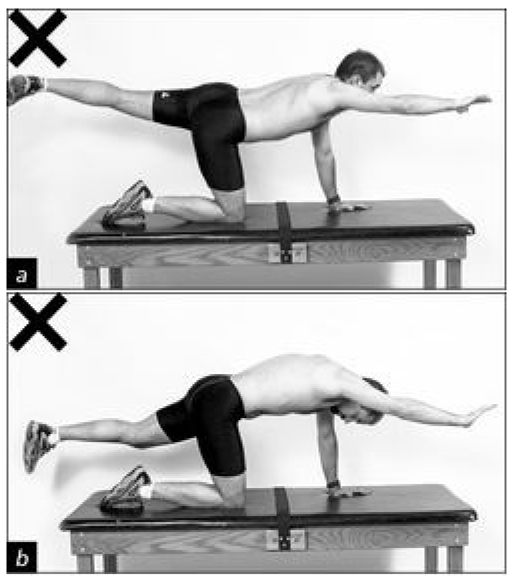
Side Bridge, Advanced

The advanced technique to enhance the motor challenge of the side bridge requires transferring from one elbow to the other while abdominally bracing (a-c) rather than repeatedly hiking the hips off the floor into the bridge position. Ensure that the client’s rib cage is braced to the pelvis and that this rigidity is maintained through the full roll from one side to the other (a, b). You may cue the patient by placing the heel of your hand on the iliac crest with your fingers touching the rib cage. This ensures that the pelvis does not lead the rib cage during rolling and makes the exercise more challenging and safer for the spine. Poor form due to spine twist is shown in c. Still higher levels of activation would be reached with the feet on a labile surface (Vera-Garcia, Grenier, and McGill, 2000). This technique is reserved for the athletic back.





Remember to emphasize abdominal bracing and a neutral spine throughout all versions of the final of the big three exercises—the bird dog. Poor form includes hip hiking or any other configuration that causes deviation (twist, flexion, or lateral bending) to the spine (see a and b).



Common mistakes made in the bird dog are (a) hiking the hip, which twists the spine, and (b) not achieving a neutral spine. A flexed posture is shown here.

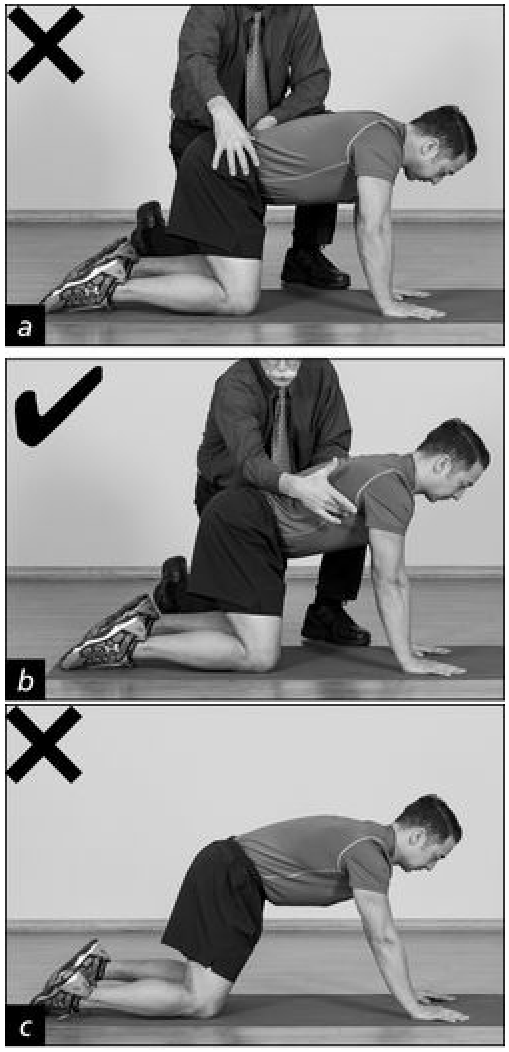
Photos from Stuart McGill

Remedial Through Advanced Bird Dog

Recall that the basic bird dog trains the extensor muscles, even though the abdominals are consciously activated to control and maintain the neutral spine posture. As always, progression based on each patient’s unique needs and responses to exercise, along with insistence on correct form, is indispensable to ensuring that the patient experiences strength and endurance gains without injury.

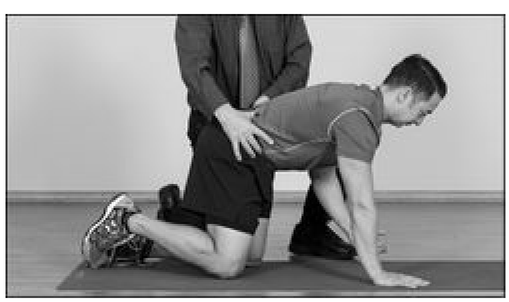
Bird Dog, Remedial

The starting position is on the hands and knees with the hands under the shoulders and the knees directly under the hips (a). For the patient with a very deconditioned back, this exercise involves simply lifting a hand or knee about an inch (2.5 cm) off the floor (b). After the patient is able to raise a hand or knee without pain, it is appropriate to progress to raising the opposite hand and knee simultaneously. Starting the exercise with good form is important to reduce pain and enhance tolerance. Here, a patient is starting in a flexed spine posture (a). Corrections include more lordosis, “life the tail,” and manually cuing up the rib cage (b). Another poor beginning posture is not having the knees under the hips and the hands under the shoulders (c).



Bird Dog, Beginner

The progression continues with raising one leg or one arm at a time.



Bird Dog, Intermediate

The intermediate bird dog is achieved when the patient is able to raise the opposite arm and leg simultaneously. Raising either the arm or the leg past horizontal should be avoided. The objective is to be able to hold the limbs parallel to the floor for about 6 to 8 seconds. Good form includes a neutral spine with no spine motion and abdominal bracing. Also, here is a cue to push the heel away and make a fist to enhance muscular engagement and reduce the tendency for hip hiking.

Bird Dog, Advanced

To develop the motor control challenge further, the patient should not rest by placing the hand and knee to the floor after each holding repetition. Rather, after extending the normal bird dog position (a), the patient should sweep the floor with the hand and knee so that no weight is borne by either. The patient is shown beginning the sweep (b), at the innermost point of the sweep (c), and coming back out of the sweep (d, e). The patient then extends the active limbs back out into the bird dog position. This technique also allows muscles to reoxygenate with each sweep cycle (McGill, Hughson, and Parks, 2000). Finally, the raised hand and arm should be stiffened with cocontraction including the shoulder. With focus on the upper back, these muscles (thoracic longissimus and iliocostalis) achieve a higher level of contraction. The patient should continue the progression by pushing the heel away. Note how this facilitates high activation in the gluteal and low back muscles on the side of the elevated leg.


a
b
c
d


e

Special Conditions

Thoracic kyphosis. As some people age, they develop thoracic kyphosis—a flexed thoracic spine (see figure 10.9a). However, younger people also develop kyphosis through habitually poor posture, coupled with too much sitting during computer and cell phone use. Some athletes tend to become kyphotic, such as long-distance runners and some back-dominant athletes such as rowers.

The stretch described here was developed by the great Czech neurologist Karol Lewit and based on proprioceptive neuromuscular facilitation (PNF). The clinician is on one knee while the patient adopts a full kneel. The patient's fingers are interlaced behind his head and uses a hip hinge placing his elbows on the clinician's thigh. (see figure 10.9a, b). This is held for about 10 seconds and the 5 pound push is released and the clinician draws the elbow forward extending the thoracic spine (c). The patient can learn to do this on his own, replacing the clinician with a chair (d). Upon releasing the 5 pound elbow force, he draws his buttocks back to his heels.

Figure 10.9 (a) The clinician shows how the fingers are interlaced and placed behind the head and neck. (b) The patient places his elbows on the clinician's thigh while the clinician ensures the patient has a neutral lumbar spine. The patient then pushes down through the elbows with 5 pounds (2.3 kg) of force for about 10 seconds. (c) As the patient releases the effort, the clinician pulls the elbows forward, cuing the motion with the hand over the thoracic region. No force is applied with this hand. This is repeated for perhaps 3 or 4 cycles as long as the thoracic spine releases in each cycle. The clinician should help the patient in standing because he may feel unnerved with his newfound height increase. (d) The patient can be coached to do this on his own by replacing the clinician with a chair or similar object. Upon releasing the tension, the pelvis moves towards the feet to create the thoracic extension.

Arthritic stenosis. As many people age, particularly those who had careers characterized by lots of spine motion (e.g., plumbers, recreational soccer players), they tend to develop arthritic changes in the vertebrae. This narrows the canals for the spinal cord and roots. Longer-duration standing and walking usually causes motor loss. The usual approach is to teach these patients how to stay in spine flexion. We have very good success with those who have failed this approach by doing the opposite. More flexion in daily activity is often counterproductive because the posture results in more spine loading, not less. We provide spine extension drills (figure 10.10) and corrective interval walking programs (figures 10.11 and 10.12).

Figure 10.10 Extension drills may be simply prone lying (a pillow may be needed under the pelvis).

Figure 10.11 This drill prepares the patient with stenosis for walking by assisting him to achieve a more stress-free standing posture. (a , b) Arm walking starts with the patient placing one hand above the other and literally walking the hands up the wall. (c) Cuing the hips to move toward the wall helps the patient achieve the desired upright posture**.**

Figure 10.12 (a, b) The patient begins walking, and just as discomfort starts, the patient moves to the strategically placed park bench (or other such object). (c, d) Standing with the hips close to the bench, load is applied down the stiffened arms to add traction to a tolerably extended spine. This posture is held as long as it is comfortable but no longer than about 1 minute. Hopefully, symptoms are lessened or eliminated. Now the patient repeats the walk until the next cycle of pain begins. The patient repeats the training session each day, slowly adding more intervals. This is a technique to achieve pain-free walking in cumulative intervals.


b
c

Claudication. Claudication is often confused with stenosis; however, it is a symptom of peripheral arterial disease. In claudication, atherosclerotic plaque buildup narrows the arteries, starving the working leg muscles of oxygen, causing leg pain. Once again, a walking program is recommended (Hirsch et al., 2005) based on tolerable intervals (Brunelle and Mulgrew, 2011). Basically, walking is performed until pain becomes moderate. The patient sits until the symptoms resolve; then resumes walking. The goal is to continue this cycling for up to 60 minutes 3 to 5 times week.

Knee or hip replacement. People who have had knee or hip replacement have difficulty moving to the floor for exercise. Figure 10.13 shows a patient standing, braced on a table or countertop, performing the bird dog.

Figure 10.13 Those with knee and hip replacement can perform the bird dog while standing and braced on a countertop or table. Techniques similar to the floor versions of the exercise are used—for example, fist clenching, heel push, abdominal brace, hand and foot squares. The person must maintain good form with a neutral spine.

Overweight. Unfortunately, reality television shows in which overweight people are belittled and injured influence the public, but provide a poor example. These patients have no doubt been conditioned to fail by many previous weight loss attempts. They need a support structure to comply with the clinical exercise prescription. Success will depend first on their ability to curb their overeating of poor food choices. Then simple exercise progressions are commenced with good form (corrected walking and simple starting levels of the spine-sparing drills). Interestingly, I have not found very heavy people with spine instability because their girth is naturally stabilizing. Thus, we focus more on performing work to enhance endurance and strength in the patterns of movement noted earlier. Core work, one-legged exercises, and stretching are de-emphasized.

A Final Note

Paying attention to detail is the hallmark of the successful clinician–patient relationship. Pain triggers identified using the assessment protocol in chapter 9 must be eliminated. Then the therapeutic exercises appropriately progressed and regressed to match the current tolerance of the patient will establish a decline in symptoms. This will serve many patients well and end their days of pain. Those working with people who desire more athletic abilities will need to read on in the next chapter.

Chapter 11
Advanced Exercises

The beginner's program described in chapter 10 should be sufficient for daily spine health. Several situations, however, may call for further training:

- Certain occupational tasks with specific demands that require unique preparation and training
- Athletic endeavors that demand higher challenges of low back training (although this is achieved with much higher risk of tissue damage from overload)
- Some patients who, as they progress with the isometric stabilization exercises described in chapter 10, want to continue increasing the challenge

Athletic performance, or simply increasing ability, may be enhanced with specific exercises. However, performance can also be ruined by exercises that cause imbalances between performance variables, and injury. Joints need more resilience when called upon to bear more load. Speed must be developed along with an enhanced ability to steer the forces through the body linkage. But what are the variables that increase leg speed or the speed of a tennis racket? There are many, including enhanced core stiffness to increasing distal limb speed. Mobility at the distal joints needs tuning. Creating strength in pulses enhances limb speed, which implies that training the rate of muscle force development and relaxation is important (McGill et al., 2010). Studies linking specific fitness variables with performance can be surprising, such as those we have conducted with occupational groups (McGill, Frost, and Crosby, 2013; McGill et al., 2013) and sporting groups (e.g., McGill, Andersen, and Horne, 2012). Choosing the best exercise tool to achieve each performance while considering the person's current level of competency, injury history, and body type is not a trivial task; it requires a great degree of knowledge and skill.

We have quantified the spine loading and athleticism developed during strongman tasks (McGill, McDermott, and Fenwick, 2008), kettlebell swinging (McGill and Marshall, 2012), hula hooping (McGill and Andersen, 2015), powerlifting (Cholewicki, McGill, and Norman, 1991), and mixed martial arts (McGill et al., 2010), to name just a few. All of these approaches have a place but the best program for an individual is focused. Our case studies of the great athletes revealed that the best performers were rarely those who tested as the strongest. The best performers, rather, had superior technique. They listened to their bodies and preserved their joints. Many used quite novel techniques to enhance the full spectrum of motor skill and strength and speed variables in planned programs. These programs cleverly scheduled rest periods alongside enhanced restoration techniques. The successful athletes worked on the aspects that would create the largest enhancement in performance.

Finally, we are not born equal. Many studies have documented the genetically determined differences in response to training. There are both responders and nonresponders. A technique that enhances the squat performance in one person may have no effect on another. The second person using that technique has wasted effort that could have been directed in a more productive way. This is not to discourage program design for enhancing performance, but rather to illustrate that working with an athlete with back pain to become a champion requires every bit of knowledge I have. With each of my athlete consulting cases, I proceed realizing that I will need to follow principles, but the best approach is not always obvious.

This chapter briefly introduces a few examples of how to increase low back challenges safely, addresses specific worker and athletic concerns regarding advanced training, and provides some ideas about the next steps to take in the study of low back rehabilitation and training. The goal of this book is to help clinicians and people with back disorders reduce pain and enhance function. However, advanced training is a complex topic that cannot be adequately addressed in a single chapter. See Ultimate Back Fitness and Performance (McGill, 2014) for a detailed treatment of advanced exercise for the back.

Chapter 10 covers exercises that accomplish the goals of the first three stages of progressing exercise. This chapter introduces a few notions about stages 4 and 5. Following is a recap of the stages as introduced in chapter 8:

- Stage 1: Establish quality movement engrams (motion patterns, motor patterns, and corrective exercise).
    - Identify perturbed patterns and develop appropriate corrective

exercise.
    - Address basic movement patterns through to complex-activity patterns.
    - Address basic balance challenges through to complex and specific balance challenges.
- Stage 2: Build whole-body and joint stability (focus on spine stability here).
    - Build stability while sparing the joints.
    - Ensure sufficient stability commensurate with the demands of the task.
    - Apply these patterns to the performance of daily activities.
- Stage 3: Increase endurance.
    - Address basic endurance training to ensure the capacity needed for stabilization.
    - Address activity-specific endurance (duration, intensity).
    - Increase the ability to repeat pristine movement patterns without fatigue-induced compromise.
    - Build the base for eventual performance training (only in those with this goal).
- Stage 4: Build strength.
    - Spare the joints while maximizing the neuromuscular compartment challenge.
    - Progress to skill movements.
- Stage 5: Develop speed, power, and agility.
    - Develop ultimate performance based on the foundation laid in stages 1 through 4.
    - Focus on optimizing elastic energy storage and recovery.
    - Employ the techniques of superstiffness. Stiffen the core and unleash the athleticism of the hips, shoulders, and distal joints.

Safely Increasing Challenges

To optimize patient safety, you must be aware of issues associated with training on labile surfaces and with machines, and how to progress the big three exercises described in chapter 10 (curl-up, side bridge, and bird dog) to their highest level. Some guidance is provided here.

Labile Surfaces and Resistance Training Machines

Not all clinicians are aware of the factors they should take into account when deciding whether to prescribe exercise using labile surfaces or machines. Following are guidelines about both of these.

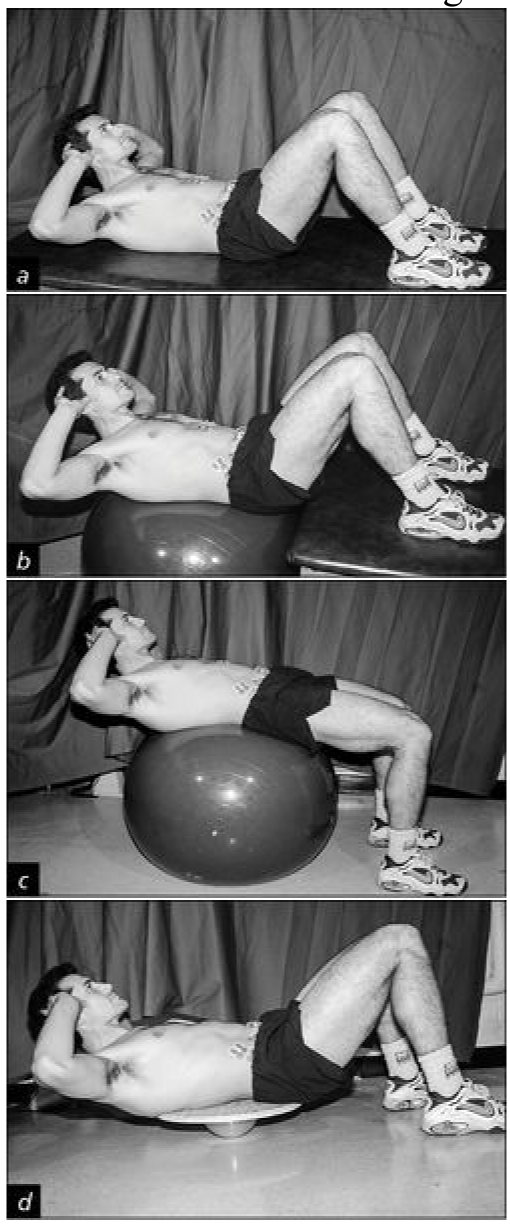
- Training with labile surfaces. Challenges to the spine during daily activity include maintaining stability during static, steady-state postures, unexpected loading events, and planned dynamic or ballistic movement. This has motivated some clinicians to recommend exercising on labile surfaces such as gym balls and suspension straps. Certainly, these labile surfaces challenge the motor system to meet the dynamic tasks of daily living or specific athletic activities and can be very helpful for advanced training. But might this type of training be of concern for some patients? Our recent quantification of elevated spine loads and muscle coactivation when performing a curl-up on labile surfaces (Vera-Garcia, Grenier, and McGill, 2000) suggests that the rehabilitation program should begin on stable surfaces. In this case, we assessed the simple curl-up for the effect of a labile surface on muscle activation patterns (see figure 11.1, a-d). Simply moving from a stable surface to a labile surface caused much more cocontraction, which in many cases virtually doubled the spine load (see figure 11.2). The practice of placing patients on labile surfaces early in the rehabilitative program can delay improvement by causing exacerbating spine loads. We therefore suggest beginning exercises on a stable surface and establishing a positive slope to improvement. Introduce labile surfaces judiciously only once the patient has achieved spine stability and sufficiently restored load-bearing capacity, and can tolerate additional compression. This principle can be extended to sitting. Sitting on a gym ball greatly elevates spine load through increased muscle coactivation. For this reason, those without back issues should avoid prolonged sitting on gym balls, and patients should use them only once they have achieved spine stability and increased load-bearing capacity.

There is a time and a place for labile surfaces. Pushing and pulling exercises need to be incorporated into any advanced back conditioning program. Comparing pushes and pulls on stable surfaces and with suspension straps documents the additional muscle challenges and corresponding spine and joint loads (McGill et al., in press a and b). More coordination is needed to control the direction of force application with labile surfaces, which is an essential skill. The cost is that labile surfaces limit the brute strength developed because the weakest component defines the balance of the other joint axes (which may also be an asset). Joint loading is also higher as a result of the extra cocontraction of many muscles needed for control.

Training with machines and equipment. Generally, the goal of establishing stabilizing motor patterns requires the person to support body weight and coordinate the stabilization of all joints involved in the task. In other words, it is a whole-body, even whole-person, endeavor. Many training machines, on the other hand, are made to isolate a specific joint. Because neither workers nor athletes perform their tasks in this artificially stabilized manner, these types of motor patterns may not be transferable and, worse, may cause inappropriate motor and motion patterns. The only instances in which machines that isolate joints can be helpful is when an injury to a specific body part requires its protection during rehabilitative training. Some incorrectly believe that isolating a joint somehow reduces the loading and thus reduces the risk. Consider, for example, the typical extensor benches or Roman chair, which generally impose twice the muscle loading than results from performing the bird dog, because both the right and left sides of the lumbar and the thoracic extensors are activated (see figure 11.3). Patients who must use machines should consider certain back-sparing techniques. Ensuring that all movement occurs about the hip joint, rather than the spine, would be an example of such a technique.

For the purposes of this discussion, cables to weight stacks are not considered machines because they do not isolate joint motion and add resistance to whole-body motion patterns. One exercise that enhances the ability to stabilize the spine is the cable pull-down. This exercise, when the handlebar is brought down to the chest (rather than the back, which is the traditional technique), challenges the latissimus dorsi and other stabilizers.

Figure 11.1 The simple curl-up was assessed for the effects of a labile surface on muscle activation patterns. The percentage of maximal voluntary contraction (%MVC) in the rectus abdominis and external and internal obliques caused by simply moving from (a) a stable surface to (b-d) varying types of labile surfaces is shown in figure 11.2.



Photos from Stuart McGill

Figure 11.2 The %MVC caused by each of the postures shown in figure 11.1. A curl-up with the body over a ball and the feet on the floor (figure 11.1c) virtually doubles the abdominal muscle activation of a curl-up on a stable surface (figure 11.1a) and, correspondingly, the spine load. Note that the %MVC required of the three muscles studied is also much higher in curl-ups with the body over a ball and the feet on a bench (figure 11.1b) and with the body on a wobble board (figure 11.1d) rather than on a stable surface. Clearly, a gym ball can be wonderful for advanced training but is contraindicated for many patients.

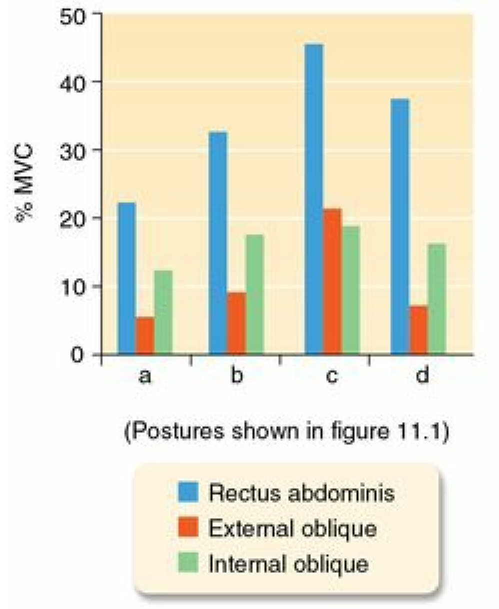


Figure 11.3 Typical back extensor devices impose generally twice the muscle loading of the bird dog because both the right and left sides of the lumbar and the thoracic extensors are activated. These may be necessary for some types of performance training, but they are ill-advised for most patients because of the spine compression and motion. Enhancing strength for people without pain would be wiser if they eliminated the spine movement when under load, and rotated about the hips.

Photos from Stuart McGill

Safely Progressing Back Exercises

The curl-ups, side bridges, and bird dogs pictured in chapter 10 can be made more challenging and yet still incorporate the safety features noted. After explaining these advanced forms of the big three exercises, I present an additional advanced back exercise.

Advanced Back Exercises

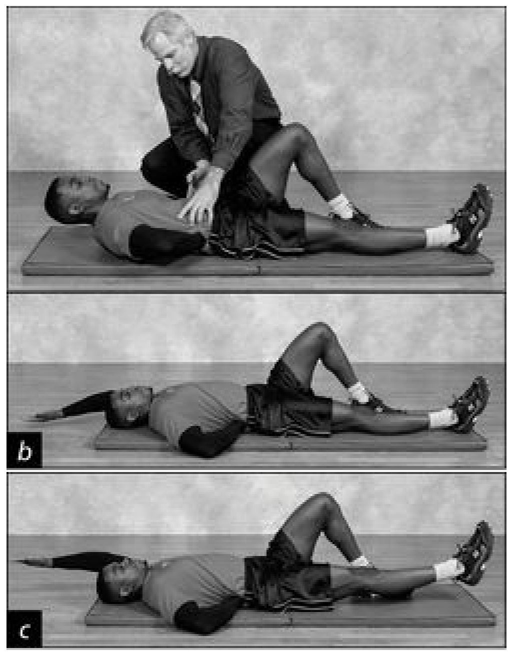
Once the patient has mastered the advanced curl-up, side bridge, and bird dog described in chapter 10, instruction to try the following may be appropriate.

Curl-Up, Highest Level

1. The patient braces the abdomen (if necessary, review the instructions).
2. The patient curls up against the brace, but not any higher than in other

forms of the exercise.

3. In the up position and while maintaining bracing, the patient takes a few deep breaths (a). This level of curl-up would be challenging for the toughest NFL linebacker!
4. The abdominal wall is cued for bracing and stiffening. Plyometrics may then be added in the dead bug position (b, c) with short-range but rapid hip and shoulder flexion. All motion must occur in the hip and shoulder joints, not in the braced spine.

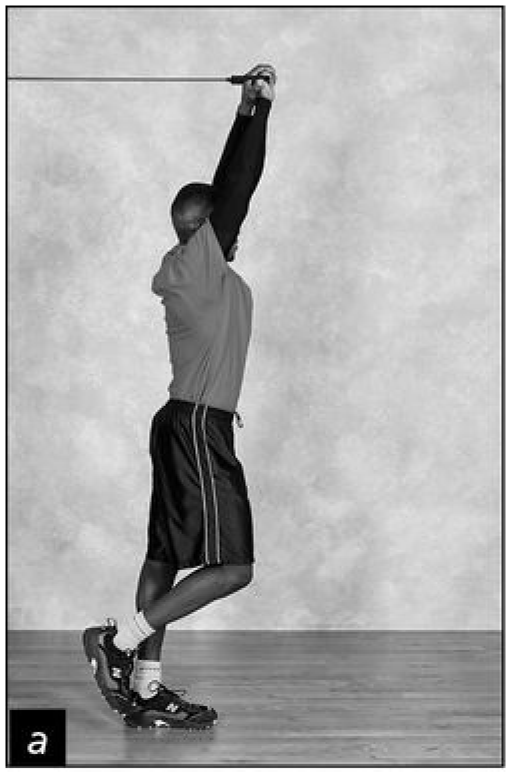


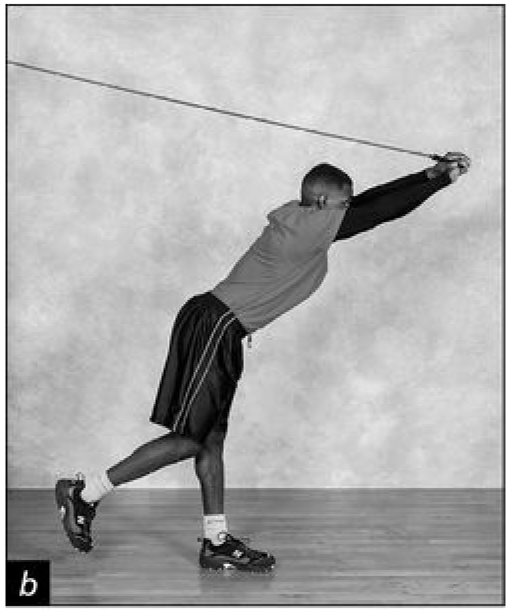
Side Bridge, Highest Level; Stir the Pot; and Overhead Cable Pull

The side bridge can be made more challenging by incorporating the roll described in chapter 10, while also dynamically contracting and relaxing the abdominal wall while sustaining the position.

Stir the pot is an advanced core exercise for those who desire a longer endurance challenge. (a, b) Correct and (c) incorrect techniques (motion in the spine rather than containing motion about the shoulder joints) are shown here.

The patient or athlete can then begin abdominal work in a standing posture. A family of exercises that very cleverly trains the entire anterior chain comprises the overhead cable pull. Here the motion is focused about the hips; there is no spine motion (see photos).





The overhead cable pull is considered an athletic progression for abdominal exercise (a). However, it is more than simply an abdominal exercises because it enhances stiffness, strength, and control of the entire anterior chain. Note that all motion is about the hips (b).

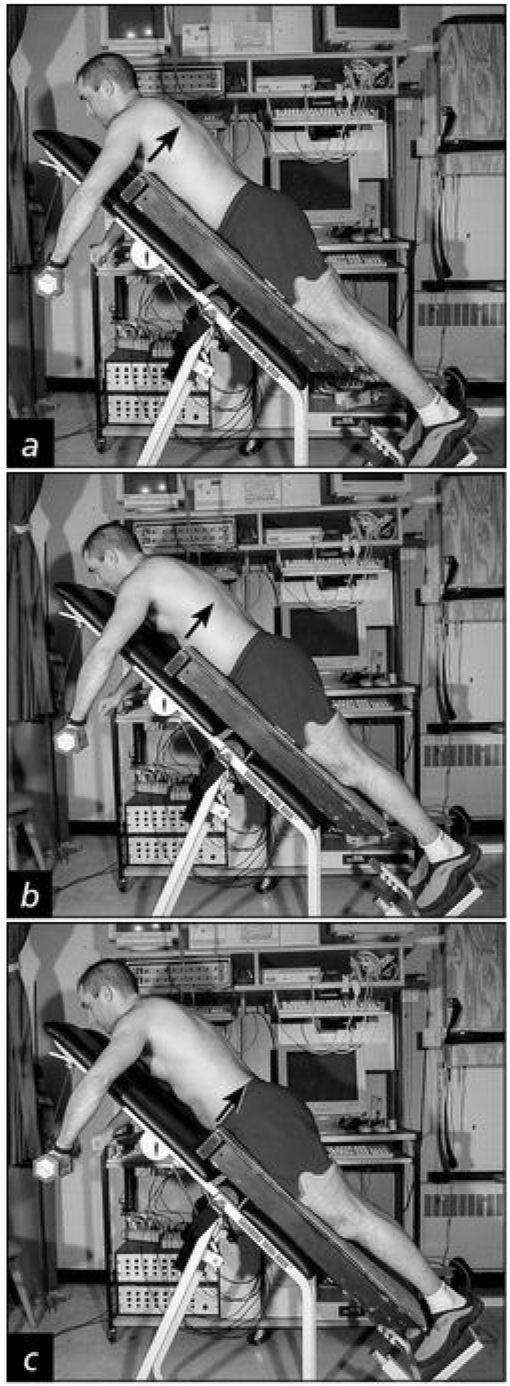
Bird Dog, Highest Level

The bird dog is more demanding when the patient consciously stretches the hand out forward and the foot out behind—all the while ensuring that spine motion (particularly bending) does not take place. Enhancing the back and gluteal muscle is achieved by pushing the heel away and making a fist on the elevated limb side. **To enhance certain forms of performance** and if the patient has no exacerbation of symptoms, small wrist and ankle weights may be allowed. Plyometrics are added as the hand and foot draw opposing squares, as follows: The person begins in the starting position, and then moves the hand and the foot down and away from the body, and then downward. Just as the hand and foot are brought toward the body midline, the torso is consciously braced and a ballistic short-range extensor contraction occurs at the shoulder and hip. Once again, no motion must occur in the spine.


Educating All Back Extensor Muscle Motor Units

When searching for ways to obtain maximal myoelectric activity from the back extensor muscles, we discovered that isometric back extensor exercises do not recruit the full pool of motor units. Many more motor units fire with some extensor motion. As a result of this discovery, this back extensor exercise, which builds strength and endurance and helps to hypertrophy the muscles, was developed. It is used by some of the strongest athletes in the world to educate the motor units in the back musculature to fully recruit and fire.

While the athlete is lying prone on a bench with the torso supported on a movable stiff pad, place a weight in one hand. The edge of the pad is under the mid rib cage. The cantilevered portion of the spine is slightly flexed and then extended, combined with some slight twist back to neutral (a). The object is not to actually twist but to focus activation on one side of the extensors—to use mental imagery (chapter 10) to assist in activating the maximal number of available motor units. The spine never extends past neutral. After a set, place the weight in the athlete's other hand and have the athlete repeat the exercise. Then move the pad downward (perhaps about the level of the navel) so that a greater portion of the torso is cantilevered (b). Have the athlete repeat the sets. Move the pad farther down the athlete's body (perhaps to about the pelvis), leaving more of the torso cantilevered, and have the athlete repeat the entire process (c). The sharp edge of the pad is better for imaging the edge and focusing on the corresponding section of muscle.



Photos from Stuart McGill

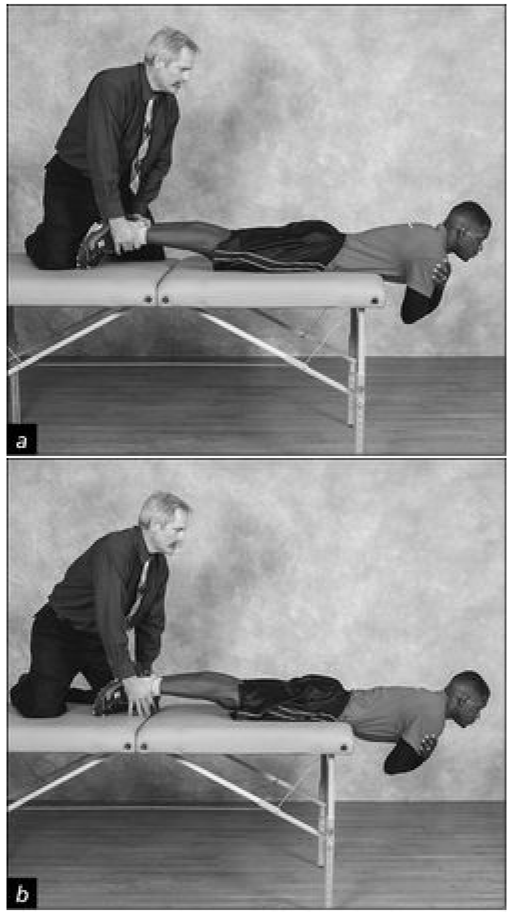
Squat With Overhead Resistance

Moving to a standing posture, the progression continues with a wonderful exercise that challenges the back and hip extensor mechanism. The squat with overhead, self-generated hand resistance grooves a squat pattern, with very robust recruitment of back and shoulder musculature for stability and stiffness.

(a) The squat with overhead resistance requires one hand to push into the other causing contraction throughout the entire upper back, shoulders, and chest. (b) The squat uses the hip hinge, and the hips track along a 45° path (no spine motion).

Back Extensor Exercise Using a Bench or Table

Another variation of the exercise for educating all back extensor muscle motor units involves performing the extension motion over a table edge. The feet are secured, and the torso is progressively moved farther over the edge with each repetition (a, b).


a
b

Occupational and Athletic Work Hardening

Certain workers and athletes encounter demands that require training that we would never recommend to patients unless absolutely necessary. Anyone considering going beyond the exercises described previously should be fully aware of the increased risks of back reinjury that the exercises in this section pose. These exercises, which were developed for workers or occupational athletes, should be restricted to the groups they were developed for because they present a level of risk that is unacceptable for any but those willing to assume it for the sake of athletic or work performance.

Once again, choosing the appropriate challenge involves a blend of clinical art and science. Too little load will not produce the training adaptation, and too much will break tissue down. Listen to the body, and provide adequate rest.

Low Back Exercises for High-Performance Workers or Athletes

In general, we stage training of high-performance workers and athletes with the same steps used in our rehabilitation approach, except that two more steps are added. We begin in stage 1 by identifying the essential motions and establishing appropriate motion and motor patterns. Stage 2 is directed toward ensuring joint and whole-body stabilizing patterns. Stage 3 is to develop muscle endurance around these patterns. Stage 4 is directed to enhancing strength. And, finally, stage 5 is to establish power. Unfortunately, too many exceptional athletes are trained, or given a rehabilitation program based on strength and power, without an adequate foundation of stabilizing motion and motor patterns. They end up with back problems and are referred to us.

In this section we briefly consider exercises that may be helpful to either workers with demanding jobs or athletes. The requirements of the activities are not excessive for those who have adequately prepared by mastering all of the exercises previously discussed in the text. Specifically, these people should have mastered spine position awareness and be able to produce low back stabilizing patterns, but may need more work to move through the ranges of motion specific to their tasks. In addition, they may need more strength and endurance.

Qualifying the Worker or Athlete

The clinical decisions involved in staging a worker or athlete through the progression of challenge is an art that can be assisted with scientific data. Those who are looking for a set recipe, however, will fail at creating a successful program for an individual. Generally, we blend the worker's or athlete's current exercise status and history of injury with our own knowledge of spine tissue loads that result from various activities and our knowledge of injury mechanisms to qualify someone for a specific exercise progression. We then put our educated guess to the test and monitor the person's progress to ensure the maintenance of a positive slope of improvement in both symptoms and function.

Lumbar Stability With Elevated Simultaneous Physiological Work Rates

Although some people can maintain spine stability over all sorts of activities, including those that require the stabilizing musculature to assist with physiological challenges such as elevated breathing, others cannot (McGill, Sharratt, and Seguin, 1995). We have noted a compromise in the ability to stabilize more often postinjury (McGill et al., 2003), although we have also detected that compromise in so-called virgin backs. Interestingly, tall athletes tend to be poorer in their ability to cocontract the abdominal muscles to ensure sufficient stability during high work rates and highly challenged breathing than their shorter counterparts. This perception appears to be supported by the most recent evidence showing that taller workers have a greater likelihood of having perturbed motor patterns while breathing heavily and holding spine loads (McGill et al., 2003).

Consider the warehouse worker, firefighter, or football player who must work at a high physiological rate that results in deep and elevated lung ventilation. The inability to maintain constant cocontraction in the abdominal wall (i.e., the muscles tend to relax during deep inhalation) is an indicator of compromised spine stability, particularly when heavy external loads that demand a stable spine are required. These people must develop stabilizing motor patterns that transfer to all activities. Later in this section is an exercise we devised to train these people to maintain abdominal bracing while breathing heavily.

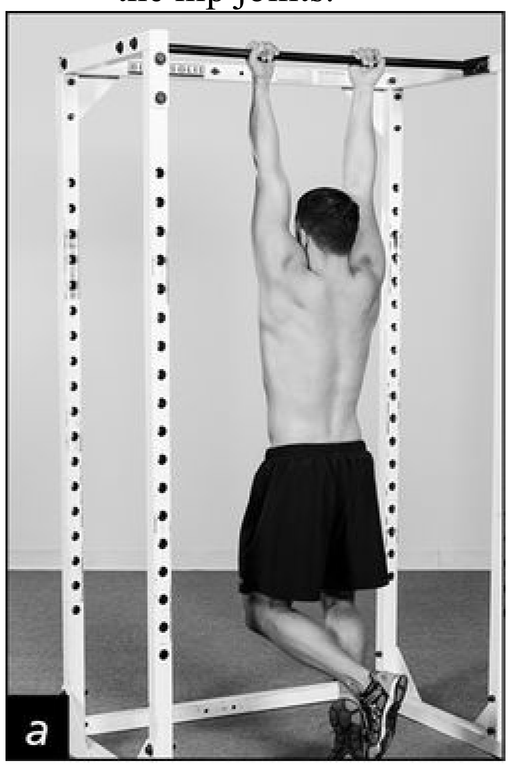
Push, Pull, Lift, Squat, Lunge, Carry, and Torsional Capability

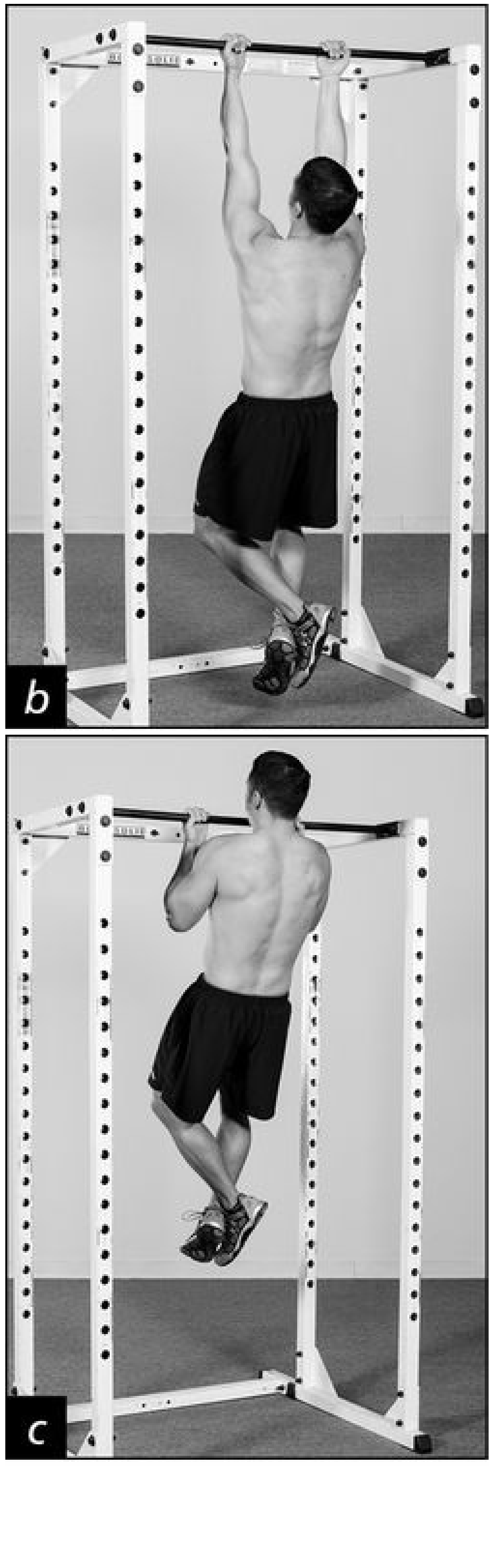
Progressions in these movement patterns are essential for creating a balanced foundation for enhanced pain-free performance. Pulls usually begin with pull-ups. Emphasis is placed on a full and concentrated hand grip, centrated scapulae, a prestiffened core, and so on. Better form, with fewer repetitions but more effort within the few repetitions, characterizes the beginning of each exercise progression. Push patterns generally begin with push-ups. For example, the bench press would never be recommended to someone who could not perform 20 fast and pristine push-ups. Lifts begin with the shortstop squat of weight elevated on blocks. One-legged kettlebell lifts may be added to the progression to challenge balance and precise spine control. Here the core is stiffened, grip effort is emphasized, and all motion occurs about the stance-leg hip. Kettlebell swings may be added for those with sufficient shear stability or stiffness.

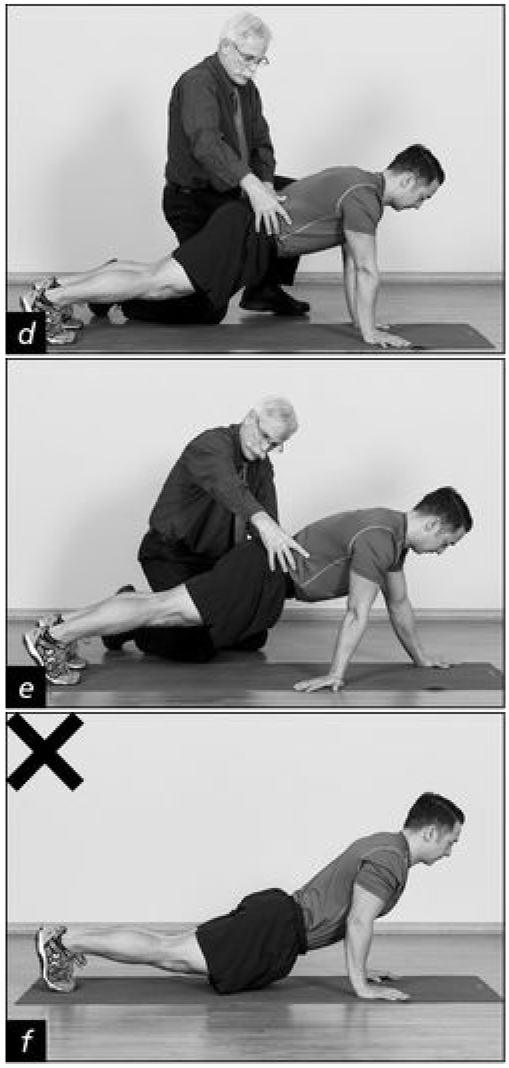
Lunges may incorporate hand loads, but the person is screened for a sufficiently tight pelvic ring (e.g., a history of sacroiliac pain would disqualify the person). Carries may begin with light suitcase carries and progress to farmer’s walks with appropriate coaching of core-stiffening techniques. Torsional exercises always begin with the ability to stop a twist. Athletes such as golfers, for example, greatly benefit from stop-twist exercises in both directions to enhance control and provide protection during the swing. Recall that generating torque about the twist axis imposes approximately four times the compression on the spine as an equal torque about the flexion–extension axis. This is why it is unwise to train for torsion generation until the back is quite robust and healthy.

As speed is added to the exercises, the ability to stop movement is as important as creating the movement. A balanced and successful program will address these considerations.

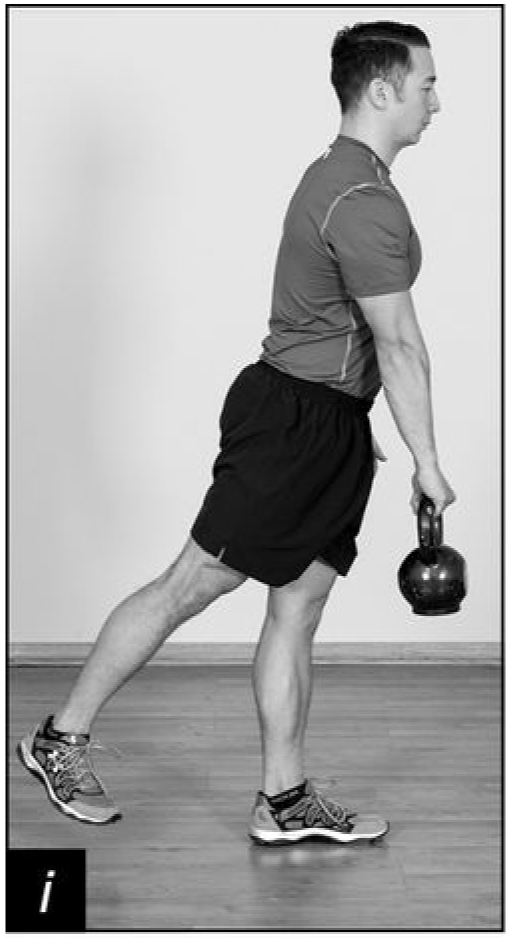
Figure 11.4 (a-c) Pull-ups are performed by first centrating the scapulae and enhancing the grip effort and core stiffness. Then the person pulses to accelerate the pull. (d-f) Conventional push-ups are performed with a neutral spine and a stiffened torso. The person pulses to accelerate the move. The staggered hand placement challenges control as the person ensures the torso does not laterally bend. Poor form is shown in photo f. (g-i) A one-legged good morning version of this exercise can be included for those interested in more hamstring challenge. The hands alternate between the right in front and the left in front. (j-l) Carrys begin in (j) suitcase style then progress to (k) racked and finally to (l) bottoms-up. (m-n) Lunges follow all of the spine-sparing principles by maintaining a neutral spine and motion occurring about the hip joints.






d
e
f


g
h


i


j

k


l
m

Special Spine Exercises for Workers and Athletes

Although the exercises in this section have been designed to minimize injury risk, you must be careful to prescribe them only to those who can already safely perform the advanced levels of the big three exercises presented in chapter 10.

Establishing Spine Stability When Breathing Is Elevated

Some athletes and workers risk spine stability only when they are breathing heavily. To train simultaneous patterns to ensure stability during heavy breathing, we have developed the following approach:

1. Ride an exercise bike at an intensity to elevate ventilation.
2. Then immediately dismount and adopt the side bridge posture on the floor.

In this position the stabilizing musculature must remain isometrically contracted; otherwise, the bridge posture is lost. Heavy ventilation is also required, however, to groove the motor patterns that coordinate diaphragm contraction and the other thoracic muscles involved in efficient lung function. The curl-up and bird dog postures are also used after vigorous stationary biking to groove stable patterning in all of the supporting muscles. This establishes spine stabilization patterns in workers and athletes alike.

Suggestion for Those Whose Back Is Worse With Sitting and Then Have Trouble Standing

To combat the cumulative stresses of sitting, we often recommend walking over rough ground for an hour or two while wearing a backpack with a 5 to 10 kg (11 to 22 lb) load placed low in the pack.

Amazingly (to some), many patients report that this works wonders. The typical forward-flexed antalgic posture requires the back extensors to be active (which imposes a substantial load penalty on a flexed spine). The backpack acts as a counterweight to extend the spine and bring the torso upright. The back extensors are no longer needed to contract, effectively removing their contribution to spine load. Wearing the backpack reduces spine loading!

Low Back Exercises Only for Athletes

Specific athletic objectives require specific training techniques. As noted earlier, however, too many patients make the mistake of looking to athletes for training exercises under the misconception that the same approach will help their own backs. This critically important notion must be emphasized again: Athletic exercises are not for enhancing back health in patients. In addition, too many athletes use bodybuilding principles for building mass. These exercises must not be emulated by patients. The following exercise examples are reserved for athletes only.

Training-Specific Athletic Maneuvers

Trunk torsional machines and resisted twisting motions are reserved for those who want to excel at very special tasks such as Olympic discus throwing. (Some track athletes with whom I have been associated clinically do not perform these exercises because they exacerbate their back symptoms.) Otherwise, the torsional moment capabilities are developed with the spine in a neutral position, with a fully braced torso musculature, but with a torsional moment challenge. The neutral position is the most robust posture for withstanding elevated spine loads; it is also the spine posture that is transferable to other activities that require torsional moments with the least risk of spine damage.

Sprinting and other power running events such as those performed by football players should be addressed here. Top-end speed for most sprinters is usually limited by the recovery of the leg in flexion (hip flexion), not a lack of hip extension power. Thus, many train the psoas according to the power philosophy—and end up with painful backs. It is critical for these athletes to up-train spine stability to support the added power and spine load during the rigorous hip flexor training required for sprinting events.

All sorts of exercises and special equipment have been developed for performance training. However, very few performance approaches have been documented with substantial scientific foundation to warrant discussion in a book such as this. However, one exception is the book Supertraining, in which the late Dr. Mel Siff synthesized and reviewed an impressive number of studies (Siff, 2003). For example, Dr. Siff made a case for avoiding the development of motions that are guided predominantly by machines, and instead advocated for the use of cables and free weights, as well as unloaded speed training, with data-based argument. Further, substantiated principles of functional anatomy, physiology, biomechanics, and motor aspects such as facilitation were used to justify the approaches documented in Supertraining to enhance strength, power, speed, and muscle endurance. These performance objectives cannot exist without the foundation of stability, mobility, and the additional principles documented in this book. Although there is no question that these principles and exercises are excellent for performance, there is a high resultant spine load. Some backs simply will not tolerate some of these higher-demand exercises—not everyone can tolerate the rigors of training needed to be a champion, regardless of the soundness of the scientific technique. The exercise variations are endless and are outside of the scope of this book.

Finally, with respect to designing workouts, the big three exercises can be performed at the beginning of the workout to establish the stable motor patterns for the rest of the training session. These exercises also enhance core stiffness that lasts for a period of time (Lee and McGill, 2015) to enhance both safety and performance. Some trainers also like to finish the session with these exercises if they desire to take the exertions to fatigue.

Training to Squat and Power Clean

Squats and power cleans are good exercises for developing power for athletic performance, but they have limitations. Many top athletes are referred to us who have enormous sagittal plane strength, yet show great weakness in nonsagittal strength. They have overemphasized the Olympic lifts for power without training power for lateral motion, turns, and so on. Furthermore, these lifts create a lot of damaged backs. Many younger athletes do not have the hip structure to begin the pull with the hip flexion required to have a neutral spine at the beginning of the pull. Serious squat training for us always includes one-legged squats, the bowler's squat, stiff-legged sled drags, hip airplanes, and many more. This is a very involved topic that is discussed in more detail in Ultimate Back Fitness and Performance (McGill, 2014).

The instructions for performing squats and power cleans from our perspective are very simple: If a perfect starting position cannot be obtained, other means are needed. Many great athletes cannot do these lifts. Never sacrifice form for lifting more weight. Year after year I give this advice to young athletes, and year after year a substantial proportion of them ruin their backs by not heeding this simple guideline. As the old saying goes, It's amazing how much your parents learned as you grew older.

Very few people in North America can perform traditional squats and power cleans well, lifting the bar off the floor with a low risk of injury. The Eastern Europeans are technical masters, and the technique is well covered in McGill (2014). It is interesting to see the emphasis placed on establishing the correct motion and motor patterns long before substantial weight is attempted. Young Eastern European athletes spend years developing the form by lifting broomsticks. Only when form is perfect is strength increased and weight added to the bar. Generally, preserving the neutral lumbar spine will solve many of the safety issues, but this depends on the ability to take the shoulders and hips to extremes in the range of motion.

Some have found that placing the weight bar on blocks to raise the starting height improves the utility of this exercise for athletes other than competitive weightlifters. This way a lifter can accomplish a fast lift without the larger back loads associated with the initial crouched posture needed to pull from the floor. In this power exercise, speed is important; participants are advised to train slow to be slow, train fast to be fast. If you are interested in these exercises, the actual weight kinematics for the full power clean and the coordination of the leg muscles, torso muscles, and those involved in the shoulder pull can be found in extensive detail in McGill (2014).

Performance exercise is so much more than lifting weights. In many cases the way the training programs are organized causes detriments to performance. For example, one high-performance athlete came for a consult wondering why, after training the squat with a bar on his back, he was able to squat more load but his vertical jump—which had been his original objective for training—had decreased. I had to show him that he had trained himself to be slow! Clearly, his objectives were not matched to the training approach.

If the objective is speed, the person should not endurance train or train slowly. Speed comes from cyclic ballistic contraction and relaxation. Thus, many athletes need to train speed of relaxation more than speed of contraction. Performance enhancements come from a refinement of the balance of qualities. A strong person who lacks balance and falls over when performing a one-legged squat may be fine in a weight room but a disaster on the football field. Strength, speed, and power all demand the foundation of appropriate motion and motor patterns, balance, and regional range of motion within the three-dimensional context of athletic motion. Training to achieve optimal performance is the topic of Ultimate Back Fitness and Performance (McGill, 2014), which is a companion text to this one for the real expert.

A Final Note

Rehabilitation approaches are continuing to embrace techniques that consider notions of torso stability and components involving posture, motor patterning, and appropriate progressive challenge. Many groups continue to work to do the following:

- Understand the contributions to stability of various components of the anatomy at particular joints—and the ideal ways to enhance their contributions.
- Understand the magnitudes and patterns of muscle activation required to achieve sufficient stability while sparing the joints.
- Identify the best methods to reeducate faulty motor control systems to both achieve sufficient stability and reduce the risk of inappropriate motor patterns occurring in the future.
- Develop motor patterns for optimal performance in athletes.

Collegial efforts between scientists and clinicians continue to develop the scientific foundation to justify better low back injury prevention and rehabilitation approaches. Much remains to be done.

Epilogue

Now you have another perspective to employ in your efforts to prevent and rehabilitate back disorders. I hope that from now on, when you read that painful backs just happen, or that they cannot be diagnosed to guide therapy, or that they are solely the function of psychological factors or the compensation system, you will pause and consider that you are reading the musings of those who have reached the limit of their underdeveloped expertise.

Not that anyone has all the answers. With each experiment we perform, we may obtain some new insight, but generally we are confronted with many more questions. So, with each experiment we become relatively more aware of what we don’t know.

Consider the viewpoint of this text and blend it with your own clinical wisdom and experiences. The very best clinicians with whom I have had the pleasure of working had wonderful clinical skills and insights, but also a solid scientific foundation.

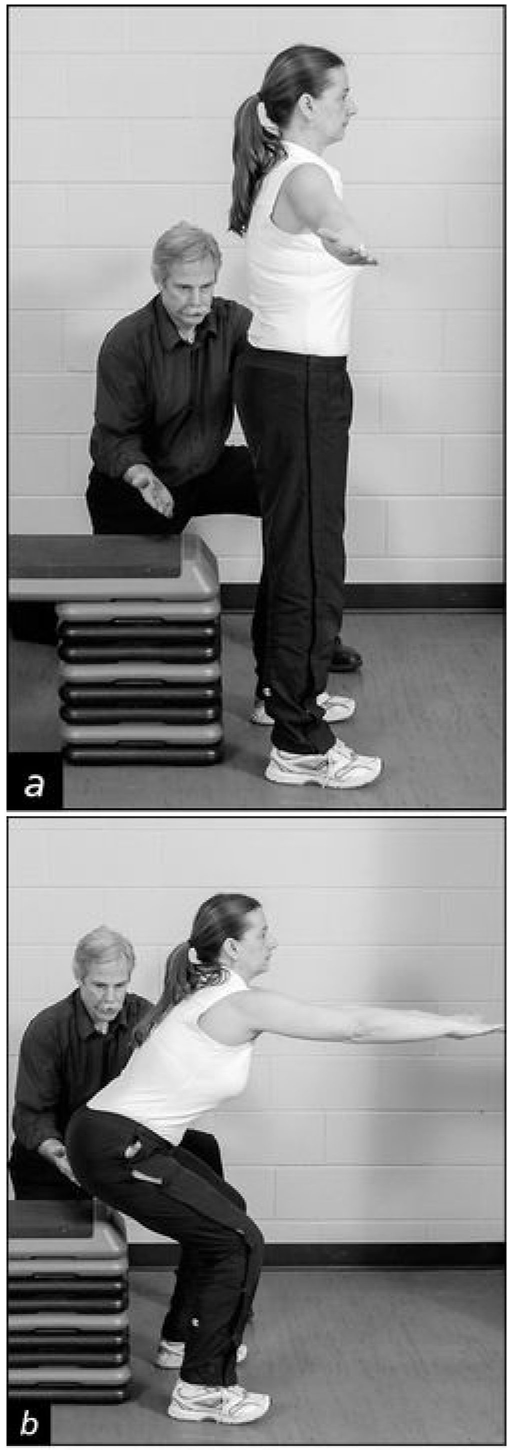
To the clinicians—I wish the confidence to continue with what you know works and the inspiration and leadership to try new approaches when things are problematic. To the students of clinical disciplines and those involved in the continuing development of the scientific foundation—there are so many more exciting experiments to perform on this fascinating part of our human anatomy, the low back. May we all enjoy the continuing journey.

Handouts for Patients or Clients

With great reservation I present these handouts of cues to use when prescribing exercises for patients. As I have mentioned, handing out pamphlets showing back exercises ensures only mediocre practice. To avoid such practice and to use these handouts effectively, you must select appropriately designed exercises for a patient only after you have carefully assessed that patient. Further, you must fine-tune each exercise for the patient by working with him or her to adjust posture, motion, and motor patterns to eliminate pain. This tuning is essential for making the exercise most tolerable and beneficial. During the workshopping of each exercise, you identify problems or limitations and follow up by designing and trying appropriate corrections with the patient. You may reproduce each handout for use with your clients, but you may also access blank copies of these handouts in the web resource available at www.HumanKinetics.com/LowBackDisorders. The handouts in the web resource include fields in which you may type customized cues and steps for each patient as well as his or her unique dosage. Not everyone will do every exercise. Exercises progress at various rates. Add new exercises only one at a time.

Success at this stage will elevate you to the elite level of practice—but you need to consider all principles in this book. First, work to remove all exacerbators of the pain. Then consider and evaluate the patient's tolerance and capabilities and stay within those parameters, avoid injury mechanisms, and correct perturbed patterns.

Potty Squat



Example Cues

1. Stand with feet shoulder-width apart, arms to sides, and hands supinated.
2. Sniff air and appropriately stiffen the torso.
3. Squat the pelvis back, controlling the knees so they do not drift forward beyond the toes. The arms move forward during this descent and the hands roll over into pronation.
4. To begin the ascent, “spread the floor,” cueing the gluteal muscles.
5. Think of pulling the hips forward to encourage the hip hinge.
6. During the ascent, extend the arms while supinating the hands.

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

Flossing, Beginner

Example Cues

1. If a bulging disc is the cause of radiating neural symptoms, begin with the appropriate posture to reduce the bulge.
2. Find a sitting posture that minimizes pain and maximizes the reflexes.
3. Floss one leg and the head and neck 10 cycles, never pushing to the range that triggers pain or symptoms.
4. Repeat with the other leg and assess reaction immediately after the

session and the following day.

5. If symptoms are not worse, continue the 10 reps per leg 2 or 3 times per day.
6. If symptoms worsen, stop immediately.

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

Cat–Camel

Example Cues

1. Separate knees to free up the hips.
2. Cycle between flexion and extension of the entire spine (cervical, thoracic, lumbar) no more than 8 reps.
3. Do not push the end range because this is not a stretch. It's the motion that is therapeutic.
4. If you wish, add lateral bending motion cycles.

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

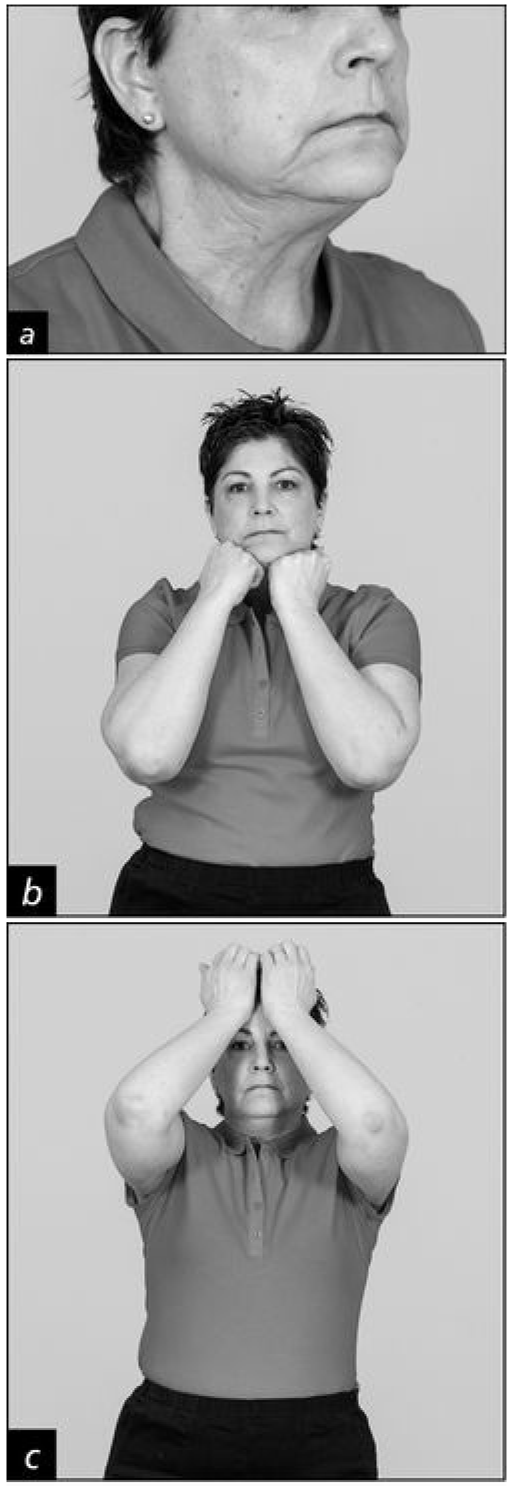
Curl-Up, Beginner

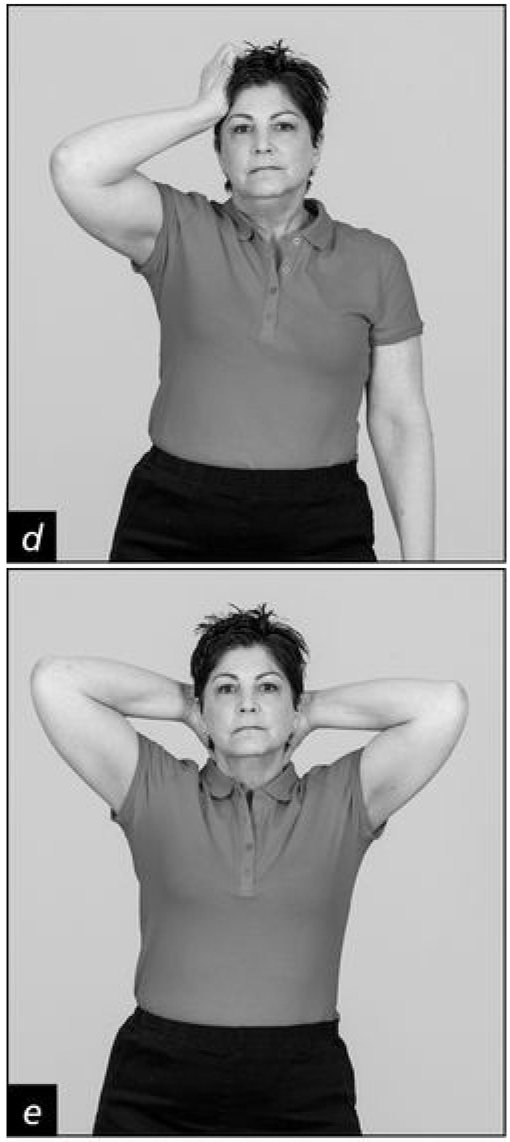
Example Cue

Mildly contract the abdominal muscles and the lifting effort to ensure there is no pain.

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

Isometric Exercises for the Neck





Example Cues

1. Position the head and neck in a neutral posture.
2. Maintaining this posture, bring the fists underneath the chin.
3. Push tongue to roof of mouth behind front teeth.
4. Push up with the fists, matching the resistance with the neck muscles; do not allow motion.
5. Note that just the exercise shown in a may be sufficient.

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

Curl-Up, Intermediate

Example Cues

1. Prebrace the abdominal muscles.
2. Lift elbows.
3. Raise head and shoulders slightly.
4. Hold 8 seconds (2 breaths).
5. Repeat 6 times (set 1), rest 20 seconds, repeat 4 reps (set 2), rest, repeat 2 reps (set 3).

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

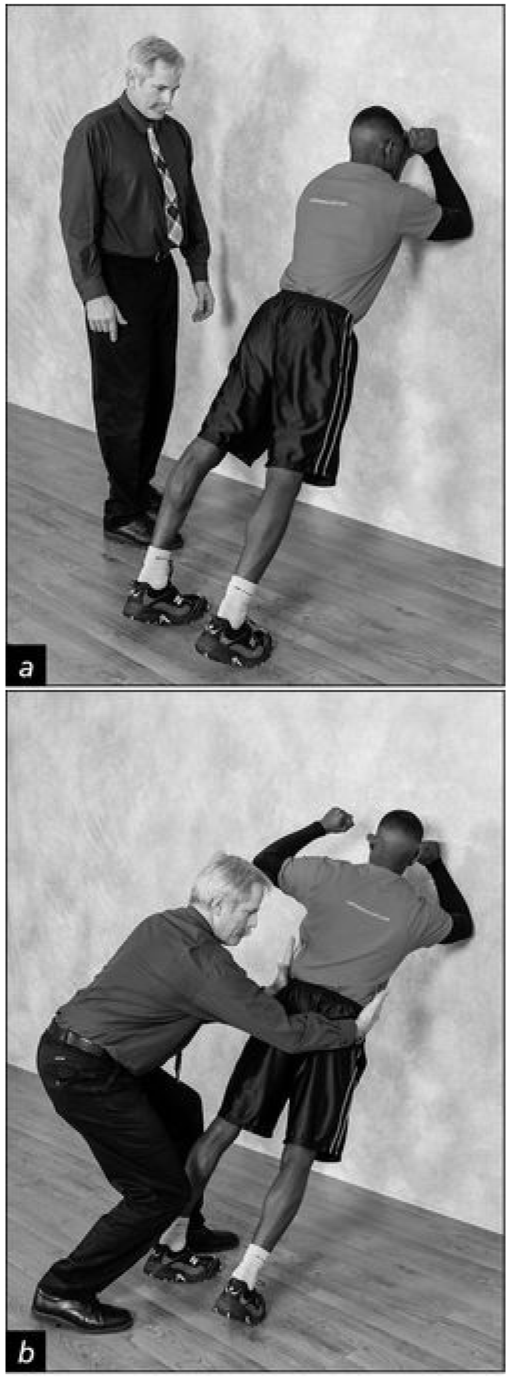
Curl-Up, Advanced

Example Cues

1. Push tongue to roof of mouth.
2. Add full-tide breathing while maintaining the appropriate abdominal brace.

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

Remedial Side Bridge for Deconditioned, Chronic Patient



a

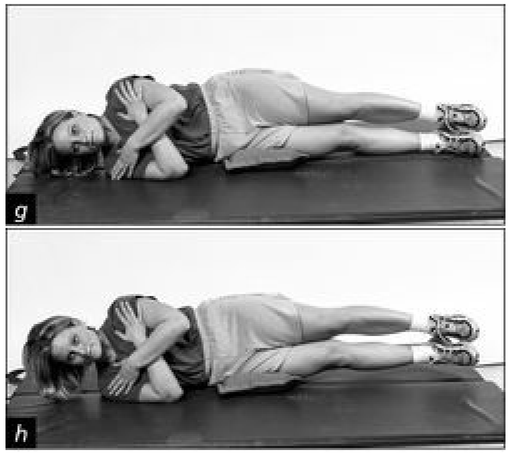
b

Example Cues

1. The challenge of effort and pain control begins with adjusting the distance from the feet to the wall.
2. Keep the spine aligned in the neutral curves but allow slight variations to reduce pain.

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

Remedial Side Bridge for Painful Shoulders 1

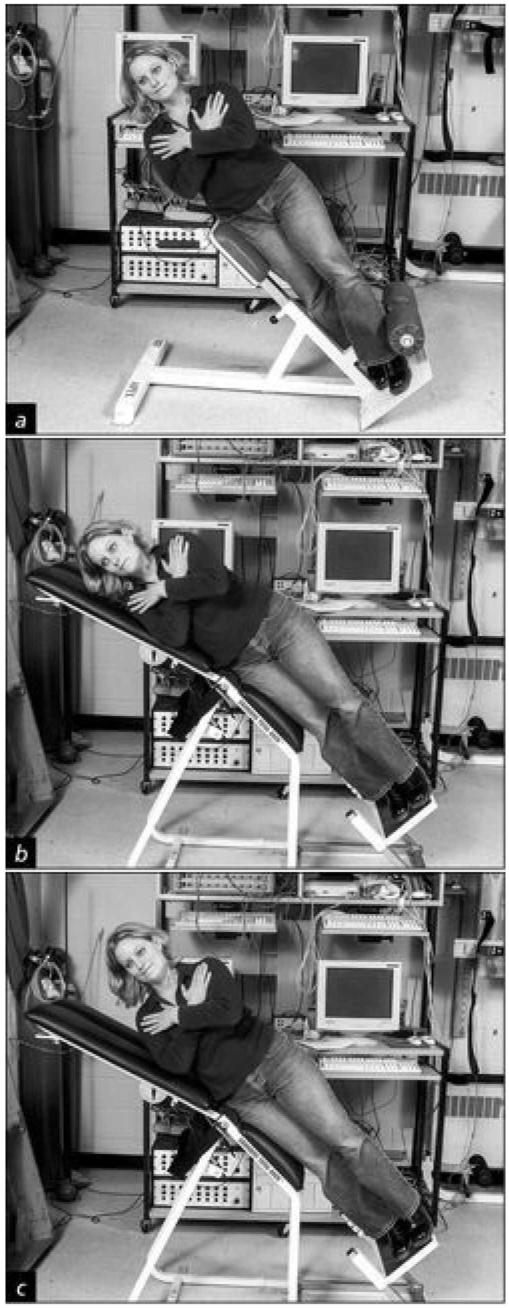


Example Cues

1. Adjusting a folded towel under the hip or between the waist and floor may increase the pain tolerance.
2. Lift the legs from the floor and no further.
3. Prebrace the torso appropriately.

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

Remedial Side Bridge for Painful Shoulders 2



Follow the same cues as other versions of the remedial side bridge.

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

Side Bridge, Beginner

Example Cues

1. Assume a neutral spine in the starting posture by adjusting the pelvis in the frontal plane.
2. To pull up into the bridge posture, extend through the hips in a squat motion.
3. Pull down with the hand, cupping the shoulder and pulling the elbow across the chest.
4. Tune the stiffening of the torso with mental focus on the latissimus dorsi and abdominal wall.

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

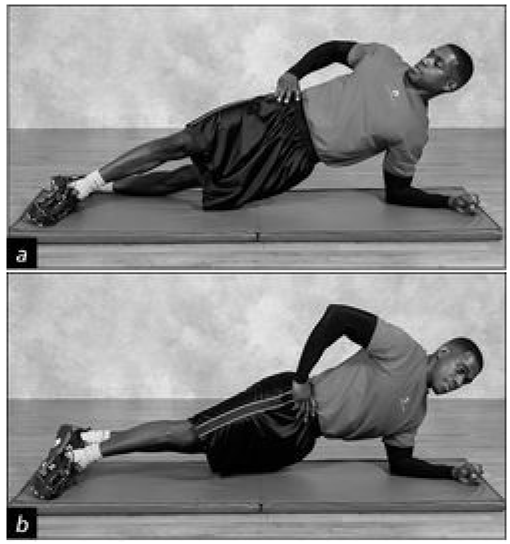
Side Bridge, Intermediate

Example Cue

Think about neutral spine postures: “Pull the hips through” is the most common cue.

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

Side Bridge, Intermediate Variation

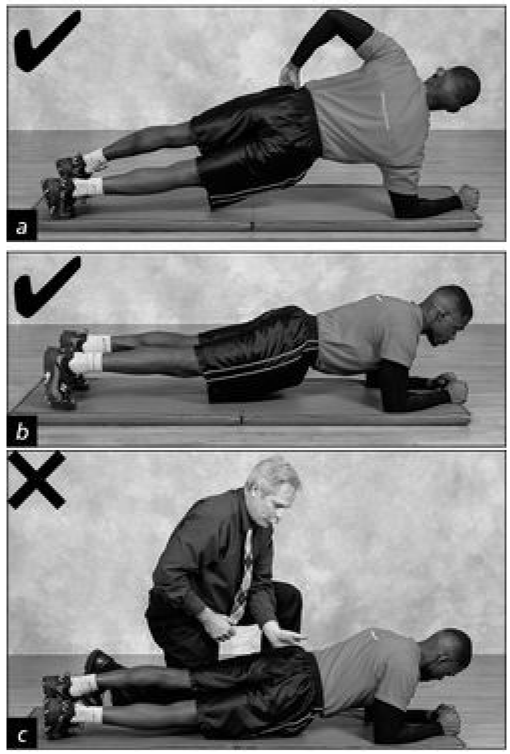


Example Cues

1. Lock the rib cage to the pelvis and focus on the latissimus dorsi initiating the turn rather than the spine twisting the muscles.
2. Roll back and forth over the elbow to train the multiple neuromuscular compartments in the abdominal wall.

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

Side Bridge, Advanced

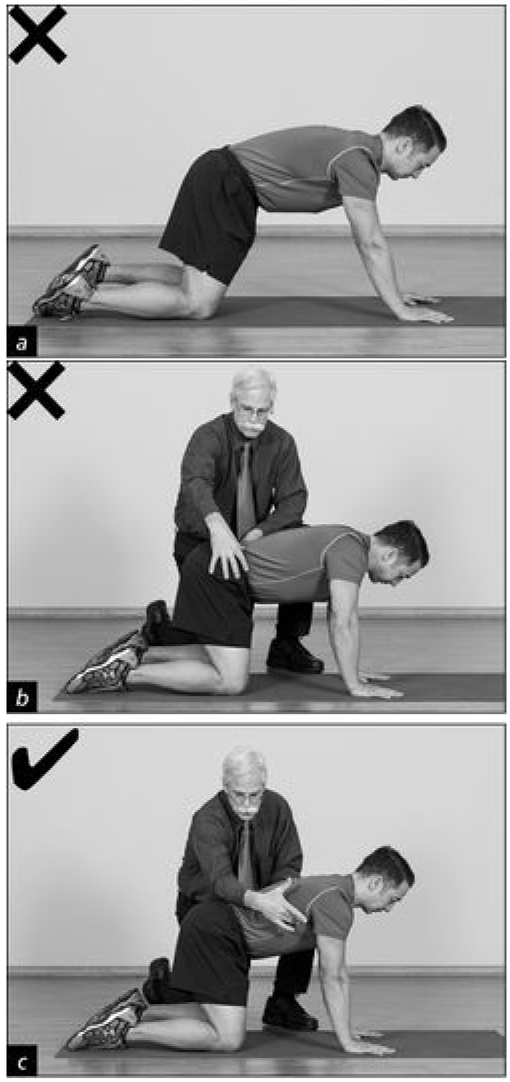


Example Cues

1. Use all previous cues plus focus more on locking the rib cage to the pelvis.
2. Note that the spine twist in c is poor form.

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

Bird Dog, Remedial



Example Cues

1. Tune the initial spine curve to eliminate pain triggers.
2. Poor starting posture (a) is corrected (b, c).

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

Bird Dog, Beginner

Focus on modifying the posture and motion to eliminate pain.
Example Cues

1. Starting posture: Restore a neutral curve to the spine.
2. Position the knees under the hips and the hands under the shoulders.
3. Tune the level of bracing to reduce or eliminate pain.
4. Lift just one hand.

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

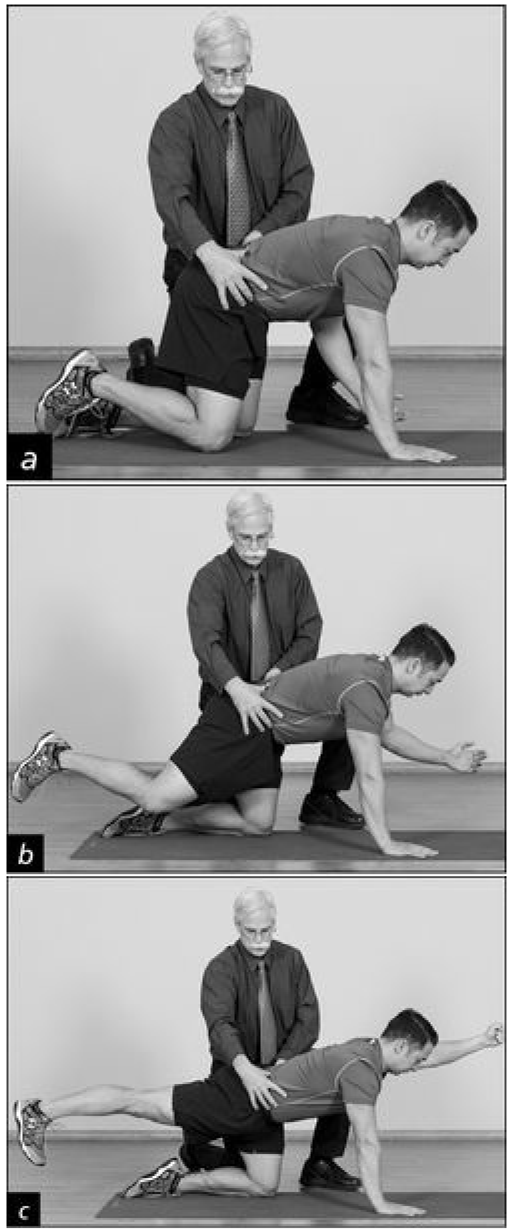
Bird Dog, Intermediate

Example Cues

1. Make a fist and squeeze and push the heel away.
2. Hold for 10 seconds, then sweep the floor with the hands and knees.

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

Bird Dog, Advanced

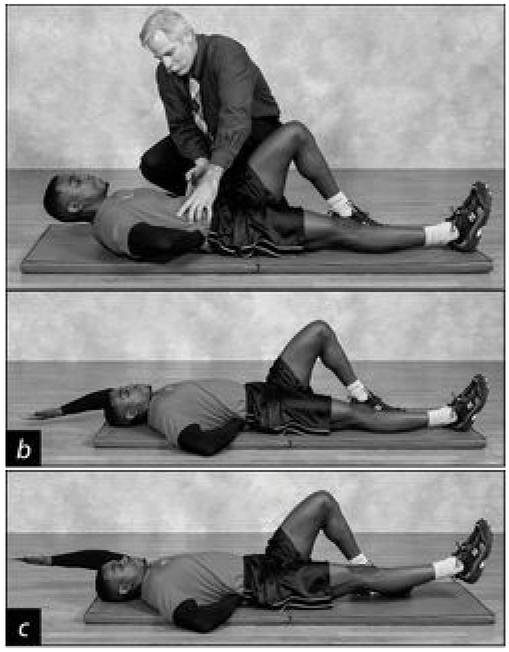


Example Cue

If your torso twists on the leg lift, reduce the height of the leg and instead push the heel away.

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

Curl-Up, Highest Level



Example Cues

1. Begin with the modified curl-up, then progress to a modified dead bug.
2. Here the core is prestiffened and small, but quick motion occurs about the shoulder and opposite side hip joint.
3. No spine motion occurs.
4. Keep one hand supporting the lumbar spine.

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

Bird Dog, Highest Level

Example Cues

1. Focus on motion in the shoulder and hip joints while stiffening the spine.
2. Draw squares with one hand and one foot.

From S. McGill, 2016, Low back disorders, 3rd ed. (Champaign, IL: Human Kinetics).

Appendix

A.1 Raw Cross-Sectional Areas (mm²) (Standard Deviation in Parentheses) Measured Directly From MRI Scans

	VERTEBRAL LEVEL					
Muscles	**L5**	**L4**	**L3**	**L2**	**L1**	**T12**
R rectus abdominis	787 (250)	750 (207)	670 (133)	712 (239)	576 (151)	
L rectus abdominis	802 (247)	746 (181)	693 (177)	748 (240)	514 (99)	
R external oblique		915 (199)	1,276 (171)	1,158 (222)		
L external oblique		992 (278)	1,335 (213)	1,351 (282)		
R internal oblique		903 (83)	1,515 (317)	1,055 (173)		
L internal oblique		900 (115)	1,424 (310)	1,027 (342)		
R trans. abdominis	119 (22)	237 (82)	356 (110)	596 (50)		
L trans. abdominis	175 (57)	224 (48)	376 (115)	646 (183)		
R abdominal wall*	1,104 (393)	2,412 (418)	3,269 (422)	3,051 (463)		
L abdominal wall*	1,146 (377)	2,420 (475)	3,329 (468)	3,111 (556)		
R longissimus thor.			747 (162)	1,175 (370)	1,248 (228)	1,095 (222)
L longissimus thor.			782 (129)	1,089 (251)	1,180 (184)	1,258 (347)
R iliocostalis lumb.			1,368 (341)	1,104 (181)	1,181 (316)	921 (339)
L iliocostalis lumb.			1,395 (223)	1,150 (198)	1,158 (247)	835 (400)
R multifidus			447 (271)	343 (178)	290 (96)	289 (66)
L multifidus			472 (269)	366 (157)	324 (95)	312 (76)
R latissimus dorsi			232 (192)	429 (202)	717 (260)	1,014 (264)
L latissimus dorsi			256 (217)	372 (161)	682 (260)	960 (310)
R erector mass**	905 (331)	2,151 (539)	2,831 (458)	2,854 (547)	2,615 (405)	2,614 (584)
L erector mass**	986 (338)	2,234 (476)	2,933 (382)	2,833 (456)	2,723 (428)	2,601 (559)
R psoas	1,606 (198)	1,861 (347)	1,594 (369)	1,177 (285)	513 (329)	330 (210)
L psoas	1,590 (244)	1,820 (272)	1,593 (291)	1,211 (298)	488 (250)	462 (190)
R quadratus lumb.		725 (209)	701 (212)	552 (192)	392 (249)	320 (197)
L quadratus lumb.		625 (249)	746 (167)	614 (189)	404 (220)	326 (5)
Disc area	1,360 (276)	1,459 (270)	1,415 (249)	1,332 (294)	1,334 (285)	1,241 (166)
Total area	52,912 (9,123)	51,813 (9,845)	54,286 (8,702)	55,834 (8,112)	59,091 (6,899)	63,287 (9,153)

*Abdominal wall includes external and internal oblique and transverse abdominis.

**Erector mass includes longissimus thoracis, iliocostalis lumborum, and multifidus.

	VERTEBRAL LEVEL						
Muscles	T11	T10	T9	T8	T7	T6	T5
R rectus abdominis							
L rectus abdominis							
R external oblique							
L external oblique							
R internal oblique							
L internal oblique							
R trans. abdominis							
L trans. abdominis							
R abdominal wall*							
L abdominal wall*							
R longissimus thor.	938 (49)						
L longissimus thor.	938 (21)						
R iliocostalis lumb.	556 (234)						
L iliocostalis lumb.	551 (170)						
R multifidus	331 (89)	351 (90)	312 (97)				
L multifidus	327 (80)	353 (53)	355 (73)				
R latissimus dorsi	1,254 (281)	1,368 (330)	1,458 (269)	1,581 (159)	1,764 (289)	1,876 (432)	2,477 (246)
L latissimus dorsi	1,102 (316)	1,239 (257)	1,417 (293)	1,582 (281)	1,697 (189)	2,013 (422)	2,596 (721)
R erector mass**	1,832 (282)	1,690 (210)	1,413 (304)	1,049 (201)	842 (165)	777 (189)	743 (70)
L erector mass**	2,041 (285)	1,722 (279)	1,471 (351)	1,129 (100)	879 (114)	779 (95)	675 (76)
R psoas							
L psoas							
R quadratus lumb.							
L quadratus lumb.							
Disc area	1,133 (124)	1,015 (125)	933 (112)	798 (91)	797 (104)	741 (80)	671 (82)
Total area	59,249 (7,272)	61,051 (7,570)	61,732 (6,960)	65,794 (5,254)	67,782 (3,982)	66,410 (2,372)	69,337 (2,233)

*Abdominal wall includes external and internal oblique and transverse abdominis.

**Erector mass includes longissimus thoracis, iliocostalis lumborum, and multifidus.

A.2 Raw Lateral Distances (mm) Between Muscle Centroids and Intervertebral Disc Centroid (Standard Deviation in Parentheses)

	VERTEBRAL LEVEL					
Muscles	**L5**	**L4**	**L3**	**L2**	**L1**	**T12**
R rectus abdominis	32 (5)	38 (7)	43 (7)	46 (8)	37 (8)	
L rectus abdominis	−33 (6)	−36 (7)	−38 (8)	−43 (7)	−35 (17)	
R external oblique		125 (13)	130 (10)	140 (5)		
L external oblique		−120 (9)	−125 (9)	−133 (7)		
R internal oblique		109 (11)	116 (8)	123 (9)		
L internal oblique		−103 (9)	−112 (8)	−121 (11)		
R trans. abdominis	99 (1)	108 (11)	122 (9)	117 (9)		
L trans. abdominis	−101 (1)	−101 (9)	−107 (7)	−109 (9)		
R abdominal wall*	102 (8)	113 (12)	119 (8)	123 (9)		
L abdominal wall*	−102 (9)	−115 (14)	−114 (7)	−120 (9)		
R longissimus thor.			22 (4)	32 (2)	32 (6)	30 (2)
L longissimus thor.			−19 (5)	−30 (6)	−37 (12)	−34 (4)
R iliocostalis lumb.			52 (4)	58 (4)	68 (10)	65 (7)
L iliocostalis lumb.			−48 (6)	−60 (10)	−65 (9)	−67 (7)
R multifidus			11 (1)	13 (4)	13 (3)	10 (3)
L multifidus			−14 (7)	−12 (3)	−11 (3)	−11 (2)
R latissimus dorsi			102 (8)	108 (8)	122 (12)	129 (10)
L latissimus dorsi			−104 (15)	−107 (9)	−117 (11)	−128 (7)
R erector mass**	22 (6)	34 (7)	40 (4)	42 (4)	44 (5)	42 (3)
L erector mass**	−21 (5)	−33 (6)	−38 (5)	−41 (6)	−41 (7)	−40 (4)
R psoas	54 (4)	50 (3)	44 (3)	39 (2)	32 (3)	32 (3)
L psoas	−54 (4)	−48 (4)	−42 (3)	−38 (3)	−31 (3)	−32 (2)
R quadratus lumb.		81 (5)	75 (6)	63 (5)	46 (6)	46 (11)
L quadratus lumb.		−78 (12)	−73 (4)	−64 (5)	−50 (6)	−47 (5)
Total area	0 (2)	1 (3)	−2 (4)	−1 (3)	−1 (4)	0 (3)

*Abdominal wall includes external and internal oblique and transverse abdominis.

**Erector mass includes longissimus thoracis, iliocostalis lumborum, and multifidus.

	VERTEBRAL LEVEL						
Muscles	T11	T10	T9	T8	T7	T6	T5
R rectus abdominis							
L rectus abdominis							
R external oblique							
L external oblique							
R internal oblique							
L internal oblique							
R trans. abdominis							
L trans. abdominis							
R abdominal wall*							
L abdominal wall*							
R longissimus thor.							
L longissimus thor.	29 (1)						
R iliocostalis lumb.	−36 (7)						
L iliocostalis lumb.	61 (4)						
R multifidus	−67 (11)						
L multifidus	8 (2)	11 (2)	12 (2)				
R latissimus dorsi	−12 (2)	−12 (2)	−15 (10)				
L latissimus dorsi	129 (9)	140 (9)	141 (8)	145 (7)	146 (7)	153 (7)	153 (4)
R erector mass**	−129 (10)	−137 (9)	−139 (8)	−143 (6)	−147 (10)	−153 (5)	−151 (5)
L erector mass**	34 (4)	34 (4)	32 (4)	31 (7)	30 (4)	25 (5)	27 (2)
R psoas	−40 (3)	−36 (3)	−35 (4)	−33 (6)	−31 (2)	−29 (3)	−27 (6)
L psoas							
R quadratus lumb.							
L quadratus lumb.							
Total area	1 (3)	0 (2)	0 (2)	2 (1)	2 (1)	1 (3)	2 (3)

*Abdominal wall includes external and internal oblique and transverse abdominis.

**Erector mass includes longissimus thoracis, iliocostalis lumborum, and multifidus.

A.3 Raw Anteroposterior Distances (mm) Between Muscle Centroids and Intervertebral Disc Centroid (Standard Deviation in Parentheses)

	VERTEBRAL LEVEL					
Muscles	L5	L4	L3	L2	L1	T12
R rectus abdominis	81 (16)	73 (14)	79 (13)	91 (14)	109 (8)	
L rectus abdominis	80 (15)	73 (14)	81 (14)	92 (14)	112 (6)	
R external oblique		35 (10)	20 (14)	28 (12)		
L external oblique		32 (18)	19 (11)	28 (11)		
R internal oblique		41 (12)	25 (9)	36 (17)		
L internal oblique		41 (17)	26 (12)	40 (16)		
R trans. abdominis	55 (0)	28 (11)	22 (11)	36 (6)		
L trans. abdominis	50 (5)	30 (14)	23 (10)	44 (5)		
R abdominal wall*	58 (16)	31 (12)	17 (12)	30 (15)		
L abdominal wall*	59 (17)	32 (13)	20 (12)	31 (11)		
R longissimus thor.			−61 (6)	−62 (7)	−60 (7)	−60 (6)
L longissimus thor.			−61 (5)	−63 (6)	−60 (7)	−59 (8)
R iliocostalis lumb.			−57 (7)	−61 (7)	−62 (5)	−59 (7)
L iliocostalis lumb.			−57 (7)	−61 (6)	−61 (4)	−58 (6)
R multifidus			−55 (7)	−55 (6)	−52 (6)	−51 (3)
L multifidus			−53 (7)	−56 (5)	−51 (5)	−50 (3)
R latissimus dorsi			−45 (16)	−47 (12)	−47 (10)	−39 (8)
L latissimus dorsi			−43 (17)	−46 (10)	−46 (7)	−37 (8)
R erector mass**	−64 (6)	−61 (5)	−61 (5)	−61 (5)	−59 (5)	−56 (5)
L erector mass**	−63 (5)	−61 (5)	−61 (5)	−62 (5)	−60 (4)	−57 (5)
R psoas	18 (9)	1 (5)	−7 (5)	−9 (5)	−11 (6)	−14 (2)
L psoas	19 (8)	2 (4)	−6 (4)	−8 (2)	−11 (4)	−11 (1)
R quadratus lumb.		−36 (9)	−37 (6)	−37 (6)	−35 (4)	−31 (6)
L quadratus lumb.		−31 (5)	−34 (6)	−36 (5)	−34 (4)	−32 (8)
Total area	1 (10)	−2 (9)	1 (8)	9 (8)	18 (5)	24 (7)

*Abdominal wall includes external and internal oblique and transverse abdominis.

**Erector mass includes longissimus thoracis, iliocostalis lumborum, and multifidus.

	VERTEBRAL LEVEL						
Muscles	T11	T10	T9	T8	T7	T6	T5
R rectus abdominis							
L rectus abdominis							
R external oblique							
L external oblique							
R internal oblique							
L internal oblique							
R trans. abdominis							
L trans. abdominis							
R abdominal wall*							
L abdominal wall*							
R longissimus thor.	−56 (4)						
L longissimus thor.	−52 (4)						
R iliocostalis lumb.	−57 (1)						
L iliocostalis lumb.	−56 (2)						
R multifidus	−47 (5)	−49 (4)	−48 (2)				
L multifidus	−47 (5)	−47 (3)	−47 (2)				
R latissimus dorsi	−32 (7)	−24 (7)	−22 (7)	−18 (9)	−17 (6)	−12 (3)	−17 (5)
L latissimus dorsi	−28 (9)	−23 (7)	−19 (7)	−17 (7)	−15 (8)	−11 (7)	−19 (3)
R erector mass**	−54 (4)	−54 (4)	−52 (4)	−52 (3)	−52 (4)	−47 (4)	−50 (3)
L erector mass**	−52 (4)	−52 (4)	−51 (4)	−51 (3)	−51 (4)	−46 (5)	−50 (3)
R psoas							
L psoas							
R quadratus lumb.							
L quadratus lumb.							
Total area	29 (8)	30 (9)	32 (7)	36 (7)	37 (6)	32 (10)	34 (5)

Glossary

anisotropic—A description of a material whose behavior depends on the direction of the applied load. For example, adult bone is stronger in compression than in tension.

antagonist—A muscle that opposes the function of another in terms of torque generation but that may be necessary to ensure a stable joint.

breaking strain—The strain at which tissue fails.

buckle—An abnormally large rotation at a single joint, resulting in very large passive tissue forces. The spine may experience several modes of unstable behavior. The column may buckle under compressive loads if sufficient stiffness is not present in the rotational degrees of freedom.

dermatome—An area of skin supplied with afferent nerve fibers by a single spinal nerve root.

discogenic —Relating to or originating from the disc (e.g., discogenic pain or discogenic disorders).

eigenvalue—The element of the Hessian matrix that, if less than zero, indicates the potential for unstable behavior.

eigenvector—The mode and site of unstable behavior, and the deficit that allowed the instability to develop.

electromyography—The measurement of the electromyogram, electrical activity associated with muscle contraction.

engram—The encoded representation of a movement, which resides in the motor cortex or the spinal cord. Most repeated movements arise from "running the tape," or engram. The movement may then be modified by reflexes if the normal pattern is disturbed.

etiology—The analysis and description of the cause of injury or disease.

failure tolerance—The load at which a tissue fails.

lordosis—The natural curvature of the neutral lumbar spine; hypolordosis occurs with a flexed lumbar spine; hyperlordosis occurs with an extended lumbar spine.

moment—Moment of force; occurs when a force is applied through a distance about an axis to create rotational effort.

moment arm—The perpendicular distance between the axis of rotation and a force, which creates a moment.

myoelectric—Of or relating to an electrical signal obtained from contracting muscle.

myotome—An electrical signal obtained from contracting muscle.

neural arch—The structure that encloses the spinal cord at each vertebra; includes the pedicles and laminae.

neutral lordosis—When the lumbar spine is neither flexed nor extended.

NIOSH—National Institute for Occupational Safety and Health (United States).

nociception—The sensation of pain with a nociceptor (pain-sensing organ).

odds ratio—The increase in the odds of an event occurring.

osteophytes—Bony spurs that grow on the stress points of a vertebra; usually indicative of an unstable joint.

palpation—Using the hands to feel for spinal motion or pathology.

pars interarticularis —The part of the vertebra that connects the facet articulating surface to the neural arch and often is the site of fracture.

pathomechanics —Mechanics associated with pathology.

Poisson's effect—A material property of an elastic sheet in which strain longitudinally produces a proportional lateral strain.

provocative , or functional, diagnosis—A diagnosis based on tasks that exacerbate symptoms.

provocative testing—Provoking tissues with movement or direct load to elicit pain.

psychophysical—A description of variables that are associated with psychological or social aspects of a person.

psychosocial—A description of variables that influence behavior that may include psychological traits such as the perception of control over a task and social traits such as how people are viewed by others.

spondylolisthesis—A fracture of the pars interarticularis that causes an anterior shear displacement of the superior vertebra (usually L4 or L5).

strain—A change in length normalized to rest length (dimensionless ratio) and expressed as a percentage.

subfailure —An applied load that is less than the load required to cause damage if applied once.

synergist—A muscle that assists another in the same functional objective.

tolerance—A load value above which injury occurs. It is modulated by repetition, duration, rest, and so on.

transmissible vector—The transmissible property of any force vector is due to its having an effect along the line on which it is directed. A perpendicular line can be drawn from the force vector to any body joint to determine the moment arm, or the potential of that force to cause a moment.

viscoelastic—The property of being both elastic and viscous to absorb the shock of loads.

viscosity—Friction as a function of velocity; it always moves in the direction of motion or impending motion.

yield point—The load at which a tissue begins to experience damage.

References and Additional Readings

Preface

McGill, S.M. (2015) Back mechanic: The step-by-step McGill method to fix back pain. Backfitpro, Inc. (www.backfitpro.com)

Chapter 1: Introduction to the Issues and Scientific Approach Unique to This Book

Abdu, W.A., Lurie, J.D., Spratt, K., et al. (2009) Degenerative spondylolisthesis: Does fusion method influence outcome: Four-year results of the patient outcome research trial, Spine, 34: 2351-2360.

Adams, M.A., and Hutton, W.C. (1985) Gradual disc prolapse. Spine, 10: 524.

Adams, M.A., Hutton, W.C., and Stott, J.R.R. (1980) The resistance to flexion of the lumbar intervertebral joint. Spine, 5: 245.

American Medical Association. (1990) Guides to the evaluation of permanent impairment (3rd edition).

American Medical Association. (2008) Sixth edition guides to the evaluation of permanent Impairment: Clarifications and corrections.

Arts, M.P., Brand, R., van den Akker, M.E., et al. (2009) Tubular diskectomy versus conventional diskectomy for sciatica: A randomized controlled trial. Journal of the American Medical Association, 302: 149-158.

Ashmen, K.J., Swanik, C.B., and Lephart, S.M. (1996) Strength and flexibility characteristics of athletes with chronic low-back pain. Journal of Sport Rehabilitation, 5: 275-286.

Axler, C.T., and McGill, S.M. (1997) Choosing the best abdominal exercises based on knowledge of tissue loads. Medicine & Science in Sports & Exercise, 29: 804-811.

Balkovic, C. and McGill, S.M. (2012) Extent of nucleus pulposus migration in the annulus of intervertebral discs exposed to cyclic flexion only versus cyclic flexion and extension. Clinical Biomechanics, 27: 766-770.

Battie, M.C., Bigos, S.J., Fisher, L.D., Spengler, D.M., Hansson, T.H., Nachemson, A.L., and Wortley, M.D. (1990) The role of spinal flexibility in back pain complaints within industry: A prospective study. Spine, 15: 768-773.

Biering-Sorensen, F. (1984) Physical measurements as risk indicators for

low-back trouble over a one-year period. Spine, 9: 106-119.
Bogduk, N., Derby, R., April, C., Louis, S., and Schwarzer, R. (1996) Precision diagnosis in spinal pain. In: Campbell, J. (Ed.), Pain 1996— An updated review (pp. 313-323). Seattle: IASP Press.
Brennan, G.P., Fritz, J.M., Hunter, S.J., Thackeray, A., Delitto, A., and Erhard, R.E. (2006) Identifying subgroups of patients with acute/subacute "nonspecific" low back pain: Results of a randomized clinical trial. Spine, 31 (6): 623-631.
Brinckmann, P. (1985) Pathology of the vertebral column. Ergonomics, 28: 235-244.
Brinckmann, P., Biggemann, M., and Hilweg, D. (1989) Prediction of the compressive strength of human lumbar vertebrae. Clinical Biomechanics, 4 (Suppl. 2): S1-S27.
Brown, S.H., and McGill, S.M. (2005) Muscle force-stiffness characteristics influence joint stability. Clinical Biomechanics, 20 (9): 917-922.
Brown, S., McGill, S.M. (2008a) How the inherent stiffness of the in-vivo human trunk varies with changing magnitude of muscular activation. Clinical Biomechanics, 23 (1): 15-22.
Brown, S., McGill, S.M. (2008b) Co-activation alters the linear versus non-linear impression of the EMG-torque relationship of trunk muscles. Journal of Biomechanics, 41: 491-497.
Brown, S., and McGill, S.M. (2009) Transmission of muscularly generated force and stiffness between layers of the rat abdominal wall. Spine, 34 (2): E70-E75.
Buchbinder, R., Osborne, R., Ebeling, P., et al. (2009) A randomized trial of vertebroplasty for osteoporotic vertebral fractures. New England Journal of Medicine, 361: 557-568.
Burton, A.L., Tillotson, K.M., and Troup, J.D.G. (1989) Prediction of low back trouble frequency in a working population. Spine, 14: 939-946.
Callaghan, J.P., Howarth, S., and Beach, T. (2012) Validation of occupational estimates of cumulative low back load. Occupational Ergonomics, 10 (3): 113-124.
Carey, T.S., Freburger, J.K., Holmes, G.M., Castel, L., Darter, J., Agans, J., Kalsbeek, W., and Jackman, A. (2009) A long way to go: Practice patterns and evidence in chronic low back pain care. Spine, 34 (7): 718-724.
Carragee, E.J., Deyo, R.A., Kovacs, F.M., Wilco, P., Lurie, J.D., Urriyia, G., Corbin, T.P., and Schonene, M.L. (2009) Clinical research: Is the spine

field a mine field? Spine, 34 (5): 423-430.
Cholewicki, J., and McGill, S.M. (1994) EMG Assisted Optimization: A hybrid approach for estimating muscle forces in an indeterminate biomechanical model. Journal of Biomechanics, 27 (10): 1287-1289.
Cholewicki, J., and McGill, S.M. (1995) Relationship between muscle force and stiffness in the whole mammalian muscle: A simulation study. Journal of Biomechanical Engineering, 117: 339-342.
Cholewicki, J., and McGill, S.M. (1996) Mechanical stability of the in vivo lumbar spine: Implications for injury and chronic low back pain. Clinical Biomechanics, 11 (1): 1-15.
Cholewicki, J., McGill, S.M., and Norman, R.W. (1995) Comparison of muscle forces and joint load from an optimization and EMG assisted lumbar spine model: Towards development of a hybrid approach. Journal of Biomechanics, 28 (3): 321-331.
Chou, R., Deyo, R.A., and Jarvik, J.G. (2012) Appropriate use of lumbar imaging for evaluation of low back pain. Radiologic Clinics of North America, 50 (4): 569.
Cibulka, M.T., Sinacore, D.R., Cromer, G.S., and Delitto, A. (1998) Unilateral hip rotation range of motion asymmetry in patients with sacroiliac joint regional pain. Spine, 23 (9): 1009-1015.
Curatolo, M., Bogduk, N., Ivancic, P., McLean, S., and Siegmund, G. (2011) The role of tissue damage in whiplash associated disorders: Discussion paper, Spine, 36 (25 suppl): S309-S315.
Currie, S.R., and Wang, J.L. (2004) Chronic back pain and major depression in the general Canadian population. Pain, 107: 54-60.
Deyo, R.A. (1998, August) Low back pain. Scientific American, 49-53.
Ferguson, S.A., and Marras, W.S. (1997) A literature review of low back disorder surveillance measures and risk factors. Clinical Biomechanics, 12 (4): 211-226.
Finch, P. (1999, November 11-14) Spinal pain—An Australian perspective. In Proceedings of the 13th World Congress of the International Federation of Physical Medicine and Rehabilitation, Washington, DC, 243-246.
Fritz, J.M., Cleland, J.A., and Childs, J.D. (2007) Subgrouping patients with low back pain: Evolution of a classification approach to physical therapy. Journal of Orthopaedic and Sports Physical Therapy, 37 (6): 290.
Fordyce, W.E. (Ed.). (1995) Back pain in the workplace. Seattle: IASP Press.
Galbusera, F., Schmidt, H., Neidlinger-Wilke, C., Gottschalk, A., and Wilke,

H.J. (2011) The mechanical response of the lumbar spine to different combinations of disc degenerative changes investigated using randomized poroelastic finite element models. European Spine Journal, 20 (4): 563-571.
Gallagher, S., and Heberger, J.R. (2013) Examining the interaction of force and repetition on musculoskeletal disorder risk: A systematic literature review. Human Factors: The Journal of the Human Factors and Ergonomics Society, 55 (1): 108-124.
Gibson, E.S., Martin, R.H., and Terry, C. (1980) Incidence of low back pain and pre-placement X-ray screening. Journal of Occupational Medicine, 22: 515.
Granata, K.P., and Marras, W.S. (1993) An EMG-assisted model of loads on the lumbar spine during asymmetric trunk extensions. Journal of Biomechanics, 26: 1429-1438.
Grenier, S.G., and McGill, S.M. (2008) When exposed to challenged ventilation, those with a history of LBP increase spine stability relatively more than healthy individuals. Clinical Biomechanics, 23 (9): 1105-1111.
Grundy, P.F., and Roberts, C.J. (1984, August 4) Does unequal leg length cause back pain? A case control study. Lancet, 256-258.
Gunning, J.L., Callaghan, J.P., and McGill, S.M. (2001) The role of prior loading history and spinal posture on the compressive tolerance and type of failure in the spine using a porcine trauma model. Clinical Biomechanics, 16 (6): 471-480.
Hadler, N. (2001) The bane of the aging worker [Editorial]. Spine, 26 (12): 1309-1310.
Hadler, N.M., Tait, R.C., and Chibnall, J.T. (2007) Back pain in the workplace. Journal of the American Medical Association, 297 (14): 1594-1596.
Hellsing, A.L. (1988) Tightness of hamstring and psoas major muscles. Upsala Journal of Medical Science, 93: 267-276.
Hellum, C., Berg, L., Gjertsen, O., Johnsen, L.G., Neckelmann, G., Storheim, K., Keller, A., Grundnes, O., and Espeland, A. (2012) Adjacent level degeneration and facet arthropathy after disc prosthesis surgery or rehabilitation in patients with chronic low back pain and degenerative disease. Spine, 37 (25): 2063-2073.
Heneweer, H., Vanhees, L., and Picavet, H.S.J. (2009) Physical activity and low back pain: A U-shaped relation? Pain, 143 (1): 21-25.

Hirsch, C., Ingelmark, B.E., and Miller, M. (1963-64) The anatomical basis for low back pain. Acta Orthopaedica Scandinavica, 1 (33).

Hoikka, V., Ylikoski, M., and Tallroth, K. (1989) Leg-length inequality has poor correlation with lumbar scoliosis: A radiological study of 100 patients with chronic low back pain. Archives of Orthopaedic Trauma Surgery, 108: 173-175.

Howarth, S.J., Allison, A.E., Grenier, S., Cholewicki, J., and McGill, S.M. (2004) On the implications of interpreting the stability index: A spine example. Journal of Biomechanics, 37 (8): 1147-1154.

Hsu, K., Zucherman, J.F., Derby, R., White, A.H., Goldthwaite, N., and Wynne, G. (1988) Painful lumbar end plate disruptions. A significant discographic finding. Spine, 13 (1): 76-78.

Ikeda, D., and McGill, S.M. (2012) Can altering motions, postures and loads provide immediate low back pain relief: A study of four cases investigating spine load, posture and stability. Spine, 37 (23): E1469-E1475.

Itz, C.J., Geurts, J.W., van Kleef, M., and Nelemans, P. (2012) Clinical course of non-specific low back pain: A systematic review of prospective cohort studies set in primary care, European Journal of Pain. doi:10.1002/j.1532-2149.2012.00170.x

Jager, M., Luttman, A., and Laurig, W. (1991) Lumbar load during one-handed bricklaying. International Journal of Industrial Ergonomics, 8: 261-277.

Kavcic, N., Grenier, S., and McGill, S. (2004a) Determining the stabilizing role of individual torso muscles during rehabilitation exercises. Spine, 29 (11): 1254-1265.

Kavcic, N., Grenier, S.G., and McGill, S.M. (2004b) Quantifying tissue loads and spine stability while performing commonly prescribed low back stabilization exercises. Spine, 29 (20): 2319-2329.

Kirkaldy-Willis, W.H. (1998) The three phases of the spectrum of degenerative disease. In: Managing low back pain (2nd ed.). New York: Churchill Livingstone.

Lord, S.M., Barnsley, L., Wallis, B.J., McDonald, G.J., and Bogduk, N. (1996) Percutaneous radio frequency neurotomy for chronic cervical zygapophyseal joint pain. New England Journal of Medicine, 335: 1721-1726.

Luoto, S., Heliovaara, M., Hurri, H., and Alarenta, M. (1995) Static back endurance and the risk of low back pain. Clinical Biomechanics, 10: 323-

324.
Maitland, G. (1987) The Maitland concept: Assessment, examination, and treatment by passive movement. In: Twomey, L., and Taylor, J. (Eds.), Physical therapy of the low back. New York: Churchill Livingstone.
McGill, S.M. (1988) Estimation of force and extensor moment contributions of the disc and ligaments at L4/L5. Spine, 12: 1395-1402.
McGill, S.M. (1992) A myoelectrically based dynamic 3-D model to predict loads on lumbar spine tissues during lateral bending. Journal of Biomechanics, 25 (4): 395-414.
McGill, S.M. (1996) A revised anatomical model of the abdominal musculature for torso flexion efforts. Journal of Biomechanics, 29 (7): 973-977.
McGill, S.M. (1997) Biomechanics of low back injury: Implications on current practice and the clinic [Invited paper]. Journal of Biomechanics, 30 (5): 456-475.
McGill, S.M., and Brown, S. (1992) Creep response of the lumbar spine to prolonged lumbar flexion. Clinical Biomechanics, 7: 43.
McGill, S.M., Grenier, S., Preuss, R.P., and Brown, S. (2003) Previous history of LBP with work loss is related to lingering effects in psychosocial, physiological, and biomechanical characteristics. Ergonomics, 46 (7): 731-746.
McGill, S.M., Juker, D., and Axler, C. (1996) Correcting trunk muscle geometry obtained from MRI and CT scans of supine postures for use in standing postures. Journal of Biomechanics, 29 (5): 643-646.
McGill, S.M., Juker, D., and Kropf, P. (1996) Appropriately placed surface EMG electrodes reflect deep muscle activity (psoas, quadratus lumborum, abdominal wall) in the lumbar spine. Journal of Biomechanics, 29 (11): 1503-1507.
McGill, S.M., and Norman, R.W. (1985) Dynamically and statically determined low back moments during lifting. Journal of Biomechanics, 18 (12): 877-885.
McGill, S.M., and Norman, R.W. (1986) The Volvo Award for 1986: Partitioning of the L4/L5 dynamic moment into disc, ligamentous and muscular components during lifting. Spine, 11 (7): 666-678.
McGill, S.M., and Norman, R.W. (1987a) Effects of an anatomically detailed erector spinae model on L4/L5 disc compression and shear. Journal of Biomechanics, 20 (6): 591-600.

McGill, S.M., and Norman, R.W. (1987b) An assessment of intra-abdominal pressure as a viable mechanism to reduce spinal compression. Ergonomics, 30 (11): 1565-1588.

McGill, S.M., and Norman, R.W. (1988) The potential of lumbodorsal fascia forces to generate back extension moments during squat lifts. Journal of Biomedical Engineering, 10: 312-318.

McGill, S.M., Patt, N., and Norman, R.W. (1988) Measurement of the trunk musculature of active males using CT scan radiography: Implications for force and moment generating capacity about the L4/L5 joint. Journal of Biomechanics, 21 (4): 329-341.

McGill, S.M., Santaguida, L., and Stevens, J. (1993) Measurement of the trunk musculature from T6 to L5 using MRI scans of 15 young males corrected for muscle fibre orientation. Clinical Biomechanics, 8: 171-178.

McGill, S.M., Seguin, J., and Bennett, G. (1994) Passive stiffness of the lumbar torso about the flexion-extension, lateral bend and axial twist axes: The effect of belt wearing and breath holding. Spine, 19 (6): 696-704.

McGill, S.M., Thorstensson, A., and Norman, R.W. (1989) Non-rigid response of the trunk to dynamic axial loading: An evaluation of current modelling assumptions. Clinical Biomechanics, 4: 45-50.

McGill, S.M., and Yingling, V. (1999) Traction may enhance the imaging of spine injuries with plane radiographs: Implications for the laboratory versus the clinic. Clinical Biomechanics, 14 (4): 291-295.

Mendelson, G. (1982) Not cured by a verdict: Effect of a level settlement on compensation claimants. Medical Journal of Australia, 2: 219-230.

Nachemson, A.L. (1992) Newest knowledge of low back pain: A critical look. Clinical Orthopaedics and Related Research, 279: 8-20.

Nguyen, T.H., Randolph, D.C., Talmage, J., Succop, P., and Travis, R. (2011) Long term outcomes of lumbar fusion among workers' compensation subjects: A historical cohort study. Spine, 36 (4): 320-331.

Niemelainen, R., Battié, M.C., Gill, K., and Videman, T. (2008) The prevalence and characteristics of thoracic magnetic resonance imaging findings in men. Spine, 33 (23): 2552-2559.

Ortiz, A.O., and Bordia, R. (2011) Injury to the vertebral endplate-disk complex associated with osteoporotic vertebral compression fractures. American Journal of Neuroradiology, 32 (1): 115-120.

Parks, K.A., Crighton, K.S., Goldford, R.J., and McGill, S.M. (2003) On the validity of ratings of impairment for low back disorders. Spine, 28 (4):

380-384.
Parks, K.A., Crichton, K.S., Goldford, R.J., and McGill, S.M. (2003) A comparison of lumbar range of motion with functional ability scores on low back pain patients: Assessment of the validity of range of motion. Spine, 28 (4): 380-384.
Porter, R.W. (1987) Does hard work prevent disc protrusion? Clinical Biomechanics, 2: 196-198.
Radanov, B.P., Sturzenegger, M., DeStefano, G., and Schinrig, A. (1994) Relationship between early somatic, radiological, cognitive and psychosocial findings and outcome during a one-year follow up in 117 patients suffering from common whiplash. British Journal of Rheumatology, 33: 442-448.
Raftry, S.M., and Marshall, P.W. (2012) Does a 'tight' hamstring predict low back pain reporting during prolonged standing? Journal of Electromyography and Kinesiology, 22 (3): 407-411.
Rose, S.J. (1989) Physical therapy diagnosis: Role and function. Physical Therapy, 69: 535-537.
Saal, J.A., and Saal, J.S. (1989) Nonoperative treatment of herniated lumbar intervertebral disc with radiculopathy: An outcome study. Journal of Biomechanics, 14: 431-437.
Santaguida, L., and McGill, S.M. (1995) The psoas major muscle: A three-dimensional mechanical modelling study with respect to the spine based on MRI measurement. Journal of Biomechanics, 28 (3): 339-345.
Savage, R.A., Whitehouse, G.H., and Roberts, N. (1997) The relationship between the magnetic resonance imaging appearance of the lumbar spine and low back pain, age and occupation in males. European Spine Journal, 6: 106-114.
Siepe, C.J., Zelenkov, P., Sauri-Barraza, J-C., Szeimies, U., Grubinger, T., Tepass, A., Stabler, A., and Mayer, M. (2010) The fate of facet joint and adjacent level disc degeneration following total lumbar disc replacement: A prospective clinical, x-ray, and magnetic resonance imaging investigation, Spine, 35 (22): 1991-2003.
Smeets, R.J., Vlaeyen, J.W., Kester, A.D., and Knottnerus, J.A. (2006) Reduction of pain catastrophizing mediated the outcome of both physical and cognitive-behavioral treatment in chronic low back pain, Journal of Pain 7 (4): 261-271.
Solomonow, M. (2012) Neuromuscular manifestations of viscoelastic tissue

degradation following high and low risk repetitive lumbar flexion. Journal of Electromyography and Kinesiology, 22 (2): 155-175.
Spitzer, W.O. (1993) Low back pain in the workplace: Attainable benefits not attained [Editorial]. British Journal of Industrial Medicine, 50: 385-388.
Staal, J.B., de Bie, R.A., de Vet, H.C., Hildebrandt,J., and Nelemans, P. (2009) Injection therapy for subacute and chronic low back pain: An updated Cochrane review. Spine, 34 (1): 49-59.
Stevenson, J.M., Weber, C.L., Smith, J.T., Dumas, G.A., and Albert, W.J. (2001) A longitudinal study of the development of low back pain in an industrial population. Spine, 26 (12): 1370-1377.
Stroyer, J., and Jensen, L.D. (2008) The role of physical fitness as a risk indicator of increased low back pain intensity among people working with physically and mentally disabled persons: A 30-month prospective study. Spine, 33 (5): 546-554.
Suni, J., Rinne, M., Natri, A., Statistisian, M.P., Parkkari, J., and Alaranta, H. (2006) Control of the lumbar neutral zone decreases low back pain and improves self-evaluated work ability: A 12-month randomized controlled study. Spine, 31 (18): E611-E620.
Suni, J.H., Taanila, H., Mattila, V.M., Ohrankammen, O., Yourinen, P., Pihlajamaki, H., and Parkkari, J. (2013) Neuromuscular exercise and councilling decrease absenteeism due to low back pain in young conscripts, Spine, 38 (5): 375-384.
Sutarno, C., and McGill, S.M. (1995) Iso-velocity investigation of the lengthening behaviour of the erector spinae muscles. European Journal of Applied Physiology and Occupational Physiology, 70 (2): 146-153.
Taylor, J.R., Twomey, L.T., and Corker, M. (1990) Bone and soft tissue injuries in post-mortem lumbar spines. Paraplegia, 28: 119-129.
Teasell, R.W. (1997) The denial of chronic pain. Journal of Pain Research Management, 2: 89-91.
Thompson, E.N. (1997) Back pain: Bankrupt expertise and new directions. Journal of Pain Research Management, 2: 195-196.
Valkenburg, H.A., and Haanen, H.C.M. (1982) The epidemiology of low back pain. In: White, A.A., and Gordon, S.L. (Eds.), Symposium on idiopathic low back pain. St. Louis: Mosby.
Van Dillen, L.R., Bloom, N.J., Gombatto, S.P., and Susco, T.M. (2008) Hip rotation range of motion in people with and without low back pain who participate in rotation-related sports. Physical Therapy in Sport, 9 (2): 72-

81.
Van Nieuwenhuyse, A., Crombez, G., Burdorf, A., Verbeke, G., Masschelein, R., Moens, G., and Mainaux, P. (2009) Physical characteristics of the back are not predictive of low back pain in healthy workers: A prospective study. BMC Musculoskeletal Disorders, 10 (2): doi: 10.1186/1471-2474-10-2.
Vera Garcia, F., Moreside, J., and McGill, S.M. (2009) MVC techniques to normalize trunk muscle EMG in healthy women. Journal of Electromyography and Kinesiology, 20: 10-16.
Videman, T., Battie, M.C., Gill, K., Manninen, H., Gibbons, L.E., and Fisher, L.D. (1995) Magnetic resonance imaging findings and their relationships in the thoracic and lumbar spine: Insights into the etiopathogenesis of spinal degeneration. Spine, 20 (8): 928-935.
Wassenaar, M., van Rijn, R.M., van Tulder, M.W., et al. (2012) Magnetic resonance imaging for diagnosing lumbar spinal pathology in adult patients with low back pain or sciatica: A diagnostic systematic review. European Spine Journal, 21 (2): 220-227.
Weber, H. (1983) Lumbar disk herniation: A controlled prospective study with ten years of observation. Spine, 8: 131.
Woo, S.L.-Y., Gomez, M.A., and Akeson, W.H. (1985) Mechanical behaviors of soft tissues: Measurements, modifications, injuries, and treatment. In: Nahum, H.M., and Melvin, J. (Eds.), Biomechanics of trauma (pp. 109-133). Norwalk, CT: Appleton-Century-Crofts.
Zhao, F., Pollintine, P., Hole, B.D., Dolan, P., and Adams, M.A. (2005) Discogenic origins of spine instability. Spine, 30: 2621-2630.

Chapter 2: Epidemiological Studies and What They Really Mean

Adams, M., Bogduk, N., Burton, K., and Dolan, P. (2002) The biomechanics of back pain. Edinburgh: Churchill Livingstone.
Andersson, G.B. (1981) Epidemiologic aspects of low back pain in industry. Spine, 6: 53-60.
Andersson, G.B. (1991) The epidemiology of spinal disorders. In: J.W. Frymoyer (Ed.), The adult spine: Principles and practice (chapter 8). New York: Raven Press.
Arendt-Nielson, L., Graven-Neilson, T., Svarrer, H., and Svensson, P. (1995) The influence of low back pain on muscle activity and coordination during gait. Pain, 64: 231-240.
Battie, M.C., Haynor, D.R., Fisher, L.D., Gill, K., Gibbons, L.E., and

Videman, T. (1995) Similarities in degenerative findings on magnetic resonance images of the lumbar spines of identical twins. Journal of Bone and Joint Surgery, 77-A: 1662-1670.

Battie, M.C., Videman, T., Kaprio, J., et al. (2009) The twin spine study: Contributions to a changing view of disc degeneration. The Spine Journal, 9: 47-59.

Beneck, G.J., and Kulig, K. (2012) Multifidus atrophy is localized and bilateral in active persons with chronic unilateral low back pain. Archives of Physical Medicine and Rehabilitation, 93 (2): 300-306.

Biering-Sorensen, F. (1984) Physical measurements as risk indicators for low-back trouble over a one-year period. Spine, 9: 106-119.

Bigos, S.J., Battie, M.C., Spengler, D.M., Fisher, L.D., Fordyce, W.E., Hansson, T.H., Nachemson, A.L., and Wortley, M.D. (1991) A prospective study of work perceptions and psychosocial factors affecting the report of back injury. Spine, 16: 1-6.

Bigos, S.J., Holland, J., Holland, C., Webster, J.S., Battie, M., and Malmgren, J.A. (2009) High-quality controlled trials on preventing episodes of back problems: Systematic literature review in working-age adults. The Spine Journal, 9: 147-168.

Bigos, S.J., Spengler, D.M., Martin, N.A., Zeh, A., Fisher, L., Nachemson, A., and Wang, M.H. (1986) Back injuries in industry: A retrospective study. II. Injury factors. Spine, 11: 246-251.

Bogduk, N., Derby, R., April, C., Louis, S., and Schwarzer, R. (1996) Precision diagnosis in spinal pain. In: Campbell, J. (Ed.), Pain 1996— An updated review (pp. 313-323). Seattle: IASP Press.

Brennan, G.P., Fritz, J.M., Hunter, S.J., Thackeray, A., Delitto, A., and Erhard, R.E. (2007) Identifying subgroups of patients with acute/subacute "nonspecific" low back pain. Spine, 31: 623-631.

Brereton, L., and McGill, S.M. (1999) Effects of physical fatigue and cognitive challenges on the potential for low back injury. Human Movement Science, 18: 839-857.

Burton, A.K., Symonds, T.L., Zinzen, E., et al. (1996) Is ergonomics intervention alone sufficient to limit musculoskeletal problems in nurses. Occupational Medicine, 47: 25-32.

Burton, A.K., Tillotson, K.M., Symonds, T.L., Burke, C., and Mathewson, T. (1996) Occupational risk factors for the first onset of low back trouble: A study serving police officers. Spine, 21: 2621.

Burton, A.K., Tillotson, K.M., and Troup, J.D.G. (1989) Prediction of low back trouble frequency in a working population. Spine, 14: 939-946.

Butler, D.S. (1991) Mobilization of the nervous system. Melbourne: Churchill Livingstone.

Butler, D.S. (2000) The sensitive nervous system. Adelaide, Australia: Noigroup Publications.

Butler, H.L., Lariviere, C., Hubley-Kozey, C.L., and Sullivan, M.J. (2010) Directed attention alters the temporal activation patterns of back extensors during trunk flexion–extension in individuals with chronic low back pain. European Spine Journal, 19 (9): 1508-1516.

Callaghan, J., and McGill, S.M. (2001) Intervertebral disc herniation: Studies on a porcine model exposed to highly repetitive flexion/extension motion with compressive force. Clinical Biomechanics, 16 (1): 28-37.

Cholewicki, J., and McGill, S.M. (1996) Mechanical stability of the in vivo lumbar spine: Implications for injury and chronic low back pain. Clinical Biomechanics, 11 (1): 1-15.

Coenen, P., Kingma, I., Boot, C.R., Twisk, J.W., Bongers, P.M., and van Dieën, J.H. (2013) Cumulative low back load at work as a risk factor of low back pain: A prospective cohort study. Journal of Occupational Rehabilitation, 23 (1): 11-18.

Currie, S.L., and Wang, J.L. (2004) Chronic back pain and major depression in the general Canadian population. Pain, 107: 54-60.

Dankaerts, W., O'Sullivan, P., Burnett, A., and Straker, L. (2006) Altering patterns of superficial trunk muscle activation during sitting in non-specific chronic low back pain patients. Spine, 31: 2017-2013.

Delitto, A., Erhard, R.E., and Bowling, R.W. (1995) A treatment-based classification approach to acute low back syndrome: Identifying and staging patients for conservative treatment. Physical Therapy, 75: 470-489.

Durall, C.J., Udermann, B.E., Johansen, D.R., Gibson, B., Reineke, D.M., and Reutman, P. (2009) The effects of preseason trunk muscle training on low back pain occurrence in women collegiate gymnasts. Journal of Strength and Conditioning Research, 23 (1): 86-92.

Edwards, R.R., Kronfli, T., Hawthornthwaite, J.A., Smith, M.T., McGuire, L., and Page, G.G. (2008) Association of catastrophising with interleukin-6 responses to acute pain. Pain, 140 (1): 135-144.

Ferguson, S.A., Allread, W.G., Burr, D.L., Heaney, C., and Marras, W.S. (2012) Biomechanical, psychosocial and individual risk factors predicting

low back functional impairment among furniture distribution employees. Clinical Biomechanics, 27 (2): 117-123.
Ferguson, S.A., and Marras, W.S. (1997) A literature review of low back disorder surveillance measures and risk factors. Clinical Biomechanics, 12 (4): 211-226.
Fersum, K.V., Dankaerts, W., and O'Sullivan, P.B. (2009) Integration of sub-classification strategies in RCT's evaluating manual therapy treatment and exercise therapy for non-specific chronic low back pain: A systematic review. British Journal of Sports Medicine. doi:10.1136/bjsm.2009.063289
Freeman, S., Mascia, A., and McGill, S.M. (2013) Arthrogenic neuromuscular inhibition: A foundational investigation of existence in the hip joint. Clinical Biomechanics, 28: 171-177.
Fordyce, W.E. (Ed.) (1995) Back pain in the workplace. Seattle: IASP Press.
Fordyce, W.E. (1996) Response to Thompson/Merskey/Teasell letters. Pain, 65: 112-114.
Fritz, J.M., Cleland, J.A., and Childs, J.D. (2007) Subgrouping patients with low back pain: Evolution of a classification approach to physical therapy. Journal of Orthopaedic & Sports Physical Therapy, 37 (6): 290-302.
Gallagher, K.M., Nelson-Wong, E., and Callaghan, J.P. (2011) Do individuals who develop transient low back pain exhibit different postural changes than non-pain developers during prolonged standing? Gait and Posture 34 (4): 490-495.
Gamsa, A. (1990) Is emotional status a precipitator or a consequence of pain? Pain, 42: 183-195.
Gatchel, R.J., Polatin, P.B., and Mayer, T.G. (1995) The dominant role of psychosocial risk factors in the development of chronic low back pain disability. Spine, 20: 2702-2709.
Gordon, S.I., Yang, K.H., Mayer, P.J., Mace, A.H.J., Kish, V.I., and Radin, E.L. (1991) Mechanism of disc rupture—A preliminary report. Spine, 16: 450-456.
Gordon, W.A. (2010) Perspectives on rehabilitation research, Archives of Physical Medicine and Rehabilitation, 91: 169-172.
Grabiner, M.D., Koh, T.J., and Ghazawi, A.E. (1992) Decoupling of bilateral excitation in subjects with low back pain. Spine, 17: 1219-1223.
Hadler, N. (2001) The bane of the aging worker [Editorial]. Spine, 26 (12): 1309-1310.
Hadler, N.M., Tait, R.C., and Chibnall, J.T. (2007) Back pain in the

workplace. Journal of the American Medical Association, 297 (14): 1594-1596.
Helmhout, P.H., Harts, C.C., Viechtbauer, W., and Staal, J.B. (2008) Isolated lumbar extensor strengthening versus regular physical therapy in an army working population with nonacute low back pain: A randomized controlled trial. Archives of Physical Medicine and Rehabilitation, 89: 1675-1685.
Herrin, G.A., Jaraiedi, M., and Anderson, C.K. (1986) Prediction of overexertion injuries using biomechanical and psychophysical models. American Industrial Hygiene Association Journal, 47: 322-330.
Hewett, T.E., Lindenfeld, T.N., Riccobene, J.V., and Noyes, F.R. (1999) The effect of neuromuscular training on the incidence of knee injury in female athletes: A prospective study, American Journal of Sports Medicine, 27: 699-706.
Hicks, G.E., Fritz, J.M., Delitto, A., and McGill, S.M. (2005) Preliminary development of a clinical prediction rule for determining which patients with low back pain will respond to a stabilization exercise program. Archives of Physical Medicine and Rehabilitation, 86 (9): 1753-1762.
Hides, J., Gilmore, C., Stanton, W., and Bohlscheid, E. (2008) Multifidus size and symmetry among chronic LBP and healthy asymptomatic subjects. Manual therapy, 13 (1): 43-49.
Hides, J.A., Stokes, M.J., Saide, M., Jull, G.A., and Cooper, D.H. (1994) Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. Spine, 19: 165-177.
Hodges, P.W., and Richardson, C.A. (1996) Inefficient muscular stabilization of the lumbar spine associated with low back pain. Spine, 21: 2640-2650.
Hodges, P.W., and Richardson, C.A. (1999) Altered trunk muscle recruitment in people with low back pain with upper limb movement at different speeds. Archives of Physical Medicine and Rehabilitation, 80: 1005-1012.
Ikeda, D., and McGill, S.M. (2012) Can altering motions, postures and loads provide immediate low back pain relief: A study of four cases investigating spine load, posture and stability. Spine, 37 (23): E1469-E1475.
Jager, M., Luttmann, A., and Laurig, W. (1991) Lumbar load during one-handed bricklaying. International Journal of Industrial Ergonomics, 8: 261-277.
Jonsson, H., Bring, G., Rauschning, W., and Shalstedt, B. (1991) Hidden cervical spine injuries in traffic accident victims with skull fractures. Journal of Spinal Disorders, 4 (3): 251-263.

Kelsey, J.L. (1975) An epidemiological study of the relationship between occupations and acute herniated lumbar intervertebral discs. International Journal of Epidemiology, 4: 197-205.

Kobayashi, Y., Kurata, J., Sekiguchi, M., Kokubun, M., Akaishizawa, T., Chiba, Y., Konno, S., and Kikuchi, S. (2009) Augmented cerebral activation by lumbar mechanical stimulus in chronic low back pain patients. Spine, 34 (22): 2431-2436.

Lepine, J.P., and Briley, M. (2004) The epidemiology of pain in depression. Human Psychopharmacology, 19: S3-S7.

Liira, J., Shannon, H.S., Chambers, L.W., and Haives, T.A. (1996) Long term back problems and physical work exposures in the 1990 Ontario health survey. American Journal of Public Health, 86 (3): 382-387.

Lis, A.M., Black, K M., Korn, H., and Nordin, M. (2007) Association between sitting and occupational LBP. European Spine Journal, 16 (2): 283-298.

Lord, S.M., Barnsley, L., Wallis, B.J., McDonald, G.J., and Bogduk, N. (1996) Percutaneous radiofrequency neurotomy for chronic cervical zygapophyseal joint pain. New England Journal of Medicine, 335: 1721-1726.

Luoto, S., Helioraara, M., Hurri, H., and Alavanta, M. (1995) Static back endurance and the risk of low back pain. Clinical Biomechanics, 10: 323-324.

Mannion, A.F., Junge, A., Taimela, S., Muntener, M., Lorenzo, K., and Dvorak, J. (2001) Active therapy for chronic low back pain. Part 3: Factors influencing self-rated disability and its change following therapy. Spine, 26: 920-929.

Marras, W. (2008) The working back. Hoboken, New Jersey: Wiley Interscience.

Marras, W.S., Davis, K.G., Heaney, C.A., Maronitis, A.B., and Allread, W.G. (2000) The influence of psychosocial stress, gender, and personality on mechanical loading of the lumbar spine. Spine, 25: 3045-3054.

Marras, W.S., Lavender, S.A., Leurgens, S.E., et al. (1993) The role of dynamic three-dimensional trunk motion in occupationally related low back disorders: The effects of workplace factors, trunk position and trunk motion characteristics on risk of injury. Spine, 18: 617-628.

Marras, W.S., Lavender, S.A., Leurgans, S.E., Fathallah, F.A., Ferguson, S.A., Allread, W.G., and Rajulu, S.L. (1995) Biomechanical risk factors

for occupationally related low back disorders. Ergonomics, 38: 377-410.
Marshall, P.W.M., Patel, H., and Callaghan, J.P. (2011) Gluteus medius strength, endurance, and co-activation in the development of low back pain during prolonged standing. Human Movement Science, 30 (1): 63-73.
McCall, I.W., Park, W.M., and O'Brien, J.P. (1979) Induced pain referral from posterior lumbar elements in normal subjects. Spine, 4: 441-446.
McGill, S.M. (1997) The biomechanics of low back injury: Implications on current practice in industry and the clinic. Journal of Biomechanics, 30: 465-475.
McGill SM. (1998) Low back exercises: Evidence for improving exercise regimens [Invited paper]. Physical Therapy, 78 (7): 754-765.
McGill, S.M. (2011a) Is a postural-structural-biomechanical model, within manual therapies, viable: AJBMT debate [Invited response]. Journal of Bodywork and Movement Therapy, 15 (2): 150-152.
McGill, S.M. (2011b) Occupational lifting is not related to low back pain [Letter to editor regarding Wei et al.]. The Spine Journal, 11: 365.
McGill, S.M. (2013) On the issue of clinical test reliability [Invited commentary]. Archives of Physical Medicine and Rehabilitation, 94: 1635-1637.
McGill, S.M., Grenier, S., Preuss, R.P., and Brown, S. (2003) Previous history of LBP with work loss is related to lingering effects in psychosocial, physiological, and biomechanical characteristics. Ergonomics, 46 (7): 731-746.
McGill, S.M., Sharratt, M.T., and Seguin, J.P. (1995) Loads on spinal tissues during simultaneous lifting and ventilatory challenge. Ergonomics, 38: 1772-1792.
McLean, S., and Beaulieu, M.L. (2010) Complex integrative morphological and mechanical contribution to ACL injury risk. Exercise and Sport Science Review, 38 (4): 192-200.
McLean, D., Pearce, N., Walls, C.B., and Wigley, R.D. (2011) The "Twin study" and the misunderstanding of epidemiology that clouds occupational associations and low back disorder. New Zealand Medical Journal, 124 (1339).
Melzack, R., and Wall, P.D. (1983) The challenge of pain. New York: Basic Books.
Mendelson, G. (1982) Not cured by a verdict: Effect of a level settlement on compensation claimants. Medical Journal of Australia, 2: 219-230.

Moneta, G.B., Viderman, T., Kaivanto, K., et al. (1994) Reported pain during lumbar discography as a function of anular ruptures and disc degeneration. A reanalysis of 833 discograms. Spine 19: 1968-1974.

Morl, F., and Bradl, I. (2013) Lumbar posture and muscular activity while sitting during office work. Journal of Electromyography and Kinesiology, 23 (2): 362-368.

National Institute for Occupational Safety and Health (NIOSH). (1981) Work practices guide for manual lifting. NIOSH Publication No. 81-122. Washington, DC: U.S. Department of Health and Human Services (DHHS).

Nelson-Wong, E., and Callaghan, J.P. (2010) Is muscle coactivation a causal factor for low back pain development during standing? A multifactorial approach for early identification of atrisk individuals. Journal of Electromyography and Kinesiology, 20 (2): 256-263.

Norman, R., Wells, R., Neumann, P., Frank, P., Shannon, H., and Kerr, M. (1998) A comparison of peak vs. cumulative physical work exposure risk factors for the reporting of low back pain in the automotive industry. Clinical Biomechanics, 13: 561-573.

Peyron, R., Laurent, B., and Garcia-Larrea, L. (2000) Functional imaging of brain responses to pain. A review and meta-analysis. Clinical Neurophysiology, 30: 263-288.

Pope, M.H. (1989) Risk indicators in low back pain. Annals of Medicine, 21: 387-392.

Porter, R.W. (1987) Does hard work prevent disc protrusion? Clinical Biomechanics, 2: 196-198.

Porter, R.W. (1992) Is hard work good for the back? The relationship between hard work and low back pain-related disorders. International Journal of Industrial Ergonomics, 9: 157-160.

Punnett, L., Fine, L.J., Keyerserling, W.M., Herrin, G.D., and Chaffin, D.A. (1991) Back disorders and non-neutral trunk postures of automobile assembly workers. Scandinavian Journal of Work Environment and Health, 17: 337-346.

Radanov, B.P., Sturzenegger, M., DeStefano, G., and Schinrig, A. (1994) Relationship between early somatic, radiological, cognitive and psychosocial findings and outcome during a one-year follow-up in 117 patients suffering from common whiplash. British Journal of Rheumatology, 33: 442-448.

Rafeemanesh, E., Jafari, Z., Kashani, F., and Rahimpour, F. (2013) A study on job postures and musculoskeletal illnesses in dentists. International Journal of Occupational Medicine and Environmental Health, 26 (4): 615-620.

Rainville, J., Sobel, J.B., Hartigan, C., and Wright, A. (1997) The effect of compensation involvement on the reporting of pain and disability by patients referred for rehabilitation of chronic low back pain. Spine, 22: 2016-2024.

Rantanen, J., Hurme, M., Falck, B., et al. (1993) The lumbar multifidus muscle five years after surgery for a lumbar intervertebral disc herniation. Spine, 18: 568-574.

Roudsari, B., and Jarvik, J.G. (2010) Lumbar spine MRI for low back pain: Indications and yield. American Journal of Roentgenology, 195 (3): 550-559.

Roy, T.C., Lopez, H.P., and Piva, S.R. (2013) Loads worn by soldiers predict episodes of low back pain during deployment in Afghanistan. Spine, 28 (15): 1310-1317.

Schwarzer, A.C., Wang, S., O'Driscoll, D., Harrington, T., Bogduk, N., and Laurent, R. (1995) The ability of computed tomography to identify a painful zygapophyseal joint in patients with low back pain. Spine, 20 (8): 907-912.

Sihvonen, T., Lindgren, K., Airaksinen, O., and Manninen, H. (1997) Movement disturbances of the lumbar spine and abnormal back muscle electromyographic findings in recurrent low back pain. Spine, 22: 289-295.

Silfies, S.P., Mehta, R., Smith, S.S., and Karduna, A.R. (2009) Differences in feedforward trunk muscle activity in subgroups of patients with mechanical low back pain. Archives of Physical Medicine and Rehabilitation, 90 (7): 1159-1169.

Skargren, E.I., Carlsson, P.G., and Oberg, B.E. (1998) One-year follow-up comparison of the cost and effectiveness of chiropractic and physiotherapy as primary management for back pain. Spine, 23 (17): 1875-1884.

Smeets, R.J., Vlaeyen, J.W., Kester, A.D., and Knottnerus, J.A. (2006) Reduction of pain catastrophising mediates the outcome of both physical and cognitive-behavioural treatment in chronic low back pain. Journal of Pain, 7 (4): 261-271.

Snook, S.H. (1982) Low back pain in industry. In: White, A.A., and Gordon, S.L. (Eds.), Symposium on idiopathic low back pain. St. Louis: Mosby.

Spearling, N.M., and Connelly, L.B. (2011) Is compensation "bad for health"? A systematic meta-review. Injury, 42: 15-24.

Sterling, M., Jull, G., and Wright, A. (2001) The effect of musculoskeletal pain on motor activity and control. Journal of Pain, 2 (3): 135-145.

Suni, J., Rinne, M., Natri, A., Statistisian, M.P., Parkkari, J., and Alaranta, H. (2006) Control of the lumbar neutral zone decreases low back pain and improves self-evaluated work ability: A 12-month randomized controlled study. Spine, 31 (18): E611-E620.

Suni, J.H., Taanila, H., Mattila, V.M., Ohrankammen, O., Vuorinen, P., Pihlajamaki, H., and Parkkari, J. (2013) Neuromuscular exercise and counseling decrease absenteeism due to low back pain in young conscripts. Spine, 38: 375-384.

Taylor, J.R., Twomey, L.T., and Corker, M. (1990) Bone and soft tissue injuries in post-mortem lumbar spines. Paraplegia, 28: 119-129.

Teasell, R.W. (1997) The denial of chronic pain. Journal of Pain Research Management, 2: 89-91.

Teasell, R.W., and Shapiro, A.P. (1998) Whiplash injuries: An update. Journal of Pain Research Management, 3: 81-90.

Thacker, M.A., Clark, A.K., et al. (2007) Pathophysiology of peripheral neuropathic pain: Immune cells and molecules. Anesthesia & Analgesia, 105: 838-847.

Thompson/Merskey/Teasell/Fordyce. (1996) Letters published in Pain, 65: 111-114.

Troup, J.D.G., Foreman, T.K., Baxter, C.E., and Brown, D. (1987) The perception of back pain and the role of psychological tests of lifting capacity. Spine, 12: 645-657.

Troup, J.D.G., Martin, J.W., and Lloyd, D.C.E.F. (1981) Back pain in industry—A prospective study. Spine, 6: 61-69.

U.S. Department of Labor. (1982) Back injuries associated with lifting (Bulletin 2144). Washington, DC: U.S. Government Printing Office.

Van der Windt, D., Hay, E., Jellema, P., and Main, C. (2008) Psychosocial interventions for low back pain in primary care: Lesson learned from recent trials. Spine, 33 (1): 81-89.

Van Middelkoop, M., Rubinstein, S.M., Verhagen, A.P., Ostelo, R.W., Koes, B.W., and van Tulder, M.W. (2010) Exercise therapy for chronic nonspecific low-back pain. Best Practice & Research Clinical Rheumatology, 24: 193-204.

Vanharanta, H., Sacks, B.L., Spivey, M.A., et al. (1987) The relationship of pain provocation to lumbar disc deterioration as seen by CT/discography. Spine, 12: 295-298.

Videman, T., Nurminen, M., and Troup, J.D. (1990) Lumbar spinal pathology in cadaveric material in relation to history of back pain, occupation and physical loading. Spine, 15: 728-740.

Waddell, G. (1987) A new clinical model for the treatment of low back pain. Spine, 12: 632-644.

Waddell, G., Bircher, M., Finlayson, D., and Main, C.J. (1984) Symptoms and signs: Physical disease or illness behaviour? British Medical Journal, 289: 739-741.

Waddell, G., McCulloch, J.A., Kummell, E., and Venner, R.M. (1980) Non-organic signs in low back pain. Spine, 5: 117-125.

Wallis, B.J., Lord, S.M., and Bogduk, N. (1997) Resolution of psychological distress of whiplash patients following treatment by radiofrequency neurotomy: A randomized double-blind, placebo controlled study. Pain, 73: 15-22.

Wang, S., and McGill, S.M. (2008) Links between the mechanics of ventilation and spine stability. Journal of Applied Biomechanics 24 (2): 166-174.

Wang, Y., Videman, T., and Battie, M.C. (2012) Lumbar vertebral endplate lesions: Associations with disc degeneration and back pain history [ISSLS prize winner]. Spine, 37 (17): 1490-1496.

Werneke, M., and Hart, D. (2001) Centralization phenomenon as a prognostic factor for chronic low back pain and disability. Spine, 26 (7): 758-765.

White, A.A., and Gordon, S.L. (1982) Synopsis: Workshop on idiopathic low back pain. Spine, 7: 141-149.

Woolf, C.J. (2010) Central sensitization: Implications for the diagnosis and treatment of pain. Pain, 152: S2-S15.

Yates, J.P., and McGill, S.M. (2011) The effect of vibration and posture on the progression of intervertebral disc herniation. Spine, 36 (5): 386-392.

Yates, J., Giangregorio, L., and McGill, S.M. (2010) The influence of international disc shape on the pathway of posterior/posterior lateral partial herniation. Spine, 35 (7): 734-739.

Yingling, V.R., and McGill, S.M. (2000) Anterior shear of spinal motion segments: Kinematics, kinetics and resulting injuries observed in a porcine model. Spine, 24 (18): 1882-1889.

Zedka, M., Prochazka, A., Knight, B., Gillard, D., and Gauthier, M. (1999) Voluntary and reflex control of human back muscles during induced pain. Journal of Physiology, 520 (2): 591-604.

Chapter 3: Functional Anatomy of the Lumbar Spine

Adams, M., and Dolan, P. (1995) Recent advances in lumbar spinal mechanics and their clinical significance. Clinical Biomechanics, 10 (1): 3.

Adams, M.A., and Dolan, P. (2005) Spine biomechanics. Journal of Biomechanics, 38: 1972-1983.

Adams, M.A., and Hutton, W.C. (1982) Prolapsed intervertebral disc: A hyperflexion injury. Spine, 7: 184.

Adams, M.A., and Hutton, W.C. (1985) Gradual disc prolapse. Spine, 10: 524.

Adams, M.A., Stefanakis, M., and Dolan, P. (2010) Healing of a painful intervertebral disc should not be confused with reversing disc degeneration: Implications for physical therapies for discogenic back pain. Clinical Biomechanics, 25: 961-971.

Adams, P., and Muir, H. (1976) Qualitative changes with age of proteoglycans of human lumbar discs. Annals of the Rheumatic Diseases, 35: 289.

Allison, G.T., Morris, S.L., and Lay, B. (2008) Feedforward responses of transversus abdominis are directionally specific and act asymmetrically: Implications for core stability theories. Journal of Orthopaedic & Sports Physical Therapy, 38 (5): 228-237.

Al-Rawahi, M., Luo, J., Pollintine, P., Dolan, P., and Adams, M.A. (2011) Mechanical function of vertebral body osteophytes as revealed by experiments on cadaveric spine. Spine, 36 (10): 770-777.

Aly, T., and Fuji, G. (2013) Hip morphology: Comparative study between Egyptians and Japanese adults. Journal of Musculoskeletal Research, 16 (03) Abstract only.

Amonoo-Kuofi, H.S. (1983) The density of muscle spindles in the medial, intermediate, and lateral columns of human intrinsic postvertebral muscles. Journal of Anatomy, 136: 509-519.

Andersson, E.A., Oddsson, L., Grundstrom, O.M., Nilsson, J., and Thorstensson, A. (1996) EMG activities of the quadratus lumborum and erector spinae muscles during flexion-relaxation and other motor tasks. Clinical Biomechanics, 11: 392-400.

Andersson, E., Oddsson, L., Grundstrom, H., and Thorstensson, A. (1995)

The role of the psoas and iliacus muscles for stability and movement of the lumbar spine, pelvis and hip. Scandinavian Journal of Medicine and Science in Sports, 5: 10-16.

Aultman, C.D., Drake, J., Callaghan, J.P., and McGill, S.M. (2004) The effect of static torsion on the compression strength of the spine: An invitro analysis using a porcine spine model. Spine, 29 (15): E304-309.

Aultman, C.D., Scannell, J., and McGill, S.M. (2005) Predicting the direction of nucleus tracking in bovine spine motion segments subjected to repetitive flexion and simultaneous lateral bend. Clinical Biomechanics, 20: 126-129.

Avela, J., Finni, T., Liikavainio, T., Neimela, E., and Komi, P. (2003) Neural and mechanical responses of the triceps surae muscle group after 1 hour of repeated fast passive stretches. Journal of Applied Physiology, 96: 2325-2332.

Balkovec, C., Carstensen, M., Leung, A., and McGill, S.M. (2014) A preliminary investigation into the morphology of trabecular bone damage associated with intervertebral disc herniation. Journal of Spine and Neurosurgery, 3 (6): doi 10.4172/2325-9701.1000162.

Balkovec, C., and McGill, S.M. (2012) Extent of nucleus pulposus migration in the annulus of intervertebral discs exposed to cyclic flexion only versus cyclic flexion and extension. Clinical Biomechanics, 27: 766-770.

Balkovec, C., Vernengo, J., and McGill, S.M. (2014) The use of a novel injectable hydrogel nucleus pulposus replacement in restoring the mechanical properties of cyclically fatigued porcine intervertebral discs. Journal of Biomechanical Engineering, 35 (6): 61004-61005.

Barker, P.J., Hapuarachchi, K.S., Ross, J.A., Sambaiew, E., Ranger, T.A., and Briggs, C.A. (2014) Anatomy and biomechanics of gluteus maximus and the thoracolumbar fascia at the sacroiliac joint. Clinical Anatomy, 27 (2): 234-240.

Bedzinski, R. (1992) Application of speckle photography methods to the investigations of deformation of the vertebral arch. In: Little, E.G. (Ed.), Experimental mechanics. New York: Elsevier.

Benson, R.T., Tavares, S.P., Robertson, S.C., Sharp, R., and Marshall, R.W. (2010) Conservatively treated massive prolapsed discs: A 7-year follow-up. Annals of the Royal College of Surgeons of England, 92: 147-153.

Black, J.D.J., and Stevens, E.D. (2001) Passive stretching does not protect against acute contraction-induced injury in mouse EDL muscle. Journal of Muscle Research and Cell Motility, 22: 301-310.

Bogduk, N. (1980) A reappraisal of the anatomy of the human lumbar erector spinae. Journal of Anatomy, 131 (3): 525.

Bogduk, N. (1983) The innervation of the lumbar spine. Spine, 8: 286.

Bogduk, N., and Engel, R. (1984) The menisci of the lumbar zygapophyseal joints: A review of their anatomy and clinical significance. Spine, 9: 454.

Bogduk, N., and Macintosh, J.E. (1984) The applied anatomy of the thoracolumbar fascia. Spine, 9: 164.

Bogduk, N., and Twomey, L. (1991) Clinical anatomy of the lumbar spine (2nd ed.). New York: Churchill Livingstone.

Brinckmann, P., Biggemann, M., and Hilweg, D. (1988) Fatigue fracture of human lumbar vertebrae. Clinical Biomechanics, 3 (Suppl. 1): S1-S23.

Brinckmann, P., Biggemann, M., and Hilweg, D. (1989) Prediction of the compressive strength of human lumbar vertebrae. Clinical Biomechanics, 4 (Suppl. 2).

Brown, S., Gregory, D., and McGill, S.M. (2008) Vertebral and plate fractures as a result as a result of high rate pressure loading in the nucleus of the young porcine spine. Journal of Biomechanics, 41 (1): 122-127.

Brown, S., and McGill, S.M. (2009) Transmission of muscularly generated force and stiffness between layers of the rat abdominal wall. Spine, 34 (2): E70-E75.

Brown, S., and McGill, S.M. (2008) An ultrasound investigation into the morphology of the human abdominal wall uncovers complex deformation patterns during contraction. European Journal of Applied Physiology, 104 (6): 1021-1030.

Brown, S.H.M., and McGill, S.M. (2010) A comparison of ultrasound and electromyography measures of force and activation to examine the mechanics of abdominal wall contraction. Clinical Biomechanics, 25: 115-123.

Butler, D.S. (1989) Adverse mechanical tension in the nervous system: A model for assessment and treatment, Australian Journal of Physiotherapy, 35: 227-238.

Butler, D.S. (2000) The sensitive nervous system. Australia: Noigroup Publications.

Callaghan, J.P., and McGill, S.M. (2001) Intervertebral disc herniation: Studies on a porcine model exposed to highly repetitive flexion/extension motion with compressive force. Clinical Biomechanics, 16 (1): 28-37.

Carr, D., Gilbertson, L., Frymeyer, J., Krag, M., and Pope, M. (1985) Lumbar paraspinal compartment syndrome: A case report with physiologic and anatomic studies. Spine, 10: 816.

Cavanaugh, J.M. (1995) Neural mechanisms of lumbar pain. Spine, 20 (16): 1804.

Cavanaugh, J.M., Ozaktay, C.A., Yamashita, T., and Hing, A.I. (1996) Lumbar facet pain: Biomechanics, neuroanatomy and neurophysiology. Journal of Biomechanics, 29: 1117-1129.

Cholewicki, J., Juluru, K., and McGill, S.M. (1999) The intra-abdominal pressure mechanism for stabilizing the lumbar spine. Journal of Biomechanics, 32 (1): 13-17.

Cresswell, A.G., Oddsson, L., and Thorstensson, A. (1994) The influence of sudden perturbations on trunk muscle activity in intraabdominal pressure while standing. Experimental Brain Research, 98: 336-341.

Cripton, P., Berlemen, U., Visarino, H., Begeman, P.C., Nolte, L.P., and Prasad, P. (1995) Response of the lumbar spine due to shear loading. In: Injury prevention through biomechanics (p. 111). Detroit: Wayne State University.

Crossman, K., Mahon. M., Watson, P., Oldham, J., and Cooper, R. (2004) Chronic low back pain-associated paraspinal muscle dysfunction is not the result of a constitutionally determined adverse fibre-type composition. Spine, 29 (6): 628-634.

D'Ambrosia, P., King, K., Davidson, B., Zhou, B., Lu, Y., and Solomonow, M. (2010) Pro-inflammatory cytokines expression increases following low and high magnitude cyclic loading of lumbar ligaments. European Spine Journal. doi:10.1007/s00586-010-1371-4

Dickey, J.P., Pierrynowski, M.R., and Bednar, D.A. (1996, May 25) Deformation of vertebrae in vivo—Implications for facet joint loads and spinous process pin instrumentation for measuring sequential spinal kinematics. Presented at the Canadian Orthopaedic Research Society, Quebec City.

Drake, J.D., Aultman, C.D., McGill, S.M., and Callaghan, J.P. (2005) The influence of static axial torque in combined loading on intervertebral joint failure mechanics using a porcine model. Clinical Biomechanics, 20 (10): 1038-1045.

Farfan, H.F. (1973) Mechanical disorders of the low back. Philadelphia: Lea and Febiger.

Fowles, J.R., Sale, D.G., and MacDougall, J.D. (2000) Reduced strength after

passive stretch in the human plantar flexors. Journal of Applied Physiology, 89: 1179-1188.
Freeman, S., Mascia, A., and McGill, S.M. (2013) Arthrogenic neuromuscular inhibition: A foundational investigation of existence in the hip joint. Clinical Biomechanics, 28: 171-177.
Fyhrie, D.P., and Schaffler, M.B. (1994) Failure mechanisms in human vertebral cancellous bone. Bone, 15 (1): 105-109.
Gallois, J., and Japoit, T. (1925) Architecture intérieure des vertèbres du point de vue statique et physiologique. Rev Chir (Paris), 63: 687-708.
Ge, W., and Pickar, J.G. (2012) The decreased responsiveness of lumbar muscle spindles to a prior history of spinal muscle lengthening is graded with the magnitude of change in vertebral position. Journal of Electromyography and Kinesiology, 22: 814-820.
Gliedt, J.A., and Scali, F. (2012) Femoroacetabular inpingement syndrome. Topics in Integrated Health Care, 3 (2): ID 3.2004.
Goel, V.K., Monroe, B.T., Gilbertson, L.G., and Brinckmann, P. (1995) Interlaminar shear stresses and laminae-separation in a disc: Finite element analysis of the L3-L4 motion segment subjected to axial compressive loads. Spine, 20 (6): 689.
Goel, V.K., Wilder, D.G., Pope, M.H., and Edwards, W.T. (1995) Controversy: Biomechanical testing of the spine: Load controlled versus displacement controlled analysis. Spine, 20: 2354-2357.
Gordon, S.J., et al. (1991) Mechanism of disc rupture—A preliminary report. Spine, 16: 450.
Gracovetsky, S., Farfan, H.F., and Lamy, C. (1981) Mechanism of the lumbar spine. Spine, 6 (1): 249.
Gunning, J.L., Callaghan, J.P., and McGill, S.M. (2001) The role of prior loading history and spinal posture on the compressive tolerance and type of failure in the spine using a porcine trauma model. Clinical Biomechanics, 16 (6): 471-480.
Guyton, A.C. (1981) Sensory receptors and their basic mechanisms of action. In: Textbook of medical physiology (6th ed., p. 588). Philadelphia: W.B. Saunders.
Hardcastle, P., Annear, P., and Foster, D. (1992) Spinal abnormalities in young fast bowlers. Journal of Bone and Joint Surgery, 74B (3): 421.
Henke, K.G., Sharratt, M.T., Pegelow, D., and Dempsey, J.A. (1988) Regulation of end-expiratory lung volume during exercise. Journal of

Applied Physiology, 64: 135-146.
Heylings, D.J.A. (1978) Supraspinous and interspinous ligaments of the human lumbar spine. Journal of Anatomy, 123: 127.
Hides, J.A., Stokes, M.J., Saide, M., Jull, G.A., and Cooper, D.H. (1994) Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. Spine, 19 (2): 165-172.
Hodges, P.W., and Richardson, C.A. (1996) Inefficient muscular stabilisation of the lumbar spine associated with low back pain: A motor control evaluation of transversus abdominis. Spine, 21: 2640-2650.
Howe, J.F., Loeser, J.D., and Calvin, W.H. (1977) Mechanosensitivity of dorsal root ganglia and chronically ignored axons: A physiological basis for the radicular pain of nerve root compression. Pain, 3: 25.
Hubbard, R.D., and Winkelstein, B.A. (2005) Transient cervical nerve root compression in the rat induces bilateral forepaw allodynia and spinal glial activation: Mechanical factors in painful neck injuries. Spine, 30: 1924-1932.
Hubbard, R.D., Chen, Z., and Winkelstein, B.A (2007) Transient cervical nerve root compression modulates pain: Load thresholds for allodynia and sustained changes in spinal neuropeptide expression. Journal of Biomechanics, 41 (3): 677-685.
Inoue, K., et al. (2000) Prevalence of hip osteoarthritis and acetabular dysplasia in French and Japanese adults Rheumatology, 39 (7): 745-748.
Jiang, H.J., Russell, G., Raso, J., Moreau, M.J., Hill, D.J., and Bagnall, K.M. (1995) The nature and distribution of the innervation of human supraspinal and interspinal ligaments. Spine, 20: 869-876.
Juker, D., McGill, S.M., and Kropf, P. (1998) Quantitative intramuscular myoelectric activity of lumbar portions of psoas and the abdominal wall during cycling. Journal of Applied Biomechanics, 14 (4): 428-438.
Juker, D., McGill, S.M., Kropf, P., and Steffen, T. (1998) Quantitative intramuscular myoelectric activity of lumbar portions of psoas and the abdominal wall during a wide variety of tasks. Medicine & Science in Sports & Exercise, 30 (2): 301-310.
Kader, D.F., Wardlaw, D., and Smith, F.W. (2000) Correlation between the MRI changes in the lumbar multifidus muscles and leg pain. Clinical Radiology, 55 (2): 145-149.
Kandel, E.R., Schwartz, J.H., and Jessell, T.M., (Eds.). (2000) Principles of neural science (4th ed.). New York: McGraw-Hill (Health Professions

Division).
Keller, T.S., Ziv, I., Moeljanto, E., and Spengler, D.M. (1993) Interdependence of lumbar disc and subdiscal bone properties: A report of the normal and degenerated spine. Journal of Spinal Disorders, 6 (2): 106-113.
Kelly, M., and Robinson, C.M. (2003) Fractures of the thoracolumbar vertebrae from sledging: A recurrent British winter problem. Injury, 1906: 1-2.
King, A.I. (1993) Injury to the thoraco-lumbar spine and pelvis. In: Nahum, H.M., and Melvin, J. (Eds.), Accidental injury, biomechanics and prevention. New York: Springer.
Kirkaldy-Willis, W.H., and Burton, C.V. (1992) Managing low back pain (3rd ed.). New York: Churchill Livingstone.
Kobayashi, S., Kenichi, T., Takafumi, Y., Kousuke, A., Miyazaki, T., Guerrero, A., and Hisatoshi, B. (2010) Pathomechanisms of sciatica in lumbar disc herniation: Effect of periradicular adhesive tissue on electrophysiological values by intraoperative straight leg raising test. Spine, 35 (22): 2004-2014.
Le Cara, E., Marcus, R., Dempsey, A., Hoffman, M., and Hebert, J. (2014) Morphology vs. function: The relationship between lumbar multifidus intramuscular adipose tissue and muscle function among patients with low back pain. Archives of Physical Medicine and Rehabilitation, 10: 1848-1842.
Lehman, G., and McGill, S.M. (2001) Quantification of the differences in EMG magnitude between upper and lower rectus abdominis during selected trunk exercises. Physical Therapy, 81: 1096-1101.
Lehman, G., and McGill, S.M. (1999a) The importance of normalization in the interpretation of surface electromyography: A proof of principle. Journal of Manipulative and Physiological Therapeutics, 22 (7): 444-446.
Lehman, G., and McGill, S.M. (1999b) Influence of chiropractic manipulation on lumbar kinematics and EMG during simple and complex tasks: A case study. Journal of Manipulative and Physiological Therapeutics, 22 (9): 576-581.
Loder, R.T., and Skopelja, E.N. (2011) The epidemiology and demographics of hip dysplasia. ISRN Orthopaedics. http://dx.doi.org/10.5402/2011/238607
Lotz, J.C., and Chin, J.R. (2000) Intervertebral disc cell death is dependent on

the magnitude and duration of spinal loading. Spine, 25 (12): 1477-1483.

Lu, W.W., Luk, K.D.K., Cheung, K.M.C., Fang, D., Holmes, A.D., and Leong, J.C.Y. (2001, June 19-23) Energy absorption of human vertebral body under fatigue loading. In: Abstracts, International Society for Study of the Lumbar Spine, Edinburgh, Scotland.

Lucas, D., and Bresler, B. (1961) Stability of the ligamentous spine. Tech. Report No. 40, Biomechanics Laboratory, University of California, San Francisco.

Macintosh, J.E., and Bogduk, N. (1987) The morphology of the lumbar erector spinae. Spine, 12 (7): 658.

Macintosh, J.E., Bogduk, N., and Gracovetsky, S. (1987) The biomechanics of the thoracolumbar fascia. Clinical Biomechanics, 2: 78.

Markolf, K.L., and Morris, J.M. (1974) The structural components of the intervertebral disc. Journal of Bone and Joint Surgery, 56A (4): 675.

Marras, W.S., and Granata, K.P. (1997) Changes in trunk dynamics and spine loading during repeated exertions. Spine, 22: 2564-2570.

Marras, W.S., Jorgensen, M.J., Granata, K.P., and Waind, B. (2001) Female and male trunk geometry: Size and prediction of the spine loading trunk muscles derived from MRI. Clinical Biomechanics, 16: 38-46.

Marshall, L., and McGill, S.M. (2010) The role of axial torque/twist in disc herniation. Clinical Biomechanics, 25 (1): 6-9.

McGill, S.M. (1991a) Electromyographic activity of the abdominal and low back musculature during the generation of isometric and dynamic axial trunk torque: Implications for lumbar mechanics. Journal of Orthopaedic Research, 9: 91.

McGill, S.M. (1991b) The kinetic potential of the lumbar trunk musculature about three orthogonal orthopaedic axes in extreme postures. Spine, 16 (7): 809-815.

McGill, S.M. (1992) A myoelectrically based dynamic 3-D model to predict loads on lumbar spine tissues during lateral bending. Journal of Biomechanics, 25 (4): 395.

McGill, S.M. (1996) A revised anatomical model of the abdominal musculature for torso flexion efforts. Journal of Biomechanics, 29 (7): 973-977.

McGill, S.M. (1997) Biomechanics of low back injury: Implications on current practice and the clinic [Invited paper]. Journal of Biomechanics, 30 (5): 465-475.

McGill, S.M. (2014) Ultimate back fitness and performance. Backfitpro Inc. (www.backfitpro.com)
McGill, S.M. (2014) Ultimate back fitness and performance (5th ed.). Waterloo, ON: Backfitpro. (www.backfitpro.com)
McGill, S.M. (2015) Back mechanic: The step-by-step McGill method to fix back pain. Backfitpro (www.backfitpro.com)
McGill, S.M., Hughson, R.L., and Parks, K. (2000) Changes in lumbar lordosis modify the role of the extensor muscles. Clinical Biomechanics, 15 (1): 777-780.
McGill, S.M., Juker, D., and Axler, C. (1996) Correcting trunk muscle geometry obtained from MRI and CT scans of supine postures for use in standing postures. Journal of Biomechanics, 29 (5): 643-646.
McGill, S.M., Juker, D., and Kropf, P. (1996a) Appropriately placed surface EMG electrodes reflect deep muscle activity (psoas, quadratus lumborum, abdominal wall) in the lumbar spine. Journal of Biomechanics, 29 (11): 1503-1507.
McGill, S.M., Juker, D., and Kropf, P. (1996b) Quantitative intramuscular myoelectric activity of quadratus lumborum during a wide variety of tasks. Clinical Biomechanics, 11 (3): 170-172.
McGill, S.M., and Norman, R.W. (1987) Effects of an anatomically detailed erector spinae model on L4/L5 disc compression and shear. Journal of Biomechanics, 20 (6): 591.
McGill, S.M., and Norman, R.W. (1988) The potential of lumbodorsal fascia forces to generate back extension moments during squat lifts. Journal of Biomedical Engineering, 10: 312.
McGill, S.M., Patt, N., and Norman, R.W. (1988) Measurement of the trunk musculature of active males using CT scan radiography: Duplications for force and moment generating capacity about the L4/L5 joint. Journal of Biomechanics, 21 (4): 329.
McGill, S.M., Santaguida, L., and Stevens, J. (1993) Measurement of the trunk musculature from T6 to L5 using MRI scans of 15 young males corrected for muscle fibre orientation. Clinical Biomechanics, 8: 171.
McLain, R.F., and Pickar, J.G. (1998) Mechanoreceptor endings in human thoracic and lumbar facet joints. Spine, 23: 168-173.
Miyazaki, T., Takafumi, Y., Kubota, M., Nomura, E., Mwaka, E., Baba, H., Kobayashi, S., Meir, A., Kokubo, Y., and Takeno, K. (2009) Ultrastructural analysis on lumbar disc herniation using surgical

specimens: Role of neovascularization and macrophages in hernias. Spine, 34 (7): 655-662.
Modic, M.T., Steinberg, P.M., Ross, J.S., Masaryk, T.J., and Carter, J.R. (1988) Degenerative disk disease: Assessment of changes in vertebral body marrow with MR imaging. Radiology, 166 (1 Pt 1): 193-199.
Moreside, J., and McGill, S.M. (2011) Quantifying normal 3D hip range of motion in healthy adult males with clinical and laboratory tools: Hip mobility restrictions appear to be plane specific. Clinical Biomechanics, 26: 824-829.
Moreside, J.M., and McGill, S.M. (2012) Hip joint ROM improvements using 3 different interventions. Journal of Strength and Conditioning Research, 26 (5): 1265-1273.
Moreside, J.M., Vera-Garcia, F., and McGill, S.M. (2008) Neuromuscular independence of abdominal wall muscles as demonstrated by middle-eastern style dancers. Journal of Electromyography and Kinesiology, 18: 527-537.
Myers, T.W. (2014) Anatomy trains: Myofascial meridians for manual and movement therapists. Churchill Livingstone.
Nachemson, A.L. (1960) Lumbar interdiscal pressure. Acta Orthopaedica Scandinavica (Suppl. 43).
Nachemson, A. (1966) The load on lumbar discs in different positions of the body. Clinical Orthopaedics and Related Research, 45: 107.
Nitz, A.J., and Peck, D. (1986) Comparison of muscle spindle concentrations in large and small human epaxial muscles acting in parallel combinations. American Surgeon, 52: 273-277.
Norman, R.W., Wells, R., Neumann, P., Frank, J., Shannon, H., and Kerr, M. (1998) A comparison of peak vs. cumulative physical work exposure factors for the reporting of low back pain in the automotive industry. Clinical Biomechanics, 13: 561-573.
Noyes, F.R., De Lucas, J.L., and Torvik, P.J. (1994) Biomechanics of ligament failure: An analysis of strain-rate sensitivity and mechanisms of failure in primates. Journal of Bone and Joint Surgery, 56A: 236.
Olmarker, K., Rydevik, B., and Holm, S. (1989) Edema formation in spinal nerve roots induced by experimental graded compression. Spine, 14 (6): 569-573.
Ozaktay, A.C., et al. (1994) Effects of carrageenan-induced inflammation in rabbit lumbar facet joint capsule and adjacent tissue. Journal of

Neuroscience Research, 20: 355.
Paalanne, N., Jaakko, N., Karppinen, J., Taimela, S., Mutanen, P., Takatalo, J., Korpelainen, R., and Tervonen, O. (2011) Assessment of association between low back pain and paraspinal muscle atrophy using opposed-phase magnetic resonance imaging: A population-based study among young adults. Spine, 36 (23): 1961-1968.
Park, R.J., Tsao, H., Cresswell, A.G., and Hodges, P.W. (2013) Changes in direction-specific activity of psoas major and quadratus lumborum in people with recurring back pain differ between muscle regions and patient groups. Journal of Electromyography and Kinesiology, 23 (3): 734-740.
Porterfield, J.A., and DeRosa, C. (1998) Mechanical low back pain: Perspectives in functional anatomy. Philadelphia: W.B. Saunders.
Ranawat, V.S., Dowell, J.K., and Heywood-Waddington, M.B. (2003) Stress fractures of the lumbar pars interarticularis in athletes: A review based on long-term results of 18 professional cricketers. Injury, 34 (12): 915-919.
Richardson, C., Jull, G., Hodges, P., and Hides, J. (1999) Therapeutic exercise for spinal segmental stabilization in low back pain. Edinburgh: Churchill Livingstone.
Rissanen, P.M. (1960) The surgical anatomy and pathology of the supraspinous and interspinous ligaments of the lumbar spine with special reference to ligament ruptures. Acta Orthopaedic Scandinavica (Suppl. 46).
Roaf, R. (1960) A study of the mechanics of spinal injuries. Journal of Bone and Joint Surgery, 42B: 810.
Roberts, S., Menage, J., and Urban, J.P.G. (1989) Biochemical and structural properties of the cartilage end-plate and its relationship to the intervertebral disc. Spine, 14: 166.
Santaguida, P., and McGill, S.M. (1995) The psoas major muscle: A three dimensional mechanical modelling study with respect to the spine based on MRI measurement. Journal of Biomechanics, 28: 339-345.
Scannell, J., and McGill, S.M. (2005, May 26-28) Spinal disc prolapse caused by flexion can be reduced by extension: An intro study of disc mechanics. Waterloo, ON: Canadian Biomaterials Society.
Scannell, J.P., and McGill, S.M. (2009) Disc prolapse: Evidence of reversal with repeated extension. Spine, 34 (4): 344-350.
Sharma, M., Langrama, N.A., and Rodriguez, J. (1995) Role of ligaments and facets in lumbar spine stability. Spine, 20 (8): 887.
Silfies, S.P., Mehta, R., Smith, S.S., and Karduna, A.R. (2009) Differences in

feedforward trunk muscle activity in subgroups of patients with mechanical low back pain. Archives of Physical Medicine and Rehabilitation, 90 (7): 1159-1169.
Silva, M.J., and Gibson, L.J. (1997) Modeling the mechanical behaviour of vertebral trabecular bone: Effects of age-related changes in microstructure. Bone, 21: 191-199.
Sirca, A., and Kostevc, V. (1985) The fibre type composition of thoracic and lumbar paravertebral muscles in man. Journal of Anatomy, 141: 131.
Skrzpiec, D., Tarala, M., Pollintine, P., Dolan, P., and Adams, M.A. (2007) When are intervertebral discs stronger than their adjacent vertebrae? Spine, 32 (22): 2455-2461.
Solomonow, M., Zhou, B., Harris, M., Lu, Y., and Baratta, R.V. (2000) The ligamento-muscular stabilizing system of the spine. Spine, 23: 2552-2562.
Styf, J. (1987) Pressure in the erector spinae muscle during exercise. Spine, 12: 675.
Tampier, C., Drake, J., Callaghan, J., and McGill, S.M. (2007) Progressive disc herniation: An investigation of the mechanism using radiologic, histochemical and microscopic dissection techniques. Spine, 32 (25): 2869-2874.
Tesh, K.M., Dunn, J., and Evans, J.H. (1987) The abdominal muscles and vertebral stability. Spine, 12 (5): 501.
Twomey, L., and Taylor, J.R. (1987) Physical therapy of the low back. New York: Churchill Livingstone.
Vera-Garcia, F.J., Grenier, S.G., and McGill, S.M. (2000) Abdominal response during curl-ups on both stable and labile surfaces. Physical Therapy, 80 (6): 564-569.
Vera Garcia, F.J., Moreside, J.M., and McGill, S.M. (2011) Abdominal muscle activation changes if the purpose is to control pelvis motion or thorax motion. Journal of Electromyography and Kinesiology, 21: 893-903.
Veres, S.P., Robertson, P.A., and Broom, N.D. (2009) The morphology of acute disc herniation: A clinically relevant model defining the role of flexion. Spine, 34 (21): 2288-2296.
Videman, T., Nurminen, M., and Troup, J.D.G. (1990) Lumbar spinal pathology in cadaveric material in relation to history of back pain, occupation and physical loading. Spine, 15 (8): 728.
Ward, S.R., Kim, C.W., Eng, C.M., Gottschalk, L.J., Tomiya, A., Garfin, S.,

and Lieber, R. (2009b) Architectural analysis and intraoperative measurements demonstrate the unique design of the multifidus muscle for lumbar spine stability. Journal of Bone and Joint Surgery, 91(1): 176-185.
Ward, S.R., Tomiya, A., Regev, G., Thacker, B., Benzl, R., and Lieber, R. (2009a) Passive mechanical properties of the lumbar multifidus supports its role as a stabilizer. Journal of Biomechanics, 42(10): 1384-1389.
Watkins, J. (1999) Structure and function of the musculoskeletal system. Champaign, IL: Human Kinetics.
Wilder, D.G., Pope, M.H., and Frymoyer, J.W. (1988) The biomechanics of lumbar disc herniation and the effect of overload and instability. Journal of Spinal Disorders, 1 (1): 16.
Winkelstein, B.A., Rutkowski, M.D., Sweitzer, S.M., Pahl, J.L., and DeLeo, J.A. (2001) Nerve injury proximal or distal to the DRG induces similar spinal glial activation and selective cytokine expression but differential behavioral responses to pharmacologic treatment. Journal of Comparative Neurology 439: 127-139.
Yahia, L.H., Newman, N., and Rivard, C.H. (1988) Neurohistology of the lumbar spine. Acta Orthopaedic Scandinavica, 59: 508-512.
Yates, J.P., Giangregorio, L., and McGill, S.M. (2010) The influence of intervertebral disc shape on the pathway of posterior/posterior lateral partial herniation. Spine, 35 (7): 734-739.
Yates, J.P., and McGill, S.M. (2011) The effect of vibration and posture on the progression of intervertebral disc herniation. Spine, 36 (5): 386-392. 1-16.
Yingling, V.R., Callaghan, J.P., and McGill, S.M. (1999) The porcine cervical spine as a reasonable model of the human lumbar spine: An anatomical, geometrical and functional comparison. Journal of Spinal Disorders, 12 (5): 415-423.
Yingling, V.R., and McGill, S.M. (1999a) Mechanical properties and failure mechanics of the spine under posterior shear load: Observations from a porcine model. Journal of Spinal Disorders, 12 (6): 501-508.
Yingling, V.R., and McGill, S.M. (1999b) Anterior shear of spinal motion segments: Kinematics, kinetics and resulting injuries observed in a porcine model. Spine, 24 (18): 1882-1889.
Young, S., Aprill, C., and Laslett, M. (2003) Correlation of clinical examination characteristics with three sources of chronic low back pain. The Spine Journal, 3: 460-465.

Zielinski, K.A., Henry, S.M., Ouellette-Morton, R.H., and DeSamo, M.J. (2013) Lumbar multifidus muscle thickness does not predict patients with low back pain who improve with trunk stabilization exercises. Archives of Physical Medicine and Rehabilitation. doi:10.1016/j.apmr.2012.12.001

Zhi, S., Fan, S., Xie, Q., Suyou, L., Liu, J., Wang, C., and Zhao, F. (2014) Spontaneous resorption of lumbar disc herniation is less likely when Modic changes are present. Spine, 39 (9): 736-744.

Chapter 4: Normal and Injury Mechanics of the Lumbar Spine

Adams, M., and Dolan, P. (1995) Recent advances in lumbar spinal mechanics and their clinical significance. Clinical Biomechanics, 10 (1): 3.

Adams, M.A., Dolan, P., and Hutton, W.C. (1987) Diurnal variations in the stresses on the lumbar spine. Spine, 12 (2): 130.

Adams, M.A., and Hutton, W.C. (1985) Gradual disc prolapse. Spine, 10: 524.

Adams, M.A., and Hutton, W.C. (1988) Mechanics of the intervertebral disc. In: Ghosh, P. (Ed.), The biology of the intervertebral disc. Boca Raton, FL: CRC Press.

Adams, M.A., McNally, D.S., Chinn, H., and Dolan, P. (1994) Posture and the compressive strength of the lumbar spine. Clinical Biomechanics, 9: 5-14.

Aggrawall, N.D., Kavr, R., Kumar, S., and Mathur, D.N. (1979) A study of changes in the spine in weightlifters and other athletes. British Journal of Sports Medicine, 13: 58-61.

Albert, H.B., Sorensen, J.S., Christensen, B.S., and Manniche, C. (2013) Antibiotic treatment in patients with chronic low back pain and vertebral bone edema (Modic type 1 changes): A double-blind randomized clinical controlled trial of efficacy. European Spine Journal, 22 (4): 697-707.

Arendt-Nielson, L., Graven-Neilson, T., Svarrer, H., and Svensson, P. (1995) The influence of low back pain on muscle activity and coordination during gait. Pain, 64: 231-240.

Ashton-Miller, J.A., and Schultz, A.B. (1988) Biomechanics of the human spine and trunk. In: Pandolf, K.B. (Ed.), Exercise and sport science reviews (Vol. 16), American College of Sports Medicine Series. New York: Macmillan.

Aultman, C.D., Scannell, J., and McGill, S.M. (2005) Predicting the direction of nucleus tracking in porcine spine motion segments subjected to repetitive flexion and simultaneous lateral bend. Clinical Biomechanics,

20: 126-129.
Axler, C., and McGill, S.M. (1997) Low back loads over a variety of abdominal exercises: Searching for the safest abdominal challenge. Medicine & Science in Sports & Exercise, 29 (6): 804-811.
Balkovec, C., Carstensen, M.H., Leung, A., and McGill, S.M. (2014) A preliminary investigation into the morphology of trabecular bone damage associated with intervertebral disc herniation. Journal of Spine and Neurosurgery, 3 (6). doi:10.4172/2325-9701.1000162
Balkovec, C., and McGill, S.M. (2012) Extent of nucleus pulposus migration in the annulus of intervertebral discs exposed to cyclic flexion only versus cyclic flexion and extension. Clinical Biomechanics, 27: 766-770.
Banerjee, P., Brown, S., Howarth, S., and McGill, S.M. (2009) Torso and hip muscle activity and resulting spine load and stability while using the Profitter 3-D Cross Trainer. Journal of Applied Biomechanics, 25: 73-84.
Barton, C.J., Coyle, J.A., and Tinley, P. (2009) The effect of heel lifts on trunk muscle activation during gait: A study of young healthy females. Journal of Electromyography and Kinesiology, 19: 598-606.
Bearn, J.G. (1961) The significance of the activity of the abdominal muscles in weight lifting. Acta Anatomica, 45: 83.
Berkson, M.H., Nachemson, A.L., and Shultz, A.B. (1979) Mechanical properties of human lumbar spine motion segments. Part II: Responses in compression and shear: Influence of gross morphology. Journal of Biomechanical Engineering, 101: 53.
Bogduk, N. (1980) A reappraisal of the anatomy of the human lumbar erector spinae. Journal of Anatomy, 131 (3): 525.
Bogduk, N., Derby, R., Aprill, C., Louis, S., and Schwartzer, R. (1996) Precision diagnosis in spinal pain. In: Campbell, J. (Ed.). Pain 1996— An updated view (pp. 313-323). Seattle: IASP Press.
Bogduk, N., and Macintosh, J.E. (1984) The applied anatomy of the thoracolumbar fascia. Spine, 9: 164.
Brinckmann, P. (1985) Pathology of the vertebral column. Ergonomics, 28: 235-244.
Brinckmann, P., Biggemann, M., and Hilweg, D. (1988) Prediction of the compressive strength of human lumbar vertebrae. Clinical Biomechanics, 4 (Suppl. 2).
Buckwalter, J.A. (1995) Spine update: Ageing and degeneration of the human intervertebral disc. Spine, 20: 1307-1314.

Burnett, A.F., Khangure, M., Elliot, B.C., Foster, D.H., Marshall, R.N., and Hardcastle, P. (1996) Thoracolumbar disc degeneration in young fast bowlers in cricket. A follow-up study. Clinical Biomechanics, 11: 305-310.

Butler, D., Trafimow, J.H., Andersson, G.B.J., McNiell, T.W., and Hackman, M.S. (1990) Discs degenerate before facets. Spine, 15: 111-113.

Callaghan, J.P., Gunning, J.L., and McGill, S.M. (1998) Relationship between lumbar spine load and muscle activity during extensor exercises. Physical Therapy, 78 (1): 8-18.

Callaghan, J.P., and McGill, S.M. (2001a) Intervertebral disc herniation: Studies on a porcine model exposed to highly repetitive flexion/extension motion with compressive force. Clinical Biomechanics, 16 (1): 28-37.

Callaghan, J.P., and McGill, S.M. (2001b) Low back joint loading and kinematics during standing and unsupported sitting. Ergonomics, 44 (4): 373-381.

Callaghan, J.P., Patla, A.E., and McGill, S.M. (1999) Low back three-dimensional joint forces, kinematics and kinetics during walking. Clinical Biomechanics, 14: 203-216.

Carr, D., Gilbertson, L., Frymeyer, J., Krag, M., and Pope, M. (1985) Lumbar paraspinal compartment syndrome: A case report with physiologic and anatomic studies. Spine, 10: 816.

Casthanhero, R., Duarte, M., and McGill, S.M. (2014) Corrective sitting strategies: An examination of muscle activity and spine load. Journal of Electromyography and Kinesiology, 24 (1): 114-119.

Chin, J.R., Liebenberg, E., Colliou, O.K., and Lotz, J.C. (2007) Biological and mechanical consequences of transient intervertebral disc bending. European Spine Journal, 16 (11): 1899-1906.

Cholewicki, J., Juluru, K., and McGill, S.M. (1999) The intra-abdominal pressure mechanism for stabilizing the lumbar spine. Journal of Biomechanics, 32 (1): 13-17.

Cholewicki, J., Juluru, K., Radebold, A., Panjabi, M.M., and McGill, S.M. (1999) Lumbar spine stability can be augmented with an abdominal belt and/or increased intra-abdominal pressure. European Spine Journal, 8: 388-395.

Cholewicki, J., and McGill, S.M. (1992) Lumbar posterior ligament involvement during extremely heavy lifts estimated from fluoroscopic measurements. Journal of Biomechanics, 25 (1): 17.

Cholewicki, J., and McGill, S.M. (1996) Mechanical stability of the in vivo lumbar spine: Implications for injury and chronic low back pain. Clinical Biomechanics, 11 (1): 1-15.

Cholewicki, J., McGill, S.M., and Norman, R.W. (1991) Lumbar spine loads during lifting extremely heavy weights. Medicine & Science in Sports & Exercise, 23 (10): 1179-1186.

Cripton, P., Berlemen, U., Visarino, H., Begeman, P.C., Nolte, L.P., and Prasad, P. (1995) Response of the lumbar spine due to shear loading. In: Injury prevention through biomechanics (p. 111). Detroit: Wayne State University.

Crisco, J.J., and Panjabi, M.M. (1990) Postural biomechanical stability and gross muscular architecture in the spine. In: Winters, J., and Woo, S. (Eds.), Multiple muscle systems (p. 438). New York: Springer-Verlag.

D'Ambrosia, P., King, K., Davidson, B., Zhou, B., Lu, Y., and Solomonow, M. (2010) Pro-inflammatory cytokines expression increases following low and high magnitude cyclic loading of lumbar ligaments. European Spine Journal. doi:10.1007/s00586-010-1371-4

Dangaria, T.R., and Naesh, O. (1998) Changes in cross-sectional area of psoas major muscle in unilateral sciatica caused by disc herniation. Spine, 23 (8): 928-931.

Danneels, L.A., Vanderstraeten, G.G., Cambier, D.C., Witrouw, E.E., Bourgois, J., Dankaerts, W., and De Cuyper, H.J. (2001) Effects of three different training modalities on the cross sectional area of the lumbar multifidus muscle in patients with chronic low back pain. British Journal of Sports Medicine, 35: 186-191.

Davis, P.R. (1959) The causation of herniae by weight-lifting. Lancet, 2: 155.

Duncan, N.A., and Ahmed, A.M. (1991) The role of axial rotation in the etiology of unilateral disc prolapse: An experimental and finite-element analysis. Spine, 16: 1089-1098.

Dunk, N., Kedgley, A., Jenkyn, T., and Callaghan, J. (2009) Evidence of a pelvis-driven flexion pattern: Are the joints of the lower lumbar spine fully flexed in seated postures? Clinical Biomechanics, 24: 164-168.

Farfan, H.F. (1973) Mechanical disorders of the low back. Philadelphia: Lea and Febiger.

Fenwick, C.M.J., Brown, S.H.M., and McGill, S.M. (2009) Comparison of different rowing exercises: Trunk muscle activation, and lumbar spine motion, load and stiffness. Journal of Strength and Conditioning Research. 23 (5): 1408-1417.

Flint, M.M. (1965) Abdominal muscle involvement during performance of various forms of sit-up exercises: Electromyographic study. American Journal of Physical Medicine, 44: 224-234.

Freeman, S., Karpowicz, A., Gray, J., and McGill, S. (2006) Quantifying muscle patterns during various forms of the push-up: Implications for spine loading and stability. Medicine & Science in Sports & Exercise, 38 (3): 570-577.

Freeman, S., Mascia, A., and McGill, S.M. (2013) Arthrogenic neuromuscular inhibition: A foundational investigation of the hip joint. Clinical Biomechanics, 28: 171-177.

Fyhrie, D.P., and Schaffler, M.B. (1994) Failure mechanisms in human vertebral cancellous bone. Bone, 15 (1): 105-109.

Gallagher, K.M., Howarth, S.J., and Callaghan, J.P. (2010) Effects of anterior shear displacement rate on the structural properties of the porcine cervical spine. Journal of Biomechanical Engineering, 132 (9): 091004.

Gardner-Morse, M., Stokes, I.A.F., and Laible, J.P. (1995) Role of the muscles in lumbar spine stability in maximum extension efforts. Journal of Orthopaedic Research, 13: 802-808.

Gordon, S.J., et al. (1991) Mechanism of disc rupture—A preliminary report. Spine, 16: 450.

Grabiner, M.D., Koh, T.J., and Ghazawi, A.E. (1992) Decoupling of bilateral excitation in subjects with low back pain. Spine, 17: 1219-1223.

Gracovetsky, S., Farfan, H.F., and Lamy, C. (1981) Mechanism of the lumbar spine. Spine, 6 (1): 249.

Green, J., Grenier, S., and McGill, S.M. (2002) Low back stiffness is altered with warmup and bench rest: Implications for athletes. Medicine & Science in Sports & Exercise, 34 (7): 1076-1081.

Grenier, S.G., and McGill, S.M. (2007) Quantification of lumbar stability using two different abdominal activation strategies. Archives of Physical Medicine and Rehabilitation, 88 (1): 54-62.

Grenier, S.G., and McGill, S.M. (2008) When exposed to challenged ventilation, those with a history of LBP increase spine stability relatively more than healthy individuals. Clinical Biomechanics, 23 (9): 1105-1111.

Grenier, S., Preuss, R., Scannel, J., Brown, S., and McGill, S.M. (2001) Correlates of occupational low back troubles: Clues for better evaluation and rehabilitation. Association of Canadian Ergonomists annual meeting. Montreal, Quebec. October 3-5: 159-160.

Grew, N.D. (1980) Intra-abdominal pressure response to loads applied to the torso in normal subjects. Spine, 5 (2): 149.

Gunning, J.L., Callaghan, J.P., and McGill, S.M. (2001) The role of prior loading history and spinal posture on the compressive tolerance and type of failure in the spine using a porcine trauma model. Clinical Biomechanics, 16 (6): 471-480.

Hadler, N.M. (1991) Insuring against work capacity from spinal disorders. In: Frymoyer, J.W. (Ed.), The adult spine (pp. 77-83). New York: Raven Press.

Haig, A.J., LeBreck, D.B., and Powley, S.G. (1995) Paraspinal mapping: Quantified needle electromyography of the paraspinal muscles in persons without low back pain. Spine, 20 (6): 715-721.

Halpern, A.A., and Bleck, E.E. (1979) Sit-up exercises: An electromyographic study. Clinical Orthopaedics and Related Research, 145: 172-178.

Hardcastle, P., Annear, P., and Foster, D. (1992) Spinal abnormalities in young fast bowlers. Journal of Bone and Joint Surgery, 74B (3): 421.

Herrin, G.A., Jaraiedi, M., and Anderson, C.K. (1986) Prediction of overexertion injuries using biomechanical and psychophysical models. American Industrial Hygiene Association Journal, 47: 322-330.

Hides, J.A., Richardson, C.A., and Jull, G.A. (1996) Multifidus muscle recovery is not automatic following resolution of acute first episode low back pain. Spine, 21: 2763-2769.

Hilkka, R., Mattsson, T., Zitting, A., Wickstrom, G., Hanninen, K., and Waris, P. (1990) Radiographically detectable degenerative changes of lumbar spine among concrete reinforcement workers and house painters. Spine, 15, 114-119.

Hodges, P.W., and Richardson, C.A. (1996) Inefficient muscular stabilisation of the lumbar spine associated with low back pain: A motor control evaluation of transversus abdominis. Spine, 21: 2640-2650.

Hodges, P.W., and Richardson, C.A. (1999) Altered trunk muscle recruitment in people with low back pain with upper limb movement at different speeds. Archives of Physical Medicine and Rehabilitation, 80: 1005-1012.

Holm, S., and Nachemson, A. (1983) Variations in the nutrition of the canine intervertebral disc induced by motion. Spine, 8: 866-874.

Holmes, A.D., Hukins, D.W.L., and Freemont, A.J. (1993) End-plate displacement during compression of lumbar vertebra-disc-vertebra

segments and the mechanism of failure. Spine, 18: 128-135.
Hukins, D.W.L., Aspden, R.M., and Hickey, D.S. (1990) Thoracolumbar fascia can increase the efficiency of the erector spinae muscles. Clinical Biomechanics, 5 (1): 30.
Ikeda, D., and McGill, S.M. (2012) Can altering motions, postures and loads provide immediate low back pain relief: A study of four cases investigating spine load, posture and stability. Spine, 37 (23): E1469-E1475.
Jager, M., Luttman, A., and Laurig, W. (1991) Lumbar load during one-handed bricklaying. International Journal of Industrial Ergonomics, 8: 261-277.
Jayson, M., and Dixon, A. (1970) Intra-articular pressure in rheumatoid arthritis of the knee III: Pressure changes during joint use. Annals of the Rheumatic Diseases, 29: 401-408.
Jette, M., Sidney, K., and Cicutti, N. (1984, Sept.-Oct.) A critical analysis of sit-ups: A case for the partial curl-up as a test of muscular endurance. Canadian Journal of Physical Education and Recreation: 4-9.
Jorgensen, K., and Nicolaisen, T. (1987) Trunk extensor endurance: Determination and relation to low back trouble. Ergonomics, 30: 259-267.
Juker, D., McGill, S.M., and Kropf, P. (1998) Quantitative intramuscular myoelectric activity of lumbar portions of psoas and the abdominal wall during cycling. Journal of Applied Biomechanics, 14 (4): 428-438.
Juker, D., McGill, S.M., Kropf, P., and Steffen, T. (1998) Quantitative intramuscular myoelectric activity of lumbar portions of psoas and the abdominal wall during a wide variety of tasks. Medicine & Science in Sports & Exercise, 30 (2): 301-310.
Kaneda, K., Sato, D., Wakabayashi, H., and Nomura, T. (2009) EMG activity of hip and trunk muscles during deep water running. Journal of Electromyography and Kinesiology, 19: 1064-1070.
Kavcic, N., Grenier, S., and McGill, S.M. (2004) Determining the stabilizing role of individual torso muscles during rehabilitation exercises, Spine, 29(11): 1254-1265.
Kelsey, J.L. (1975) An epidemiological study of the relationship between occupations and acute herniated lumbar intervertebral discs. International Journal of Epidemiology, 4: 197-205.
Kirkaldy-Willis, W.H., and Burton, C.V. (1992) Managing low back pain (3rd ed.). New York: Churchill Livingstone.
Kornberg, M. (1988) MRI diagnosis of traumatic Schmorl's nodes. Spine, 13:

934-935.
Krag, M.H., Byrne, K.B., Gilbertson, L.G., and Haugh, L.D. (1986, August 25-27) Failure of intraabdominal pressurization to reduce erector spinae loads during lifting tasks (p. 87). In Proceedings of the North American Congress on Biomechanics, Montreal.
Krag, M.H., Seroussi, R.E., Wilder, D.G., and Pope, M.H. (1987) Internal displacement distribution from in vitro loading of human thoracic and lumbar spinal motion segments: Experimental results and theoretical predictions. Spine, 12 (10): 1001.
Kubo, M., Holt, K.G., Saltzman, E., and Wagenaar, R.C. (2006) Changes in axial stiffness of the trunk as a function of walking speed. Journal of Biomechanics, 39: 750-757.
Kumar, S. (1990) Cumulative load as a risk factor for back pain. Spine, 15: 1311-1316.
Lett, K., and McGill, S.M. (2006) Pushing and pulling: Personal mechanics influence spine loads. Ergonomics, 49 (9): 895-908.
Lotz, J.C., and Chin, J.R. (2000) Intervertebral disc cell death is dependent on the magnitude and duration of spinal loading. Spine, 25 (12): 1477-1483.
Lucas, D., and Bresler, B. (1961) Stability of the ligamentous spine. Tech. Report No. 40, Biomechanics Laboratory, University of California, San Francisco.
Macintosh, J.E., and Bogduk, N. (1987) The morphology of the lumbar erector spinae. Spine, 12 (7): 658.
Macintosh, J.E., Bogduk, N., and Gracovetsky, S. (1987) The biomechanics of the thoracolumbar fascia. Clinical Biomechanics, 2: 78.
Marras, W.S., Ferguson, S.A., Lavender, S.A., Splittstoesser, R.E., and Yang, G. (2013) Cumulative spine loading and clinically meaningful declines in low back function. Human Factors, 56 (1): 29-43.
Marras, W.S., Lavender, S.A., Leurgens, S.E., et al. (1993) The role of dynamic three-dimensional trunk motion in occupationally related low back disorders: The effects of workplace factors, trunk position and trunk motion characteristics on risk of injury. Spine, 18: 617-628.
McGill, S.M. (1987) A biomechanical perspective of sacro-iliac pain. Clinical Biomechanics, 2 (3): 145-151.
McGill, S.M. (1991) The kinetic potential of the lumbar trunk musculature about three orthogonal orthopaedic axes in extreme postures. Spine, 16 (7): 809-815.

McGill, S.M. (1997) Biomechanics of low back injury: Implications on current practice and the clinic [Invited paper]. Journal of Biomechanics, 30 (5): 465-475.

McGill, S.M. (1998) Low back exercises: Evidence for improving exercise regimens [Invited paper]. Physical Therapy, 78 (7): 754-765.

McGill, S.M. (2014) Ultimate back fitness and performance. Waterloo, ON: Backfitpro Inc. (www.backfitpro.com)

McGill, S.M., Andersen, J., and Horne, A. (2012) Predicting performance and injury resilience from movement quality and fitness scores in a basketball player population. Journal of Strength and Conditional Research, 26 (7): 1731-1739.

McGill, S.M., and Axler, C.T. (1996) Changes in spine height throughout 32 hours of bedrest: Implications for bedrest and space travel on the low back. Archives of Physical Medicine and Rehabilitation, 38 (9): 925-927.

McGill, S.M., Belore, M., Crosby, I., and Russell, C. (2010) Comparison of two methods to quantify torso flexion endurance. Occupational Ergonomics, 9: 55-61.

McGill, S.M., and Brown, S. (1992) Creep response of the lumbar spine to prolonged lumbar flexion. Clinical Biomechanics, 7: 43.

McGill, S.M., and Callaghan, J.P. (1999) Impact forces following the unexpected removal of a chair while sitting. Accident Analysis and Prevention, 31: 85-89.

McGill, S.M., Cambridge, E., and Andersen, J. (2014) Muscle activity and spine load during pulling exercises: Influence of stable and labile contact surfaces and technique coaching. Journal of Electromyography and Kinesiology, 24 (5): 652-665.

McGill, S.M., Cannon, J., and Andersen, J. (2014a) Analysis of pushing exercises: Muscle activity and spine load while contrasting techniques on stable surfaces with labile suspension strap training system. Journal of Strength and Conditioning Research, 28 (i): 105-116.

McGill, S.M., Cannon, J., and Anderson, J. (2014b) Muscle activity and spine load during anterior chain whole body linkage exercises: The body saw, hanging leg raise and walkout from a pushup. Journal of Sports Sciences. doi:10.1080/026 40414.2014.946 437

McGill, S.M., Cannon, J., and Andersen, J. (submitted) Physiological and biomechanical mechanisms in hula hooping: Caloric expenditure and spine loads.

McGill, S.M., Frost, D., Andersen, J., Crosby, I., and Gardiner, D. (2013a) Movement quality and links to measures of fitness in firefighters. Work, 45 (3): 357-366.

McGill, S.M., Frost, D., Lam, T., Findlay, T., Darby, K., and Andersen, J. (2013b) Fitness and movement quality of emergency task force police officers: A database with comparison to populations of emergency services personnel, athletes and the general public [Invited paper]. International Journal of Industrial Ergonomics. http://dx.doi.org/10.1016/j.ergon.2012.11.013

McGill, S.M., Grenier, S., Bluhm, M., Preuss, R., Brown, S., and Russell, C. (2003) Previous history of LBP with work loss is related to lingering effects in biomechanical physiological, personal, and psychosocial characteristics. Ergonomics, 46 (7): 731-746.

McGill, S.M., Juker, D., and Axler, C. (1996) Correcting trunk muscle geometry obtained from MRI and CT scans of supine postures for use in standing postures. Journal of Biomechanics, 29 (5): 643-646.

McGill, S.M., Juker, D., and Kropf, P. (1996) Quantitative intramuscular myoelectric activity of quadratus lumborum during a wide variety of tasks. Clinical Biomechanics, 11 (3): 170-172.

McGill, S.M., Karpowicz, A., and Fenwick, C. (2009a) Ballistic abdominal exercises: Muscle activation patterns during a punch, baseball throw, and a torso stiffening manoeuvre. Journal of Strength and Conditioning Research, 23 (3): 898-905.

McGill, S.M., Karpowicz, A., and Fenwick, C. (2009b) Exercises for the torso performed in a standing posture: Motion and motor patterns. Journal of Strength and Conditioning Research, 23 (2): 455-464.

McGill, S.M., and Kippers, V. (1994) Transfer of loads between lumbar tissues during the flexion relaxation phenomenon. Spine, 19 (19): 2190.

McGill, S.M., and Marshall, L.W. (2012) Kettlebell swing, snatch, and bottoms-up carry: Back and hip muscle activation, motion, and low back loads. Journal of Strength and Conditioning Research, 26 (1): 16-27.

McGill, S.M., Marshall, L., and Andersen, J. (2013) Low back loads while walking and carrying: Comparing the load carried in one hand or in both hands. Ergonomics, 56 (2): 293-302.

McGill, S.M., McDermott, A., and Fenwick, C.M. (2009) Comparison of different strongman events: Trunk muscle activation and lumbar spine motion, load, and stiffness. Journal of Strength and Conditioning Research,

23 (4): 1148-1161.
McGill, S.M., and Norman, R.W. (1987) Reassessment of the role of intraabdominal pressure in spinal compression. Ergonomics, 30 (11): 1565.
McGill, S.M., and Norman, R.W. (1988) The potential of lumbodorsal fascia forces to generate back extension moments during squat lifts. Journal of Biomedical Engineering, 10: 312.
McGill, S.M., Sharratt, M.T., and Seguin, J.P. (1995) Loads on spinal tissues during simultaneous lifting and ventilatory challenge. Ergonomics, 38: 1772-1792.
McGill, S.M., van Wijk, M., Axler, C.T., and Gletsu, M. (1996) Spinal shrinkage: Is it useful for evaluation of low back loads in the workplace? Ergonomics, 39 (1): 92-102.
McGill, S.M., Yingling, V.R., and Peach, J.P. (1999) Three-dimensional kinematics and trunk muscle myoelectric activity in the elderly spine: A database compared to young people. Clinical Biomechanics, 14 (6): 389-395.
McGlashen, K.M., Miller, J.A.A., Shultz, A.B., and Anderson, G.B.J. (1987) Load displacement behaviour of the human lumbosacral joint. Journal of Orthopedic Research, 5: 488.
McKenzie, R.A. (1979) Prophylaxis in recurrent low back pain. New Zealand Medical Journal, 89: 22.
Mens, J., van Dijke, G.H., Pool-Goudzwaard, A., van der Hulst, V., and Stam, H. (2006) Possible harmful effects of high intra-abdominal pressure on the pelvic girdle. Journal of Biomechanics, 39: 627-635.
Mody, G.M., and Cassim, B. (1997) Rheumatologic manifestations of malignancy. Current Opinions in Rheumatology, 9: 75-79.
Moreside, J.M., and McGill, S.M. (2012) How do elliptical machines differ from walking: A study of torso motion and muscle activity. Clinical Biomechanics, 27: 738-743.
Moreside, J.M., Vera-Garcia, F.J., and McGill, S.M. (2007) Trunk muscle activation patterns, lumbar compressive forces and spine stability when using the body blade. Physical Therapy, 87 (2): 153-163.
Morris, J.M., Lucas, D.B., and Bresler, B. (1961) Role of the trunk in stability of the spine. Journal of Bone and Joint Surgery, 43A: 327-351.
Nachemson, A. (1966) The load on lumbar discs in different positions of the body. Clinical Orthopaedics and Related Research, 45: 107.
Nachemson, A., Andersson, G.B.J., and Schultz, A.B. (1986) Valsalva

manoeuvre biomechanics: Effects on lumbar trunk loads of elevated intra-abdominal pressure. Spine, 11 (5): 476.
Nachemson, A.L., and Morris, J.M. (1964) In vivo measurements of intradiscal pressure. Journal of Bone and Joint Surgery, 46A: 1077.
National Institute for Occupational Safety and Health (NIOSH). (1981) Work practices guide for manual lifting. NIOSH Publication No. 81-122. Washington, DC: U.S. Department of Health and Human Services (DHHS).
Nelson-Wong, E., and Callaghan, J. (2014) Transient low back pain development during standing predicts future clinical low back pain in previously asymptomatic individuals. Spine, 39 (6): E379-E383.
Norman, R., Wells, R., Neumann, P., Frank, P., Shannon, H., and Kerr, M. (1998) A comparison of peak vs. cumulative physical work exposure risk factors for the reporting of low back pain in the automotive industry. Clinical Biomechanics, 13: 561-573.
Noyes, F.R., De Lucas, J.L., and Torvik, P.J. (1994) Biomechanics of ligament failure: An analysis of strain-rate sensitivity and mechanisms of failure in primates. Journal of Bone and Joint Surgery, 56A: 236.
Nutter, P. (1988) Aerobic exercise in the treatment and prevention of low back pain. State of the Art Review of Occupational Medicine, 3: 137.
Oganov, V.S., Rakhmanov, A.S., Novikov, V.E., Zatsepin, S.T., Rodionova, S.S., and Cann, C. (1991) The state of human bone tissue during space flight. Acta Astronomy, 213: 129-133.
Pearcy, M.J., Portek, J., and Shepherd, J. (1984) Three-dimensional X-ray analysis of normal measurement in the lumbar spine. Spine, 9: 294.
Pearcy, M.J., and Tibrewal, S.B. (1984) Axial rotation and lateral bending in the normal lumbar spine measured by three-dimensional radiography. Spine, 9: 582.
Peterson, D. (2013) Proposed performance standards for the plank and inclusion consideration into the navy's physical readiness test. Strength and Conditioning Journal, 35 (5): 22-26.
Phillips, P.E. (1997) Viral arthritis. Current Opinions in Rheumatology, 9: 337-344.
Rantanen, J., Hurme, M., Falck, B., et al. (1993) The lumbar multifidus muscle five years after surgery for a lumbar intervertebral disc herniation. Spine, 18: 568-574.
Reilly, T., Tynell, A., and Troup, J.D.G. (1984) Circadian variation in human

stature. Chronobiology International, 1: 121.
Richardson, C., Jull, G., Hodges, P., and Hides, J. (1999) Therapeutic exercise for spinal segmental stabilization in low back pain. Edinburgh: Churchill Livingstone.
Roaf, R. (1960) A study of the mechanics of spinal injuries. Journal of Joint and Bone Surgery, 42B: 810.
Rohlmann, A., Zander, T., Graichen, F., and Schmidt, H. (2014) How does the way a weight is carried affect spinal loads? Ergonomics, 57(2): 262-270.
Ross, J.K., Bereznik, D., and McGill, S.M. (1999) Atlas-axis facet asymmetry: Implications for manual palpation. Spine, 24 (12): 1203-1209.
Rossignol, M., Stock, S., Patry, L., and Armstrong, B. (1997) Carpal tunnel syndrome: What is attributable to work? The Montreal study. Occupational and Environmental Medicine, 54: 519-523.
Roy, S.H., De Luca, C.J., Emley, M., and Buijs, R.J.C. (1995) Spectral electromyographic assessment of back muscles in patients with low back pain undergoing rehabilitation. Spine, 20: 38-48.
Samii, K., Cassinotti, P., de Freudenreich, J., Gallopin, Y., Le Fort, D., and Stalder, H. (1996) Acute bilateral carpal tunnel syndrome associated with the human parvovirus B19 infection. Clinical Infectious Diseases: An Official Publication of the Infectious Diseases Society of America, 22: 162-164.
Santaguida, P., and McGill, S.M. (1995) The psoas major muscle: A three-dimensional mechanical modelling study with respect to the spine based on MRI measurement. Journal of Biomechanics, 28: 339-345.
Schultz, A.B., Warwick, D.N., Berkson, M.H., and Nachemson, A.L. (1979) Mechanical properties of human lumbar spine motion segments. Part I: Response in flexion, extension, lateral bending and torsion. Journal of Biomechanical Engineering, 101: 46.
Siddall, P.J., and Cousins, M.J. (1997) Spine pain mechanisms. Spine, 22: 98-104.
Sidorkewicz, N., Cambridge, E., and McGill, S.M. (accepted 2014) Examining the effects of altering hip angle on gluteus medius and tensor fascia latae interplay during common non-weight bearing hip rehabilitation exercises. Clinical Biomechanics, 29 (9): 971-976.
Sidorkewicz, N., and McGill, S.M. (2015) Documenting female spine motion during coitus with a commentary on the implications for the low back pain

patient. European Spine Journal, 24 (3): 513-520.
Sidorkewicz, N., and McGill, S.M. (2014b) Male spine motion during coitus: Implications for the low back pain patient. Spine, 39 (20): 1633-1639.
Sihvonen, T., Lindgren, K., Airaksinen, O., and Manninen, H. (1997) Movement disturbances of the lumbar spine and abnormal back muscle electromyographic findings in recurrent low back pain. Spine, 22: 289-295.
Slyfield, C.R., Tkachenko, E.V., Fischer, et al. (2012) Mechanical failure begins preferentially near resorption cavities in human vertebral cancellous bone under compression. Bone, 50 (6): 1281-1287.
Snijders, C.J., Hermens, P.F.G., and Kleinrensink, G.J. (2006) Functional aspects of cross-legged sitting with special attention to piriformis muscles and sacroiliac joints. Clinical Biomechanics, 21: 116-121.
Snook, S.H., Webster, B.S., McGorry, R.W., Fogleman, M.T., and McCann, K.B. (1998) The reduction of chronic nonspecific low back pain through the control of early morning lumbar flexion. Spine, 23: 2601-2607.
Spencer, D.L., Miller, J.A.A., and Schultz, A.B. (1985) The effects of chemonucleolysis on the mechanical properties of the canine lumbar disc. Spine, 10: 555.
Stokes, M., and Young, A. (1984) The contribution of reflex inhibition to arthrogenous muscle weakness. Clinical Science, 67: 7-14.
Tanaka, N., An, H.S., Lim, T-H., Fujiwara, A., Jeon, C., and Haughton, V.M. (2001) The relationship between disc degeneration and flexibility of the lumbar spine. Spine, 1: 47-56.
Tesh, K.M., Dunn, J., and Evans, J.H. (1987) The abdominal muscles and vertebral stability. Spine, 12 (5): 501.
Thomson, K.D. (1988) On the bending moment capability of the pressurized abdominal cavity during human lifting activity. Ergonomics, 31 (5): 817.
Troup, J.D.G., Martin, J.W., and Lloyd, D.C.E.F. (1981) Back pain in industry—A prospective study. Spine, 6: 61-69.
Van Wingerden J-P., Vleeming, A., and Ronchetti, I. (2008) Differences in standing and forward bending in women with chronic low back pain or pelvic girdle pain: Indications for physical compensation strategies. Spine, 33 (11): E334-E341.
Vernon-Roberts, B., and Pirie, C.J. (1973) Healing trabecular microfractures in the bodies of lumbar vertebrae. Annals of the Rheumatic Diseases, 32: 406-412.
Videman, T., Nurminen, M., and Troup, J.D.G. (1990) Lumbar spinal

pathology in cadaveric material in relation to history of back pain, occupation and physical loading. Spine, 15 (8): 728.
Vogt, L., Pfeifer, K., and Banzer, W. (2003) Neuromuscular control of walking with chronic low back pain, Manual Therapy, 8 (1): 21-28.
Wagnac, E., Arnoux, P.J., Garo, A., and Aubin, C.E. (2012) Finite element analysis of the influence of loading rate on a model of the full lumbar spine under dynamic loading conditions. Medical and biological engineering & computing, 50 (9): 903-915.
Wang, Y., Videman, T., and Battié, M.C. (2012) ISSLS prize winner: Lumbar vertebral endplate lesions: Associations with disc degeneration and back pain history. Spine, 37 (17): 1490-1496.
Watkins, J. (1999) Structure and function of the musculoskeletal system. Champaign, IL: Human Kinetics.
White, A.A., and Panjabi, M.M. (1978) Clinical biomechanics of the spine. Philadelphia: J.B. Lippincott.
Wilder, D.G., Pope, M.H., and Frymoyer, J.W. (1988) The biomechanics of lumbar disc herniation and the effect of overload and instability. Journal of Spinal Disorders, 1 (1): 16.
Woo, S.L.-Y., Gomez, M.A., and Akeson, W.H. (1985) Mechanical behaviors of soft tissues: Measurements, modifications, injuries, and treatment. In: Nahum, H.M., and Melvin, J. (Eds.), Biomechanics of trauma (pp. 109-133). Norwalk, CT: Appleton-Century-Crofts.
Woolf, C.J., Bennett, G.J., Doherty, M., Dubner, R., Kidd, B., Koltzenburg, M., Lipton, R., Loeser, J.D., Payne, R., and Torebjork, E. (1998) Towards a mechanism-based classification of pain? Pain, 77: 227-229.
Yingling, V.R., and McGill, S.M. (1999a) Anterior shear of spinal motion segments: Kinematics, kinetics and resulting injuries observed in a porcine model. Spine, 24 (18): 1882-1889.
Yingling, V.R., and McGill, S.M. (1999b) Mechanical properties and failure mechanics of the spine under posterior shear load: Observations from a porcine model. Journal of Spinal Disorders, 12 (6): 501-508.

Chapter 5: Myths and Realities of Lumbar Spine Stability

Allison, G.T. (2012) Abdominal muscle feedforward activation in patients with chronic low back pain is largely unaffected by 8 weeks of core stability training. Journal of Physiotherapy, 58 (3): 200.
Bergmark, A. (1987) Mechanical stability of the human lumbar spine. Doctoral dissertation, Department of Solid Mechanics, Lund University,

Sweden.
Biering-Sorensen, F. (1984) Physical measurements as risk indicators for low back trouble over a one year period. Spine, 9: 106-119.
Brooks, C., Kennedy, S., and Marshall, P. (2012) Specific trunk and general exercise elicit similar changes in anticipatory postural adjustments in patients with chronic low back pain. Spine, 37 (25): E1543-E1550.
Brown, S.H., and McGill, S.M. (2005) Muscle force-stiffness characteristics influence joint stability. Clinical Biomechanics, 20 (9): 917-922.
Brown, S., and McGill, S.M. (2008a) Co-activation alters the linear versus non-linear impression of the EMG-torque relationship of trunk muscles. Journal of Biomechanics, 41: 491-497.
Brown, S., and McGill, S.M. (2008b) How the inherent stiffness of the in-vivo human trunk varies with changing magnitude of muscular activation. Clinical Biomechanics, 23 (1): 15-22.
Brown, S., and McGill, S.M. (2008c) An ultrasound investigation into the morphology of the human abdominal wall uncovers complex deformation patterns during contraction. European Journal of Applied Physiology, 104 (6): 1021-1030.
Brown, S., and McGill, S.M. (2009a) The intrinsic stiffness of the invivo lumbar spine in response to a variety of quick releases: Implications for reflexive requirements. Journal of Electromyography and Kinesiology, 19 (5): 727-736.
Brown, S., and McGill, S.M. (2009b) Transmission of muscularly generated force and stiffness between layers of the rat abdominal wall. Spine, 34 (2): E70-E75.
Brown, S.H.M., and McGill, S.M. (2010a) A comparison of ultrasound and electromyography measures of force and activation to examine the mechanics of abdominal wall contraction. Clinical Biomechanics, 25: 115-123.
Brown, S.H.M., and McGill, S.M. (2010b) The relationship between trunk muscle activation and trunk stiffness: Examining a non-constant stiffness gain. Computer Methods in Biomechanics and Biomedical Engineering, 13 (6): 829-835.
Childs, J.D., Teyhen, D.S., Casey, P.R., et al. (2010) Effects of traditional sit-up training versus core stabilization exercises on short-term musculoskeletal injuries in U.S. Army soldiers: A cluster randomized trial. Physical Therapy, 90 (10): 1404-1412.

Cholewicki, J., and McGill, S.M. (1996) Mechanical stability of the in vivo lumbar spine: Implications for injury and chronic low back pain. Clinical Biomechanics, 11 (1): 1-15.

Cholewicki, J., Simons, A.P.D., and Radebold, A. (2000) Effects of external trunk loads on lumbar spine stability. Journal of Biomechanics, 33 (11): 1377-1385.

Crisco, J.J., and Panjabi, M.M. (1992) Euler stability of the human ligamentous lumbar spine. Part I: Theory and Part II: Experiment. Clinical Biomechanics, 7: 27-32.

Gardner-Morse, M., Stokes, I.A.F., and Laible, J.P. (1995) Role of the muscles in lumbar spine stability in maximum extension efforts. Journal of Orthopaedic Research, 13: 802-808.

Grenier, S.G., and McGill, S.M. (2007) Quantification of lumbar stability using two different abdominal activation strategies. Archives of Physical Medicine and Rehabilitation, 88 (1): 54-62.

Grenier, S.G., and McGill, S.M. (2008) When exposed to challenged ventilation, those with a history of LBP increase spine stability relatively more than healthy individuals. Clinical Biomechanics, 23 (9): 1105-1111.

Gubler, D., Mannion, A., Schenk, P., Gorelick, M., Helbling, D., Gerber, H., Toma, V., and Sprott, H. (2010) Ultrasound image doppler imaging reveals no delay in abdominal muscle feed-forward activity during rapid arm movements in patients with chronic low back pain. Spine, 35 (16): 1506-1513.

Hebert, J.J., Koppenhaver, S.L., Magel, J.S., and Fritz, J.M. (2010) The relationship of transverse abdominis and lumbar multifidus activation and prognostic factors for clinical success with a stabilization exercise program: A cross-sectional study. Archives of Physical Medicine and Rehabilitation, 91: 78-85.

Hodges, P., McGill, S.M., et al. (2013a) Integrated clinical approach to motor control interventions in low back and pelvic pain. In: Hodges, P., Cholewicki, J., and van Dieen, J. (Eds.), Spinal control: The rehabilitation of back pain. London: Churchill Livingstone.

Hodges, P., McGill, S.M., and Hides, J. (2013b) Motor control of the spine and changes in pain: Debate about the extrapolation from research observations of motor control strategies to effective treatments for back pain, In: Hodges, P., Cholewicki, J., and van Dieen, J. (Eds.), Spinal control: The rehabilitation of back pain. London: Churchill Livingstone.

Hoffer, J., and Andreassen, S. (1981) Regulation of soleus muscle stiffness in premamillary cats. Journal of Neurophysiology, 45: 267-285.
Ikeda, D., and McGill, S.M. (2012) Can altering motions, postures and loads provide immediate low back pain relief: A study of four cases investigating spine load, posture and stability. Spine, 37 (23): E1469-E1475.
Ikeda, D., and McGill, S.M. 2013. Assessing joint stability from eigan values obtained from multi-channel EMG—A spine example. In: Naik, E. (Ed.), Applications, challenges and advancements in electromyography signal processing.
Koumantakis, G.A., Watson, P.J., and Oldham, J.A. (2005) Trunk muscle stabilization training plus general exercise versus general exercise only: Randomized controlled trial of patients with recurrent low back pain. Physical Therapy, 85 (3): 209-225.
Lee, B., and McGill, S.M. (2015) Effect of long term isometric training on core/torso stiffness. Journal of Strength and Conditioning Research, 29(6): 1515-1526.
Liebenson, C., Karpowicz, A., Brown, S., Howarth, S., and McGill, S.M. (2009) The active straight leg raise test and lumbar spine stability. Physical Medicine and Rehabilitation, 1 (6): 530-535.
Lucas, D., and Bresler, B. (1961) Stability of the ligamentous spine. In: Tech report No. 40, Biomechanics Laboratory, University of California, San Francisco.
McGill, S.M. (2001) Low back stability: From formal description to issues for performance and rehabilitation [Invited review]. Exercise and Sports Science Reviews, 29 (1): 26-31.
McGill, S.M. (2011) Is a postural-structural-biomechanical model, within manual therapies, viable: AJBMT debate [Invited response]. Journal of Bodywork and Movement Therapy, 15 (2): 150-152.
McGill, S.M. (2013) Opinions on the links between back pain and motor control: The disconnect between clinical practice and research. In: Hodges, P., Cholewicki, J., and van Dieen, J. (Eds.), Spinal control: The rehabilitation of back pain. London: Churchill Livingstone. McGill, S.M., Sharratt, M.T., and Seguin, J.P. (1995) Loads on the spinal tissues during simultaneous lifting and ventilatory challenge. Ergonomics, 38 (9): 1772-1792.
O'Sullivan, P., Twomey, L.T., and Allison, G.T. (1997) Altered pattern of abdominal muscle activation in chronic back pain patients. Australian

Journal of Physiotherapy, 43: 91-98.
Oxland, T.R., Panjabi, M.M., Southern, E.P., and Duranceau, J.S. (1991) An anatomic basis for spinal instability: A porcine trauma model. Journal of Orthopaedic Research, 9: 452-462.
Richardson, C., Jull, G., Hodges, P., and Hides, J. (1999) Therapeutic exercise for spinal segmental stabilization in low back pain. Edinburgh: Churchill Livingstone.
Silfies, S.P., Mehta, R., Smith, S.S., and Karduna, A.R. (2009) Differences in feedforward trunk muscle activity in subgroups of patients with mechanical low back pain. Archives of Physical Medicine and Rehabilitation, 90: 1159-1169.
Unsgaard-Tondel, M., Lund, N., Magnussen, J., and Vasseljen, O. (2012) Is activation of transverse abdominis and obliquus internus abdominis associated with long term changes in chronic low back pain? A prospective study with 1-year follow-up. British Journal of Sports Medicine, 46 (10): 729-734.
Vasseljen, O., Unsgaard-Tondel, M., Westad, C., and Mork, P.J. (2012) Effect of core stability exercises on feed-forward activation of deep abdominal muscle in chronic low back pain: A randomized controlled trial. Spine, 37 (13): 1101-1108.
Wagner, H., Anders, C. Puta, C., Petrovitch, A., Morl, F., Schilling, N., Witte, H., and Blickhan, R. (2005) Musculoskeletal support of lumbar spine stability. Pathophysiology, 12: 257-265.
Wang, S., and McGill, S.M. (2008) Links between the mechanics of ventilation and spine stability. Journal of Applied Biomechanics, 24 (2): 166-174.
Wong, A.Y.L., Parent, E.C., Funabashi, M., Stanton, T.R., and Kawchuk, G.N. (2013) Do various baseline characteristics of transversus abdominis and lumbar multifidus predict clinical outcomes in non-specific low back pain? A systematic review. Pain. http://dx.doi.org/10.1016/j.pain.2013.07.010

Chapter 6: LBD Risk Assessment

Adams, M.A., and Hutton, W.C. (1988) Mechanics of the intervertebral disc. In: Ghosh, P. (Ed.), The biology of the intervertebral disc. Boca Raton, FL: CRC Press.
Bobick, T.G., Belard, J.L., Hisao, M., and Wassell, J.T. (2001) Physiological effects of back belt wearing during asymmetric lifting. Applied

Ergonomics, 32: 541-547.
Cholewicki, J., and McGill, S.M. (1992) Lumbar posterior ligament involvement during extremely heavy lifts estimated from fluoroscopic measurements. Journal of Biomechanics, 25 (1): 17-28.
Cholewicki, J., and McGill, S.M. (1994) EMG assisted optimization: A hybrid approach for estimating muscle forces in an indeterminate biomechanical model. Journal of Biomechanics, 27: 1287-1289.
Cholewicki, J., and McGill, S.M. (1996) Mechanical stability of the in vivo lumbar spine: Implications for injury and chronic low back pain. Clinical Biomechanics, 11 (1): 1-15.
Ciriello, V.M., and Snook, S.H. (1995) The effect of back belts on lumbar muscle fatigue. Spine, 20 (11): 1271-1278.
Dul, J., Douwes, M., and Smitt, P. (1994) Ergonomic guidelines for the prevention of discomfort of static postures can be based on endurance data. Ergonomics, 37: 807-815.
Ferguson, S.A., Marras, W.S., and Burr, D. (2005) Workplace design guidelines for asymptomatic vs. low back injured workers. Applied Ergonomics, 36 (1): 85-95.
Granata, K.P., Marras, W.S., and Davis, K.G. (1997) Biomechanical assessment of lifting dynamics, muscle activity and spinal loads while using three different style lifting belts. Clinical Biomechanics, 12 (2): 107-115.
Grood, E.S., and Suntay, W.J. (1983) A joint coordinate system for the clinical description of three-dimensional motions: Application to the knee. Journal of Biomechanical Engineering, 105: 136- 144.
Harman, E.A., Rosenstein, R.M., Frykman, P.N., and Nigro, G.A. (1989) Effects of a belt on intraabdominal pressure during weight lifting. Medicine & Science in Sports & Exercise, 2 (12): 186-190.
Honsa, K., Vennettelli, M., Mott, N., Silvera, D., Niechwiej, E., Wagar, S., Howard, M., Zettel, J., and McGill, S.M. (1998) The efficacy of the NIOSH hand-to-container coupling factor. Proceedings of the 30th Annual Conference of the Human Factors Association of Canada, p. 253.
Hunter, G.R., McGuirk, J., Mitrano, N., Pearman, P., Thomas, B., and Arrington, R. (1989) The effects of a weight training belt on blood pressure during exercise. Journal of Applied Sport Science Research, 3 (1): 13-18.
Ikeda, D., and McGill, S.M. (2012) Can altering motions, postures and loads

provide immediate low back pain relief: A study of four cases investigating spine load, posture and stability. Spine, 37 (23): E1469-E1475.
Karwowski, W. (1991) Psychophysical acceptability and perception of load heaviness by females. Ergonomics, 34 (4): 487-496.
Karwowski, W. (1992) Comments on the assumption of multiplicity of risk factors in the draft revisions to NIOSH lifting guide. In: Kumar, S. (Ed.), Advances in industrial ergonomics and safety. London: Taylor and Francis.
Karwowski, W., and Pongpatanasuegsa, N. (1989) The effect of color on human perception of load heaviness. In: Mital, A. (Ed.), Advances in industrial ergonomics and safety (pp. 673-678). London: Taylor and Francis.
Kraus, J.F., Brown, K.A., McArthur, D.L., Peek-Asa, C., Samaniego, L., and Kraus, C. (1996) Reduction of acute low back injuries by use of back supports. International Journal of Occupational and Environmental Health, 2: 264-273.
Lander, J.E., Hundley, J.R., and Simonton, R.L. (1992) The effectiveness of weight belts during multiple repetitions of the squat exercise. Medicine & Science in Sports & Exercise, 24 (5): 603-609.
Lantz, S.A., and Schultz, A.B. (1986) Lumbar spine orthosis wearing. I: Restriction of gross body motion. Spine, 11 (8): 834-837.
Leamon, T.B. (1994) Research to reality in a critical review of the validity of various criteria to the prevention of occupationally induced low back pain disability. Ergonomics, 37 (12): 1959-1974.
Marras, W. (2008) The working back. Hoboken, New Jersey: Wiley Interscience.
Marras, W.S., Fine, L.J., Ferguson, S.A., and Waters, T.R. (1999) The effectiveness of commonly used lifting assessment methods to identify industrial jobs associated with elevated risk of low-back disorders. Ergonomics, 42 (1): 229-245.
Marras, W.S., Lavender, S.A., Leurgens, S.E., et al. (1993) The role of dynamic three-dimensional trunk motion in occupationally related low back disorders: The effects of workplace factors, trunk position and trunk motion characteristics on risk of injury. Spine, 18: 617-628.
Marras, W.S., Lavender, S.A., Leurgans, S.E., Fathallah, F.A., Ferguson, S.A., Allread, W.G., and Rajulu, S.L. (1995) Biomechanical risk factors for occupationally related low back disorders. Ergonomics, 38: 377-410.
Marras, W.S., and Sommerich, C.M. (1991a) A three-dimensional motion

model of loads on the lumbar spine. I: Model structure. Human Factors, 32: 123-137.
Marras, W.S., and Sommerich, C.M. (1991b) A three-dimensional motion model of loads on the lumbar spine. II: Model structure. Human Factors, 32: 139-149.
McGill, S.M. (1992) A myoelectrically based dynamic three-dimensional model to predict loads on lumbar spine tissues during lateral bending. Journal of Biomechanics, 25: 395-414.
McGill, S.M. (1993) Abdominal belts in industry: A position paper on their assets, liabilities and use. American Industrial Hygiene Association Journal, 54 (12): 752-754.
McGill, S.M. (1997) The biomechanics of low back injury: Implications on current practice in industry and the clinic. Journal of Biomechanics, 30: 465-475.
McGill, S.M. (2014) Ultimate back fitness and performance. Waterloo, ON: Backfitpro, Inc. (www.backfitpro.com)
McGill, S.M., and Norman, R.W. (1986) Partitioning of the L4/L5 dynamic moment into disc, ligamentous and muscular components during lifting. Spine, 11: 666-677.
McGill, S.M., and Norman, R.W. (1987) Reassessment of the role of intra-abdominal pressure in spinal compression. Ergonomics, 30 (11): 1565-1588.
McGill, S., Norman, R.W., and Sharratt, M.T. (1990) The effect of an abdominal belt on trunk muscle activity and intra-abdominal pressure during squat lifts. Ergonomics, 33 (2): 147-160.
McGill, S., Norman, R., Yingling, V., Wells, R., and Neumann, P. (1998) Shear happens! Suggested guidelines for ergonomists to reduce the risk of low back injury from shear loading. 30th Annual Conference of the Human Factors Association of Canada, Mississauga, Canada, pp. 157-161.
McGill, S.M., Seguin, J.P., and Bennett, G. (1994) Passive stiffness of the lumbar torso in flexion, extension, lateral bend and axial twist: The effect of belt wearing and breath holding. Spine, 19 (6): 696-704.Mitchell, L.V., Lawler, F.H., Bowen, D., Mote, W., Asundi, P., and Purswell, J. (1994) Effectiveness and cost-effectiveness of employer-issued back belts in areas of high risk for back injury. Journal of Occupational Medicine, 36 (1): 90-94.
National Institute for Occupational Safety and Health (NIOSH). (1981) Work

practices guide for manual lifting. NIOSH Publication No. 81-122. Washington, DC: U.S. Department of Health and Human Services (DHHS).
Norman, R., Wells, R., Neumann, P., Frank, P., Shannon, H., and Kerr, M. (1998) A comparison of peak vs. cumulative physical work exposure risk factors for the reporting of low back pain in the automotive industry. Clinical Biomechanics, 13: 561-573.
Potvin, J.R. (2012a) Predicting maximum acceptable efforts for repetitive tasks: An equation based on duty cycle. Human Factors, 54 (2): 175-188. doi:10.1177/0018720811424269
Potvin, J.R. (2012b) An equation to predict maximum acceptable loads for repetitive tasks based on duty cycle: Evaluation with lifting and lowering tasks. Work, 41: 397-400. doi:10.3233/WOR-2012-0189-397
Potvin, J.R. (2014) Comparing the revised NIOSH lifting equation to the psychophysical, biomechanical and physiological criteria used in its development. International Journal of Industrial Ergonomics, 44 (2): 246-252.
Snook, S.H. (1978) The ergonomics society—The Society's Lecture 1978. Ergonomics, 21 (12): 963- 985.
Snook, S.H., and Ciriello, V.M. (1991) The design of manual handling tasks: Revised tables of maximum acceptable weights and forces. Ergonomics, 34 (9): 1197-1213.
Waters, T.R., Putz-Anderson, V., Garg, A., and Fine, L.J. (1993) Revised NIOSH equation for the design and evaluation of manual lifting tasks. Ergonomics, 36 (7): 749-776.

Chapter 7: Reducing the Risk of Low Back Injury

Aaras, A. (1994) The impact of ergonomic intervention on individual health and corporate prosperity in a telecommunications environment. Ergonomics, 37 (10): 1679-1696.
Adams, M.A., and Hutton, W.C. (1981) The relevance of torsion to the mechanical derangement of the lumbar spine. Spine, 6: 241-248.
Adams, M.A., and Hutton, W.C. (1988) Mechanics of the intervertebral disc. In: Ghosh, P. (Ed.), The biology of the intervertebral disc. Boca Raton, FL: CRC Press.
Aultman, C.D., Drake, J., Callaghan, J.P., and McGill, S.M. (2004) The effect of static torsion on the compression strength of the spine: An invitro analysis using a porcine spine model. Spine, 29 (15): E304-309.

Autier, M., Lortie, M., and Gagnon, M. (1996) Manual handling techniques: Comparing novices and experts. International Journal of Industrial Ergonomics, 17: 419-429.

Baldwin, M.L., Johnson, W.G., and Butler, R.J. (1996) The error of using returns-to-work to measure the outcomes of health care. American Journal of Industrial Medicine, 29 (6): 632-641.

Biering-Sorensen, F. (1984) Physical measurements as risk indicators for low-back trouble over a one-year period. Spine, 9: 106-119.

Bobick, T.G., Belard, J.L., Hisao, M., and Wassell, J.T. (2001) Physiological effects of back belt wearing during asymmetric lifting. Applied Ergonomics, 32: 541-547.

Brinkmann, P., Biggemann, M., and Hilweg, D. (1989) Prediction of the compressive strength of human lumbar vertebrae. Clinical Biomechanics, 4 (Suppl. 2): S1-S27.

Burnett, A.F., Khangure, M., Elliot, B.C., Foster, D.H., Marshall, R.N., and Hardcastle, P. (1996) Thoracolumbar disc degeneration in young fast bowlers in cricket. A follow-up study. Clinical Biomechanics, 11: 305-310.

Burstein, A.H., and Frankel, W.H. (1968) The viscoelastic properties of some biological material. Annals of New York Academy of Science, 146: 158-165.

Cady, L.D., Bischoff, D.P., O'Connell, E.R., Thomas, P.C., and Allan, J.H. (1979) Strength and fitness and subsequent back injuries of firefighters. Journal of Occupational Medicine, 21: 269.

Callaghan, J.P., and McGill, S.M. (2001a) Low back joint loading and kinematics during standing and unsupported sitting. Ergonomics, 44 (3): 280-294.

Callaghan, J., and McGill, S.M. (2001b) Intervertebral disc herniation. Studies on a porcine model exposed to highly repetitive flexion/extension motion with compressive force. Clinical Biomechanics, 16 (1): 28-37.

Calmels, P., Queneau, P., Hamonet, C., Le Pen, C., Maurel, F., Lerouvrear, C., and Thoumie, P. (2009) Effectiveness of a lumbar belt in subacute low back pain: An open, multicentric, and randomized clinical study. Spine, 34 (3): 215-220.

Carter, D.R. (1985) Biomechanics of bone. In: Nahum, H.M., and Melvin, J. (Eds.), Biomechanics of trauma. Norwalk, CT: Appleton–Century-Crofts.

Cholewicki, J., Juluru, K., and McGill, S.M. (1999) The intraabdominal pressure mechanisms for stabilizing the lumbar spine. Journal of

Biomechanics, 32: 13-17.
Ciriello, V.M., and Snook, S.H. (1995) The effect of back belts on lumbar muscle fatigue. Spine, 20 (11): 1271-1278.
Drake, J.D., Aultman, C.D., McGill, S.M., and Callaghan, J.P. (2005) The influence of static axial torque in combined loading on intervertebral joint failure mechanics using a porcine model. Clinical Biomechanics, 20 (10): 1038-1045.
Farfan, H.F., Cossette, J.W., Robertson, G.H., Wells, R.V., and Kraus, H. (1970) The effects of torsion on the lumbar intervertebral joints: The role of torsion in the production of disc degeneration. Journal of Bone and Joint Surgery, 52A (3): 469-497.
Farmer, M.E., Locke, B.Z., Moscicki, E.K., Dannenburg, A.L., Larson, D.B., and Radloff, L.S. (1988) Physical activity and depressive symptoms. The NHANES I epidemiologic follow-up study. American Journal of Epidemiology, 128: 1340-1351.
Frank, J.W., Brooker, A.S., DeMaio, S.E., et al. (1996) Disability resulting from occupational LBP: Part II. What do we know about secondary prevention? A review of the scientific evidence on prevention after disability begins. Spine, 21: 2918-2917.
Frost, D., Andersen, J., Lam, T., Findlay, T., Darby, K., and McGill, S.M. (2012) The relationship between general measures of fitness, passive range of motion and whole body movement quality. Ergonomics, 1-16.
Frost, D.M., Beach, T.A., Callaghan, J.P., and McGill, S.M. (2011) Movement screening for performance: What information do we need to guide exercise progression? Journal of Strength and Conditioning Research, 25: S2-S3.
Frost, D.M., Beach, T.A.C., Callaghan, J.P., and McGill, S.M. (2012) Using the functional movement screen to evaluate the effectiveness of training. Journal of Strength and Conditioning Research, 26 (6): 1620-1630.
Frost, D.M., Beach, T.A.L., Callaghan, J., and McGill, S.M. (2015) FMS scores change with performer's knowledge of the grading criteria: Are general whole body movement screens capturing "dysfunction"? doi:10.1519/JSC.0b013e3182a95343
Frost, D.M., Beach, T.A.L., Callaghan, J., and McGill, S.M. (in press) The influence of load and speed on individual's movement behaviour. Journal of Strength and Conditioning Research.
Frost, D.M., Beach, T.A.L., McGill, S.M., and Callaghan, J. (2014) The

predictive value of general movement tasks in assessing occupational task performance. Work (June 24).
Frymoyer, J.W., Pope, M.H., Clements, J.H., Wilder, D.G., MacPherson, B., and Ashikaga, T. (1983) Risk factors in low back pain. Journal of Bone and Joint Surgery, 65A: 213-218.
Gagnon, M. (2003) The efficacy of training for three manual handling strategies based on the observation of expert and novice workers. Clinical Biomechanics, 18: 601-611.
Garg, A., and Herrin, G. (1979) Stoop or squat: A biomechanical and metabolic evaluation. American Institute of Industrial Engineers Transactions, 11: 293-302.
Granata, K.P., Marras, W.S., and Davis, K.G. (1997) Biomechanical assessment of lifting dynamics, muscle activity and spinal loads while using three different style lifting belts. Clinical Biomechanics, 12 (2): 107-115.
Green, J., Grevier, S., and McGill, S.M. (2002) Low back stiffness is altered with warm-up and bench rest: Implications for athletes. Medicine & Science in Sports & Exercise, 34 (7): 1076-1081.
Grieve, D.W. (1975) Dynamic characteristics of man during crouch and stoop lifting. Biomechanics IV (eds. Nelson, R.C., and Morehouse, C.A.) (pp. 19-29) Baltimore: University Park Press.
Haex, B. (2004) Back and bed: ergonomic aspects of sleeping. CRC press.
Hamilton, W.F., Woodbury, R.A., and Harper, H.T. (1944) Arterial, cerebrospinal and venous pressures in man during cough and strain. American Journal of Physiology, 141: 42-50.
Harman, E.A., Rosenstein, R.M., Frykman, P.N., and Nigro, G.A. (1989) Effects of a belt on intraabdominal pressure during weight lifting. Medicine and Science in Sports and Exercise, 2 (12): 186-190.
Haslam, D., McCartney, N., McKelvie, R., and MacDougall, D. (1988) Direct measurements of arterial blood pressure during formal weight lifting in cardiac patients. Journal of Cardiopulmonary Rehabilitation, 8: 213-225.
Hughes, J.R. (1984) Psychological effects of habitual aerobic exercise: A critical review. Preventive Medicine, 13: 66-78.
Hunter, G.R., McGuirk, J., Mitrano, N., Pearman, P., Thomas, B., and Arrington, R. (1989) The effects of a weight training belt on blood pressure during exercise. Journal of Applied Sport Science Research, 3 (1): 13-18.

Jackson, M., Solomonow, M., Zhou, B., Baratta, R.V., and Harris, M. (2001) Multifidus EMG and tension-relaxation recovery after prolonged static lumbar flexion. Spine, 26 (7): 715-723.

Kinney, S.E., Callaghan, J., and McGill, S.M. (1996) Lumbar spine movement and muscle activity using the golfer's lifting technique. In: Evidence-based ergonomics, 28th Annual Conference of the Human Factors Association of Canada, Kitchener, ON, pp. 73-78.

Kraus, J.F., Brown, K.A., McArthur, D.L., Peek-Asa, C., Samaniego, L., and Kraus, C. (1996) Reduction of acute low back injuries by use of back supports. International Journal of Occupational and Environmental Health, 2: 264-273.

Krause, N., Dasinger, L.K., and Neuhauser, F. (1998) Modified work and return to work: A review of the literature. Journal of Occupational Rehabilitation, 8 (2): 113-139.

Krismer, M., Trobos, S., Hanna, R., Sollner, W., Schonthaler, C., Auckenthaler, T., and Watzdorf, M. (2001) Prevalence and risk factors of low back pain in school children: A cross sectional study. In: Abstracts, International Society for Study of the Lumbar Spine, Edinburgh, Scotland.

Lander, J.E., Hundley, J.R., and Simonton, R.L. (1992) The effectiveness of weight belts during multiple repetitions of the squat exercise. Medicine and Science in Sports and Exercise, 24 (5): 603-609.

Lantz, S.A., and Schultz, A.B. (1986) Lumbar spine orthosis wearing. I. Restriction of gross body motion. Spine, 11 (8): 834-837.

Le, B., Davidson, B., Solomonow, D., Zhou, B.H., Lu, Y., Patel, V., and Solomonow, M. (2009) Neuromuscular control of lumbar instability following static work of various loads. Muscle and Nerve, 39: 71-82.

Lett, K., and McGill, S.M. (2006) Pushing and pulling: Personal mechanics influence spine loads. Ergonomics, 49 (9): 895-908.

Linton, S.J., and van Tulder, M.W. (2001) Preventative interventions for neck and back pain problems. Spine, 26 (7): 778-787.

Loisel, P., Abenhaim, L., Durand, P., Esdaile, J.M., Suissa, S., Gosselin, L., Simard, R., Turcotte, J., and Lemaire, J. (1997) A population-based, randomized clinical trial on back pain management. Spine, 22 (24): 2911-2918.

Luoto, S., Helioraara, M., Hurri, H., and Alavanta, M. (1995) Static back endurance and the risk of low back pain. Clinical Biomechanics, 10: 323-324.

MacDougall, D., Tuxen, D., Sale, D., Moroz, J., and Sutton, J.R. (1985) Arterial blood pressure response in heavy resistance exercise. Journal of Applied Physiology, 58 (3): 785-790.
Mantysaari, M., Antila, K., and Peltonen, T. (1984) Relationship between the changes in heart rate and cardiac output during the Valsalva manoeuver. Acta Physiologica Scandinavica (Suppl.), 537: 45-49.
Marshall, L., and McGill, S.M. (2010) The role of axial torque/twist in disc herniation. Clinical Biomechanics, 25 (1): 6-9.
McCoy, M.A., Congleton, J.J., Johnston, W.L., and Jiang, B.C. (1988) The role of lifting belts in manual lifting. International Journal of Industrial Ergonomics, 2: 259-266.
McGill, S.M. (1993) Abdominal belts in industry: A position paper on their assets, liabilities and use. American Industrial Hygiene Association Journal, 54 (12): 752-754.
McGill, S.M. (1997) Biomechanics of low back injury: Implications on current practice and the clinic. Journal of Biomechanics, 30 (5): 465-475.
McGill, S.M. (1999a) Should industrial workers wear abdominal belts: Guidelines based on the recent literature [Invited paper]. International Journal of Industrial Ergonomics, 23 (5-6): 633-636.
McGill, S.M. (1999b) Update on the use of back belts in industry: More data —same conclusion. In: Karwowski, W., and Marras, W. (Eds.), The industrial ergonomics handbook. CRC Press.
McGill, S.M. (2014) Ultimate back fitness and performance. Waterloo, ON: Backfitpro Inc. (www.backfitpro.com)
McGill, S.M. (2008) On the use of weightbelts [Invited review]. NSCA Hot Topics Series, www.nsca-lift.org.
McGill, S.M. (2014) Ultimate back fitness and performance (5th ed.). Waterloo, ON: Backfitpro Inc. (www.backfitpro.com)
McGill, S.M., Andersen, J., and Horne, A. (2012) Predicting performance and injury resilience from movement quality and fitness scores in a basketball player population. Journal of Strength and Conditioning Research, 26 (7): 1731-1739.
McGill, S.M., and Brown, S. (1992) Creep response of the lumbar spine to prolonged lumbar flexion. Clinical Biomechanics, 7: 43-46.
McGill, S.M., Frost, D., and Crosby, I. (2013) Movement quality and links to measures of fitness in firefighters. Work, 45 (3): 357-366.
McGill, S.M., Frost, D., Lam, T., Findlay, T., Darby, K., and Andersen, J.

(2013) Fitness and movement quality of emergency task force police officers: A database with comparison to populations of emergency services personnel, athletes and the general public. International Journal of Industrial Ergonomics. http://dx.doi.org/10.1016/j.ergon.2012.11.013

McGill, S.M., Grenier, S., Bluhm, M., Preuss, R., Brown, S., and Russell, C. (2003) Previous history of LBP with work loss is related to lingering effects in biomechanical physiological, personal, and psychosocial characteristics. Ergonomics, 46 (7): 731-746.

McGill, S.M., and Hoodless, K. (1990) Measured and modelled static and dynamic axial trunk torsion during twisting in males and females. Journal of Biomedical Engineering, 12: 403-409.

McGill, S.M., and Kavcic, N. (2005) Transfer of the horizontal patient: The effect of a friction reducing assistive on low back mechanics. Ergonomics, 48 (8): 915-929.

McGill, S.M., and Kippers, V. (1994) Transfer of loads between lumbar tissues during the flexion relaxation phenomenon. Spine, 19 (19): 2190-2196.

McGill, S.M., Marshall, L., and Andersen, J. (2013) Low back loads while walking and carrying: Comparing the load carried in one hand or in both hands. Ergonomics, 56 (2): 293-302. doi:10.1080/00140139.2012.752528

McGill, S.M., and Norman, R.W. (1987) Reassessment of the role of intra-abdominal pressure in spinal compression. Ergonomics, 30 (11): 1565-1588.

McGill, S.M., and Norman, R.W. (1985) Dynamically and statically determined low back moments during lifting. Journal of Biomechanics, 18 (12): 877-885.

McGill, S.M., and Norman, R.W. (1987) Effects of an anatomically detailed erector spinae model on L4/L5 disc compression and shear. Journal of Biomechanics, 20 (6): 591-600.

McGill, S., Norman, R.W., and Sharratt, M.T. (1990) The effect of an abdominal belt on trunk muscle activity and intra-abdominal pressure during squat lifts. Ergonomics, 33 (2): 147-160.

McGill, S.M., Seguin, J.P., and Bennett, G. (1994) Passive stiffness of the lumbar torso in flexion, extension, lateral bend and axial twist: The effect of belt wearing and breath holding. Spine, 19 (6): 696-704.

Mitchell, L.V., Lawler, F.H., Bowen, D., Mote, W., Asundi, P., and Purswell, J. (1994) Effectiveness and cost-effectiveness of employer-issued back

belts in areas of high risk for back injury. Journal of Occupational Medicine, 36 (1): 90-94.
Nachemson, A.L. (1966) The load on lumbar discs in different positions of the body. Clinical Orthopaedics and Related Research, 45: 107-122.
Nachemson, A.L., Andersson, G.B.J., and Schultz, A.B. (1986) Valsalva maneuver biomechanics. Effects on lumbar trunk loads of elevated intra-abdominal pressures. Spine, 11 (5): 476-479.
National Institute for Occupational Safety and Health (NIOSH). (1994, July) Workplace use of back belts. Washington, DC: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention.
Parker, P.L., Crumpton-Young, L.L., and Brandon, K.M. (2000) Does abdominal body composition modulate the effects of back belts on the respiratory system? International Journal of Industrial Ergonomics, 26: 561-567.
Pope, M.H., Hanley, E.N., Matteri, R.E., Wilder, D.G., and Frymoyer, J.W. (1977) Measurement of intervertebral disc space height. Spine, 2: 282-286.
Porter, R.W. (1992) Is hard work good for the back? The relationship between hard work and low back pain-related disorders. International Journal of Industrial Ergonomics, 9: 157-160.
Potvin, J., Norman, R.W., and McGill, S. (1991) Reduction in anterior shear forces on the L4/L5 disc by the lumbar musculature. Clinical Biomechanics, 6: 88-96.
Preuss, R., Grenier, S., and McGill, S.M. (2005) Postural control of the lumbar spine in unstable sitting. Archives of Physical Medicine, 86: 2309-2315.
Rafacz, W., and McGill, S.M. (1996) Abdominal belts increase diastolic blood pressure. Journal of Occupational and Environmental Medicine, 38 (9): 925-927.
Reddell, C.R., Congleton, J.J., Huchinson, R.D., and Montgomery, J.F. (1992) An evaluation of a weightlifting belt and back injury prevention training class for airline baggage handlers. Applied Ergonomics, 23 (5): 319-329.
Reitan, M. (1941) On the movements of fluid inside the cerebrospinal space. Acta Radiologica Scandinavica, 22: 762-779.
Reyna, J.R., Leggett, S.H., Kenney, K., Holmes, B., and Mooney, V. (1995) The effect of lumbar belts on isolated lumbar muscle. Spine, 20 (1): 68-73.
Ross, C.E., and Hayes, D. (1988) Exercise and psychologic well-being in the

community. American Journal of Epidemiology, 127: 762-771.

Scannell, J., and McGill, S.M. (2003) Lumbar posture—Should, and can, it be modified? A study of passive tissue stiffness and lumbar position in activities of daily living. Physical Therapy, 83 (10): 907-917.

Schultz, A.B., Warwick, D.N., Berkson, M.H., and Nachemson, A. (1979) Mechanical properties of the human lumbar spine motion segments. Part 1: Responses to flexion, extension, lateral bending and torsion. Journal of Biomechanical Engineering, 101: 46-52.

Shirazi-Adl, A., Ahmed, A.M., and Shrivastava, S.C. (1986) Mechanical response of a lumbar motion segment in axial torque alone and combined with compression. Spine, 11 (9): 914-927.

Sidorkewicz, N., and McGill, S.M. (accepted October 2014a) Documenting female spine motion during coitus with a commentary on the implications for the low back pain patient. European Spine Journal, 24(3): 513-520.

Sidorkewicz, N., and McGill, S.M. (2014b) Male spine motion during coitus: Implications for the low back pain patient. Spine, 39 (20): 1633-1639.

Snook, S.H., Webster, B.S., McGarry, R.W., Fogleman, M.T., and McCann, K.B. (1998) The reduction of chronic nonspecific low back pain through the control of early morning lumbar flexion: A randomized controlled trial. Spine, 23 (23): 2601-2607.

Suni, J.H., Oja, P., Miilunpalo, S.I., Pasanen, M.E., Vuori, I.M., and Bos, K. (1998) Health-related fitness test battery for adults: Association with perceived health, mobility, and back function and symptoms. Archives of Physical Medicine and Rehabilitation, 79 (5): 559-569.

Suni, J.H., Taanila, H., Mattila, V.M., Ohrankammen, O., Vuorinen, P., Pihlajamaki, H., and Parkkari, J. (2013) Neuromuscular exercise and counseling decrease absenteeism due to low back pain in young conscripts, Spine, 38: 375-384.

Troup, J.D.G. (1977) Dynamic factors in the analysis of stoop and crouch lifting methods: A methodological approach to the development of safe materials handling standards. Orthopedic Clinics of North America, 8 (1): 201-209.

Troup, J.D.G., and Chapman, A.E. (1969) The strength of the flexor and extensor muscles of the trunk. Journal of Biomechanics, 2: 49-62.

Troup, J.D.G., Martin, J.W., and Lloyd, D.C. (1981) Back pain in industry: A prospective survey. Spine, 6: 61-69.

Twomey, L., and Taylor, J. (1982) Flexion creep deformation and hysteresis

in the lumbar vertebral column. Spine, 7: 116-122.
Ueno, K., and Liu, Y.K. (1987) A three-dimensional nonlinear finite element model of lumbar intervertebral joint in torsion. Journal of Biomechanical Engineering, 109: 200-209.
van der Molen, H.F., Sluiter, J.K., Hulshof, C., Vink, P., and Frings-Dresen, M. (2005) Effectiveness of measures and implementation strategies in reducing physical work demands due to manual handling at work. Scandinavian Journal of Work and Environmental Health, 31 (Suppl. 2): 75-87.
Van Poppel, M.N.M., Koes, B.W., van der Ploeg, T., et al. (1998) Lumbar supports and education for the prevention of low back pain in industry: A randomized controlled trial. Journal of the American Medical Association, 279: 1789-1794.
Videman, T., Nurminen, M., and Troup, J.D. (1990) Lumbar spinal pathology in cadaveric material in relation to history of back pain, occupation and physical loading. Spine, 15: 728-740.
Viggiani, D., Noguchi, M., Gruevski, K., De Carvalho, D., and Callaghan, J. (2012), Effect of wallet size on trunk angles, seat pressure, and discomfort. Canadian Journal of Kinesiology, 6 (2): 14.
Walsh, N.E., and Schwartz, R.K. (1990) The influence of prophylactic orthoses on abdominal strength and low back injury in the work place. American Journal of Physical Medicine and Rehabilitation, 69 (5): 245-250.
Wassell, J.T., Gardner, L.I., Landsittel, D.P., Johnston, J.J., and Johnston, J.M. (2000) A prospective study of back belts for prevention of back pain and injury. Journal of the American Medical Association, 284 (21): 2727-2734.
Wilder, D.G., Pope, M.H., and Frymoyer, J.W. (1988) The biomechanics of lumbar disc herniation and the effect of overload and instability. Journal of Spinal Disorder, 1: 16-32.
Winkel, J., and Westgaard, R.H. (1996) A model for solving work-related musculoskeletal problems in a profitable way [Editorial]. Applied Ergonomics, 27: 71-78.
Woo, S.L.-Y., Gomez, M.A., and Akeson, W.H. (1985) Mechanical behaviors of soft tissues: Measurements, modifications, injuries, and treatment. In: Nahum, H.M., and Melvin, J. (Eds.), Biomechanics of trauma (pp. 109-133) Norwalk, CT: Appleton-Century-Crofts.

Yassi, A., Cooper, J.E., Tate, R.B., Gerlach, S., Muir, M., Trottier, J., and Massey, K. (2001) A randomized controlled trial to prevent patient lift and transfer injuries of health care workers. Spine, 26 (16): 1739-1746.

Young, R.J. (1979) The effect of regular exercise on cognitive functioning and personality. British Journal of Sports Medicine, 13 (3): 110-117.

Zhang, Y., Sun, Z., Zhang, Z., Liu, J., and Guo, X. (2009) Risk factors for lumbar intervertebral disc herniation in a Chinese population: A case-control study. Spine, 34 (25): E918-E922.

Chapter 8: Building Better Rehabilitation Programs for Low Back Injuries

Adams, M.A., and Dolan, P. (1995) Recent advances in lumbar spine mechanics and their clinical significance. Clinical Biomechanics, 10: 3-19.

Adams, M.A., and Dolan, P. (2005) Spine biomechanics. Journal of Biomechanics, 38: 1972-1983.

Alaranta, H., Hurri, H., Heliovaara, M., Soukka, A., and Harju, R. (1994) Nondynamometric trunk performance tests: Reliability and normative data. Scandinavian Journal of Rehabilitation Medicine, 26: 211-215.

Biering-Sorensen, F. (1984) Physical measurements as risk indicators for low back trouble over a one-year period. Spine, 9: 106-109.

Bridger, R.S., Orkin, D., and Henneberg, M. (1992) A quantitative investigation of lumbar and pelvic postures in standing and sitting: Interrelationships with body position and hip muscle length. International Journal of Industrial Ergonomics, 9: 235-244.

Brown, S., and McGill, S.M. (2008) How the inherent stiffness of the in-vivo human trunk varies with changing magnitude of muscular activation. Clinical Biomechanics, 23 (1): 15-22.

Brown, S.H.M., and McGill, S.M. (2010) The relationship between trunk muscle activation and trunk stiffness: Examining a non-constant stiffness gain. Computer Methods in Biomechanics and Biomedical Engineering, 13 (6): 829-835.

Brown, S.H.M., Vera-Garcia, F.J., and McGill, S.M. (2007) Effects of abdominal bracing on the externally pre-loaded trunk: Implications for spine stability. Spine, 31: E387-398.

Butler, D.S., and Moseley, L.S. (2013) Explain pain. Adelaide, Australia: Noigroup Publications.

Cady, L.D., Bischoff, D.P., O'Connell, E.R., et al. (1979) Strength and fitness and subsequent back injuries in firefighters. Journal of

Occupational Medicine, 21 (4): 269-272.
Callaghan, J.P., Patla, A.E., and McGill, S.M. (1999) Low back three-dimensional joint forces, kinematics and kinetics during walking. Clinical Biomechanics, 14: 203-216.
Faas, A. (1996) Exercises: Which ones are worth trying, for which patients, and when? Spine, 12 (24): 2874-2879.
Findley, T.W. (2011) Fascia research from a clinician/scientist's perspective. International Journal of Therapeutic Massage & Bodywork, 4 (4): 1.
Freeman, S., Mascia, A., and McGill, S.M. (2013) Arthrogenic neuromuscular inhibition: A foundational investigation of existence in the hip joint. Clinical Biomechanics, 28: 171-177.
Fritz, J.M., Whitman, J.M., and Childs, J.D. (2005) Lumbar spinal segmental mobility assessment: An examination of validity for determining intervention strategies for patients with low back pain. Archives of Physical Medicine and Rehabilitation, 86: 1745-1752.
George, S.Z., Fritz, J.M., Bialosky, J.E., and Donald, D.A. (2003) The effect of a fear-avoidance–based physical therapy intervention for patients with acute low back pain: Results of a randomized clinical trial. Spine, 28 (23): 2551-2560.
Grenier, S., and McGill, S.M. (2007) Quantification of lumbar stability using two different abdominal activation strategies. Archives of Physical Medicine and Rehabilitation, 88 (1): 54-62.
Hides, J.A., Jull, G.A., and Richardson, C.A. (2001) Long-term effects of specific stabilizing exercises for first-episode low back pain. Spine, 26: E243-248.
Holmstrom, E., and Moritz, U. (1992) Trunk muscle strength and back muscle endurance in construction workers with and without back disorders. Scandinavian Journal of Rehabilitation Medicine, 24: 3-10.
Juker, D., McGill, S.M., Kropf, P., and Steffen, T. (1998) Quantitative intramuscular myoelectric activity of lumbar portions of psoas and the abdominal wall during a wide variety of tasks. Medicine & Science in Sports & Exercise, 30 (2): 301-310.
Koes, B.W., Bouter, L.M., Beckerman, H., et al. (1991) Physiotherapy exercises and back pain: A blinded review. British Medical Journal, 302: 1572-1576.
Koumantakis, G.A., Watson, P.A., and Oldham, J. (2005) Trunk muscle stabilization training plus general exercise versus general exercise only:

Randomized controlled trial of patients with recurrent low back pain. Physical Therapy, 85 (3): 209-225.
Krag, M.H., Seroussi, R.E., Wilder, D.G., and Pope, M.H. (1987) Internal displacement distribution from in vitro loading of human thoracic and lumbar spinal motion segments: Experimental results and theoretical predictions. Spine, 12 (10): 1001.
Lee, B., and McGill, S.M. (2015) Effect of long term isometric training of core/torso stiffness. Journal of Strength and Conditioning Research, 29(6): 1515-1526.
Leino, P., Aro, S., and Hasan, J. (1987) Trunk muscle function and low back disorders. Journal of Chronic Disease, 40: 289-296.
Lewis, C.L., Sahrmann, S.A., and Moran, D.W. (2009) Effect of position and alteration in synergist muscle force contribution on hip forces when performing hip strengthening exercises. Clinical Biomechanics, 24 (1): 35-42.
Linton, S.J., and van Tulder, M.W. (2001) Preventative interventions for neck and back pain problems. Spine, 26 (7): 778-787.
Luoto, S., Heliovaara, M., Hurri, H., et al. (1995) Static back endurance and the risk of low back pain. Clinical Biomechanics, 10: 323-324.
Manniche, C., Hesselsoe, G., Bentzen, L., et al. (1988) Clinical trial of intensive muscle training for chronic low back pain. Lancet, 24: 1473-1476.
Mannion, A.F., Taimela, S., Muntener, M., and Dvorak, J. (2001) Poor back muscle endurance: A psychological or physiological limitation? In: Abstracts, International Society for Study of the Lumbar Spine, Edinburgh, Scotland, June 19-23, p. 147.
Marras, W.S., Parnianpour, M., Ferguson, S.A., Kim, J.Y., Crowell, R.R., and Simon, S.R. (1993) Quantification and classification of low back disorders based on trunk motion. European Journal of Physical Medicine and Rehabilitation, 3: 218-235.
Mayer, T.G., Gatchel, R.J., Kishino, N., et al. (1985) Objective assessment of spine function following industrial injury: A prospective study with comparison group and one-year follow-up. Spine, 10: 482-493.
McGill, S.M. (1995) The mechanics of torso flexion: Sit-ups and standing dynamic flexion manoeuvres. Clinical Biomechanics, 10 (4): 184-192.
McGill, S.M. (2001) Low back stability: From formal description to issues for performance and rehabilitation. Exercise and Sports Science Reviews,

29 (1): 26-31.
McGill, S.M. (2014) Ultimate back fitness and performance (5th ed.). Waterloo, ON: Backfitpro Inc. (www.backfitpro.com)
McGill, S.M., and Brown, S. (1992) Creep response of the lumbar spine to prolonged full flexion. Clinical Biomechanics, 7: 43-46.
McGill, S., Frost, D., Lam, T., Finlay, T., Darby, K., and Cannon, J. (2015) Can fitness and movement quality prevent back injury in emergency task force police officers: A 5-year longitudinal trial. Ergonomics, (Epub ahead of spring).
McGill, S.M., Grenier, S., Bluhm, M., Preuss, R., and Brown, S. (2003) Previous history of LBP with work loss is related to lingering effects in biomechanical, physiological, personal and psychosocial characteristics. Ergonomics, 46 (7): 731-746.
McGill, S.M., Hughson, R., and Parks, K. (2000) Lumbar erector spinae oxygenation during prolonged contractions: Implications for prolonged work. Ergonomics, 43: 486-493.
McGill, S.M., Marshall, L., and Andersen, J. (2013) Low back loads while walking and carrying: Comparing the load carried in one hand or in both hands. Ergonomics. doi:10.1080/00140139.2012.752528
McGill, S.M., and Norman, R.W. (1992) Low back biomechanics in industry —The prevention of injury. In: Grabiner, M.D. (Ed.), Current issues of biomechanics. Champaign, IL: Human Kinetics.
McGill, S.M., Sharratt, M.T., and Seguin, J.P. (1995) Loads on spinal tissues during simultaneous lifting and ventilatory challenge. Ergonomics, 38: 1772-1792.
Moreside, J.M., and McGill, S.M. (2012) Hip joint ROM improvements using 3 different interventions. Journal of Strength and Conditioning Research, 26 (5): 1265-1273.
Nachemson, A. (1992) Newest knowledge of low back pain: A critical look. Clinical Orthopaedics, 279: 8-20.
Nicolaisen, T., and Jorgensen, K. (1985) Trunk strength, back muscle endurance and low back trouble. Scandinavian Journal of Rehabilitation Medicine, 17: 121-127.
Nutter, P. (1988) Aerobic exercise in the treatment and prevention of low back pain. Occupational Medicine, 3: 137-145.
Potvin, J.R., and Norman, R.W. (1992) Can fatigue compromise lifting safety: Proceedings NACOB II. The Second North American Congress on

Biomechanics, August 24-28, pp. 513-514.
Preuss, R., Grenier, S., and McGill, S.M. (2005) Postural control of the lumbar spine in unstable sitting. Archives of Physical Medicine, 86: 2309-2315.
Ranney, D. (1997) Chronic musculoskeletal injuries in the workplace. Philadelphia: W.B. Saunders.
Richardson, C., Jull, G., Hodges, P., and Hides, J. (1999) Therapeutic exercise for spinal stabilization in low back pain. Edinburgh: Churchill Livingstone.
Saal, J.A., and Saal, J.S. (1989) Nonoperative treatment of herniated lumbar intervertebral disc with radiculopathy: An outcome study. Spine, 14: 431-437.
Santaguida, L., and McGill, S.M. (1995) The psoas major muscle: A three-dimensional mechanical modelling study with respect to the spine based on MRI measurement. Journal of Biomechanics, 28 (3): 339-345.
Scannell, J.P., and McGill, S.M. (2009) Disc prolapse: Evidence of reversal with repeated extension. Spine, 34 (4): 344-350.
Sharma, N., Ryals, J.M., Gajewski, B.J., and Wright, D.E. (2010) Aerobic exercise alters analgesia and neurotrophion-3 synthesis in an animal model of chronic widespread pain. Physical Therapy, 90 (5): 714-725.
Sidorkewicz, N., Cambridge, E., and McGill, S.M. (2013) Can gluteus medius be targeted over TFL muscle activation during common non weight bearing hip rehabilitation exercises. Canadian Journal of Kinesiology, 6 (2): 12-13.
Snook, S.H., Webster, B.S., McGarry, R.W., Fogleman, M.T., and McCann, K.B. (1998) The reduction of chronic nonspecific low back pain through the control of early morning lumbar flexion: A randomized controlled trial. Spine, 23 (23): 2601-2607.
Solomonow, M., Zhou, B-H., Baratta, R., Burger, E., Zieske, A., and Gedalia, A. (2002) Muscular dysfunction elicited by creep of lumbar viscoelastic tissue. Journal of Electromyography and Kinesiology, 13: 381-396.
Stevenson, J.M., Weber, C.L., Smith, T., Dumas, G.A., and Albert, W.J. (2001) A longitudinal study of the development of low back pain in an industrial population. Spine, 26: 1370-1377.
Sullivan, M.S., Shaof, L.D., and Riddle, D.L. (2000) The relationship of lumbar flexion to disability in patients with low back pain. Physical Therapy, 80: 241-250.

Suni, J., Rinne, M., Natri, A., Statistisian, M.P., Parkkari, J., and Alaranta, H. (2006) Control of the lumbar neutral zone decreases low back pain and improves self-evaluated work ability: A 12-month randomized controlled study. Spine, 31 (18): E611-E620.

Troup, J.D.G., Martin, J.W., and Lloyd, D.C.E.F. (1981) Back pain in industry: A prospective survey. Spine, 6: 61-69.

Videman, T., Sarna, S., Crites-Battie, M., et al. (1995) The long-term effects of physical loading and exercise lifestyles on back-related symptoms, disability, and spinal pathology among men. Spine, 20 (6): 699-709.

Wahl, P., Bloch, W., and Schmidt, A. (2007) Exercise has a positive effect on endothelial progenitor cells, which could be necessary for vascular adaptation processes. International Journal of Sports Medicine, 28 (5): 374-380.

Chapter 9: Evaluating the Patient

Biering-Sorensen, F. (1984) Physical measurements as risk indicators for low-back trouble over a one-year period. Spine, 9: 106-119.

Casthanhero, R., Duarte, M., and McGill, S.M. (2014) Corrective sitting strategies: An examination of muscle activities and spine load. Journal of Electromyography and Kinesiology, 24(1): 114-119.

Dejanovic, A., Harvey, E., and McGill, S.M. (2012) Changes in torso muscle endurance profiles in children aged 7-14: Reference values. Archives of Physical Medicine and Rehabilitation, 93: 2295-2301.

DePalma, A.F., and Rothman, R.H. (1970) The intervertebral disc. Philadelphia: W.B. Saunders.

Dunk, N., Kedgley, A., Jenkyn, T., and Callaghan, J. (2009) Evidence of a pelvis-driven flexion pattern: Are the joints of the lower lumbar spine fully flexed in seated postures? Clinical Biomechanics, 24: 164-168.

Frost, D.M., Beach, T.A.L., Callaghan, J., and McGill, S.M. (in press) The influence of load and speed on individual's movement behaviour. Journal of Strength and Conditioning Research. .

Frost, D.M., Beach, T.A.L., Callaghan, J., and McGill, S.M. (2014a) FMS scores change with performer's knowledge of the grading criteria- Are general whole body movement screens capturing "dysfunction"? doi:10.1519/JSC.013e3182a95343

Frost, D.M., Beach, T.A.L., Callaghan, J., and McGill, S.M. (in press) The predictive value of general movement tasks in assessing occupational task performance. Work.

Frost, D.M., Crosby, I., and McGill, S.M. (in press) Firefighter injuries are not just a fireground problem. Work.

Hancock, M.J., Koes, B., Ostelo, R., and Wilco, P. (2011) Diagnostic accuracy of the clinical examination in identifying the level of herniation in patients with sciatica. Spine, 36 (11): E712-E719.

Hicks, G.E., Fritz, J.M., Delitto, A., and McGill, S.M. (2005) Preliminary development of a clinical prediction rule for determining which patients with low back pain will respond to a stabilization exercise program. Archives of Physical Medicine and Rehabilitation, 86 (9): 1753-1762.

Huntoon, E., and Huntoon, M. (2004) Differential diagnosis of low back pain. Seminars in Pain Medicine, 2 (3): 138-144.

Jeon, C.H., Chung, N.S., Lee, Y.S., Son, K.H., and Kim, J.H. (2013) Assessment of hip adductor power in foot drop patients: A simple and useful test to differentiate lumbar radiculopathy and peroneal neuropathology. Spine, 38(3): 257-263.

Juker, D., McGill, S.M., Kropf, P., and Steffen, T. (1998) Quantitative intramuscular myoelectric activity of lumbar portions of psoas and the abdominal wall during a wide variety of tasks. Medicine & Science in Sport & Exercise, 30 (2): 301-310.

LaBan, M.M. (2008) Comment on: Musculoskeletal disorders in suspected cervical radiculopathy. Archives of Physical Medicine and Rehabilitation, 88 (10): 1256-1259.

Liebenson, C., Howarth, S., Brown, S., and McGill, S. (2009) The active straight leg raise test as an indicator of lumbar spine control and stability. Physical Medicine Rehabilitiation, 1(6): 530-535.

Liebenson, C., Karpowicz, A., Brown, S., Howarth, S., and McGill, S.M. (2009) The active straight leg raise test and lumbar spine stability. Physical Medicine and Rehabilitation, 1 (6): 530-535.

McGill, S.M. (2014) Ultimate back fitness and performance. Waterloo, ON: Backfitpro Inc. (www.backfitpro.com)

McGill, S.M., Belore, M., Crosby, I., and Russell, C. (2010) Comparison of two methods to quantify torso flexion endurance. Occupational Ergonomics, 9: 55-61.

McGill, S.M., Childs, A., and Liebenson, C. (1999) Endurance times for stabilization exercises: Clinical targets for testing and training from a normal database. Archives of Physical Medicine and Rehabilitation, 80: 941-944.

McGill, S.M., Frost, D., Lam, T., Finlay, T., Darby, K., and Andersen, J. (in press) Fitness and movement quality of emergency task force police officers: An age-grouped database with comparison to populations of emergency services personnel, athletes and the general public.

McGill, S.M., Grenier, S., Bluhm, M., Preuss, R., Brown, S., and Russell, C. (2003) Previous history of LBP with work loss is related to lingering effects in biomechanical physiological, personal, and psychosocial characteristics. Ergonomics, 46 (7): 731-746.

McGill, S.M., and Yingling, V.R. (1999) Traction may enhance the imaging of spine injuries with plane radiographs: Implications for the laboratory versus the clinic. Clinical Biomechanics, 14 (4): 291-295.

Mens, J.M.A., Vleeming, A., Snijders, C.J., Stam, H.J., and Ginai, A.Z. (1999) Active straight leg raising test and mobility of the pelvic joints, European Spine Journal, 8 (6): 468-473.

Morgan, W.E. (2013) The lumbar MRI in clinic practice: A survey of lumbar MRI for musculoskeletal clinicians. Ijamsville, MD: Bethesda Spine Institute.

Nelson-Wong, E., and Callaghan, J. (2014) Transient low back pain development during standing predicts future clinical low back pain in previously asymptomatic individuals. Spine, 39 (6): E379-E383.

Nelson-Wong, E., Flynn, T., and Callaghan, J.P. (2009) Development of active hip abduction as a screening test for identifying occupational low back pain. Journal of Orthopaedic & Sports Physical Therapy 39 (9): 649-657.

Parks, K.A., Crichton, K.S., Goldford, R.J., and McGill, S.M. (2003) On the validity of ratings of impairment for low back disorders. Spine, 28 (4): 380-384.

Peach, J.P., and McGill, S.M. (1998) Classification of low back pain with the use of spectral EMG parameters during submaximum isometric fatiguing contractions and recovery. Spine, 23 (10): 1117-1123.

Peterson, D. (2013) Proposed performance standards for the plank and inclusion consideration into the navy's physical readiness test. Strength and Conditioning Journal, 35 (5): 22-26.

Preuss, R., Grenier, S., and McGill, S.M. (2005) Postural control of the lumbar spine in unstable sitting. Archives of Physical Medicine, 6: 2309-2315.

Richardson, C., Jull, G., Hodges, P., and Hides, J. (1999) Therapeutic

exercise for spinal segmental stabilization in low back pain. Edinburgh: Churchill Livingstone.
Rihn, J.A., Lee, J.Y., Khan, M., Ulibarri, J.A., Tannoury, C., Donaldson, W.F., and Kang, J.D. (2007) Does lumbar facet fluid detected on magnetic resonance imaging correlate with radiologic instability in patients with degenerative lumbar disease? Spine, 32 (14): 155-1560.
Ross, J.K., Bereznick, D., and McGill, S.M. (1999) Atlas-axis facet asymmetry: Implications for manual palpation. Spine, 24 (12): 1203-1209.
Saulino, M.F., Kornbluth, I.D., Overton, E.A., Holding, M.Y., and Freedman, M.K. (2008) Interventions in chronic pain management 3. Evaluation and management of lumbar pain syndromes. Archives of Physical Medicine and Rehabilitation, 89 (3): S47-S50.
Scannell, J.P., and McGill, S.M. (2009) Disc prolapse: Evidence of reversal with repeated extension. Spine, 34 (4): 344-350.
Sembrano, J.N., and Polly, D. W. (2008) How often is low back pain not coming from the back? Spine, 34 (1): E27-E32.
Smith, A., O'Sullivan, P., and Straker, L. (2008) Classification of sagittal thoraco-lumbo-pelvic alignment of the adolescent spine in standing and its relationship to low back pain. Spine, 33 (19): 2101-2107.
Young, S., Aprill, C., and Laslett, M. (2003) Correlation of clinical examination characteristics with three sources of chronic low back pain. The Spine Journal, 3: 460-465.

Chapter 10: Developing the Exercise Program

Andersson, E.A., Oddsson, L., Grundstrom, O.M., Nilsson, J., and Thorstensson, A. (1996) EMG activities of the quadratus lumborum and erector spinae muscles during flexion-relaxation and other motor tasks. Clinical Biomechanics, 11: 392-400.
Axler, C., and McGill, S.M. (1997) Low back loads over a variety of abdominal exercises: Searching for the safest abdominal challenge. Medicine & Science in Sports & Exercise, 29 (6): 804-811.
Biering-Sorensen, F. (1984) Physical measurements as risk indicators for low-back trouble over a one-year period. Spine, 9: 106-119.
Brunalle, C.L., and Mulgrew, J.A. (2011) Exercise for intermittent claudication. Physical Therapy, 91 (7): 997-1002.
Butler, D.S. (1999) Mobilization of the nervous system. Edinburgh: Churchill Livingstone.
Callaghan, J.P., Gunning, J.L., and McGill, S.M. (1998) Relationship

between lumbar spine load and muscle activity during extensor exercises. Physical Therapy, 78 (1): 8-18.

Cholewicki, J., and McGill, S.M. (1996) Mechanical stability of the in vivo lumbar spine: Implications for injury and chronic low back pain. Clinical Biomechanics, 11 (1): 1-15.

Hirsch, A.T., Haskal, Z.T., Hertzer, N.R., et al. (2006) ACC/AHA 2005 guidelines for the management of patients with peripheral arterial disease. Journal of the American College of Cardiology, 47: 1239-1312.

Ikeda, D., and McGill, S.M. (2012) Can altering motions, postures and loads provide immediate low back pain relief: A study of four cases investigating spine load, posture and stability. Spine, 37 (23): E1469-E1475.

Juker, D., McGill, S.M., Kropf, P., and Steffen, T. (1998) Quantitative intramuscular myoelectric activity of lumbar portions of psoas and the abdominal wall during a wide variety of tasks. Medicine & Science in Sports & Exercise, 30 (2): 301-310.

Kavcic, N., Grenier, S., and McGill, S. (2004) Determining the stabilizing role of individual torso muscles during rehabilitation exercises. Spine, 29 (11): 1254-1265.

Louis, R. (1981) Vertebroradicular and vertebromedullar dynamics. Anatomica Clinica, 3: 1-11.

Luomajoki, H., Kool, J., de Bruin, E.D., and Airaksinen, O. (2008) Movement control tests of the low back: Evaluation of the difference between patients with low back pain and healthy controls. BMC Musculoskeletal Disorders, 9: 170. doi:10.1186/1471-2472-9-170

Luoto, S., Heliovaara, M., Hurri, H., and Alaranta, M. (1995) Static back endurance and the risk of low back pain. Clinical Biomechanics, 10: 323-324.

McGill, S.M. (2014) Ultimate back fitness and performance (5th ed.). Waterloo, ON: Backfitpro Inc. (www.backfitpro.com)

McGill, S.M., Grenier, S., Bluhm, M., Preuss, R., Brown, S., and Russell, C. (2003) Previous history of LBP with work loss is related to lingering effects in biomechanical physiological, personal, and psychosocial characteristics. Ergonomics, 46 (7): 731-746.

McGill, S.M., Hughson, R., and Parks, K. (2000) Lumbar erector spinae oxygenation during prolonged contractions: Implications for prolonged work. Ergonomics, 43: 486-493.

McGill, S.M., and Karpowicz, A. (2009) Exercises for spine stabilization:

Motion/motor patterns, stability progressions and clinical technique. Archives of Physical Medicine and Rehabilitation, 90: 118-126.
Moreside, J.M., Vera-Garcia, F., and McGill, S.M. (2008) Neuromuscular independence of abdominal wall muscles as demonstrated by middle-eastern style dancers. Journal of Electromyography and Kinesiology, 18: 527-537.
Suni, J.H., Taanila, H., Mattila, V.M., Ohrankammen, O., Yourinen, P., Pihlajamaki, H., and Parkkari, J. (2013) Neuromuscular exercise and councilling decrease absenteeism due to low back pain in young conscripts. Spine, 38 (5): 375-384.
Vera-Garcia, F.J., Grenier, S.G., and McGill, S.M. (2000) Abdominal response during curl-ups on both stable and labile surfaces. Physical Therapy, 80 (6): 564-569.

Chapter 11: Advanced Exercises

Cholewicki, J., McGill, S.M., and Norman, R.W. (1991) Lumbar spine loads during lifting extremely heavy weights. Medicine & Science in Sports & Exercise, 23 (10): 1179-1186.
Lee, B., and McGill, S.M. (2015) Effect of long term isometric training of core/torso stiffness. Journal of Strength and Conditioning Research, 29(6): 1515-1526.
McGill, S.M. (2014) Ultimate back fitness and performance (5th ed.). Waterloo, ON: Backfitpro Inc. (www.backfitpro.com)
McGill, S.M., and Andersen, J. (2014) Analysis of pushing exercises: Muscle activity and spine load while contrasting techniques on stable surfaces with labile suspension strap training system. Journal of Strength and Conditioning Research, 28 (1): 105-16.
McGill, S.M., and Andersen, J. (in press) Physiological and biomechanical mechanisms in hula hooping: Caloric expenditure and spine loads.
McGill, S.M., and Andersen, J. (2015) A six week trial of hula hooping using a weighted hoop: Effects on skinfold, girths, weight and torso muscle endurance. Journal of Strength and Conditioning Research, 29 (5): 1279-1284.
McGill, S.M., Andersen, J., and Horne, A. (2012) Predicting performance and injury resilience from movement quality and fitness scores in a basketball player population. Journal of Strength and Conditioning Research, 26 (7): 1731-1739.
McGill, S.M., Cambridge, E., and Andersen, J. (2014) Muscle activity and

spine load during pulling exercises: Influence of stable and labile contact surfaces and technique coaching. Journal of Electromyography and Kinesiology, 24 (5): 652-665.

McGill, S.M., Cannon, J., and Andersen, J. (2014) Muscle activity and spine load during anterior chain whole body linkage exercises: The body saw, hanging leg raises and walkout form a pushup. Journal of Sports Sciences. doi:10.1080/026 40414.2014.946 437

McGill, S.M., Chaimberg, J., Frost, D., and Fenwick, C. (2010) The double peak: How elite MMA fighters develop speed and strike force. Journal of Strength and Conditioning Research, 24 (2): 348-357.

McGill, S.M., Frost, D., and Crosby, I. (2013) Movement quality and links to measures of fitness in firefighters. Work, 45 (3): 357-366.

McGill, S.M., Frost, D., Lam, T., Findlay, T., Darby, K., and Andersen, J. (2103) Fitness and movement quality of emergency task force police officers: A database with comparison to populations of emergency services personnel, athletes and the general public [Invited paper]. International Journal of Industrial Ergonomics. http://dx.doi.org/10.1016/j.ergon.2012.11.013

McGill, S.M., Grenier, S., Bluhm, M., Preuss, R., Brown, S., and Russell, C. (2003) Previous history of LBP with work loss is related to lingering effects in biomechanical physiological, personal, and psychosocial characteristics. Ergonomics, 46 (7): 731-746.

McGill, S.M., and Marshall, L. (2012) Kettlebell swing snatch and bottoms-up carry: Back and hip muscle activation, motion, and low back loads. Journal of Strength and Conditioning Research, 26 (1): 16-27.

McGill, S.M., McDermott, A., and Fenwick, C. (2008) Comparison of different strongman events: Trunk muscle activation and lumbar spine motion, load and stiffness. Journal of Strength and Conditioning Research, 23 (4): 1148-1161.

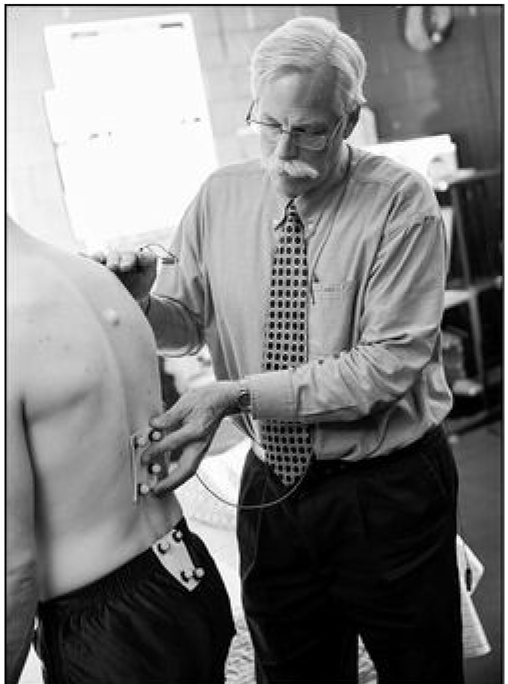
McGill, S.M., Sharratt, M.T., and Seguin, J.P. (1995) Loads on spinal tissues during simultaneous lifting and ventilatory challenge. Ergonomics, 38: 1772-1792.

Moreside, J.M., and McGill, S.M. (2012) How do elliptical machines differ from walking: A study of torso motion and muscle activity. Clinical Biomechanics, 27: 738-743.

Siff, M.C. (2003) Supertraining. Denver, CO: Supertraining Institute.

Vera-Garcia, F.J., Grenier, S.G., and McGill, S.M. (2000) Abdominal response during curl-ups on both stable and labile surfaces. Physical Therapy, 80 (6): 564-569.

About the Author



Stuart McGill, PhD, is a professor at the University of Waterloo, Ontario, Canada, and a world-renowned lecturer and expert in spine function, injury prevention, and rehabilitation. He has written more than 300 scientific publications on lumbar function, mechanisms of low back injury, investigation of rehabilitation programs matched to specific categories of back pain patients, and the formulation of work-related injury avoidance strategies. Recent publications include Back Mechanic: The Step-by-Step McGill Method to Fix Back Pain (www.backfitpro.com) written for the lay public with back pain. He has received several awards for his work, including the Volvo Bioengineering Award for Low Back Pain Research from Sweden.

McGill has been an invited lecturer at many universities and delivered more than 300 addresses to societies around the world. As a consultant, he has provided expertise on assessment and reduction of the risk of low back injury to government agencies, corporations, professional athletes and teams, and legal firms. He is one of the few scientists who, in addition to performing research, is regularly requested by the medical profession to consult with challenging patients from around the world.