Introduction to Manual Medicine: An Osteopathic Perspective

First Edition

Introduction to Manual Medicine: An Osteopathic Perspective

First Edition
Published by PBJ Publications LLC

5 Digges Drive
Newport News, Virginia 23602

Printed in the USA

ISBN (Hardback): 978-1-7351120-0-8
ISBN (ePub): 978-1-7351120-1-5
ISBN (mobi): 978-1-7351120-2-2

Disclaimer:
Care has been taken to confer accuracy of the information presented in this text and to describe generally accepted applications toward medical practice. However, the authors, editor, and publisher are not responsible for errors or omissions, or the consequences from application of the information presented in the book. Application of this information in clinical practice or other professional setting remains the responsibility of the of the practitioner; clinical treatments, surgery, manual medicine, prolotherapy, and other procedures described and recommended may not be considered standard of care in a particular location, absolute, nor universal recommendations.

The authors, editor, and publisher have exercised every effort to ensure that drug choice and dosage used in treatment examples are in accordance with current recommendations at the time of publication. However, due to changes in government regulations, ongoing research, standard of care, and future unforeseen complications with the application of over-the-counter and prescription medications, the reader is recommended to check package inserts for any drug change in indications and dosage and for warnings and precautions. It is the practitioner's responsibility to ensure the FDA status of each drug or device planned for use in clinical practice.

Simplify

“If you cannot explain it simply, you don’t understand it well enough.”
- Albert Einstein (1879-1955)

Mastery

“The expert is the one who does the basics the best.”
- Philip E. Greenman, DO, FAAO (1928-2013)

Foreword:

Finally! This is the book I was looking for when I was a medical student. It is a welcome addition to the text/reference books or articles for the beginner or the serious student of manual medicine.

Many people, if they have heard of Osteopathic manipulation at all, confuse it with Orthopedics. If asked for a definition, they often guess, "Something to do with bones?" They are partly right. The bones are commonly involved, but correctly utilized, osteopathic manipulation is much more than that. In my nearly forty years of practice, I have used osteopathic manipulation to effectively treat patients with neck pain, back pain, pelvic pain, and extremity pain for which Osteopathic manipulation is generally considered to be useful. In addition, I have treated people with the inconvenience of hiccups, asthma, diarrhea, and constipation plus the pain of headache, fallen arches, TMJ, dental malocclusion, otitis; and conditions such as edema, pneumonia, and congestive heart failure.

There is nothing magical about Osteopathic manipulation, but is does require the ability to think, a working knowledge of human anatomy, well trained hands, and a willingness to consider a new philosophy. Andrew Taylor Still, MD, the father of Osteopathic medicine, said "I give you the principles. You work out the details." This book addresses both principles and details.

Medical schools were almost non-existent at the time, but Still studied the methods and medicines that were available, however ineffective they may have been. He began to study the human body much more intently. Not just the effects of the available medicines, but he studied how the various components of the human body worked together and functioned. As a Civil War Surgeon, he saw and experienced firsthand the failings of nineteenth century medicine. He believed there must be a better way.

He developed a way of thinking about human health and illness that was unique in western medicine. His philosophy was successful in returning a person to health, even after other doctors had given up. In fact, his philosophy and methods were so successful that on at least one occasion he was arrested, and his son was actually jailed in an attempt to preserve the status quo in the medical community. He said, "My father was a progressive farmer and was always ready to lay aside an older plow if he could replace it with one better constructed for its work. All through life, I have ever been ready to buy a better plow." I believe he found a better "plow."

When I was in medical school and later when I was in practice, many of the "How To" books that were available described step-by-step procedures using a single system of osteopathic manipulation like a "cookbook." I wanted a book that would help me understand the "why" as well as the "how". I longed for a text/reference book that would provide a quick and comprehensive review of osteopathic principles and a variety of hands-on approaches to health in actual practice. I wanted the book organized by body regions so I could easily compare the different methodologies for my self and select the one best suited to the patient in front of me, without having to flip through multiple books or even chapters.

I was excited when I learned that Dr. Browder had started writing just such a book. He found that in some cases, the most sensitive diagnostic tool available to any physician, and one of the most effective and powerful tools for treatment, comes with an efficient built-in power supply and a high-speed computer to process all of the data. It is cost-effective, readily available, completely portable, and it is conveniently located at the end of your arm. This book is an instruction manual for learning how to use it.

It is the…
HUMAN HAND.

Al Turner, DO
Portland 2019

Preface:

Arrogantly, when I first began this text, I felt it would be easy to complete, professionally well received, and at most a one-to-two year venture. Much the opposite - military service and working career, marriage, and two children later, this book represents the culmination of twelve years work. I asked Dr. Stiles, in 2001, during my second year of medical school, why had he not written a book. He replied, "I write 60 books a year." I now wish he had told me the truth, it will take twelve years, it's a labor of love and not money - but really, it's a pain in the ass.

I feel privileged to have unwittingly received an excellent foundation in Osteopathic Principles and Practice (OPP) as a first- and second-year medical student while enrolled at Pikeville College School of Osteopathic Medicine (PCSOM), now Kentucky School of Osteopathic Medicine. Ed Stiles was the OPP department chair and was laser focused on his goal: to have Pikeville students fully capable of practicing Osteopathic Manipulative Medicine (OMM) upon entering third- and fourth-year clinical rotations. And so, he developed a program which taught with this goal in mind:

1. Teach principles, avoid memorization
2. Simplify differing treatment modalities through cohesion of diagnosis and treatment
3. Teach a method of putting it all together – Sequencing

My thoughts on his method are as follows:

1. **Teach principles, avoid memorization** – this allows students the freedom to develop new methods of diagnosis and treatment. I recall a Pikeville student developed a new method of localizing the sacral oblique axis by instructing his patient to cross their legs during OPP testing. Unshaken, Stiles went to the patient, used the same process and concurred that this method worked equally well. To me, this is medical perfection.

2. **Simplify differing treatment modalities through cohesion of diagnosis and treatment** – the method Stiles used during the first semester and into the beginning of the second was as follows:
    a. Basic principles
    b. Thoracic spine – diagnosis using spinal mechanics with active range of motion (ROM) testing and muscle energy treatment (ME)
    c. Lumbar spine – diagnosis using spinal mechanics with active ROM testing and MET
    d. Cervical spine – diagnosis using spinal mechanics with passive ROM and ME
    e. Sacrum and pelvis – diagnosis with Mitchell Model and ME
    f. Upper extremity – diagnosis with active ROM testing and ME
    g. Lower extremity – diagnosis with active ROM testing and ME
    h. Sequencing

    He then taught the following for the remainder of the first year:
    a. Thoracic spine – diagnosis using spinal mechanics with active ROM testing and functional treatments
    b. Lumbar spine – diagnosis using spinal mechanics with active ROM testing and functional treatments
    c. Cervical spine – diagnosis using spinal mechanics with passive ROM and functional treatments
    d. Sacrum and pelvis – diagnosis with Mitchell Model and functional treatments

e. Upper extremity – diagnosis with active ROM testing and functional treatments
f. Lower extremity – diagnosis with active ROM testing and functional treatments

Why is this brilliant? One - students learn to diagnose and treat the whole body (minus the head) in about 6 months using an extremely safe direct treatment modality. Two – during the spring semester, he repeated the diagnostic methods for the whole body, just in case you missed something, and incorporated them using indirect treatments. Importantly, Stiles "cracked the code" using spinal mechanics nomenclature developed by Fred Mitchell Sr., DO to link direct and indirect treatments (see Principles Chapter). Three - he gave us a method to determine a treatment starting point with his sequencing method. Effectively, the PCSOM student was armed with two modalities of treating the whole body after their first year and a method of determining a place to begin patient treatment. If that weren't enough, we were issued a treatment table as part of our tuition. Those of us genuinely interested in Osteopathy were unabashedly treating family, friends, and each other. Confidence grew as we achieved good and sometimes miraculous results as first year students.

In our second year, our first semester curriculum was taught in two-week segments including:

a. Thoracic spine – diagnosis using spinal mechanics with active ROM testing and high-velocity low amplitude treatment (HVLA)
b. Lumbar spine – diagnosis using spinal mechanics with active ROM testing and HVLA
c. Cervical spine – diagnosis using spinal mechanics with passive ROM and HVLA
d. Sacrum and pelvis – diagnosis with Mitchell Model and HVLA
e. Upper extremity – diagnosis with active ROM testing and HVLA
f. Lower extremity – diagnosis with active ROM testing and HVLA
g. Cranial

Second semester, second year was also taught in two-week segments and included:

a. Thoracic spine – diagnosis using spinal mechanics and correlation with tenderpoints, treatment with strain-counterstrain (S/CS).
b. Lumbar spine – diagnosis using spinal mechanics and correlation with tenderpoints, treatment with S/CS
c. Cervical spine – diagnosis using spinal mechanics and correlation with tenderpoints, treatment with S/CS
d. Sacrum and pelvis – diagnosis with Mitchell Model and correlation with tenderpoints, treatment with S/CS
e. Upper extremity – diagnosis with active ROM testing and correlation with tenderpoints, treatment with S/CS
f. Lower extremity – diagnosis with active ROM testing and correlation with tenderpoints, treatment with S/CS

By the end of our first two years we had gone through the diagnosis of the whole body four times and were competent with two direct and two indirect treatment modalities (we also had a smattering of myofascial release). Stiles did not teach for the Colleges of Osteopathic Medical Examination (COMLEX) board examination… he taught with his goal in mind.

3. **Teach a method of putting it all together – Sequencing.** To the unaware reader, most students learn hundreds of procedures but have no method to determine how to apply them during a patient visit. Stiles' method may not be the only one, but in this writer's opinion it was the

catalyst that allowed me to determine significant primary somatic dysfunction and avoid chasing pain.

Pikeville students did not recognize the brilliance of Stiles' teaching method until we attended the American Academy of Osteopathy annual retreat and began clerkship rotations; we were Osteopathic rock-stars. We diagnosed and treated with confidence. When faced with "out-of-the-box" situations, we applied principles. We were at risk of becoming arrogant.

Further thoughts:

In conceiving this book, Paul and I wanted to do what Stiles would not - produce a book that was principle based, easy to understand, and provided cohesion among treatment modalities. We wanted to also include items we felt would serve beginning students: specifically, evidenced based medicine (EBM), physiology, and orthopedic special testing. We hope the reader will appreciate the emphasis on principles and the "why" of OMM more than the how.

Whether you like it or not, EBM is part of medicine. As a practitioner of manual medicine, I grow ever tired of correcting colleagues who dismiss OMT, claiming it has no supporting clinical evidence. Likewise, I cringe when a fellow physician proclaims OMT as a panacea. Let the evidence speak for itself. If we as a profession want answers to whether or not OMT is effective for condition X, Y, or Z, then we, as a profession, need to dedicate time and money to that end.

Anatomy and physiology are cornerstone curricula for medical school. Irvin Korr, PhD, Frank Willard, PhD, Viola Fryman, DO, and others have spent their careers providing us with excellent anatomical and physiological evidence for OMT. We wanted to create a simplified overview of evidence. In this text we have relied heavily on Frank Willard's lectures and writing to provide the reader with basic neurophysiology.

-Jason Browder, DO

Acknowledgements:

To our mentors:
-Al Turner, DO, who introduced us to co-author Hollis King, DO, PhD, FAAO. Dr. Turner further welcomed Paul and I as medical and post-graduate students in his Portland, OR office, and we are honored for him to write our book's forward.
-To Bryan Knight, DO, who worked with Dr. Turner and provided him a break during our rotations with him.
-To Thomas Crow, DO, FAAO, and John Jones, DO, who I worked with in Philadelphia. Although I was a student, I was treated like a colleague, and I never forgot it. I pass this spirit along to my medical students.
-To Judith O'Connell, DO, FAAO, who provided me sanctity from Internal Medicine residency. Thank you for teaching me good cranial manual medicine.
-To Shirley Winters and David B. Winters, DO, who allowed me to work with them as an undergraduate student and propelled me into medicine.
-Ed Stiles, DO, FAAO, as above.
-Philip Greenman, DO, FAAO, my personal hero.

To those who have helped bring this text beyond its humble beginnings:
-Ray Hruby, DO, FAAO, who reviewed the text in entirety and provided an extensive critique.
-Rob Foster, our artist. Thank you for remaining patient and dedicating your free time to our purpose.
-James Thompson, DO, Cody Talbot, and Martin Peters, DO who served as our models.
-Penny Thompson who painstakingly edited this text and never once complained.

To our families:
-To my wife Diane Maddela, DO, for allowing me to indulge my passions in Osteopathy.
-Patricia Johnson for allowing Paul to take time away from your personal life to write this text with me.
-My parents William and Glenda Browder for allowing me to purse my passions and providing me a solid foundation for life.

About the Authors:

Jason P. Browder, DO, earned his undergraduate degree from the University of Virginia in 1998 and his medical degree from the University of Pikeville-Kentucky College of Osteopathic Medicine in 2003. He completed an Internal Medicine internship and residency in 2006 at Wright-Patterson Air Force Base in Dayton, Ohio.

Dr. Browder is board certified in Internal Medicine and has a special medical interest in Osteopathic Manual Medicine (OMM)**,** Internal Medicine, and Regenerative Medicine treatments such as prolotherapy and platelet rich plasma.

Prior to joining Tidewater Physician Multispecialty Group (TPMG) in 2009, Dr. Browder served as a Major in the U.S. Air Force Medical Corps at Langley Air Force Base. Throughout his military career, Dr. Browder was selected to serve in various supervisory and liaison positions, including Director of Internal Medicine, Director of the Coumadin Clinic, and Liaison to the NASA Clinics.

Dr. Browder is a member of multiple professional organizations including the American Osteopathic Association, American Academy of Osteopathy, Virginia Osteopathic Association and the American Board of Medicine. He is also an Adjunct Professor of Internal Medicine at the Edward Via School of Osteopathic Medicine, where he was named eastern-regional preceptor of the year for graduating classes of 2018 and 2020. Dr. Browder has also lectured extensively on Osteopathic Medicine and Osteopathic principles and practice.

In addition to his teaching duties, Dr. Browder serves on the TPMG Board of Directors from 2016-present. He has also been honored as a Coastal Virginia Magazine “Top Doc” for consecutive years since 2014, as voted for by his peers in the Hampton Roads medical community.

Paul Johnson, DO, is board certified in both Internal Medicine and Sports Medicine and has been practicing Sports Medicine since 2006 in the Portland/Vancouver area. He attended the University of Pikeville-Kentucky College of Osteopathic Medicine, and then onto Rush Presbyterian – St. Luke's Medical Center / Cook County Hospital, in Chicago, IL, for residency in Internal Medicine, followed by a Sports Medicine fellowship at the University of Pittsburgh.

Dr Johnson is a specialist in the non-surgical management of acute and over-use musculoskeletal injuries, Osteopathic Manual Medicine, concussion management, and ultrasound guided regenerative injections. He works closely with other medical specialists, athletic trainers, and physical therapists to provide comprehensive musculoskeletal care and rehabilitation for athletes and non-athletes alike.

His interest in sports and osteopathic medicine began in high school after experiencing sport-related injuries playing hockey and football. He believes that start-to-finish, personalized medicine with informed patients contribute to his philosophy that collaboration is the key element towards health and wellness. His relate-able, accessible, and practical approaches are what set him apart.

Hollis H. King, DO, PhD, FAAO, FCA, earned a bachelor's degree from Duke University, a master's degree in Psychology from Trinity University. He earned a PhD in Clinical Psychology from Louisiana State University. His medical training was at the University of North Texas Health Science Center, Texas College of Osteopathic Medicine. His graduate medical training was at Dallas Memorial Hospital. He has served on the Osteopathic Manipulative Medicine department faculties at Western University of Health Science, College of Osteopathic Medicine of the Pacific, the Texas College of Osteopathic Medicine, and the Andrew Taylor Still University School of Osteopathic Medicine in Arizona. He also served as the Associate Executive Director of the Osteopathic Research Center in Fort Worth.

He is currently a Clinical Professor at the University of California San Diego School of Medicine, which he joined after serving in a similar position at the University of Wisconsin, Madison. Dr. King is a Past President and Fellow of the American Academy of Osteopathy and a Fellow of the Osteopathic Cranial Academy. His private practice is at the Osteopathic Center for Children, continuing the work of Viola M. Frymann, DO.

Dr. King was the Editor in Chief of *The Science and Clinical Application of Manual Therapy,* and has authored chapters on "Osteopathic Cranial Manipulative Medicine" in the last three editions of *Foundations of Osteopathic Medicine.* He has authored and co-authored over 20 peer reviewed articles on osteopathic clinical research. He is currently conducting clinical research on the effects of osteopathic treatment on Parkinson's disease, glaucoma, traumatic brain injury and deformational plagiocephaly.

About the Artist:

Rob Foster is a freelance Visual Problem Solver, working as an illustrator, sketchnoter, painter and cartoonist from Greater Atlanta area for more than 25 years.

Finding it difficult to choose between a deep interest in biology and artistic talent, illustrator Rob Foster was one of a select few to receive a Bachelor of Science degree in Biological Illustration from the University of Florida, which allowed him to explore and excel in both. His senior projects were a children's book of insects and a drawing collection of native Florida freshwater fish. Earning an Illustration Certificate from the Portfolio Center in Atlanta followed.

Foster has completed commercial projects for national brands such as Cox Communications, Holiday Inn, Hill York Air Conditioning, Popeye's Simmons Co. and Cottrell Media for Cartoon Network.

Noteworthy creations in his freelance portfolio are:

- Illustrations and animations for the Centers for Disease Control Antimicrobial Susceptibility Testing Self Study Program, for which he won the CDC/ASTDR Communicator's Roundtable Award for Electronic Media
- Graphics depicting the scientific concept in child friendly illustrations for each exhibit in the Atlanta Botanical Garden Children's Gardens
- Graphics and logo for Georgia State Parks system
- Mural depicting local wildlife for Dunwoody Nature Center, Sandy Springs, GA
- Dinosaur hall exhibit display for Teaching Museum South in Atlanta, GA
- Interpretive graphics for Pritchett and Hull Health Publishing
- Sketchnoting, the live on-site-real-time drawing to capture concepts visually during meetings for technology conferences and companies including Home Depot and City Bank
- Book illustrations for The Paleo Diet Mom, Dr. Sarah Ballantyne
- Precise biological illustrations for use as medical posters, charts and (this?) textbooks

When not engaged at the intersection of art, science and technology, Foster can be found leading classes as an Adaptive Art Special Education teacher. He holds a Specialist in Education degree in Instructional Technology from the University of West Georgia.

Contents:

Foreword: Goals for this Text

From the perspective of the osteopathic medical profession, the teaching of manual medicine, referred to as Osteopathic Manipulative Treatment (OMT), has always been a challenge. The main reasons for this conundrum distill down to the wide diversity of modalities and the limited time available to teach all aspects of the approaches of OMT in the basic osteopathic curriculum in osteopathic medical school. When OMT is provided in an encounter with a patient who also receives standard of practice medical care for conditions like upper respiratory infections and asthma, the term Osteopathic Manipulative Medicine (OMM) is used. This book addresses both OMT and OMM.

Now that negotiations are complete between the American Osteopathic Association (AOA), the Association of Colleges of Osteopathic Medicine (AACOM) and the Accreditation Council for Graduate Medical Education (ACGME), there exists a single accreditation system with unified residency training standards for graduate medical education in the United States. Previously established and functioning within the ACGME are two new Residency Review Committees (RRCs): one for Neuromusculoskeletal Medicine (NMSM) and one for Osteopathic Principles and Practice (OPP).

Even before the completion of negotiations, it was apparent that the osteopathic medical perspective and its distinctiveness are accepted and firmly entrenched in mainstream medicine. This presents not only great opportunity, but also a challenge, as the teaching of OMT, OMM and OPP reach a much wider audience of not only osteopathic medical students and residents but also their counterparts who go through allopathic or MD training institutions. Also, both the accreditation system and unified residency training standards have inspired interest in OMT and OMM in many MD physicians. This text will serve as a reference for Continuing Medical Education (CME) courses designed for interested MDs.

The authors initiated this book project in response to their perception that there needed to be a better way to teach introductory OMT, OMM, and OPP in osteopathic medical training. Their goal is to present relevant and meaningful introductory level OMT concept and practices in the first courses taught to all who begin the study and practice of OMT and OMM. It is intended to provide the basis of curriculum for programs introducing OMT, OMM, and OPP in both undergraduate and residency programs that are still primarily allopathic or osteopathic and address the needs of allopathic medical school graduates who may enter primarily osteopathic residencies. As the chapters neared completion, content was included to help the allopathic medical students and residents see how OMT, OMM, and OPP are most applicable in any medical clinical setting.

While heavy in osteopathic nomenclature, the concepts and principles presented are perceived and intended to be universally applicable in the training of all OMT educational programs. The concepts and applications of sequencing and functional procedures presented in this text are based on the heretofore unpublished work of Edward G. Stiles, DO, FAAO. Dr. Stiles has devoted his life to bringing osteopathic principles and practice into the mainstream of healthcare, and for the most part his teachings have been passed down by word of mouth. The authors aspire to share Dr. Stiles' helpful concepts and procedures in the greater arena of OMT, as his hands-on principles are deemed by the authors to be universally applicable in all OMT training programs.

With the perspective described above in mind, this text is written with the following goals:

1. To teach OMT principles in a manner that is easy to understand and apply. The emphasis is on the "why" of the concept or procedure, not simply the "how to do" of the OMT

assessment or application. Although certain procedures are used as examples in this text, the goal is to demonstrate application of the principles.

2. To present examples that a beginning student can readily understand, and under the direction of faculty, apply in their particular training program. This text is not intended to include every specific type of OMT procedure or modality.

3. To serve as an introductory text for OMT education so that instructors may incorporate these concepts and procedures into their broader curriculum as deemed appropriate.

4. To provide text in a structured form so that principles may build on one another. This format is intended to provide a structure for presentation in the educational arena. This text may also serve as an introductory text in both undergraduate and graduate OMT education and training programs.

5. To present the concept of sequencing or "finding the key primary somatic dysfunction" that has been helpful to many students in the early stages of training. This process allows them to figure out where to start in the process of evaluating and providing service to patients and clients. However, in no manner of speaking do the authors intend the concept of sequencing to replace any aspect of OMT education. It is to be considered only as a potentially useful addition to OMT curricula.

This text focuses on a biomechanical rather than a biodynamic model. The authors believe that all professions utilizing OMT in healthcare generally accept the stated Tenets of Osteopathy. In fact, co-author King[1] stated the following, "At a recent research symposium dealing with synergistic goals in manual therapy research, the first author presented the set of osteopathic concepts as they related to research priorities. During the panel discussion, presenters from chiropractic, physical therapy, massage therapy, and body worker professions all said that their respective professions taught similar concepts within their scope of practice."[2]

These Tenets are:

1. The body is a functional unit, and the person represents a dynamic combination of body, mind, and spirit.
2. The body is capable of self-regulation, self-healing, and health maintenance.
3. Structure and function are reciprocally interrelated.
4. Rational treatment is based on the first three principles.

The authors have included sample test questions and case studies illustrative of the integration OMT principles in clinical practice. The test questions are intended as preparation for those who may have to take tests on OMT for course work or for board exam preparation.

The authors are US trained osteopathic physicians whose practices emphasize or are devoted totally to OMT. They are board certified in both the primary care disciplines of Internal Medicine (JB and PJ), Sports Medicine (PJ), Family Medicine (HK) and Neuromusculoskeletal Medicine (HK).

References:

1. King HH, Patterson MM. The concepts of osteopathic medicine: past and present. In King HH, Jänig W, Patterson M (Eds.) *The science and clinical application of manual therapy.* Edinburgh, Churchill Livingstone, 2011:1.
2. Langevin H, Goertz C, King HH**,** Khalsa P, Dryden T, Woodhouse LJ. Synergistic research goals for manual treatments of musculoskeletal and soft tissue disorders. Symposium presented at North American Research Conference on Complementary and Integrative Medicine, Minneapolis, MN, May 12-15, 2009.

Chapter 1: Principles of Manual Medicine/Therapy

Principles Presented in this Chapter:

- Somatic Dysfunction and TART Criteria
- Barrier Model of Restricted Range of Motion

Outline:

- Patient History
  - Red Flags to Performing Manual Medicine
- Physical Examination
  - Palpation and the Biomechanical Structural Exam
- Somatic Dysfunction
  - Barrier Concepts
- Principles of Treatment
  - Direct Treatments
    - High Velocity Low Amplitude
    - Muscle Energy
  - Indirect Treatments
    - Functional Procedures
    - Strain-counterstrain
    - Myofascial Release
- Limitations of Treatment Correlations Between Procedures
- Treatment Reaction
- Dosing Manual Medicine
- Contraindications to Manual Medicine
- Dosing Manual Medicine
- Treatment Goals of Manual Medicine
- Clinical Vignette
- Review Questions

Chapter 1: Principles of Manual Medicine/Therapy

This chapter contains terminology and principles necessary for communication between physicians who practice Osteopathic Manipulative Treatment (OMT). It reviews the components of a history and physical exam to provide the information that will determine if the patient is an appropriate candidate for manual medicine. Included at the end of this chapter is a clinical vignette that represents a real clinical scenario in which a patient's history ultimately guides the decision to perform manual medicine.

Components of a Complete History

- Chief Complaint
- History of Chief Compliant
- Past Medical History
- Past Surgical History
- Medications, Current and Past
- OTC Medications and Supplements
- Allergies to Medication and Food
- Social History
- Family History
- Review of Systems

Patient History

The first patient encounter begins with a complete history and physical exam (H&P). On initial interview, a SOAP note (Subjective, Objective, Assessment Plan) or a focused H&P lack the comprehensiveness needed to provide complete patient management. In addition to the chief complaint (CC) and history of chief complaint (HxCC), a patient history should include the following information: past medical history (PMHx), past surgical history (PSHx), prescription and over the counter (OTC) medications, medication and food allergies, social history, family history (FamHx), and a review of systems (ROS).

Red Flags to Performing Manual Medicine

The HxCC and subjective portion of every SOAP note should be directed towards identifying potentially harmful medical disease known as "red flags."[1] Questions regarding fever, night sweats, weight change, bone or non-mechanical pain and pruritus "cast a net" for potentially identifying cancer, infection, and endocrine, rheumatologic, and connective tissue diseases. History of muscle weakness, atrophy, anesthesia or paresthesia, or a change in bowel or bladder habits, point toward the possibility of neurogenic pathology and help eliminate the possibility of cauda equina syndrome, a medical emergency. History of trauma may define potential tissue injury and prompt a physician to order radiographic studies prior to manual medicine treatment.

Red Flags[18]:

- Low Back Pain (LBP) with Presentation <20 years
- LBP with onset of symptoms >55 years
- Trauma
- History of cancer
- History of osteoporosis or its risk factors
- B-type symptoms including fevers, night sweats, weight change, pruritus
- Non-mechanical or radicular pain
- Cauda Equina Syndrome
- Filum Terminal Syndrome
- Pain out of proportion to exam

A single red flag does not preclude the use of manual medicine, but alerts the clinician to possible underlying medical illness that should be addressed prior to initiating manual treatment. Consider the 50-year-old man who has sub-sternal chest pain, risk factors for coronary artery disease, and complaints of

middle thoracic back pain. A sensible provider would initially assess the patient for immediate and life- threatening causes of the chest pain, such as aortic dissection or acute coronary syndrome, prior to treating the musculoskeletal components of his rib cage and thoracic spine.

Physical Examination

Prior to treatment, a directed musculoskeletal exam should evaluate cranial nerves, cerebellar function, strength, deep tendon reflexes, sensation, static standing evaluation (Figure 1), and gait. This exam should be done in addition to a "biomechanical structural exam" that is focused on the areas to treat with manual medicine. The remainder of a physical exam may include head, ears, eyes, nose, throat (HEENT), neck, chest, heart, lungs, abdomen, extremities, and genitourinary when indicated.

Figure 1

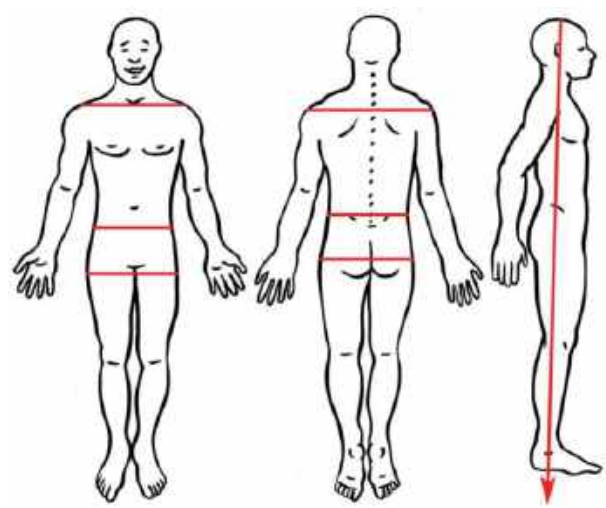


Figure 1: Static Standing Evaluation
Used to assess symmetry of posture. Ideally shoulders, iliac crest, greater trochanter should have symmetrical positions. Additionally, arches of feet should be symmetrical. On lateral examination, an imaginary plumb-line should begin at the ear lobe and move inferiorly through the acromioclavicular joint, center of gravity at the 3rd lumbar segment to the anterior portion of the lateral malleolus.

Components of a Complete Physical Exam

- HEENT
- Neck
- Chest
- Heart
- Lungs
- Abdomen
- Genitourinary
- Extremities
- Cranial Nerves
- Cerebellum
- Muscle Strength Testing
- Skin Sensation Testing
- Deep Tendon Reflex
- Static Standing Evaluation
- Gait Analysis
- Biomechanical Structural Exam

Patients presenting with seemingly benign musculoskeletal complaints may have underlying visceral or ischemic disease that has not been diagnosed and was not revealed during patient history. If musculoskeletal pathology is unresponsive to manual medicine, knowledge of viscerosomatic and somatovisceral reflexes can become an important diagnostic tool enabling clinicians to recognize medical problems that are masquerading as musculoskeletal problems. The neurophysiology chapter delineates autonomic nervous system interactions of viscerosomatic and somatovisceral reflexes. In American osteopathic medicine, autonomic nervous system interactions are considered highly relevant to the practice of Osteopathic Manipulative Medicine (OMM) and comprise a significant portion of the questions on the Comprehensive Osteopathic Medical License Examination (COMLEX) at all levels.

We advocate a thorough history and physical examination prior to the initiation of OMT. Our first job as medical care providers is to avoid harm to patients by excluding red flags on history and physical examination. A good methodology to consider is to first rule-out any life-threatening condition, then rule-out common maladies and lastly look for uncommon or "zebra" conditions. A more complete method for medical triage is provided in the lumbar spine chapter.

Palpation and the Biomechanical Structural Exam

A directed biomechanical structural exam should be included in an examination. Prior to beginning this examination, a student should understand the difference between their dominant and non-dominant eye. A dominant, or "shooting" eye, is the principle eye used to keep an object in focus when both eyes are placed on an object. Use of the dominant eye is very important since depth and height perspectives may become distorted during an examination. For instance, if the dominant eye is used to access the innominate crest height on the left and the non-dominant eye the right, a physician may errantly deceive himself or herself into diagnosing crest height asymmetry.

To determine eye dominance, fix both eyes on an object approximately two meters away through a keyhole site (such as a paper towel tube). Close one eye at a time to determine which eye maintains the object inside the keyhole (Figure 2a): this is your dominant eye.

Figure 2a & 2b

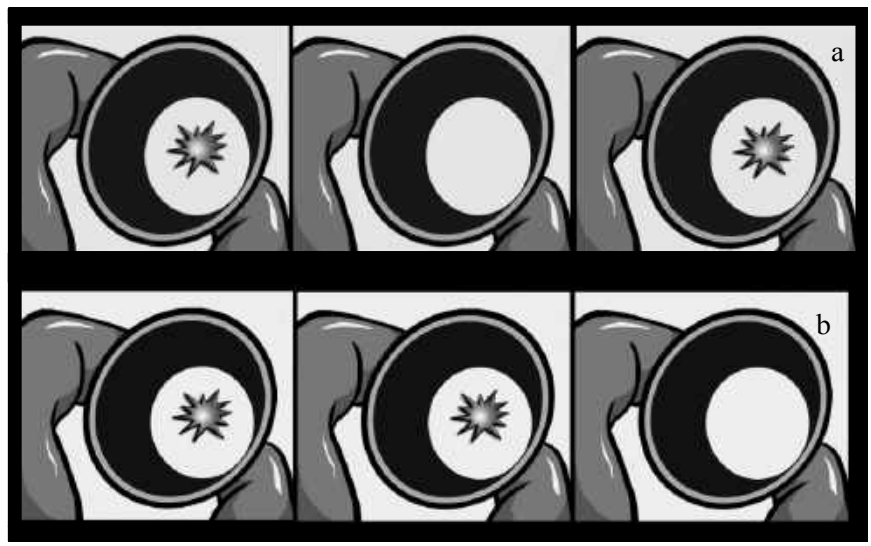


Figure 2a (upper row): Dominant Right Eye

The object is initially seen using both eyes (left picture). The dominant right eye, in this instance, will maintain a distant object in a keyhole view (right picture), whereas the non-dominant left eye will not (center picture)

Figure 2b (lower row): Dominant Left Eye

The object is initially seen using both eyes (left picture). The dominant left eye, in this instance, will maintain a distant object in a keyhole view (middle picture), whereas the non-dominant right eye will not (right picture)

The best method for checking symmetry of structures is to place the dominant eye over the center of the two structures for comparison. When a patient is lying on a table, a right eye dominant practitioner should stand over the patient, as depicted in Figure 2c, to keep their right eye over the center of the patient. A left eye dominant practitioner should stand over the patient as depicted in Figure 2d to keep their left eye over the center of the patient. The practitioner should then close their eyes and palpate using the palms as directed below to see if a proprioceptive difference exists.

Figure 2c & 2d

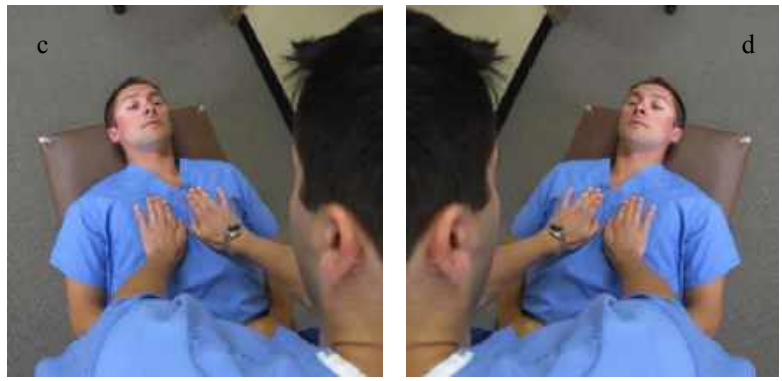


Figure 2c and 2d: Figure 2c depicts a right eye dominant provider standing over a patient to keep his right eye centered over the patient. Note how the provider stands to the left of the table to easily keep the right eye centered over the patient. Likewise, Figure 2d depicts a left eye dominant provider standing on the right of the table.

Osteopathic Principles and Practice (OPP) educators teach structural diagnosis beginning with “layer-by-layer" palpation. Most commonly, layer-by-layer is taught by resting a hand directly on the skin overlying a structure and gradually increasing pressure. Practitioners use their hands for the following diagnostic purpose:

- Palm for determining gross structural symmetry
- Finger pads for fine discrimination
- Finger joints for vibratory sense
- Extensor surface of the hand for temperature sense
- Tips of thumbs for depth perception

On static exam, students sense superficial and deep fascia as well as muscles, tendons, ligaments and bone, etc. Since muscles and tendons move readily under the skin and are therefore easier to locate, patient movements during testing also help develop palpatory skill. During biomechanical structural examination, active and passive range of motion (ROM) testing allows the practitioner to challenge the limits of flexion-extension, side bending, and rotation for comparison of one side to the other. Active ROM describes patient-initiated movement. Passive ROM describes examiner-initiated movement (without patient effort). For any joint or tissue, motion test both right and left body segments to compare. John Mennell, MD described a type

of passive ROM testing called "joint play."[1] Mennell noted that synovial joints, specifically spinal facet joints, have a small amount of pain-free unrestricted movement. Joint play is described further in the thoracic spine chapter.

Static, active, and passive ROM examinations should be performed routinely. Examine normal tissue as if practicing to use a stethoscope to listen to normal heart sound and "tissue memory" will develop. With repeated examination, students eventually discern pathological versus normal tissue.

Somatic Dysfunction

The biomechanical structural exam is directed toward identifying somatic dysfunction. The osteopathic profession defines somatic dysfunction as "impaired" or altered function of related components of the body framework system: skeletal, arthroidal, and myofascial structures, and their related vascular, lymphatic and neuronal elements."[3(pp63)] Historically, somatic dysfunction was first named "osteopathic lesion," and has also been termed "osteopathic spinal lesion," "non-allopathic lesion," "intervertebral lesion," "intervertebral dysfunction," or "segmental dysfunction." Chiropractors classically referred to the same phenomenon as "subluxation" when referenced to the spine, but now share somatic dysfunction as a common terminology. To simplify the current definition, somatic dysfunction includes any pathologic process or musculoskeletal derangement that will respond favorably to manual medicine. The current definition does not yet include visceral structures or account for possible future discoveries in fascial biomechanics.

ICD-10 Codes for Somatic Dysfunction

M99.00 – Head region, occipitocervical
M99.01 – cervical region, cervicothoracic
M99.02 – thoracic region, thoracolumbar
M99.03 – lumbar region, lumbosacral
M99.04 – sacral region, sacroiliac, sacrococcygeal
M99.05 – pelvis, hip, pubic
M99.06 – lower extremity
M99.07 – upper extremity, sternoclavicular, acromioclavicular
M99.08 – rib cage, costochondral, costovertebral, sternochondral
M99.09 – abdomen, specified NEC

Somatic dysfunction has characteristic qualities on physical exam that have prompted the traditional mnemonic TART: T = tissue texture abnormality, A= asymmetry, R = restriction of motion, T = tenderness to palpation.[3(pp69)] We point out that "tissue texture abnormality" includes fluid such as edema, fibrosis, and temperature. An important distinction worth describing is the tenderness that a practitioner elicits on exam versus patient reported pain: objective tenderness is a clinical exam finding, whereas subjective pain is part of a patient's history.

Characteristics of Somatic Dysfunction:

T – Tissue Texture Abnormality
A – Asymmetry
R – Restriction of Motion
T – Tenderness

If two structures are named in somatic dysfunction, it is assumed that the one named first is pathologic in relation to the second. For example, a sacroiliac dysfunction is a dysfunction of the sacrum that causes changes in the innominate.

Barrier Concepts

Tissue texture abnormality, asymmetry and tenderness found in somatic dysfunction were addressed in the previous section. To conceptualize

Barriers of Joint Mechanics found during ROM Testing

- Physiological Barrier: limit of normal active ROM; represents end of muscle fiber stretching
- Elastic Barrier: found during passive ROM, located between anatomical and physiological barriers; represents barrier created by joint capsule and supporting ligaments
- Anatomical Barrier: limit of normal passive ROM; represents bone contacting bone
- Pathologic Barrier: hypo/hyper-mobility of ROM

Figure 3: Normal ROM
Note the differences in range of motion (ROM) achieved by active and passive ROM. When applied to the elbow, the right side of the diagram represents extension and the left side represents flexion (or vice versa). Additionally, when applied to spinal mechanics (Chapter 2) the left and right sides can represent sidebending right or left, rotation right or left, or flexion - extension.

N = Neutral or middle

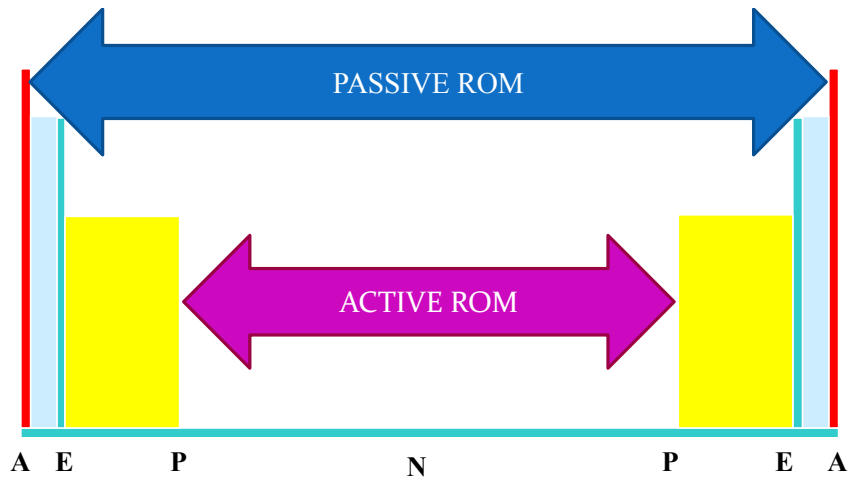
A= Anatomical barrier

E = Elastic barrier

P = Physiological barrier

restriction of motion with respect to somatic dysfunction, a barrier model developed by Paul Kimberly, DO, FAAO[3(pp10)] provides a basis to understanding diagnosis and ultimately treatment through use of motion testing. A non-pathological elbow in active ROM can extend to a certain angle; this end extension is called a physiological barrier and represents a muscular and fascial endpoint to the patient's active ROM. A physician examining that same elbow imparts passive ROM testing by extension through the physiological barrier until met by a "springy" resistance. This elastic barrier corresponds to ligament and capsule structures of the elbow. The elastic barrier has a "spring-like" sensation that with further passive extension locks as the olecranon process contacts the olecranon fossa with a bone-on-bone feel; this bony lock is named the anatomical barrier. Movement beyond the anatomical barrier results in pathological tissue trauma, fracture or dislocation. These three barriers set up a foundation for visualizing joint mechanics in normal ROM testing and barrier model as represented in Figure 3.

Figure 3



When a student begins examination, there can be uncertainty about what barrier is engaged. Practice is required to differentiate these barriers since patients' tissues vary based on age and habitus.

Now, imagine testing elbow extension and finding hypo-mobility during active and passive ROM testing of the right compared to the left. This hypo-mobility represents somatic dysfunction as represented in Figure 4 by the light blue box.

Figure 4

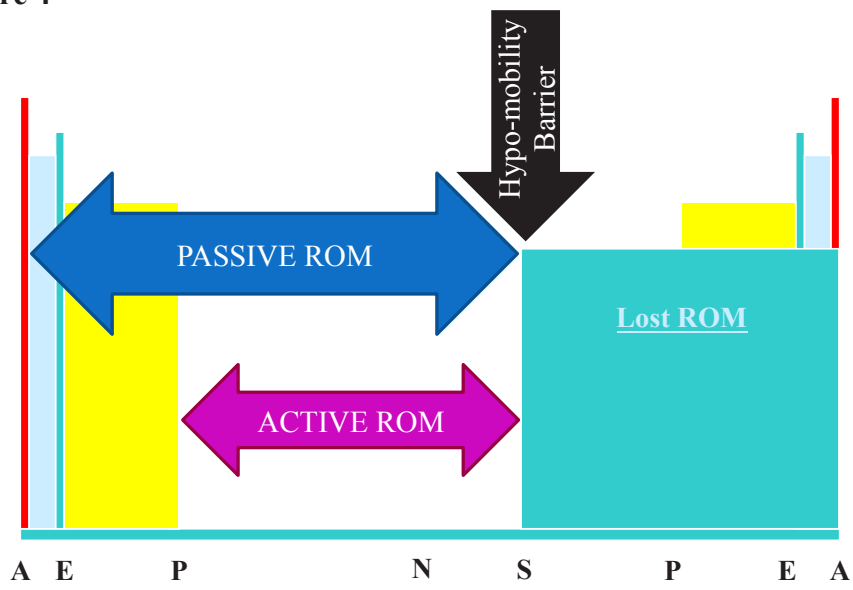


Figure 4: Hypo-mobile Somatic Dysfunction
A= Anatomical barrier
E = Elastic barrier
P = Physiological barrier
N = Neutral or middle
S = Somatic Dysfunction of hypo-mobility

When applied to an elbow with restricted extension, the right side of the diagram represents extension and the left side represents flexion. Note the decreased ROM in both active and passive ROM.

Hyper-mobility of a joint also represents somatic dysfunction and is represented in Figure 5. Examples of hyper-mobile somatic dysfunction would include grossly dislocated joints and hyper-mobile joints. Practitioners of manual medicine typically do not treat hyper-mobile joints with manual medicine as these areas normally represent compensation for hypo-mobile areas or joint instability that requires surgery or other interventions such as bracing or prolotherapy.

Figure 5

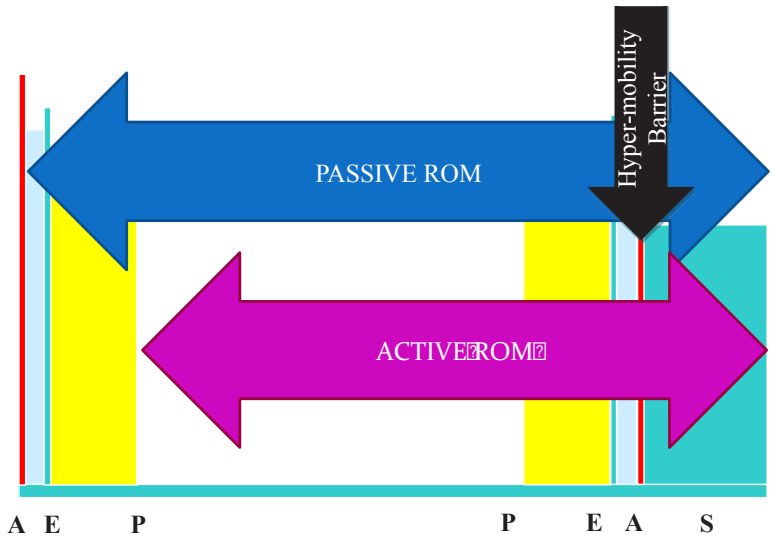


Figure 5: Hyper-mobile Somatic Dysfunction
A= Anatomical barrier
E = Elastic barrier
P = Physiological barrier
N = Neutral or middle
S = Somatic Dysfunction of hyper-mobility

When applied to an elbow with hyperextension, the right side of the diagram represents hyperextension and the left side represents normal flexion. Note the increased ROM in both active and passive extension. This would likely represent dislocation.

Principles of Treatment

Principles Treatment:
- Diagnose
- Treat
- Recheck Initial Diagnosis

As previously mentioned, hyper-mobile somatic dysfunction is typically not treated with OMT, with the exception of joint dislocation (in a relocation procedure). The remainder of this text focuses on hypo-mobile somatic dysfunction.

With the barrier model in mind, imagine treating a patient with elbow hypo-mobility using OMT. If the physician extends the elbow to the pathological "motion barrier" of somatic dysfunction and begins treatment at this barrier, they would be using a direct treatment procedure. If the physician chooses to begin treatment away from the pathological barrier in a position of ease, or by exaggerating the positional diagnosis, this would be an indirect treatment procedure (Figure 6). Using the same example, the elbow would be flexed at a position of ease during indirect treatment.

Figure 6

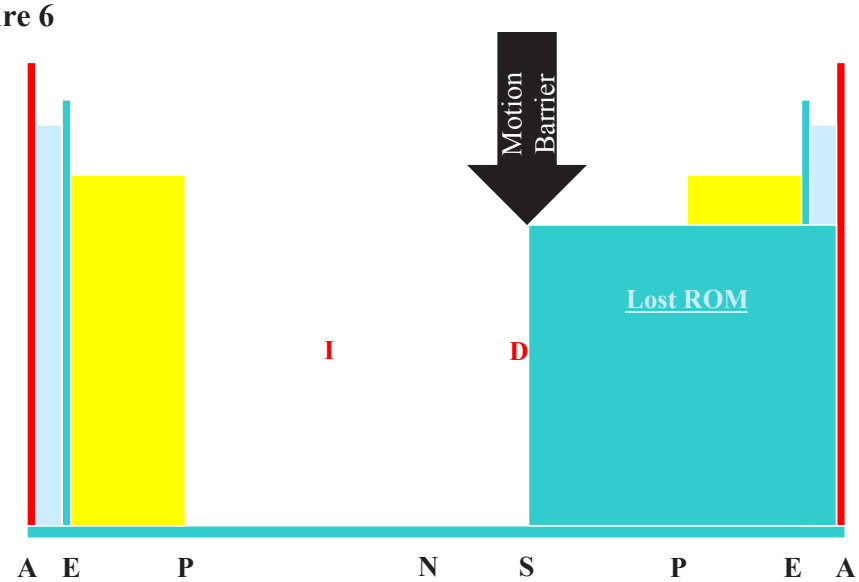


Figure 6: Comparison of Direct and Indirect Treatment Positions
A= Anatomical barrier
E = Elastic barrier
P = Physiological barrier
N = Neutral or middle
S = Somatic dysfunction of hypo-mobility

D = Conceptual starting treatment position using direct treatment.
I = Conceptual starting treatment position using indirect treatment at a point of ease or equal tissue tension.

Osteopathic nomenclature as established by the Educational Council on Osteopathic Principles (ECOP) defines direct and indirect treatments as follows:

> Direct method (D/DIR): "An osteopathic treatment in which the restrictive barrier is engaged and a final activating force is applied to correct somatic dysfunction."[3(pp35)]
>
> Indirect method (I/IND): "A manipulative treatment in which the restrictive barrier is disengaged and the dysfunctional body part is moved away from the restrictive barrier."[3(pp36)]

While the ECOP definitions are currently the best description of direct versus indirect treatment, each patient presents different somatic dysfunction patterns. Thus, certain OMT modalities, such as the Still Technique that utilizes both indirect and direct modalities, may address treatment differently. Common procedures of direct and indirect treatments are listed in the blue bar.

Direct Treatment

Since they are based on spinal mechanics, this book utilizes two direct procedures as representative examples of OMT: high velocity-low amplitude (HVLA) and muscle energy (ME). These two procedures are not the only direct treatment modalities, and some procedures can be applied directly or indirectly or in combination. For example, the "Still Technique" rediscovered and developed by Richard Van Buskirk, DO, PhD, FAAO, exclusively begins in an indirect position and then finishes with a direct-action component that moves through the restriction barrier at the end.[2(pp850)] In this book we discuss direct treatment using HVLA and ME; both modalities employ direct engagement of the barrier, a lever, a fulcrum, and an activating force as a principle of treatment.

HVLA

HVLA is commonly utilized in both osteopathic and chiropractic practice and to some degree in physical therapy. Three components are central to direct HVLA treatments: a lever, a fulcrum, and an impulse or thrust through the barrier. When using a direct thrusting technique such as HVLA, a "lock" or "end feel" is engaged at the barrier of the somatic dysfunction. This lock/end feel, while not the physiological, elastic, or anatomical barrier, can be described collectively as reaching the maximum sidebending, flexion/extension and rotational limits. This three-dimensional limit in ROM is also referred to as a closed-packed position, which in turn is interpreted as maximal tightness of the articular capsule and ligaments.

Often a joint "pop" or "crack" is noted when moving through the direct barrier. Although psychologically gratifying to the practitioner and patient, this pop does not imply success with the treatment, nor does its absence denote failure. The pop is actually cavitation that occurs in the joint capsule. Due to pressure changes in the closed pressure system of a joint (i.e., a vacuum), it is hypothesized that synovial fluid rapidly turns from a liquid to a gas state generating an audible "click". The gas state usually lasts no more than twenty minutes. It is no coincidence that many patients note relief from a HVLA pop for twenty to thirty minutes, only to have their pain return; ultimately this indicates suboptimal MM/T treatment.

Common Manual Medicine Procedural Treatment Techniques

Direct Treatments
- Articulatory (ART)
- High Velocity Low Amplitude (HVLA)
- Muscle Energy (ME)
- Myofascial Release (MFR)
- Still Technique

Indirect Treatments
- Balanced Ligamentous Tension (BLT)
- Facilitated Positional Release (FPR)
- Functional Method
- Function Procedure
- Ligamentous Articular Strain (LAS)
- Myofascial Release (MFR)
- Progressive Inhibition of Neuromuscular structures (PINS)
- Strain Counterstrain (S/CS)
- Still Technique

Principles of Direct HVLA
- Lever
- Fulcrum
- Impulse

Many allopathic practitioners hesitate to prescribe HVLA techniques since they have been implicated in patient injuries (see contraindications section below). However, Triano and Schultz measured the forces produced on the spine during HVLA thrust and concluded they were no more than those involved in the lifting and twisting movements commonly encountered by air-line baggage carriers.[4] With respect to HVLA impulse, Perrin T. Wilson, DO, claimed that force used during treatment was inversely proportional to the operator's understanding of the biomechanics involved, and we agree.

Muscle Energy

Principles of direct ME
- Lever
- Fulcrum
- Isometric Contraction

Muscle energy techniques, originally developed by Fred Mitchell Sr., DO, FAAO, are very similar to HVLA; however, instead of using an impulse, muscle effort is generated by the patient and resisted by the practitioner creating an isometric contraction. The isometric contraction is usually held for three to five seconds. The patient relaxes and is moved through one restrictive barrier to the next restrictive barrier. This is the basic concept of the law of reciprocal inhibition, where the dysfunctional hypertonic muscle is induced to relax after contraction of its paired (antagonist) muscle. This process is repeated three to five times until full ROM is restored. When using muscle energy techniques, Edward Stiles, DO, FAAO, engages the direct restrictive barrier, and once in this position, moves to what he calls the feather edge by slightly releasing the direct barrier (closed-packed position) by a fraction. Using the feather edge of a barrier reduces patient pain during an isometric contraction and allows for position maintenance at the direct barrier; it is not, however, the close-packed position ideally suited for HVLA.

Indirect Treatment

When compared with direct treatment, an indirect technique begins away from the somatic dysfunction barrier. All indirect procedures begin at a position of maximal relaxation, ideally at a point where tissue is balanced in three-dimensional space (Figure 6). In comparison to the closed-packed position used in HVLA, musculoskeletal practitioners have adopted the term loose-packed position or position of ease (POE) to denote a position that is ideally suited for indirect treatment. Practitioners have also named this position the "balance point"[3(pp9), 13(pp812)] in Balanced Ligamentous Tension and the "mobile point"[3(pp30), 14(pp31)] in Strain-counterstrain (S/CS).

Placing somatic dysfunction at the POE is not always simple, and often the operator will need to carefully move through the cardinal planes of motion one at a time. Depending on the location of the somatic dysfunction, the practitioner may need to flex or extend, abduct or adduct, internally or externally rotate, side-bend, and add compression or distraction until balance is achieved. Often the tissue will soften and balance with the slightest change

in position. Commonly, the patient will have significant reduction in objective pain when the POE is engaged. A subtle difference in this treatment position may exist if the practitioner is focused on treating fascial structures versus treating muscular or bony structures. Three of the most commonly used indirect treatments include: strain-counterstrain (S/CS), functional procedures and myofascial release (MFR).

Steps to Treatment:
1. Diagnose
2. Treat
3. Recheck original diagnosis

Functional Procedures

"Functional Methods" were originally described by William L. Johnston, DO, FAAO,[15(pp27)] after collaboration with a New England study group during the 1950's. The group used palpatory diagnosis to describe the POE in what Johnston coined "six-degrees of freedom."[15(pp162), 16(pp73-74)] Six degrees of freedom describes the passive rotary tests flexion/extension, sidebending, and axial rotation and the translatory tests left/right, anterior/posterior and cephalad/caudad, that Johnston used to determine the POE. Once in the POE, Johnston used respiratory assistance to initiate treatment that would resolve the somatic dysfunction and carry the patient back to neutral.

Separately while a medical student in 1962, Edward Stiles, DO, FAAO, learned an indirect procedure he termed "Functional" from the grandson of A.T. Still, MD, George Laughlin, DO.[17] Laughlin learned the procedure from William Garner Sutherland, DO, who claimed to have learned it from Still himself. In 1989 Stiles described Functional Treatments using spinal mechanics nomenclature developed by Fred Mitchell Sr., DO, FAAO, (which is further described in the Thoracic Spine chapter). Like Johnston, Stiles begins at the POE, but then introduces a compressive or distractive force to initiate treatment which carries the patient back to neutral.

When using Functional Treatments, the initial POE will move or change; the operator maintains the balanced tension in three-dimensional space as it changes. Because of the changing POE, Stiles refers to his initial treatment position as the dynamic balance point (DBP). As it moves and changes, there is no predictable direction of movement as the tissues moves through the cardinal planes. Many practitioners therefore refer to this patient-practitioner interaction as "the dance" where the practitioner is following the tissue's (patient's) lead. The challenge of performing Functional Treatments is in observing and following the tissue as it changes and not anticipating where the tissue should move during the treatment.

Dynamic Balance Point: ideal starting position of Stiles' "Functional" treatments where the practitioner balances structures in three-dimensional balanced tension.

During Functional Treatment, the DBP may cease movement while maintained in a balanced compression; classically this is described as a still-

Still-Point: a temporary interruption of the primary respiratory mechanism as may occur during treatment with manual medicine.

point.[3(pp68)] A still-point does not necessarily indicate the resolution of treatment; it may persist for seconds or minutes. A single treatment may involve one or multiple still-points. As practitioner awareness grows, changes become apparent in the tissue surrounding the somatic dysfunction while in a still-point. The patient will commonly have involuntary myoclonic contractions or pulsations while in a still-point. Some practitioners feel the primary respiratory mechanism (see glossary or cranial chapter) move through the tissue. Often the patient will notice tissue change adjacent or distal to the still-point.

Clinically differentiating the completion of a Functional Treatment requires experience. The direction Stiles gave his beginning students was to "challenge the tissues" by adding a slight compression or distraction while in a still-point. With compression or distraction three things may occur: the tissues will continue to move, remain in the still-point, or relax. If they relax, the treatment is done; if they continue to move or remain still, the treatment is not complete. Most functional treatments resolve within 30 seconds to two minutes.

We have chosen to include Functional Treatments in this text using the spinal mechanics naming system developed at Michigan State University. Stiles successfully used this teaching method from 1997-2006 while at Pikeville College School of Osteopathic Medicine because it mirrored the naming used in ME and HVLA and served as a bridge to S/CS. To pay homage to all physicians who developed this indirect treatment we simply call this treatment method "Functional Procedures."

Of historic note, Richard Van Buskirk, DO, FAAO describes the Still-Laughlin Technique[2(pp850)] and credits Dr. Stiles as the modern purveyor of the treatment. As students of Dr. Stiles while at Pikeville College School of Osteopathic Medicine (now called KYCOM) from 1998-2003, authors PJ and JB note that Stiles' Functional contained only indirect treatment components, and he never named it "Still-Laughlin Technique," a name Van Buskirk himself coined. Again, Van Buskirk's "Still Technique" begins at POE and then moves through the restrictive barrier of somatic dysfunction ultimately containing indirect and then direct components.

Strain-Counterstrain

"Strain-counterstrain" is a system of indirect treatment developed by Lawrence Jones, DO, FAAO. S/CS is founded on locating tender-points (TP) and placing them at a point of maximal comfort, which is usually at a POE Jones called the "mobile point." Ideally, reduction of tenderness should be a

minimum of 70%.[5(pp8)] Classically, S/CS positions are held for 90 seconds for all but ribcage TP are held for 120 seconds.[5(pp8)] If somatic dysfunction resolves prior to this time period, the treatment is completed. Sometimes, pulsations in the tissue occur that Jones termed "therapeutic pulse." In this instance, the treatment is continued at least until the therapeutic pulse is completed. After treatment, the patient remains relaxed and the practitioner moves the patient slowly out of the treatment position. A practitioner rechecks tenderness to confirm treatment success; a reduction in pretreatment tenderness is judged successful.

TP are characterized by several criteria: they are less than 1cm, edematous, non-radiating, tender to palpation only when palpated, and present in predictable locations such as ligaments, tendons, and muscle. Their presence results in restricted ROM and muscle weakness/inhibition. Since TP exhibit tenderness, asymmetry, restricted ROM, and tissue texture change, they are considered somatic dysfunction. Many practitioners believe that TP are identical to trigger points, which were first described by Janet Travell, MD; however, these seemingly similar phenomena have distinct differences.

Trigger points are described as either active or latent depending on their chronicity. Active trigger points are characterized by the following: focal, discrete, hyperirritable spots located in a taut band of skeletal muscle which produce referred pain and tenderness.[6] Classically, they cause pain with and without palpation. They exhibit a local twitch response that is a contraction of the muscle with resultant dimpling of the skin as the muscle fibers spasm in response to stimuli.[7] The twitch response is usually elicited by needle insertion into the trigger point or by perpendicular "snapping" palpation in the direction of muscle fibers.

In contrast, latent trigger points do not cause tenderness without palpation, but do restrict ROM and cause muscle weakness. Due to similarity between latent trigger points and TP, many believe they are the same.

Myofascial Release

Indirect myofascial release (MFR) has similar components to S/CS and Functional Procedures. Ideally, a patient's muscle, fascial, and ligamentous structures are placed in a POE. The practitioner either maintains this position or adds compressive forces and follows the tissues in three-dimensional space as they change. As in Functional Procedures, tissues may enter still-points as they unwind to neutral.

MFR is an important treatment procedure in manual medicine that we readily employ in practice. We acknowledge that fascial treatment shares a common thread of all practitioners of manual medicine: Physical Therapy, Chiropractic, Massage/Bodywork, and Osteopathic Practitioners/Physicians. We exclude it from the diagnosis and treatment for the remainder of this text

Characteristics of Tender Points:

- Less than 1cm
- Edematous
- Non-tender when not palpated
- Tender when palpated
- Non-radiating
- Do not refer pain
- Located in muscle insertion zones and ligaments
- May result in muscle inhibition and restricted ROM

Characteristics of Active Trigger Points:

- Focal
- Tender with and without palpation
- Refer pain and tenderness
- Local twitch response
- Located in taut band of skeletal muscle

because it does not easily follow the biomechanical model and spinal mechanics nomenclature. Please refer to research from the International Fascia Research Congress at www.fasciacongress.org for a more in-depth view of MFR.

Limitations of Treatment Correlations Between Procedures

As typically presented in osteopathic training, throughout this text we have used two direct and two indirect procedures as representative OMT procedures. As the reader will observe, there is good reliability in correlation of the aforementioned procedures in the cervical, thoracic, lumbar spine, pelvis and ribs, but less consistency in other areas. In these instances, we present two, instead of four, treatment options. With regard to S/CS, tender points occur in muscle, tendon and ligament and thus may not correlate with spinal biomechanics nomenclature. Some believe that the work of Jones' anterior tender points in the thoracic, lumbar, and pelvis are not actual anatomical representations of the somatic dysfunction, but rather they are reflex points used to identify and treat the dysfunction. This is especially apparent as we approach the extremities.

The nomenclature for specific somatic dysfunctions becomes increasingly inconsistent and difficult to correlate between the different procedures. This is where we adhere to the basic principles of treatment presented in this book. For example, direct procedures always begin at the restrictive barrier and involve a lever, a fulcrum, and a force, while indirect procedures require initial positioning away from the barrier in a position of ease. Ultimately, the goal of manual medicine is restoration of normal range of motion in areas of hypomobility or hypertonic muscles.

Treatment Reaction

Commonly after a treatment, the patient will experience pain or headache for up to 24-72 hours. This is termed a treatment reaction. Usually, pain is manifested as a tolerable, local, or diffuse muscle ache or pain similar to beginning a new workout program. Post-treatment pain likely results from lactic acid release by relaxation of tonic muscles and postural adjustment as inhibited muscles reactivate. Resulting clinical symptoms may include increased thirst, and if not adequately rehydrated, a sensation of a "hang-over" or a spinal headache also may follow.

Sometimes patients experience a severe treatment reaction and report feeling like they have been "run over by a truck" following treatment. Other patients feel as if they have influenza. Although treatment reactions this severe are rare, they do occur. Patients should be informed of possible treatment reactions. If a patient has no contraindication, a single dose of ibuprofen or naproxen and oral rehydration will often prevent post-treatment pain.

Dosing Manual Medicine

The challenge of manual medicine is identifying key somatic dysfunctions (discussed in the sequencing chapter). These "key lesions," also known as primary somatic dysfunction, cause compensatory changes in adjacent and distant structures or secondary somatic dysfunction. Compensatory structures sometimes require 24-72 hours to return to homeostasis; this is the same time period in which treatment reactions occur. From the osteopathic perspective, ideally, dosing of manual medicine/treatment (MM/T) should occur in intervals of four to seven days. Chiropractic, Massage, and Physical Therapy dosing is based on the preference of the individual provider. A patient may require five treatment sessions for up to four weeks to perceive results from MM/T. However usually, one or two treatments produce a substantial subjective reduction in pain. Success should not be measured exclusively by absence of pain, since often a patient will have improvement in ROM and function prior to pain relief.

Contraindications to Manual Medicine

While it is generally accepted that there are few absolute contraindications to MM/T, there are certain circumstances (in addition to "red flags") when providers should consider deferring treatment or withholding HVLA thrusting techniques. These include patients with rheumatoid arthritis, Trisomy 21, carotid stenosis, spondylolisthesis, intracranial bleed, and bony fusion.

Patients with rheumatoid arthritis and Trisomy 21 can have cruciform ligament laxity and therefore an increased incidence of dens subluxation into the spinal cord during cervical flexion of the first cervical vertebrae (C1) in relation to the second (C2). Unilateral carotid stenosis can cause stroke when a singly patent carotid artery is narrowed by cervical sidebending or rotation, especially during cervical extension. A grade III or IV spondylolisthesis is at risk of further subluxation and ultimately spinal cord compression.

Relative Contraindications to Manual Medicine:

- Rheumatoid Arthritis
- Trisomy 21
- Unilateral carotid stenosis
- Grade III and IV spondylolisthesis
- Acute stroke
- Intracranial bleed
- Bony fusion

Care should be taken to avoid maneuvers that increase cervical or lumbar lordosis when spinal fracture is suspected or known. Once fusion of pelvic or spinal structures has occurred through therapeutic surgical fusion or during pathological bony fusion, as in ankylosing spondylitis, thrusting maneuvers may cause pathologic fracture. Finally, acute intracranial bleed due to subdural, subarachnoid or epidural bleeding are at risk of expanding with treatment directed at the head. Much discussion exists about whether or not treatment should be performed on patients with known invasive cancer. The fear is that manual medicine will allow direct, lymphatic or hematogenous spread of cancer. It is generally considered that treatment to the area immediately surrounding the cancer is contraindicated;[8(pp428)] however areas without known or suspected cancer may be considered for manual medicine.

Serious adverse events from manipulation include: intervertebral disk herniation, cauda equina syndrome, and vertebrobasilar accidents. Estimates of severe complications are 1 per 400,000 to 1 per 2 million manipulations; this number includes practitioners from Chiropractic, Massage/Rolfing, and Osteopathy, etc. Typically, serious events occur with HVLA thrusting techniques or during treatments that utilize cervical extension in superior cervical segments (C0-C3).[9, 10] By comparison, over-the-counter use of NSAIDs results in approximately 107,000 hospitalizations for gastrointestinal complications and 16,500 deaths in arthritic patients annually. Compared to non-NSAID users, these numbers translate to 1 GI-related death in 500 patients per year with daily NSAID use.[11] The multiple treatment modalities demonstrated in this text allow the practitioner safe and efficacious alternatives to HVLA.

Treatment Goals of Manual Medicine

Manual medicine is one component of total health care and is most effective in the long term when combined with diet, exercise and lifestyle modification. As practitioners of manual medicine, we can direct patients toward their ultimate goal- restoration of health.

One goal of manual medicine is to restore ROM lost by somatic dysfunction and in doing so restore function in postural balance.[12] Patients will often state that the purpose in manual medicine is to put one structure “back into place” in relation to another. Studies have demonstrated that HVLA impulses produce vertebral motions estimated to be 0.1 cm and 1.8 degrees,[5] indicating that if structures are misaligned, they are so minimally. A better analogy to consider is a door with rusty hinges – the door is not knocked off its hinges, it simply will not fully open or close.

Stiles further describes the objective of manual medicine with the following equation:

Host + Disease = Illness

Using this equation, consider a patient with bacterial pneumonia. The bacteria represent the "disease" component of the equation and the patient the "host" component. Somatic dysfunction, if present, is a component limiting the health potential of the host. Manual medicine is directed at the host's somatic dysfunction, especially if restriction in inhalation, exhalation, or autonomic allostasis may have predisposed the patient to bacterial pneumonia. The primary goal is to restore motion to the ribcage, diaphragm, and thorax, and in doing so restore normal lymphatic and blood flow and reduce atelectasis. To treat the disease portion of the equation, antibiotics, oxygen and other appropriate medical treatments should be considered for complete patient care. Treating the "host" and the "disease" truly is holistic care and should be the goal of any practitioner.

References:

1. Mennell, John McMillan. *Joint Pain: Diagnosis and Treatment Using Manipulative Techniques*. Churchill, 1964.
2. Chila, Anthony G. *Foundations of Osteopathic Medicine.* Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2011.
3. Giusti, Rebecca, DO. *Glossary of Osteopathic Terminology*. Third ed. Chevy Chase, MD: American Association of Colleges of Osteopathic Medicine, 2017.
4. Kimberly, Paul E., and Steven L. Funk. *Outline of Osteopathic Manipulative Procedures: The Kimberly Manual Memorial Edition.* Marceline Mo.: Wadsworth Publishing., 2009 (First published 1980).
5. Triano, John, and Albert B. Schultz. "Loads Transmitted During Lumbosacral Spinal Manipulative Therapy." *Spine* 22, no. 17 (09 1997): 1955-964.
6. Myers, Harmon L., et al. *Clinical Application of Counter-strain*. Tucson, AZ: Osteopathic, a Division of Tucson Osteopathic Medical Foundation, 2012. Print.
7. Travell Janet G., and David G. Simons. *Myofascial Pain and Dysfunction: The Trigger Point Manual.* 1992.
8. Alvarez, David J., and Pamela G. Rockwell. "Trigger points: Diagnosis and Management." *American Family Physician,* no. 65 (04 2002): 653-61.
9. Ward, Robert C. *Foundations for Osteopathic Medicine*. Philadelphia: Lippincott Williams & Wilkins 1998.
10. "Spinal manipulation in the Treatment of Musculoskeletal Pain." Accessed July 12, 2016. http://www.uptodate.com/contents/spinal-manipulation-in-the-treatment-of-musculoskeletal-pain.
11. Peura, D.A. "Gastrointestinal Safety and Tolerability of Nonselective Nonsteroidal Anti-inflammatory Agents and Cyclooxygenase-2-selective Inhibitors. *Cleveland Clinic Journal of Medicine* 69, no. Suppl_1 (04, 2002). doi:10.3949/ccjm.69.suppl_1.si31.
12. Greenman, Philip E. *Principles of Manual Medicine.* Philadelphia: Williams and Wilkins, 2003.
13. Chila, Anthony G. *Foundations of Osteopathic Medicine.* Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2011.
14. Jones, Lawrence H., et al. *Strain-counterstrain*. Boise, ID: Jones Strain-Counterstrain, 1995.
15. Beal, Myron C., ed. Scientific Contributions of William L. Johnston, D.O., F.A.A.O. Indianapolis, IN: American Academy of Osteopathy, 1998.
16. Johnston, William L., Harry D. Friedman, and David C. Eland. Functional Methods: A Manual for Palpatory Skill Development in Osteopathic Examination and Manipulation of Motor Function. Indianapolis, IN: American Academy of Osteopathy, 2005.
17. "Indirect Techniques." YouTube. December 14, 2014. Accessed October 15, 2018. https://www.youtube.com/watch?v=d3ix4HqI63c. Edward Stiles, DO, FAAO lecture.
18. Waddell, Gordon. *The Back Pain Revolution*. Edinburg: Churchill Livingstone, 1998.

Clinical Vignette:

Patient Name: BL
May 4, 2013

CC: consult for low back pain (LBP)

HxCC: Patient is a 70yr old black female referred by her gynecologist for LBP. She states she suffered no trauma but has had some progressive LBP for over 6 months; it is 4/10 in intensity, radiates to her right pelvis and groin and feels like an ache. She addressed this pain with her gynecologist who has ordered a trans-vaginal ultrasound to rule out ovarian cancer. Patient also has hematuria for which she recently underwent cystoscopy by urology as well as computed tomography (CT) of the abdomen and pelvis per her primary care physician. She denies changes in bowel or bladder function, no paresthesia nor anesthesia, no weakness, no fevers/chills/ns, no weight loss. She only notes she has frequent urinary tract (UTI) infections despite her bladder suspension, but that her incontinence is much improved.

ROS:
Constitutional: no mental status changes, no travel or sick contacts
Eyes: no change in vision
Endocrine: no fever, night sweats, weight change
Cardiovascular: no chest pain or palpitations
Respiratory: no dyspnea, cough, or sputum
Gastrointestinal: no nausea, vomiting, diarrhea, no change in bowel movements, no blood in stool
Genitourinary: no change in urine, no dysuria, no blood in urine
Neurologic: no anesthesia, no paresthesia, no loss of consciousness, no seizures
Musculoskeletal: no weakness
Psychiatric: no homicidal or suicidal ideations, no signs of depression
Allergy: no angioedema, no urticaria, no anaphylaxis, no symptoms of seasonal allergies

PMHx/PSHx:
1. Right-total knee replacement secondary to degenerative osteoarthritis
2. Total abdominal hysterectomy for noncancerous reasons (still has ovaries)
3. Urinary Incontinence status post bladder suspension surgery
4. Primary Hypertension
5. Rheumatoid Arthritis (RA)
6. Chronic Sinusitis
7. Frequent UTIs

Allergies:
-no known drug or food allergies

MEDs:
-Norvasc 10mg daily
-Benicar 40mg daily
-Potassium chloride 20meq daily
-Remicaid injection every 6-8 wks
-Advil over the counter every 6 hours as needed

Immunizations:
-Tetanus/Diphtheria/attenuated Pertussis in 2015
-Influenza in fall 2015
-Pneumovax fall 2007, 2012

Social:
-no smoking; no alcohol; no drugs; work = retired office worker

FamHx:
Mom = cancer-right thigh with metastasis to brain, patient unsure of exact cancer type
Dad = unknown

VITALS = 120/70, 196, 72bpm, 5'4", 98.7F, BMI = 33.7
GEN = awake, alert, oriented to time, place, situation, self, in no acute distress
HEENT = normal cephalic, a-traumatic, extraocular muscles intact, no otic/nasal discharge
NECK = supple, thyroid w/o masses
HEART = regular rate, no clicks/rubs/murmurs
LUNGS = clear to auscultation, without crackles, wheeze, or rhonchi
GI = soft, non-tender, no rebound tenderness, +bowel sounds x 4 quadrants, no hepato-splenomegaly
CV = no jugular venous distention, no carotid bruits, mucus membrane moist, no edema, 2/4 peripheral pulses, no lymphadenopathy
N/M = cranial nerves 2-12 intact, 5/5 global strength, 2/4 global reflexes, cerebellum intact, sensation symmetric, gait normal
Biomechanical Exam pathology = **tender to palpation on cervical spine segment C4 on right posteriorly; Lumbar spine has somatic dysfunction. Iliac crest height on the right is elevated compared to the left by ~0.5cm in seated and standing positions. Short right leg when sitting, standing, and lying compared to the left.**

Assessment/Plan

1. Low Back Pain:
   This patient has no red flags for treatment. While she has no red flags to OMM and somatic dysfunction, given her history of RA and age, I have requested records regarding her CT of the abdomen/pelvis and records from her PCM for evaluation prior to her treatment. This is an attempt to rule-out cancer and potential complications of RA. I do not plan to perform any HVLA or high cervical flexion positioning for this patient due to age and RA.
2. Somatic Dysfunction, pelvis, lumbar spine, cervical spine:

At this time, I believe her pelvic pain could be due to a unilateral pelvic up-shear on the right. If her records do not appear contradictory to manual medicine, I will plan to focus treatment beginning at this area.

3. Rheumatoid Arthritis:

Will see her rheumatologist next week.

4. Frequent UTIs:

Since she is immune compromised by RA and Remicaid, I emphasized good hygiene. She will address this with her rheumatologist and urology.

5. Obesity:

Is likely contributory to her overall picture of health including HTN, LBP, osteoarthritis which led to her knee surgery. Encouraged this patient to begin exercise as tolerated to a target HR of 120bpm for ideally 30min for 3-5 times weekly and dietary program consisting of 1800kcal/day.

6. HTN:

Since goal BP<140/90; this is well controlled

7. Other:

F/u in 1 week, medication reconciliation done, will send a copy of this note to urology, rheumatology, and gynecology.

Jason Browder, DO

Review Questions:

1. The physiological barrier _____
    a. Represents the limit of active range of motion
    b. Represents the limit of passive range of motion
    c. Represents the capsule and supporting ligaments
    d. Represents bone on bone contact

2. On a normal standing posture exam, the imaginary, lateral, weight-bearing line runs through all of the following structure:
    a. Posterior orbit
    b. Acromioclavicular joint
    c. Second lumbar segment
    d. Posterior portion of the lateral malleolus

3. With regard to range of motion (ROM) testing, which of the following statements is correct?
    a. Active ROM describes patient initiated movement
    b. Active ROM describes operator-initiated movement
    c. Joint play is a type of active ROM

4. Which of the following statements regarding the hand during palpation is correct?
    a. Palm for determining fine structural symmetry
    b. Finger joints for vibratory sense
    c. Finger pads for gross discrimination
    d. Dorsum of hand for depth perception
    e. Tips of thumbs for temperature sense

5. A review of systems (ROS):
    a. is useful to uncover medical illness
    b. is considered an objective finding
    c. is part of the past medical history
    d. is not necessary on a follow up appointment

6. The acronym TART stands for:
    a. Tension, asymmetry, restricted ROM, and temperature change
    b. Tenderness, asymmetry, restricted ROM, and tissue texture change
    c. Tenderness, asymmetry, restricted ROM, and temperature change
    d. Tension, asymmetry, restricted ROM, and tissue texture change

7. Which of the following is a direct treatment technique?
    a. Strain Counter-Strain

b. Functional Procedures
c. Myofascial Release
d. Facilitated Positional Release

8. Treatment reactions comprise the following:
    a. Usually last 30 to 60min after treatment
    b. Indicate iatrogenic injury
    c. If severe, may mimic influenza
    d. Are due to release of uric acid

9. Relative Contraindications to manual medicine include:
    a. Osteoarthritis
    b. Bilateral carotid stenosis
    c. Hypermobility syndrome
    d. Grade I and II spondylolisthesis
    e. Intracranial bleed

10. What is the rate of severe complications with manual medicine?
    a. 1 in 1,000
    b. 1 in 10,000
    c. 1 in 100,000
    d. 1 in 1,000,000
    e. 1 in 10,000,000

11. Severe complications of OMT occur with
    a. C1 in extension
    b. C1 in flexion
    c. C2 in extension
    d. C2 in flexion

12. Active range of motion is induced by:
    a. The patient
    b. The practitioner
    c. HVLA impulse

13. Tender points (TP) are characterized by the following:
    a. greater than 1cm
    b. edematous
    c. radiating
    d. exhibit local twitch response

14. Providers should consider deferring treatment or withholding HVLA impulse for which patient:
    a. osteoarthritis
    b. trisomy 21
    c. embolic stroke
    d. joint pain

Answers:

1. A
2. B
3. A
4. B
5. A
6. B
7. C
8. C
9. E
10. D
11. B
12. A
13. B
14. B

Chapter 2: The Thoracic Spine

Principles Presented in this Chapter:

- Spinal Mechanics
- Positional Versus Restrictive Diagnosis
- Diagnosis using Spinal Mechanics
- Treatment using Spinal Mechanics

Outline:

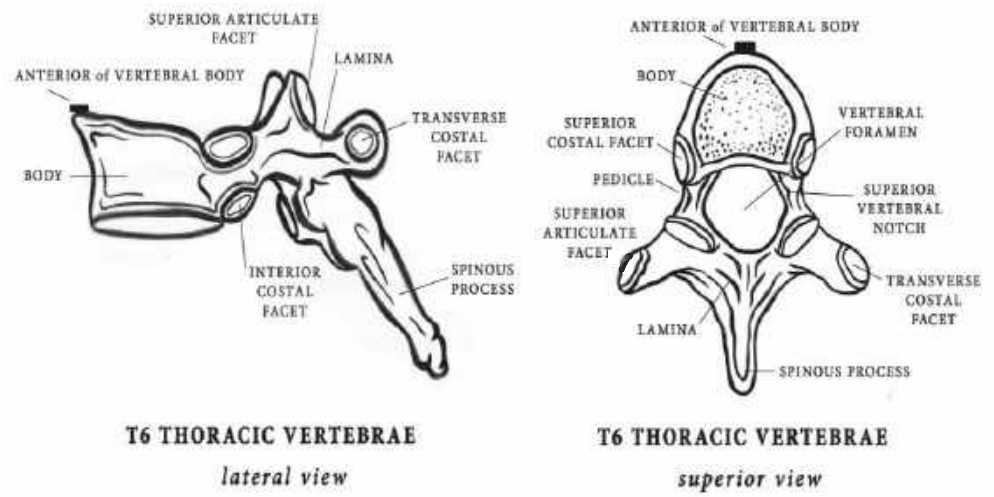
- Anatomy of the Thoracic Spine
- Biomechanics of the Thoracic Spine
    - Gross Diagnosis of Thoracic Spine – Locating the Area of Somatic Dysfunction
- Diagnosis of the Thoracic Spine
    - Etiology of Thoracic Somatic Dysfunction
- Treatment of the Thoracic Spine
    - Direct Treatment of the Thoracic Spine
    - Indirect Treatment of the Thoracic Spine
- Thoracic Spine Treatment Pearls
- Clinical Vignette
- Appendix A: Strain-counterstrain Tenderpoints
- Review Questions

Chapter 2: The Thoracic Spine

Anatomy of the Thoracic Spine

The thoracic spine chapter lays the framework for spinal mechanics and is fundamental to understanding the lumbar and cervical spine chapters. Anatomically, gaining knowledge of a few structures will lead to understanding spinal mechanics. Figure 1 provides the basic anatomy of a spinal segment.

Figure 1



When describing vertebral position in three dimensions, the anterior-superior vertebral body is the reference point for naming. Other essential structures include the transverse processes and the superior and inferior articular processes. These structures, consistently located at the level of the vertebral body, allow the practitioner to reliably palpate that segment. In contrast, the spinous process of T7, for example, extends posteriorly and inferiorly, posteriorly overlying the vertebral body of T8 below. Many anatomical variations exist for spinous processes, some with curvature right or left; if used as a landmark, the spinous process may lead to misdiagnosis. The Rule of Threes for spinous processes roughly estimates the location of thoracic spinous processes in relation to their corresponding vertebral segment. Location of the spinous process in relation to a vertebral segment is of clinical importance when using it as a lever for treatment, as in muscle energy (ME) procedures.

Figure 1: Thoracic Spinal Segment Anatomy:
The anterior-superior vertebral body is the reference point of vertebral position in three-dimensional space; determine position through palpation of the transverse processes and articular facets.

The Rule of Threes for Thoracic Spinous Processes:
T1-3 = spinous processes project posteriorly and lie at the level of their corresponding vertebral body.
T4-6 = spinous processes project posteroinferiorly, halfway between their corresponding vertebral body and the one below.
T7-9 = spinous process project inferiorly and overlay the body of the inferior segment.
T10 = same as T7-9
T11 = same as T4-6
T12 = same as T1-3

To locate articular facets, just identify the spinous process and slide the thumb to the right or left into the para-spinal "gutter." Approximately 1cm lateral to the facets lays the transverse processes and further laterally the angle of the ribs. Figure 2 has anatomical landmarks that aid in locating thoracic segments.

Figure 2

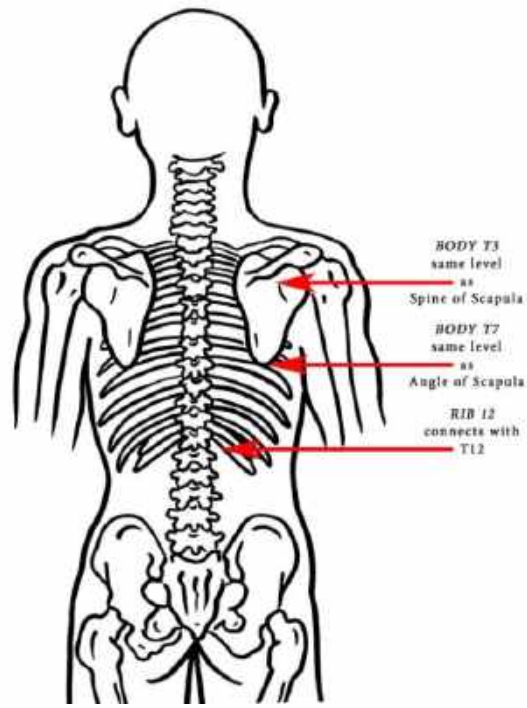


Figure 2:
Location of Thoracic Segments in Relation to other Structures.

Recalling basic anatomy, sagittal plane compartments include flexion, extension, and neutral (Figure 3). Normally, when a patient moves into flexion, facet joints should open; conversely, during extension both facets should close. This concept is clinically relevant in both diagnosis and treatment.

Figure 3

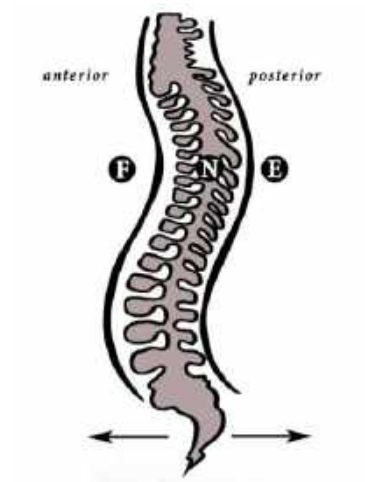


Figure 3: Sagittal Plane of Flexion, Extension and Neutral from Left Lateral Perspective:
F = Flexion
E = Extension
N = Neutral

Using "joint play," mentioned in the Basic Principles chapter, one may easily rotate a non-pathologic vertebral body right and left using the transverse processes or the articular facets. By pushing the left transverse process of T6 anteriorly, the segment rotates right. If T6 remained in this position pathologically, the positional diagnosis of rotation would be *T6 rotated right*. A positional diagnosis describes somatic dysfunction as its current location in three-dimensional space.

Positional Diagnosis:
Named for the location of somatic dysfunction in three-dimensions.

Restriction Diagnosis:
Named for the limitation in motion due to somatic dysfunction. The naming must contain either "restriction" or "restricted."

Naming *T6 restricted in left rotation* would also give the same diagnosis but depicts a restriction diagnosis. A restriction diagnosis identifies motion limitation, aka hypo-mobile somatic dysfunction. Restriction diagnoses must contain the word "restriction" or "restricted" when naming pathology.

Most osteopathic medical schools emphasize positional diagnosis, as do Comprehensive Osteopathic Medical License Examination (COMLEX) board exams based on the Educational Council of Osteopathic Principles (ECOP) recommendations. Unless otherwise specified, this text uses positional diagnosis when naming somatic dysfunction.

An important concept in communication is naming a pathological segment in relation to another. In the previous example, T6 is pathologically rotated right in relation to both T5 and T7. For simplicity and ease of communication, we refer to the pathological segment below as the one affected. Thus, T6 rotated right refers to T6 in relation to T7, **not** T6 in relation to T5.

Pathologic Spinal Segmental Diagnosis:
Segments are named for the pathological segment in question and the one below.

Coupling is the term that further describes the rotation or sidebending of a vertebral body about or along one axis that is consistently associated with its rotation or sidebending with another segment.[7(pp391)] As we will see, coupling behavior between two segments sets the framework for spinal biomechanics.

Biomechanics of the Thoracic Spine

Spinal mechanics was first widely published in *Principles of Osteopathic Technic* by Harrison H. Fryette, DO, in 1954. Three principles predict spinal motion of the lumbar and thoracic spine in the sagittal plane: flexion, extension and neutral. In his publications, Fryette introduced the following concepts that have been termed the Principles of Spinal Motion:[1(pp21-22), 12(pp43-44)]

Principles of Spinal Motion of the thoracic and lumbar spine:

1. When in neutral, sidebending produces rotation opposite that of the sidebending. The direction of rotation is toward a convexity.
2. When in flexion or extension, sidebending and rotation occur in the same direction. The direction of rotation is toward a concavity.
3. Introducing motion in one plane limits its mobility in the other two planes.

Principle I: Neutral sidebending produces rotation to the side opposite of the sidebending.

Principle II: Non-neutral rotation produces sidebending to the same direction as rotation.

Principle III: Introducing motion to a spinal joint in one plane automatically reduces its mobility in the other two planes.

The first principle of spinal motion describes the spine in a neutral position known as type I spinal motion in the ECOP glossary.[2(pp1107), 12(pp43)] According to Fryette, active or passive motion in the neutral plane that introduces right sidebending, will cause left, or opposite, vertebral rotation. In spinal mechanics, rotation that is opposite sidebending is referred to as contralateral coupling. During contralateral coupling, the vertebrae above and below also rotate and sidebend oppositely, producing spinal convexity toward the rotational direction of the segments involved (see Figure 4). The motion of sidebending precedes rotation in type I motion; thus, sidebending is documented before rotation in the abbreviation *NSR* (neutral, sidebent, rotated). Using the first principle, a neutral T6 somatic dysfunction when sidebent left, will presumptively rotate right and is abbreviated *T6 NSLRR*. The restriction diagnosis for the same pathology would be *T6 restricted in NSRRL*.

Figure 4

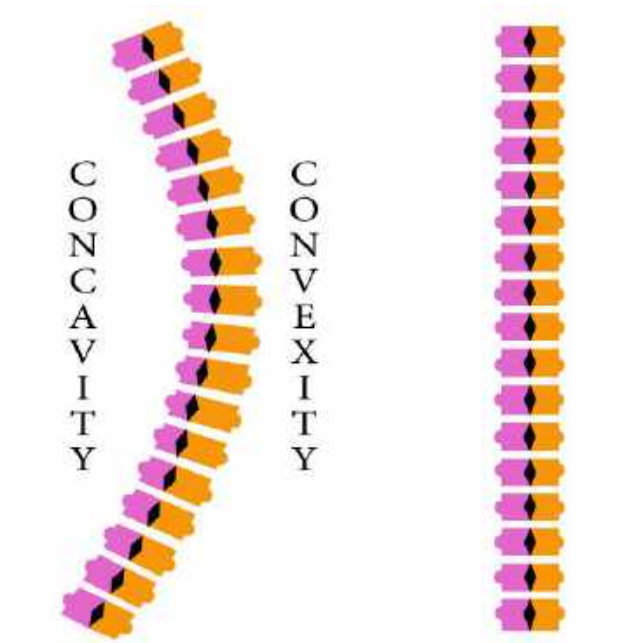


Figure 4: Type I Spinal Motion:
Orange color indicates the right side of the vertebrae and pink the left. The black ellipse represents the spinous process. Rotation and sidebending occur in opposite directions. Note that vertebral rotation is toward the convexity of the scoliotic curve.

Naming Abbreviations:

ERS (extended, rotated, sidebent)
FRS (flexed, rotated, sidebent)
NSR (neutral, sidebent, rotated)

The second principle of spinal motion, also known as type II spinal motion, describes the spine in a non-neutral position of flexion or extension. While in flexion or extension, rotational movement precedes sidebending;[2(pp1108)] hence the abbreviation *FRS* or *ERS* for flexed and extended somatic dysfunction. Fryette noted active or passive vertebral motion in a non-neutral position, couples ipsilaterally. For example, T6 in flexion when rotated left will presumptively cause left sidebending and is abbreviated FRLSL or simply FRSL.

Typically, only two segments are considered pathologic in type II somatic dysfunction. Since sidebending and rotation couple ipsilaterally, the scoliotic convexity is opposite the rotational direction of the segments involved (Figure 5).

Figure 5

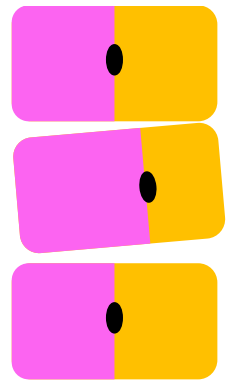


Figure 5: Type II Spinal Motion:
Orange color indicates the right side of the vertebrae and pink the left. The black ellipse represents the spinous process. Rotation and sidebending are coupled. Note that rotation of the vertebrae is toward the concavity of the scoliotic curve for this segment which is sidebent left and rotated left.

The third principle of spinal motion was actually described by Nelson in 1948.[12(pp44)] It states that as motion is introduced in one plane, the mobility in the other two is automatically reduced. That is, a patient placed in flexion will automatically have reduced capacity for sidebending or rotation. This principle is universally accepted. The relevance of the third principle, or type III spinal motion, is further explained in the diagnosis portion of this chapter.

Traditionally, type I motion is thought to wholly exist in the neutral compartment of the lumbar and thoracic spine. Fryette's research on skeleton and cadaver anatomy led him to believe that type I motion did not exist in the cervical spine. Recent *in-vivo* studies[3] have shown that contralateral coupling does occur in the cervical spine, ironically with greater frequency than ipsilateral.[4] Other studies have shown motion of the lumbar spine to be less predictable than Fryette proposed, and that in practice, contralateral and ipsilateral coupling are not limited to neutral and non-neutral compartments respectively.[5,6] With regard to the thoracic spine, a meta-analysis[7] of 56 studies found contralateral and ipsilateral coupling behavior inconsistent in any sagittal compartment. In this chapter, we present spinal diagnosis using principles of spinal motion because they were, until recently, widely accepted and ingrained in manual medicine treatment (MM/T) literature. The principles of spinal motion, despite recent evidence, are still useful as a starting point for musculoskeletal assessment and application of MM/T.

Note that the whole thumb held in a rigid-straight position is used in the spinal sweep.

Gross Diagnosis of Thoracic Spine – Locating the Area of Somatic Dysfunction

To test for somatic dysfunction in the thoracic spine, a practitioner should grossly palpate the spine for TART criteria. Several methods exist for broad evaluation.

a. One method uses the pads of digits two and three over the right and left facets. Using slight pressure, the practitioner drags their fingers caudad to feel for tenderness, asymmetry or tissues texture changes. They then return to any abnormal area and use motion testing (see the next section below) to determine specific segmental somatic dysfunction.

b. Another method utilizes type III spinal motion. This method is further explained in the sequencing chapter we term the "spinal-sweep." To test a group of right facets, the practitioner's rigid-straight thumb is placed over a group of the patient's RIGHT facets. Figure 6 demonstrates gross testing of facets T2-6. The practitioner places their right hand on the patient's right shoulder.

Figure 6

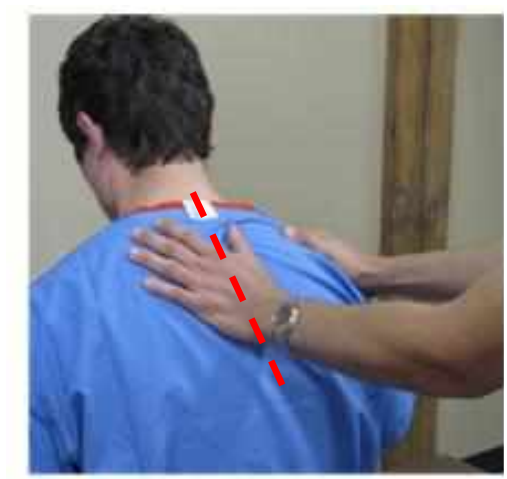


Figure 6:
Hand placement for gross testing of the facets T2-6. The locations of spinous processes are designated by the red dashed line.

As pictured, note that both the left thumb and the right hand (which introduces rotation and sidebending) remain on the right side of the patient's spinous processes when testing the right facets.

Conversely, when testing the corresponding LEFT facets (not pictured), both the right thumb and the left hand remain on the left side of the patient's spinous processes.

In one coordinated sweeping motion, the practitioner introduces extension with the thumb in a diagonal vector (Figure 7) while simultaneously introducing right sidebending and right rotation with their RIGHT hand; this motion creates a physiological "lock" of the elastic barrier.

Figure 7

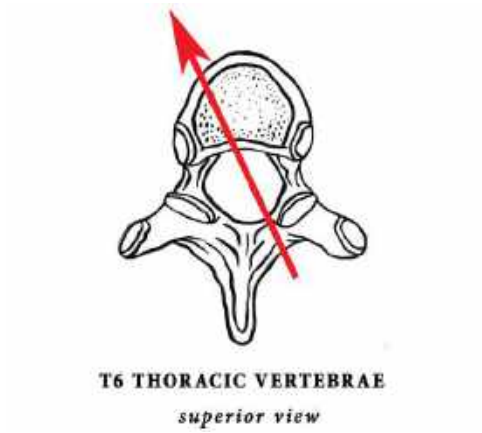
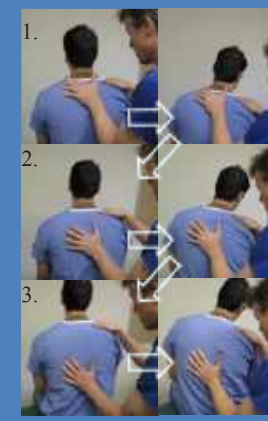


Figure 7:
The diagonal vector introduced by the left thumb as depicted in Figure 6.

Normal motion will have an end "spring" as multiple facet pairs meet their elastic barrier. If tenderness or passive ROM is reduced, somatic dysfunction is present. The practitioner then "sweeps" the thumb inferiorly to assess remaining thoracic segments.

To test a group of left facets, the RIGHT thumb is placed over a group of the patient's LEFT facets. The practitioner would place their left hand on the patient's left shoulder and repeat this process on the left facets. This spinal sweep is highly efficient as four to five segments of the spine are simultaneously evaluated.

The "spinal sweep" allows the examiner to locate the area of somatic dysfunction efficiently. Pictured below, the examiner sweeps from the upper to the lower thoracic spine on the right.



Both methods presented grossly evaluate TART criteria; the more TART criteria are present, the more likely somatic dysfunction is present[8]. Keep in mind that both methods guide a practitioner to the area of spinal somatic dysfunction, however, they do **not** provide a specific positional or restriction diagnosis.

Diagnosis of the Thoracic Spine

Once an area of thoracic pathology is suspected, determine a positional or restriction diagnosis of the segment in question. Typically, the first and second principles of spinal motion correctly diagnose the thoracic spine.[9] Accordingly, to extrapolate the sidebending, just know the sagittal compartment and the rotation.

When diagnosing vertebral somatic dysfunction, remember that we are actually evaluating a facet's ability to open and close.

With the principles of spinal motion in mind, a positional diagnosis is easily made. To begin, a practitioner monitors the transverse processes of a single thoracic segment such as T6 with their thumbs while the patient actively moves through neutral, flexion and extension. Normally, the facets should open during flexion and close during extension. When somatic dysfunction is present, it is usually a unilateral phenomenon occurring in one pair of facets.

Non-neutral pathology is noted when the facets do not open during flexion or do not close during extension. This is clinically apparent when the thumb on one transverse process, for instance the left, is more posterior during flexion or extension, indicating left rotation of the T6. Assuming validity of the second principle of spinal motion, if left rotation is observed in flexion, the diagnosis of T6 *ERLSL* (abbreviated *ERSL*) is made. For beginning practitioners, it is essential to understand that diagnosis of extended vertebrae is made during patient flexion when it is most apparent. This concept is illustrated in Figure 8a. Figure 8b and 8c demonstrate left rotation and closed facets of T6 in relation to T7.

Figure 8a

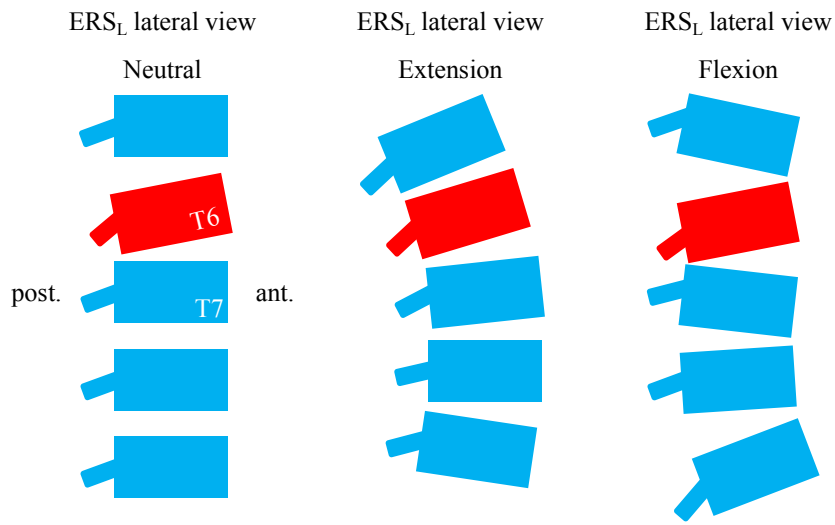


Figure 8a: T6 ERSL
The positional diagnosis of extension is not apparent when the spine is in extension. Note that the segment remains in extension when the spine is in flexion; a practitioner will notice this by the inability of the facets to open and restricted range of motion.

post. = posterior
ant. = anterior

Figure 8b

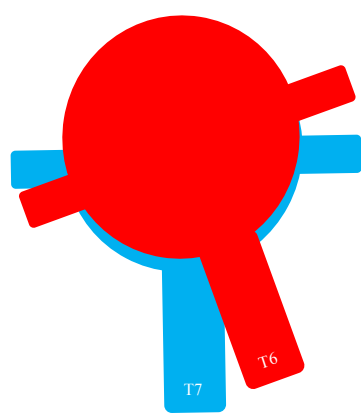


Figure 8c

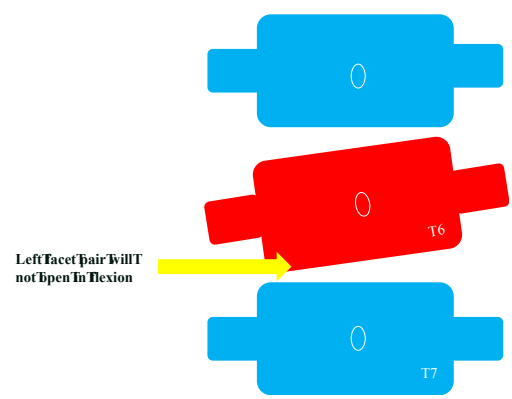


Figure 8b & 8c: Positional Diagnosis of T6 ERSL
Note left rotation of T6 in relation to T7 in Figure 8b. Also note that the left facet pair will not open as in Figure 8c.

In a separate example, a practitioner who observes a posterior thumb on one transverse process in neutral can reason that sidebending is opposite of rotation. For instance, if a posterior left thumb is present in neutral and moves symmetrically during flexion or extension, the positional diagnosis of *NSRRL* is made (assuming integrity of type 1 motion).

As mentioned previously, the assumption that principles of spinal motion are always correct is unsubstantiated by recent research. Should a practitioner choose to make no assumptions, testing coupling behavior is easily established. With a patient positioned in neutral, flexion or extension and passively sidebent right or left, the practitioner can palpate the transverse processes to determine rotation (this concept is further explained in the lumbar spine chapter). A practitioner need not reject diagnoses such as bilateral facet flexion or extension, *T6 NSRRR* or *T6 FSLRR*, to adhere to the principles of spinal motion. Further, when principles of diagnosis are understood rather than memorized, a provider can recognize and adapt when faced with unexpected clinical pathology.

Typically, the principles of spinal motion correctly diagnose the THORACIC spine.

Etiology of Thoracic Somatic Dysfunction

One question that remains unanswered is the cause of somatic dysfunction found between spinal vertebrae. Phillip Greenman, DO, FAAO, suggests that hypertonic, fourth-layer, paraspinal muscles maintain somatic dysfunction.[10] The muscles illustrated in Figure 9 include rotatores, multifidus, intertransversarii, and levator costalis.

Figure 9

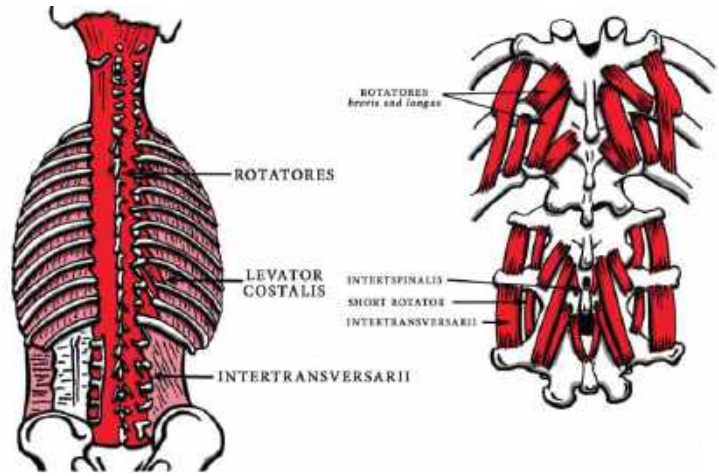


Figure 9:
Fourth layer paraspinal muscles implicated in maintaining somatic dysfunction of the spine.

Muscle Layers of the Thoracic Spine:

1st layer: trapezius, latissimus dorsi, levator scapulae, rhomboids

2nd layer: serratus posterior superior and inferior

3rd layer: iliocostalis, longissimus, and spinalis

4th layer: rotatores, intertransversarii, multifidus, and levator costalis

The mechanism by which manual medicine resolves somatic dysfunction is explained further in the neurophysiology chapter. The most important concept to understand in spinal motion is that somatic dysfunction prevents facets from opening in flexion and closing in extension; this concept, as well as the positional diagnosis, guides treatment.

Treatment of the Thoracic Spine

Before beginning this section, we emphasize that this is not a procedure manual. This introductory book teaches students to use principles of diagnosis that in turn facilitate treatment. In the manual medicine lab, instructors should call

upon their own experience and ideally teach procedures according to the principles presented in this book. Knowledge of this text's principles will ultimately allow students to craft their own treatment methods.

Direct Treatment of the Thoracic Spine

Using spinal mechanics, the positional diagnosis guides treatment of somatic dysfunction both directly and indirectly. Use the example diagnosis *T6 ERLSL* in which the left facets of T6-7 cannot open. To treat this segment with direct means, just reverse any of the components of the diagnosis. This is done by positioning T6 in flexion, right rotation, or right sidebending in relation to T7. The most efficacious direct treatments reverse all three components of the positional diagnosis. In other words, if a segment is pathologically maintained in *ERLSL*, and then placed in *FRRSR,* the patient will be directly positioned against the barrier of somatic dysfunction. In an attempt to open the facet, from here, implement a fulcrum, lever, and force. In review, the steps for direct treatment *T6 ERLSL* are:

1. Obtain the positional diagnosis *T6 ERLSL.*
2. Place a fulcrum on T7.
3. Create a lever using the spinous process of T6 or by locking down (flexing) the spine to T6.
4. Reverse the components of the positional diagnosis by placing the patient against the motion barrier in T6 FRRSR.
5. Create a force by using an impulse or isometric contraction.

These steps are the principles used in direct treatment; there is no difference if the patient is prone, supine, or sitting. See the examples provided for high-velocity, low amplitude (HVLA) and muscle energy (ME) that further extrapolate these concepts.

Indirect Treatment of the Thoracic Spine

Indirect treatments are conceptually simpler than direct treatments. Again, use the positional diagnosis *T6 ERLSL.* To treat this segment indirectly, place the patient in the position of *T6 ERLSL*. Hence, the diagnosis is the same as the indirect treatment position. To review, the steps for indirect treatment *T6 ERLSL* are:

1. Obtain the positional diagnosis *T6 ERLSL.*
2. Place the patient in the position *T6 ERLSL*.
3. Exaggerate the extension, left sidebending, and left rotation to a position of three-dimensional balance or patient comfort.

These steps are a gross simplification of the principles used during indirect treatment. For subtle differences of each technique, examples are provided for strain-counterstrain (S/CS) and functional procedures.

With regard to extended somatic dysfunction, S/CS tender points correspond to posterior tender points and the related hypertonic-fourth layer musculature of the spine. Anterior tender points along the sternum correspond to flexion dysfunction; they represent reflex points as opposed to a directly palpable vertebral segment.

SEE APPENDIX A FOR THORACIC COUNTER-STRAIN POINTS

Thoracic Spine Treatment Pearls

When faced with multiple diagnoses, a beginning student may not know where to begin treatment; a thorough method for the whole body is located in the sequencing chapter. With regard to the thoracic spine, clinical practice by Edward Stiles, Phillip Greenman and others has provided treatment "pearls" to aid in determining key areas of somatic dysfunction. These methods are listed below:

- Contralaterally coupled dysfunctions are usually compensatory; when found, look for and treat ipsilaterally coupled dysfunctions, or look for length inequality of the lower extremity (short femur or tibia, fallen arch).
- Typically, contralaterally coupled dysfunctions occur in groups of three to five segments. Treat the center segment to resolve the whole group.
- The thoracic spine has a natural kyphosis or flexion. Extended segments within this curve reduce kyphosis and are highly pathologic. Likewise, the lordotic curves of the cervical and lumbar spine with flexed segments should be treated first.
- For best results during direct treatment, reverse all three components of the positional diagnosis (i.e., with the diagnosis of *ER$_L$S$_L$*, position the patient *FR$_R$S$_R$*).
- Place the spinal segment treated at the apex of the sidebending, flexion and extension curves (see below).
- Bilateral rib cage somatic dysfunction usually indicates segmental thoracic or lower cervical spine dysfunction.
- When crossing arms across the chest for trunk control, place the arm opposite the provider on top (see below).
- Treat using direct methods first and then indirect, to integrate the dysfunctional segment with surrounding soft tissues.
- Without the history of trauma, when encountering somatic dysfunction of the thoracic spine involving multiple levels, consider viscerosomatic dysfunction in the differential diagnosis. For example, with T5-8 NS$_R$R$_L$ consider gastroesophageal reflux). Details of viscerosomatic reflex are discussed in the neurophysiology chapter.

Treatment Examples: Direct Procedures

T6 ERS$_L$ using ME

1. Place a fulcrum on T7 using the thumb and index finger of the left hand. This hand will also monitor the left facet pair opening. Have the patient cross their arms to gain control of the trunk; typically, the arm on the opposite side of the provider should be on top for optimum trunk control (in this instance, the left arm).

2. Create a lever by locking down (flexing) the spine until motion is felt on the T6 segment above T7. It is ideal to place the dysfunctional segment (T6) at the apex of the curve created by flexion (imaginary curve of the spine depicted in red).

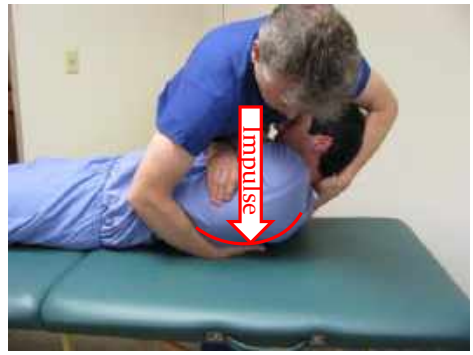
3. Further reverse the components of the positional diagnosis by sidebending right and rotating right into the position FRS$_R$. Sidebending can be achieved by pushing the T7 left. Once at the closed pack position, slightly release tension to the "feather edge" of the barrier.

4. Create an isometric contraction by instructing the patient to rotate toward the center of their body (left in this instance) while the operator resists for 3-5 seconds. Have the patient relax. Reposition at the new barrier by taking up the slack in flexion, right sidebending or right rotation. Repeat the process of isometric contraction, relaxation and movement to the new barrier three to five times.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

T6 ERS$_L$ using HVLA (Kirksville Crunch)

1. Have a supine patient cross their arms to gain control of the trunk. Typically, the arm on the opposite side of the provider should be on top for optimum trunk control (in this instance the left arm). Roll the patient toward you to gain access to the thoracic spine.
2. Place a fulcrum across T7 using the thenar eminence on the left facet pair and rest the spinous process in the palm (some prefer to use a fist and place the spinous process between the thenar eminence and the DIP joints of digits 2-4).

3. Create a lever by flexing the spine until motion is felt on the T6 segment above T7. It is ideal to place the dysfunctional segment (T6) at the apex of the curve created by flexion (imaginary curve of the spine depicted in red).
4. Further reverse the components of the positional diagnosis by sidebending right and rotating right into the position FRS$_R$. Have the patient breathe in and out to take up further slack. Feel for a locking at the closed packed position.



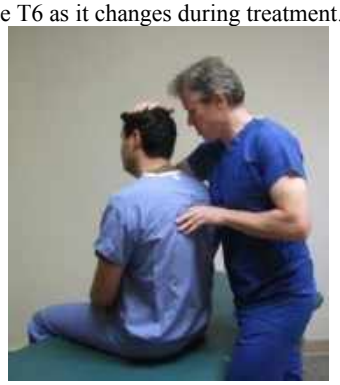
5. Create an impulse by dropping your weight directly down toward the fulcrum to open the left facets as indicated by the arrow (do not back off the barrier and then thrust). If the positional diagnosis had been *T6 FRS$_L$*, the impulse would have been diagonally toward the top of T6 vertebrae in an attempt to reverse the flexion and close the facets (see picture below).

6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Treatment Examples: Indirect Procedures

T6 ERSL using Functional Procedures

1. Place the thumb and index finger of the left hand on T6. This hand will monitor the T6 as it changes during treatment.

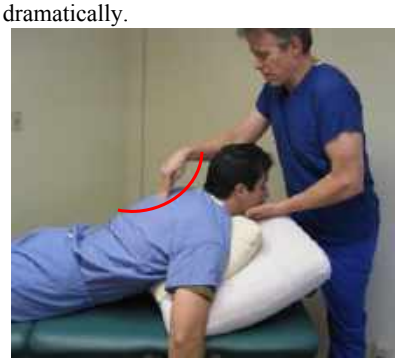


2. Place the right hand and forearm across the patient's shoulders to gain control of the trunk.
3. While sitting behind the patient and using your head as a balance-pivot, introduce extension, left sidebending and left rotation until at position T6 ERSL. As in the direct techniques, place T6 at the apex of the curve. Fine tune T6 until it is balanced in three-dimensional space by adjusting flexion-extension, sidebending and rotation to the dynamic balance point (DBP).

4. Add gentle compression from the right hand, through the thorax toward T6. Monitor the DBP as it changes using the left hand and adjust the flexion-extension, sidebending and rotation to maintain balance. If the DPB reaches a still point, wait for 10-20 seconds; if there is no change, challenge the tissues by either increasing or decreasing compression into T6. When the tissue relaxes, the treatment is finished.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction

T6 ERSL using S/CS (aka PT6 lateral-left)

1. Place the patient prone on a table. Monitor T6 with one finger. Confirm that T6 is tender on the left transverse process; tell the patient that this tenderness represents a 10 on a scale of 1-10.
2. Extend the patient using pillows or the treatment table until T6 is at the apex of the curve. Extension will usually reduce the tenderness dramatically.



3. Sidebend and rotate the patient left until tenderness is reduced to a 3 out of 10. Often the patient will ask if you are pushing in the same location or as hard as before. Sometimes, to achieve 70% comfort, you will have to sidebend or rotate the patient right for comfort. These points are called "rogue" points and occur 5-10% of the time.
4. Maintain this position for 90 seconds. You do not have to monitor the tender point during this time. If you choose to monitor, you may feel characteristic tissue changes.

5. SLOWLY, reverse sidebending and rotation, and SLOWLY reduce extension until the patient is fully prone again.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

References

1. Fryette, Harrison H., and W. Fraser. Strachan. *Principles of Osteopathic Technic.* Carmel, CA: Academy of Applied Osteopathy, 1954.
2. Chila, Anthony G. *Foundations of Osteopathic Medicine.* Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2011.
3. Capobianco, John D., and M. Protopapas. Rivera-Martinez. "Understanding the Combined Motions of the C3/C4 Vertebral Unit: A Further Look at Fryette's Model of Cervical Biomechanics." *American Academy of Osteopathy Journal,* no. 3 (12 2002): 15-30.
4. Ishii, Takahiro, et al. "Kinematics of the Cervical Spine in Lateral Bending." *Spine* 31, no. 2 (01 2006): 155-60. doi:10.1097/01.brs.0000195173.47334.1f.
5. Gibbons, P., and P. Tehan. "Muscle Energy Concepts and Coupled Motion of the Spine." *Manual Therapy* 3, no. 2 (05 1998): 95-101. doi:10.1016/s1356-689x(98)80025-8.
6. Fujii, Ryutaro, et al. "Kinematics of the Lumbar Spine in Trunk Rotation: In Vivo Three-dimensional Analysis Using Magnetic Resonance Imaging." *European Spine Journal Eur Spine J* 16, no. 11 (06, 2007): 1867-874. doi:10.1007/s00586-007-03730-3.
7. Sizer, Phillip S, Jean-Michel Brismée, and Chad Cook. "Coupling Behavior of the Thoracic Spine: A Systematic Review of the Literature." *Journal of Manipulative Physiological Therapeutics* 30, no 5 (06 2007): 390-99. doi:10.1016/j.jmpt.2007.04.009.
8. Fryer, Gary. "Muscle Energy Concepts - A need for Change." *Journal of Osteopathic Medicine* 3, no. 2 (2000): 54-59.
9. Willems Jm, Ga Jul, and Jk-F Ng. "An in Vivo Study of the Primary and Coupled Rotations of the Thoracic Spine." *Clinical Biomechanics* 11, no. 6 (09 1996): 311-16. doi:10.1016/0268-0033(96)00017-4.
10. DeStefano, Lisa A. *Greenman's Principles of Osteopathic Medicine.* Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins. 2011:223.
11. Jones, Lawrence H., et al. *Strain-Counterstrain*. Boise, ID: Jones Strain-Counterstrain, 1995.
12. Giusti, Rebecca, DO. *Glossary of Osteopathic Terminology*. Third ed. Chevy Chase, MD: American Association of Colleges of Osteopathic Medicine, 2017.

Clinical Vignette

Patient Name: SM
June 7, 2020

CC: new patient visit, headache
HxCC: Patient is a 19yr, female. She presents as a new patient complaining of three months of headaches following a motor vehicle accident (MVA) in which she hit a barrier head-on at 35mph. The patient notes nausea, sensitivity to bright lights, mild vertigo, cognitive slowing, and headaches that come on after 30minutes of studying. She also reports bizarre crawling feelings the skin of her shoulders and neck. She has been to many specialists including neurology, physical therapy and psychiatry. Of note, she has had normal studies including magnetic resonance imaging (MRI) of the head x 2, lumbar puncture with an opening pressure of 15mm H_2O, chest x-ray, electroencephalogram, computed tomography of the head, MRI cervical-spine. The patient has a history of smoking cigarettes and marijuana; she has quit tobacco and marijuana x 2 weeks with little benefit. She is currently seeing a chiropractor who is doing stretching, heat packs, and electrical stimulation.

ROS:
Constitutional: no mental status changes, no travel or sick contacts
Eyes: no change in vision
Endocrine: no fever, night sweats, weight change
Cardiovascular: no chest pain or palpitations
Respiratory: no dyspnea, cough, or sputum
Gastrointestinal: no nausea, vomiting diarrhea; no change in BM, no blood in stool
Genitourinary: no change in urine, no dysuria, no blood in urine
Neurologic: no anesthesia, no loss of consciousness, no seizures
Musculoskeletal: no weakness
Psychiatric: no homicidal or suicidal ideations, no signs of depression
Allergy: no angioedema, no urticaria, no anaphylaxis, no symptoms of seasonal allergies

PMHx/PSHx:
1. Monopolar depression
2. Car accident age 19 that left a scar on her forehead
3. Pseudo-seizures exhibited and diagnosed during the past 3months. The patient states they occur with stress.

Allergies:
-no known drug or food allergies

MEDs:
-Fluoxetine 10mg daily (was on since age 10yrs; has quit 2months prior to onset of headaches)
-Fiorcet 1 tablet every 6 hours as needed

Immunizations:
-per her primary care physician

Social:
-smoking ½ pack per day for 2 years
-no alcohol
-drugs including marijuana on weekends
-work = college student

FamHx:
Mom = monopolar depression

VITALS = 5"4", 122#, 100/62, 107bpm
GEN = awake, alert, oriented to time, place, situation, self, in no acute distress
HEENT = normal cephalic, scar 2cm across forehead, extraocular muscles intact
NECK = supple, thyroid w/o masses
HEART = regular rate, no clicks/rubs/murmurs
LUNGS = clear to auscultation, without crackles, wheeze, or rhonchi
GI = soft, non-tender, no rebound tenderness, +bowel sounds x 4 quadrants, no hepato-splenomegaly
CV = no jugular venous distention, no carotid bruits, mucus membrane moist, no edema, 2/4 peripheral pulses, no lymphadenopathy
N/M = cranial nerves 2-12 intact, 5/5 global strength, 2/4 global reflexes, cerebellum intact, sensation symmetric, gait normal, observation of lateral standing posture is normal
Biomechanical Exam = significant findings include: tender to palpation on the spinous processes from C3-T2. She has exhaled ribs 1-2 on the right. T2 ERS$_R$. C1 left-rotated.

A/P

1. Headache
2. Barré-Liéou Syndrome following MVA
3. Potentially post-concussive syndrome
4. Somatic Dysfunction of neck, thorax, ribs
5. Marijuana and Tobacco Abuse

   --no red flags to treatment; her workup has been extensive.

   --Unless this is mechanical in nature as associated with her MVA, her headaches could be factitious and secondary gain could be considered. Her symptoms of nausea, sensitivity to bright lights, vertigo, cognitive slowing, paresthesia is consistent with injury to the posterior cervical sympathetic system (aka Barré-Liéou syndrome) and potentially post-concussive syndrome. If she fails manual medicine, prolotherapy and cognitive therapy may be in order.

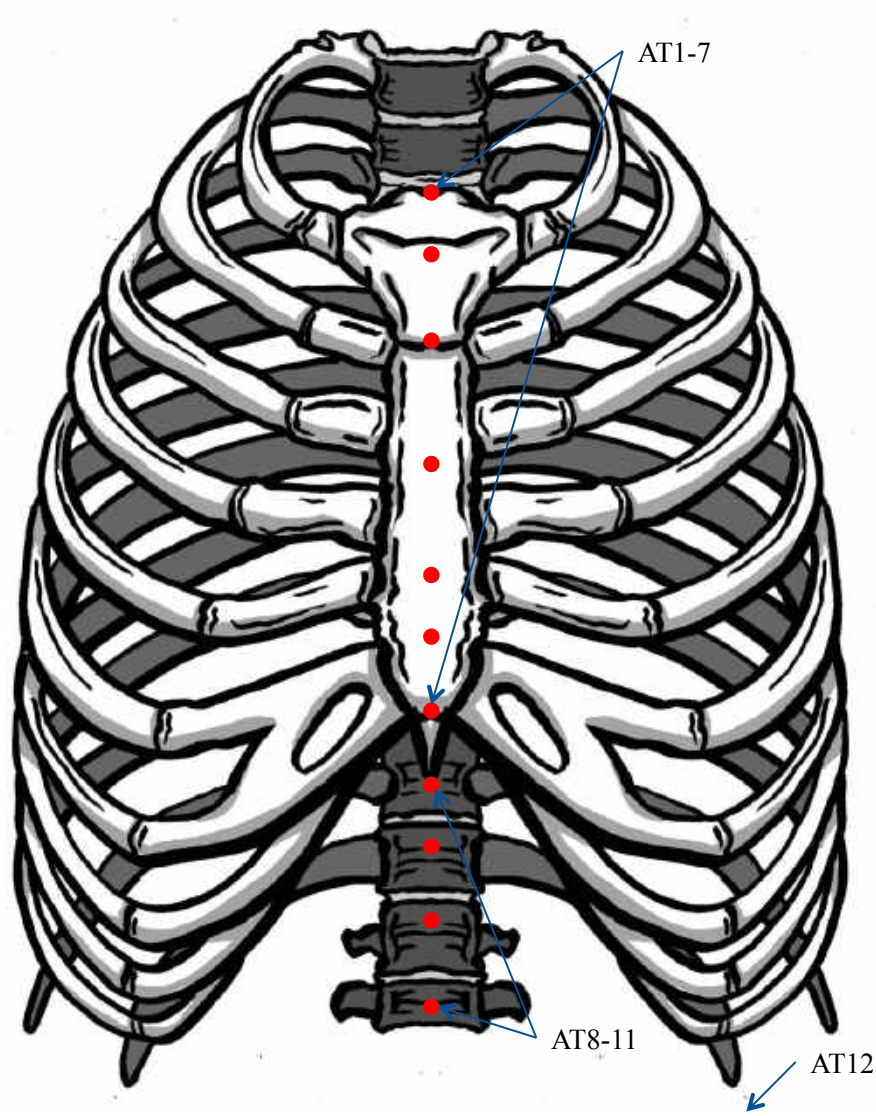
   --manual medicine performed today:

      1. Exhaled ribs 1-2 on the right treated with S-CS
      2. T2 ERS$_R$ treated with ME
      3. C1 left-rotated with ME
      4. Spinous processes from C3-T2 treated as with S-CS – note C7 is the most tender and treatment focused here

   --advised to quit smoking as it leads to chronic obstructive lung disease, multiple forms of cancer, and cardiovascular disease.

Jason Browder, DO

Appendix A: Anterior Thoracic (AT) S/CS Tender Points



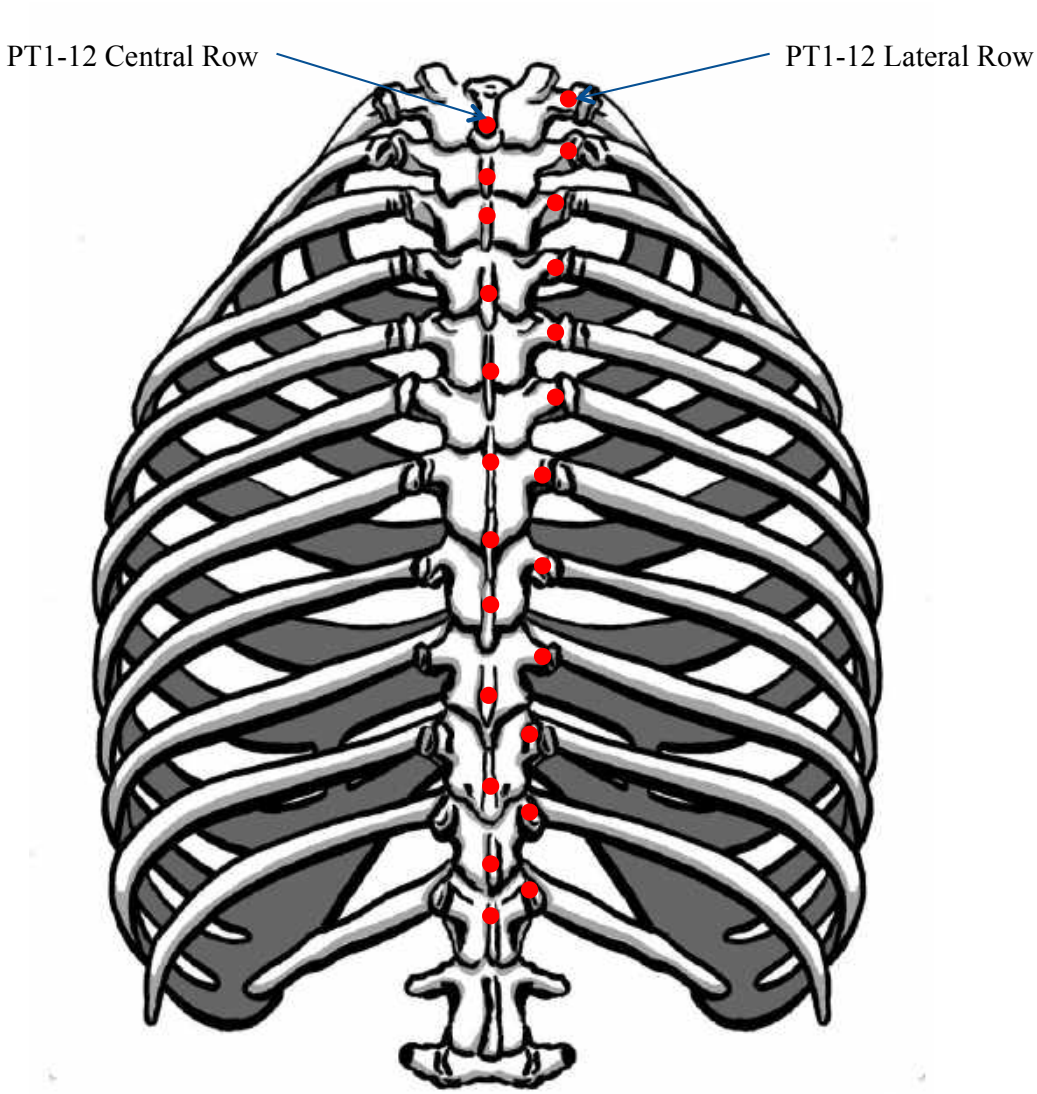
AT1-7 = along sternum and xiphoid*

AT8-11 lateral = in abdominal rectus muscles, note that A9 is superior to umbilicus and A10 is inferior to umbilicus*

AT12 = medial to the superior ilia*

*tenderpoints from Jones[11(pp62)]

Appendix A: Posterior Thoracic (PT) S/CS Tender Points



PT1-12 central = tip of spinous process of T1-T12 on each side*

PT1-12 lateral = posterior transverse process of T1-T12 bilaterally (only depicted on the right side above)*

*tenderpoints from Jones[11(pp50)]

Review Questions

1. As a patient moves into extension, he has a right posterior transverse process. Assuming the principles of spinal motion are correct in this instance, the optimum direct treatment position would be:
    a. ERS_L
    b. ERS_R
    c. FRS_L
    d. FRS_R
    e. NS_RR_L

2. The physiologic motion of the spine described by type II spinal motion:
    a. Occurs in neutral with sidebending and rotation to the same side
    b. Occurs in neutral with sidebending and rotation occurring oppositely
    c. Occurs in non-neutral with sidebending and rotation to the same side
    d. Occurs in non-neutral with sidebending and rotation occurring oppositely

3. Type I somatic dysfunctions:
    a. Are usually compensatory in nature
    b. Occur in groups of two or more vertebrae
    c. Produce rotation away from the convexity of the spinal curvature
    d. Occur in non-neutral positions

4. With your patient lying prone, application of a posterior to anterior movement on the left transverse process of T6 will cause:
    a. Flexion of T6
    b. Right rotation of T6
    c. Left rotation of T6
    d. Left rotation of T7

5. The reference point to spinal movement is:
    a. The anterior inferior portion of the vertebral body
    b. The anterior superior portion of the vertebral body
    c. The anterior middle of the vertebral body
    d. The center of the vertebral body

6. Given the positional diagnosis of T1 FRS_R, the corresponding restriction diagnosis would be:
    a. T1 restricted in ERS_L
    b. T1 restricted ERS_R
    c. T1 restricted FRS_L
    d. T1 restricted FRS_R

7. The diagnosis of T4 FRS_R is named in reference to T4 and:
    a. the segment above
    b. no other segment

c. the segment below

8. The purpose of gross spinal analysis using the spinal sweep is to:
    a. address the patient's pain compliant
    b. identify areas of greatest restriction
    c. diagnose segmental somatic dysfunction

9. The positional diagnosis of vertebral extension is made:
    a. During active patient extension
    b. During active patient flexion
    c. While the patient is in neutral

10. Which of the following are a component of a direct treatment:
    a. Placing the patient in the positional diagnosis
    b. Position of ease
    c. Lever
    d. Still point

11. When considering strain-counterstrain positioning, rogue points:
    a. Potentially represent an incorrect diagnosis
    b. Are noted when tenderness is reduced by reversal of flexion or extension
    c. Occur 35% of the time

12. Counterstrain treatment of anterior T2 somatic dysfunction:
    a. Classically takes ~120 seconds
    b. Typically occurs in a flexed position
    c. Does not typically have the TART criteria found in somatic dysfunction
    d. Involves the inferior portion of the manubrium

13. According to the principles of spinal motion, which of the following would not be a plausible diagnosis?
    a. FR_LS_L
    b. ER_RS_R
    c. NS_RR_L
    d. NR_RS_R

14. As you are examining a patient with your fingers on the posterior facets, in neutral the facets are symmetrical. As the patient moves into flexion, the left pair of facets are more posterior than the right. During extension the facets are symmetrical. Using the principles of spinal motion, what is the positional diagnosis?
    a. ERS_R
    b. ERS_L
    c. FRS_R
    d. FRS_L

15. To position the patient above for a direct treatment, you would ideally place the patient:

a. ERS_R
b. ERS_L
c. FRS_R
d. FRS_L

16. To position the patient above for indirect treatment, you would ideally place the patient:
    a. ERS_R
    b. ERS_L
    c. FRS_R
    d. FRS_L

17. You find a patient with type I somatic dysfunction involving five thoracic segments. The best method for treatment of these segments would include:
    a. Treatment of the top two vertebrae
    b. Treatment of the bottom two vertebrae
    c. Look for a type 2 dysfunction elsewhere

18. As you are examining a patient in neutral, the facets are symmetrical. As the patient moves into extension, the left pair of facets are more posterior than the right. During flexion, the facets are symmetrical. Assuming principles of spinal motion are correct, what is the restriction diagnosis?
    a. restricted in ERS_R
    b. restricted in ERS_L
    c. restricted in FRS_R
    d. restricted in FRS_L

Answers:

1. A
2. C
3. A
4. B
5. B
6. A
7. C
8. B
9. B
10. C
11. A
12. B
13. D
14. B
15. C
16. B
17. C
18. A

Chapter 3: The Lumbar Spine

Principles presented in this Chapter:

- Review of Spinal Motion
- Diagnosis using Spinal Mechanics
- Treatment using Spinal Mechanics
- Somatic Dysfunction as an Etiology of Low Back Pain
- Structural Causes of Low Back Pain
- Anatomical versus Pathological Leg Length Discrepancies

Outline:

- Anatomical Etiology of Low Back Pain
- Anatomy of the Lumbar Spine
- Spinal Biomechanics
- Diagnosis of the Lumbar Spine
- Treatment of the Lumbar Spine
    - Direct Treatment of the Lumbar Spine
    - Indirect Treatment of the Lumbar Spine
- Treatment Pearls of the Lumbar Spine
- Dirty Half Dozen of Low Back Pain
    - Muscle Imbalance
    - Anatomical Leg Length Discrepancy
- Clinical Vignette
- Appendix A: Strain-Counterstrain Tender Points
- Appendix B: Standing Postural Study – Orders
- Review Questions

Chapter 3: The Lumbar Spine

The etiology of low back pain (LBP) is multi-factorial. Osteopathic physicians have known for decades that the mind and spirit, in addition to the body, play a large role in physical health and in LBP. In the past twenty years, research has proven that psychological, social, and financial factors play a large role in LBP.[1,2(pp253-275)] Many books are dedicated to the etiology, evaluation, and treatment of LBP well beyond the scope of this text. In this chapter we evaluate structural causes of LBP, demonstrate their indistinctiveness, and set the framework for utilization of manual medicine in the treatment of LBP.

Anatomical Etiology of Low Back Pain

Nociception is the perception of pain: Pain is transmitted almost exclusively by slow-transmitting, unmyelinated A-delta and C afferent nerve fibers. Nociceptive nerve endings originate in the following lumbar structures: facet joint capsules, ligaments and fascia, the outer 1/3 of intervertebral discs, bony vertebrae, dura, nerve root sleeves, and muscle.[3(pp136-137)] The etiology of pain is most easily understood when classified into radicular, or nerve root pain, and non-nerve root pain called somatic pain.[4]

Radicular LBP is typically characterized as severe, unilateral, radiating pain to the lower extremity, foot, and toes along dermatomal distribution, however mild or even absent axially. The generator of less than 5% of LBP,[3(pp25)] it usually arises from discogenic nerve irritation (Figure 1) or spinal stenosis.

Figure 1

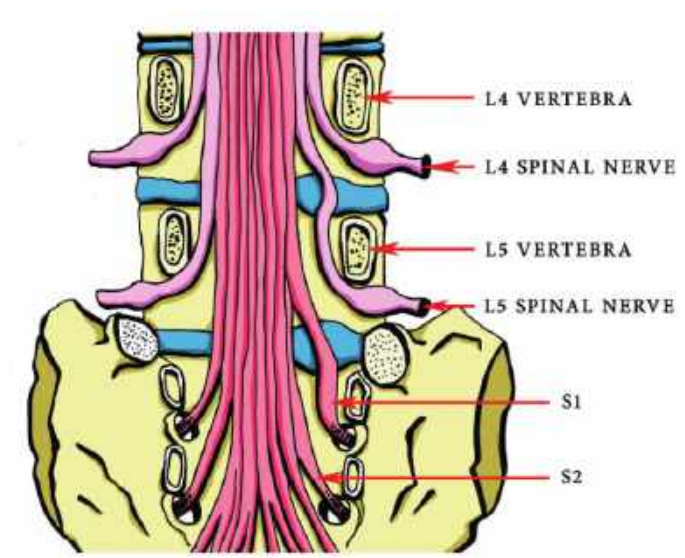


Figure 1: Bulging Lumbar Disc

Note that the L5-S1 disc bulges on the right S1 nerve root.

A patient may have peripheral neurological changes such as hyporeflexia, muscle weakness, and sensory paresthesia described as "numbness," "pins-and needles," or a "burning" sensation. Radicular symptoms usually manifest with multiple clinical findings in the involved nerve root. For instance, compression of the S1 nerve root may result in 0/4-1/4 Achilles tendon reflex, paresthesia along the posterior thigh and leg into the lateral foot, and motor weakness when walking on toes (Figure 2).

Figure 2

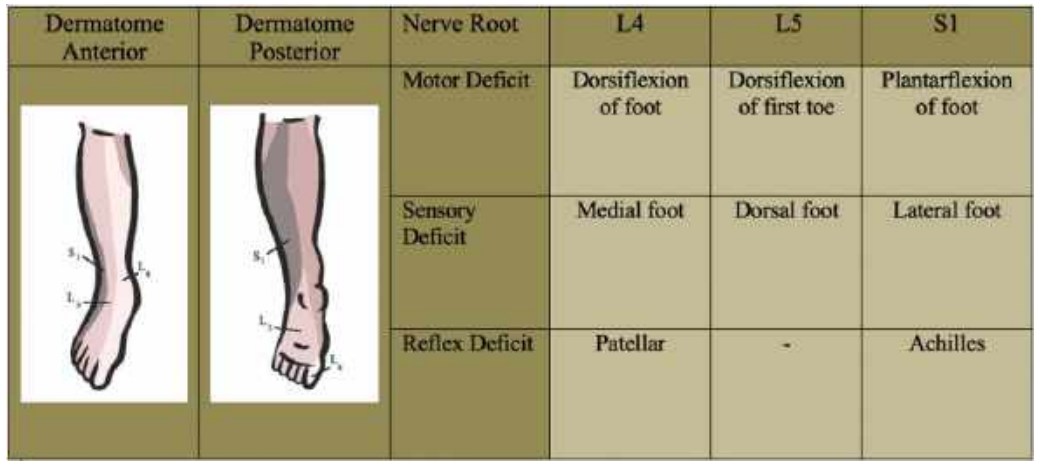


Figure 2: Neurological Findings of L4-S1 Nerve Root Compression

In severe situations, the patient may exhibit urinary retention and loss of anal sphincter control as in filum terminalis compression or cauda equina syndrome. As described in the basic principles chapter, symptoms of nerve root compression constitute a red-flag to the provider; an anatomical survey using radiological imaging, and a search for metabolic causes should be pursued prior to performing manual medicine.

Somatic pain is usually due to soft tissue injury of ligaments, fascia, dura, joint capsules, muscles, or tendinous junctions, although it may involve the spinal vertebrae or intervertebral disc.[3(pp140)] As the cause of approximately 95% of organic LBP, somatic LBP primarily localizes to the lumbar spine with referral to the buttocks or thigh, but does not extend distally. Its pain is described as an "ache" or "stiffness," with a "shooting/stabbing" sensation during certain mechanical movements. Usually, pain is relieved when lying, whereas introduction of motion to the area produces pain. The patient should have less than 30 minutes of morning stiffness.

Radicular Pain

- Mild pain in spinal area affected (cervical spine, thorax, or lumbar)
- Severe pain described as 'burning,' 'pins-and-needles,' or 'numbness' along dermatomal sensory distribution
- May cause peripheral neurological changes such as weakness or hyporeflexia

Somatic Pain

- Moderate to severe pain in spinal area affected
- Pain described as 'aching,' 'deep ache,' or 'stiffness'
- Does not cause hyporeflexia, and does not occur along dermatomal distribution
- Usually does not extend distal to the knee
- May cause weakness/muscle inhibition due to pain

Many researchers have attempted to classify somatic pain based on its quality, location, and referral patterns, giving rise to the terms "sclerotomal" and "myotomal."[5] Sclerotomal pain correlates to skeletal, arthrodial, and ligamentous structures and is described as “deep, dull, and toothache-like.” Myotomal pain refers to pain generated from acute muscle injuries and is later manifested by trigger and tender points. It is described as “achy,” “stiff,” or “crampy.” Both myotomal and sclerotomal pain have referred patterns of pain that are thought to aid in distinguishing a pain generator (Figure 3).

Figure 3

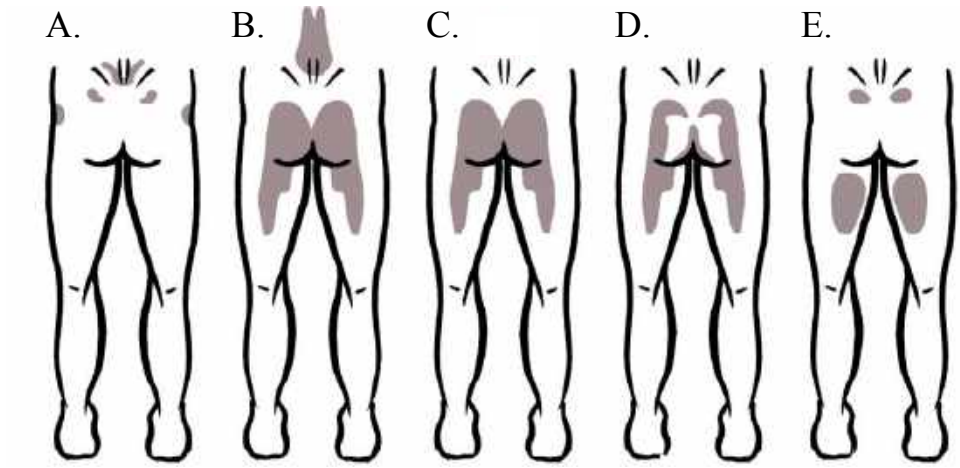


Figure 3: Patterns of Sclerotomal and Myotomal Referred Pain [2(pp551,555), 5(ppS32-S33)]

Figure 3 depicts sclerotomal patterns from the iliolumbar ligament (A), sacrospinous and sacrotuberous ligaments (E) and myotomal referral pattern from trigger points of rotators and multifidi (B), gluteus medius (C), gluteus maximus (D).

Note the referred pain similarities among sclerotomes and myotomes. Also note the referred pain similarity between sclerotomes and myotomes.

Many physicians believe they can discern the precise origin of LBP. However, as Waddell[3(pp139)] pointed out, one third of LBP experts readily admit they cannot, with absolute certainty, identify the source of LBP. When put to the test, the other two-thirds demonstrated that they too, could not identify the cause. Bogduk[4] states that sclerotomal segments overlap and are not consistent among individuals and therefore, are not clinically useful. Since movement of a single vertebral segment causes movement of ligaments, tendons, fascia, dura, joint capsules, and muscle (not to mention, movement of segments above and below), somatic referral caused by deep practitioner palpation may aid little in absolute identification of the pain generator. Superficial trigger or tender points are more easily accessible, and while their TART criteria implicate causality, they may actually represent viscerosomatic reflexes.

What then, is a practitioner of manual medicine to do? Edward Stiles, DO, FAAO, says “Pain [somatic] is the biggest liar. Do not chase pain.” Years of clinical practice have supported this statement; somatic pain is difficult to quantify, describe, and localize.

As described in the basic principles chapter, manual medicine is directed at the restoration of normal range of motion (ROM) and ultimately health. Also, as health care providers, we are mandated to "do no harm." Understanding both the limitations we face as medical providers evaluating somatic pain **and** the necessity to rule out potentially harmful causes of pain prior to prescribing manual medicine, the following principles have been formulated (see Figure 4 for flow chart):

1. Rule out red flags and contraindications to manual medicine, and in doing so, rule out radicular pain.
2. Do not chase pain. This does not negate the importance of patient history or patient drawn pain diagrams on H&P questionnaires. Pain, as communicated by patients, may be the result of a structural abnormality at a visceral or distal site, and in its absence, post-treatment may indicate treatment efficacy.
3. If pain is somatic in nature and contains TART criteria, use manual medicine to restore symmetry, function, and ROM.
4. Re-evaluate the patient at intervals of four to seven days. Reassess the etiology should red flags appear between sessions or after four to six weeks if functional improvement falters or somatic pain persists.
5. With the exclusion of red flags and contraindications to manual medicine, radiological studies are usually not necessary when somatic pain is present for less than four to six weeks.

Figure 4

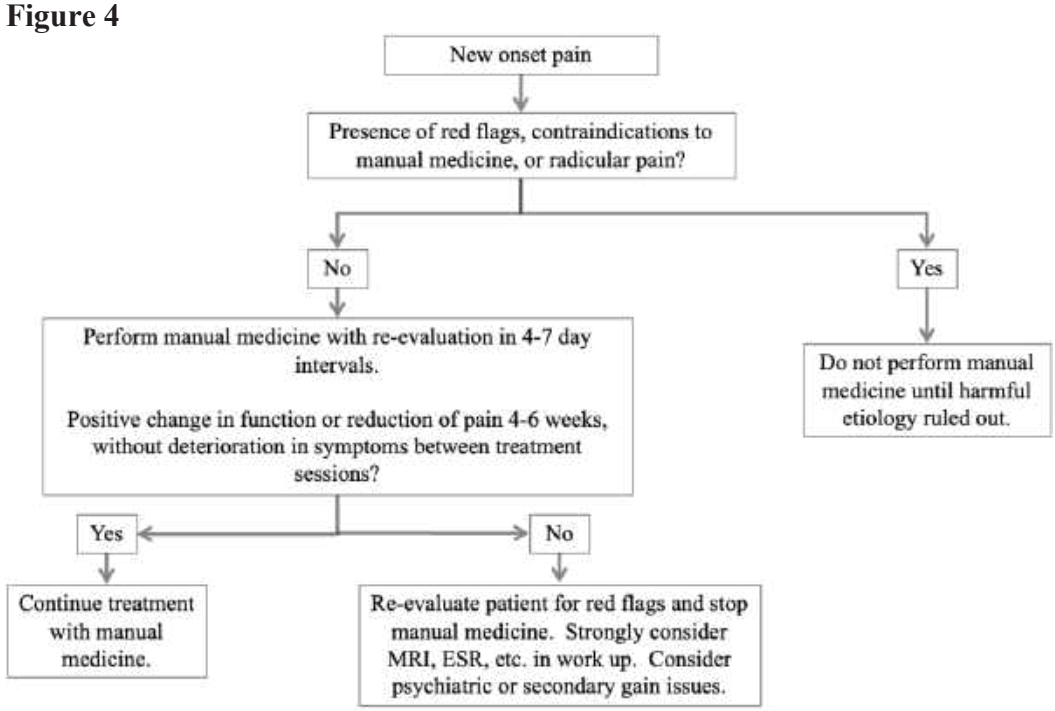


Figure 4: Pain Evaluation Flow Chart for Use of Manual Medicine
Although introduced for LBP in this chapter, this flow chart may be applied to other areas of the body as well.

Acute LBP: pain lasting less than 12 weeks.

Chronic LBP: pain lasting greater than 12 weeks.

Epidemiological studies have shown that the etiology of somatic spinal pain remains undiagnosed in 85% of cases.[6] If pain persists greater than three months, it is termed chronic and neuro-physiologically mediated and is

considered by many as a disease state.[2(pp253)] If at three months' time, the etiology cannot be determined by MRI or laboratory workup, referral for discography, radiologically guided anesthetic injections, or provocation tests may aid in determining etiology. Consideration should also be given for causes such as mental illness, secondary gain, and visceral etiologies. As sclerotomal and myotomal referred pain patterns and descriptions of somatic LBP are often not definitive in its diagnosis, we recommend evaluating lumbar spinal anatomy as it pertains to the diagnosis of somatic dysfunction.

Anatomy of the Lumbar Spine

The Anatomy of the lumbar spine is similar to that of the thoracic spine with the exception of increased vertebral body mass and facet direction. The bigger mass is necessary to carry the columnar weight of the upper body transmitted into the spine. Figure 5 displays bony structures necessary for diagnosing spinal mechanics.

Figure 5

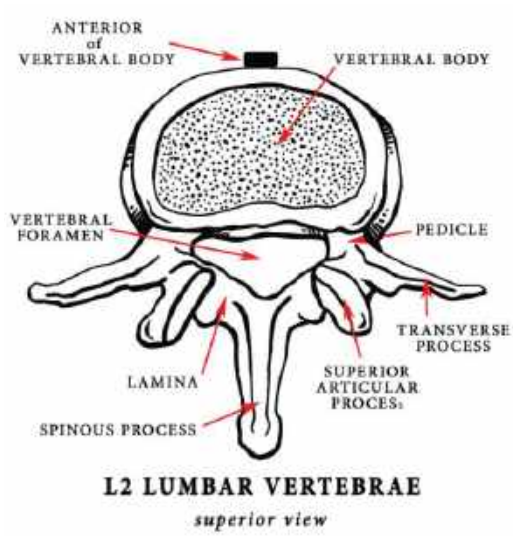


Figure 5: Lumbar Spinal Segment Anatomy
The anterior-superior vertebral body is the reference point of location for the vertebrae. Position of the vertebral body in space is most easily palpated using the transverse processes and the articular facets.

The lumbar spine's predominate motion occurs in the sagittal plane, as is evident by the vertical facing facets. As in the thoracic and cervical spines, the anterior-superior aspect of the vertebral body is the reference for positional naming. Transverse processes and the superior and inferior articular processes are used in diagnosis; joint play confirms the operator's hand placement on these structures. Due to the large muscularity of the lumbar muscles, the spinous processes are often difficult to palpate.

Spinal Biomechanics

As described in the thoracic spine chapter, for the better part of a century the principles of spinal motion have been central to understanding the diagnosis of somatic dysfunction. However, research has led to a more practical utilization

of spinal position palpatory findings that we now further describe. In review, the principles of spinal motion are:

Principle I: Neutral sidebending produces rotation to the side opposite of the side bent. This first principle is also called type I spinal motion.

Principle II: Non-neutral rotation goes to the same direction as the sidebending. The second principle is commonly referred to as type II spinal motion.

Principle III: The introduction of motion to a spinal joint in one plane automatically reduces its mobility in the other two planes. This principle is also known as type III spinal motion.

Before proceeding, it is important to point out a common misnomer in biomechanics. Practitioners will refer to type I spinal motion and contralateral coupling as if they are the same. They also refer to type II spinal motion and ipsilateral coupling interchangeably, which is also incorrect.

Type I motion refers to opposite side-bending and rotation while in neutral. Contralateral spinal coupling refers to opposite side-bending and rotation regardless of sagittal compartment. Likewise, type II spinal motion refers to ipsilateral coupled behavior in a non-neutral position. While the principles of spinal motion have been a good place to start, they do not tell the whole story.

Inherently, the third principle is irrefutable and easily demonstrated in clinical practice. We already know that type I and II motion do not hold true for the cervical spine,[7] and that although they may appear consistent, they have not undergone scientific scrutiny in the thoracic spine. In the lumbar spine, coupling biomechanics has been questioned.[8,9,10] To circumvent the coupling biomechanics controversy in flexion and extension compartments, it is practical to diagnose each of the three components separately as described in following section.

Diagnosis of the Lumbar Spine

In this section, we aim to demonstrate active (patient) diagnosis of lumbar somatic dysfunction without utilizing the principles of spinal motion. The first step is to identify segmental dysfunction using gross palpation as in this example of L3. The practitioner evaluates the inferior facet joints of L3 with their thumbs as the patient actively moves through neutral, flexion, and extension, monitoring for segmental rotation in the sagittal compartments. Ordinarily, rotation should be absent. Asymmetrical rotation, for example during extension, indicates *L3 flexion somatic dysfunction* (Figure 6a).

Figure 6a

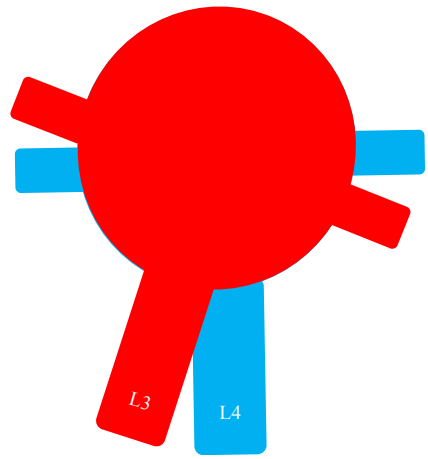


Figure 6a: L3 FRRSR Superior View

Suppose that during extension L3 is rotated right. This would yield the positional diagnosis of *L3 flexed and rotated right* (if we assume validity of the principles of spinal motion, the diagnosis would be *L3 FRRSR*).

Figure 6b

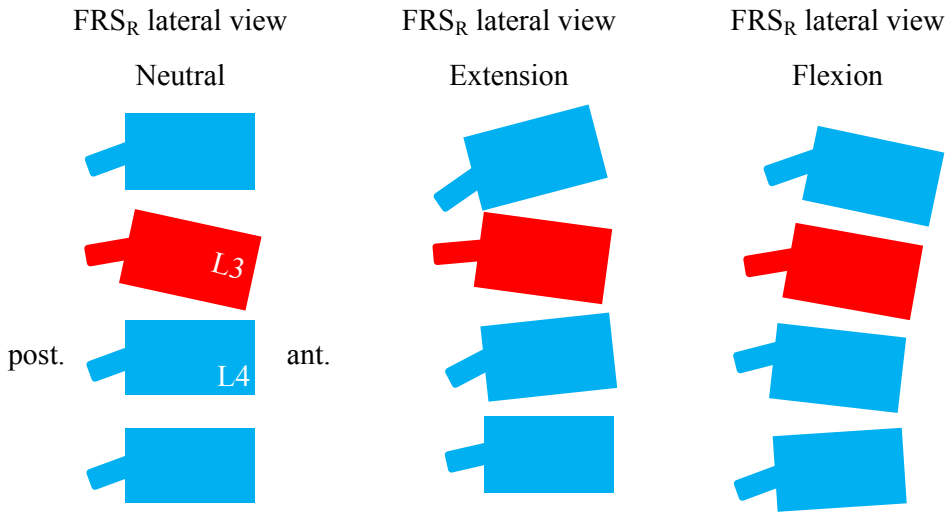
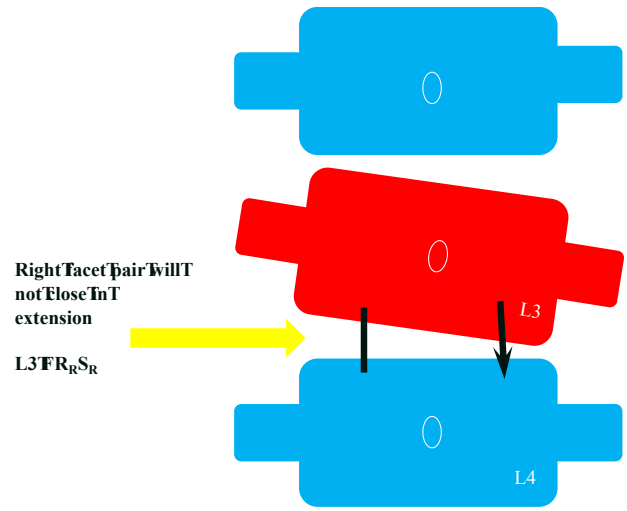


Figure 6b: L3 FRRSR Lateral View

If the patient sidebends right and the segment remains rotated right, the diagnosis is definitively named *L3 FRRSR*. If, however, the patient sidebends left and the segment remains rotated right, the diagnosis is *L3 FRRSL*. As demonstrated, ACTIVE patient side-bending is the additional step that serves to determine segment coupling with known pathologic rotation.

Figure 6c: L3 FR_RS_R Posterior View

Figure 6c



Treatment of the Lumbar Spine

Treatment of the lumbar spine follows the principles of the previous spinal chapter. It is this repetition and consistency that makes the concepts concrete, easy to understand, and simple in application.

Direct Treatment of the Lumbar Spine

Principles of Direct Treatment
- Reverse the positional diagnosis
- Fulcrum
- Lever
- Force

We continue with the example diagnosis L3 FRS_R. To treat this segment directly, just reverse any of the components of the diagnosis; if the segment is maintained in *FRS_R*, place it in *ERS_L*. From here we implement a fulcrum, lever, and force in an attempt to close the facet. Thus, the steps for direct treatment *L3 FRS_R* are:

1. Obtain the positional diagnosis *L3 FRS_R.*
2. Place a fulcrum on L4.
3. Create a lever using the spinous process of L3 or by locking down (extending) the spine to L3.
4. Reverse the components of the positional diagnosis by placing the patient against the barrier in L3 ERS_L.
5. Create a force by using an impulse or isometric contraction.

Refer to the examples provided for high velocity low amplitude (HVLA) and muscle energy (ME) that further delineate these concepts.

Indirect Treatment of the Lumbar Spine

Principles of Indirect Treatment
- Obtain the positional diagnosis
- Place patient in the positional diagnosis
- Exaggerate positional diagnosis

Again, using the positional diagnosis *L3 FRS_R,* treat this segment indirectly and place L3 in FRS_R. To review, the steps for indirect treatment *L3 FRS_R* are:

1. Obtain the positional diagnosis *L3 FRS$_R$*.
2. Place the patient in the position *L3 FRS$_R$*.
3. Exaggerate the extension using left sidebending and left rotation to a position of three-dimensional balance or patient comfort.

Follow these steps as a basis for indirect treatment of the lumbar spine: Examples of indirect procedures are provided for strain-counterstrain (S/CS) and Functional Procedures in following sections.

Treatment Pearls of the Lumbar Spine

When faced with diagnoses in multiple areas of the body or in a single region, it is difficult to determine where to begin treatment. In chapter 9, "Sequencing," we present Edward Stiles' approach to starting a manual medicine treatment (MM/T) treatment. With regard to persistent LBP, Phillip Greenman, DO, has provided six common somatic dysfunction diagnoses known as the "dirty half-dozen."

These diagnoses generate pain and should be either evaluated and dealt with or eliminated as somatic dysfunction diagnoses.[11] However, first we present some insights taught by our mentors that greatly enhance the effectiveness of our MM/T procedures and are offered here as lumbar spine treatment pearls that take into account the perspective on practical spinal biomechanics:

- Contralaterally coupled dysfunctions are usually compensatory; if found, look for and treat ipsilaterally coupled dysfunctions. Also evaluate for quadratus lumborum, ilio-psoas spasm, and leg length inequality as the cause of contralaterally coupled dysfunction.
- Recurrent FRS dysfunctions of the lumbar spine are due to ilio-psoas muscle spasm.
- Typically, contralaterally coupled dysfunctions occur in groups of three to five segments. Treat the center segment to resolve the whole group.
- The lumbar spine has a natural lordotic curve or extension. Flexed segments within this curve reduce lordosis and are highly pathologic.
- For best results during direct treatment, combine rotation and sidebending with flexion, extension, and neutral to reverse all three components of the pathological diagnosis.
- Place the spinal segment treated at the apex of the side-bending, flexion, and extension curves.
- Treat using direct methods first and **then** indirect to integrate the dysfunctional segment with surrounding soft tissues.

Dirty Half-Dozen of LBP

- Innominate Shear SD
- Pubic Shear SD
- Posterior Sacral SD
  - Unilateral Extension
  - L/R torsion
  - R/L torsion
- Type II Lumbar SD
- Short Leg Syndrome/Unlevel Sacral Base
- Muscle Imbalance

SD = Somatic Dysfunction

The Dirty Half-Dozen of LBP

In 1996 Greenman published a study[11] focusing on LBP in 183 subjects for an average of 31 months. In his study, Greenman identified and treated six common and highly pathologic causes of somatic dysfunction termed "the dirty half dozen:"

- Innominate Shear (cephalad only, caudad commonly and spontaneously correct when standing)
- Pubic Shear (cephalad and caudad)
- Posterior Sacrum (unilateral extension, left-on-right (L/R) torsion, right-on-left (R/L) torsion)
- Type II Lumbar Somatic Dysfunction
- Short Leg Syndrome/Unlevel Sacral Base (Anatomical Leg Length Discrepancy)
- Muscle Imbalance (primarily of the lower extremities)

The results of the study show that after treatment, an astonishing 75% of subjects returned to work or the normal activity of daily living.

In the pelvis chapter we detail the causes and pathology of innominate shears, pubic shears, and posterior sacral dysfunctions. In this chapter, we have identified non-neutral somatic dysfunction of the lumbar spine. The remainder of chapter 3 focuses on the other two common causes of chronic LBP: muscle imbalance and anatomical leg length discrepancy.

Muscle Imbalance

Muscle imbalance is a concept originally developed by the late Vladimir Janda, MD. Trained as a neurologist and physiatrist, Janda discovered patterns of agonist-antagonist muscles throughout the body. Muscles that tended to spasm and shorten when stressed he termed tonic. Those that tended to weaken or inhibit he named phasic. He named predictable tonic and phasic muscle patterns in the thoraco-cervical region "upper-cross syndrome," and those in the lumbo-pelvic region "lower-cross syndrome."

Janda treated muscle imbalance primarily by using exercise prescription. In Greenman's dirty half-dozen, muscle imbalance was the number one cause of chronic LBP, occurring in 90% of subjects. This section does not pay full respect to Janda, or his life's work, but briefly summarizes the concept of muscle imbalance. For a full description, please refer to Janda's work or a course in exercise prescription.[12]

Anatomical Leg Length Discrepancy

The subject of leg length discrepancy and its causes, diagnosis, and treatment are well studied but inconsistently managed. In this section we hope to identify different schools of thought and present the "short leg syndrome" as it correlates with scoliosis and LBP.

In the sacrum and pelvis chapter we identify anatomical leg length discrepancy as the result of a physically short leg, whereas, functional leg length discrepancy is due to somatic dysfunction. Causes of functional leg length discrepancy include sacral, innominate, and pubic somatic dysfunction, as well as muscle imbalance or spasm. Assuming we can correct functional leg length discrepancies with manual medicine, it is important to rule out potentially correctable causes prior to pursuing anatomical causes. While no absolute method of measuring functional leg length has been established, both prone and supine methods are performed by comparing the most distal portion of the medial malleolus. Since either measurement method should improve patient outcome, recheck the patient using the same initial method. When repeated attempts to correct functional leg length inequalities are unsuccessful, anatomical leg length should be considered; functional etiologies may represent compensatory patterns for a physically short leg. Sometimes patients have anatomical and physiological leg length discrepancies on the same side, thus presenting a clinical conundrum.

Using x-ray methods, the prevalence of anatomical leg length discrepancies has been documented at ~ 5.2mm in 90% of the population.[13] Radiographic measurements from the ASIS to the lateral or medial malleolus, as well as slit scanograms, have both been used to assess anatomical leg length.[14] No gold standard exists for measuring leg length, and when treating LBP, leg length measurement is often the prime determinant for pelvic un-leveling. Osteopathic practitioners typically do not focus on gross leg length and instead concentrate on leveling the sacral base against gravity.

Leveling the sacral base allows for perpendicular seating of the lumbar spine, and research shows it reduces lumbar scoliosis.[15] In conjunction with either a level on the radiographic cassette or a plum line running through the film (Figure 7), the stratum-of-eburnation is generally used as the radiographic horizontal landmark of the sacrum. The margin of error using the sacral base radiographically is 1-1.5mm.[16]

To level it to gravity, a heel lift is ultimately placed under the side of the low sacral base. Rarely, a patient will have asymmetrical innominates. In this instance, the patient uses an ischial tuberosity lift equivalent to the degree of un-leveling of the sacral base while seated and a heel lift while standing.

Anatomical leg length discrepancy: due to a physically short leg

Functional leg length discrepancy: due to somatic dysfunction

Etiology of Functional Leg Length Discrepancy:

- Sacral somatic dysfunction
- Innominate somatic dysfunction
- Pubic somatic dysfunction
- Muscle imbalance or spasm

Measurement of gross functional leg length is done either prone or supine. After performing manual medicine, recheck for improvement of leg length.

Figure 7: Depiction of Sacral Base Unleveling due to Anatomical Leg Length Discrepancy

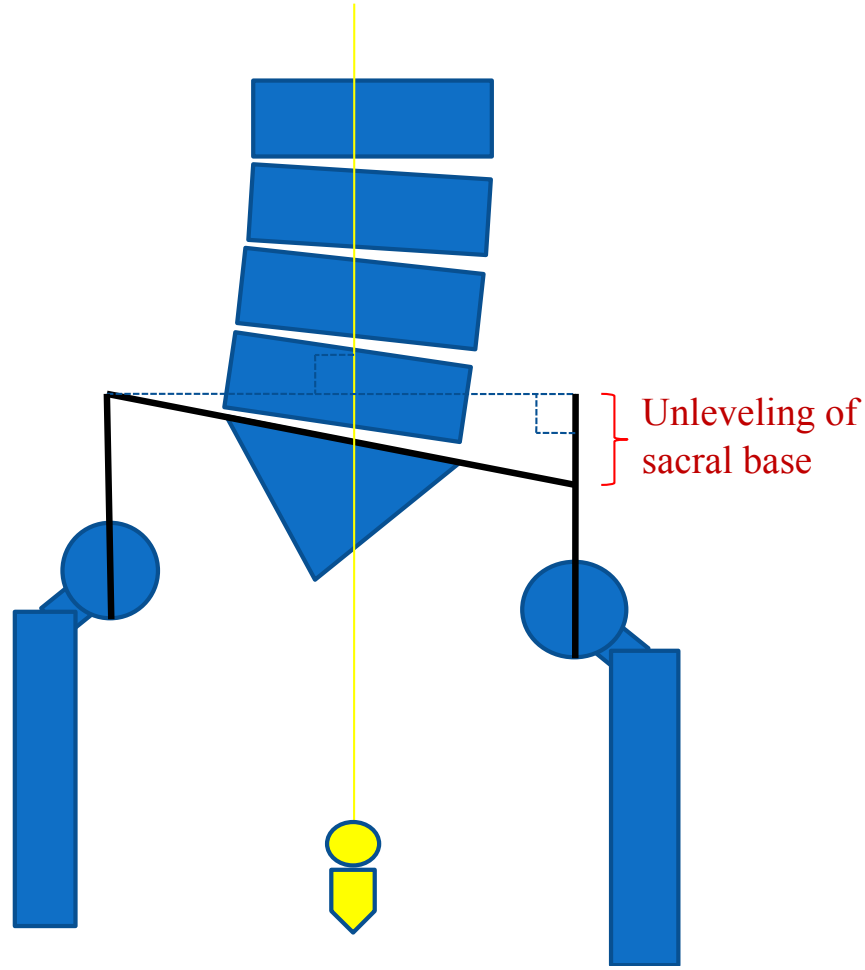
This diagram reproduces key elements found on a standing postural radiograph.[15]

A plum line, hung from the ceiling, runs through the center of the radiographic cassette; a line is drawn through the sacral stratum-of-eburnation. Lines are drawn through the center of the acetabular heads parallel to the plum line. The anatomical leg length difference is noted as the unleveling of the sacral base; thus, a heel lift would be placed on the right side of the diagram.

During the initial evaluation radiograph, the patient should be on a leveled surface and barefooted.

Please refer to the order sheet at the end of this chapter for instructions to provide when ordering this film series.

Figure 7



Using the sacral base to level the pelvis affords several advantages; it reduces both the cost of film and the amount of radiation exposure during evaluation. Further, it positions the patient in a standing rather than a prone position for radiographs, ultimately replicating the position where lift therapy is beneficial. This method also allows for reevaluation with the heel lift in use.

While virtually all authorities agree that an anatomically short leg contributes to LBP, differences in opinion exist regarding the quantity of leg length difference needed to initiate lift therapy. Waddel and other authorities believe that discrepancies less than 20mm do not require therapy.[3(pp271), 13] Most osteopathic authorities note that with a positive history of LBP, discrepancies ≥ 4mm require therapy.[17(983-997)] With refractory low back, pelvic, thoracic, or cervical pain, we begin therapy for anatomical leg length differences ≥ 4mm. This choice, while not evidenced based, eliminates one of the dirty half dozen as the etiology of pain: either the patient's function and pain will respond faorably to the lift, or they will not.

Four methods for initiating a lift have been observed in practice:[17(pp987)]

1. Make the entire height adjustment at one time.
2. Begin with 1/2 to 2/3 of the height and advance the lift height every 2-4 weeks as the patient tolerates.
3. Design the lift based on patient characteristics:
    a. Arthritic, elderly, osteoporotic, or currently in significant pain: begin with 1.6mm (1/16") and increase no faster than 1.6mm every two weeks.
    b. Supple spine, or mild to moderate pain: begin at 3.2mm (1/8") and increase by 1.6-3.2mm every two weeks.
    c. Recent loss of leg length such as fracture or hip replacement surgery - replace full length all at one time.
4. Heilg Formula (Figure 8)

Figure 8

$$\text{Initial Lift Height} = \frac{\text{Sacral Base Unleveling (in mm)}}{\text{Duration*} + \text{Compensation**}}$$

Figure 8: Heilg Formula

Duration* of pain
1= 0-10yrs
2 = 10-30yrs
3 = 30+ years

Compensation **
0 = none observed
1 = observed contralaterally coupled mechanics
2 = vertebral wedging, horizontal osseous development from endplates, or spurring

Since no study has compared these methods, practitioners should individualize lift therapy based on the patient and feedback at subsequent visits.

One additional consideration should be made when using lifts. For leg length discrepancies less than 6.3mm (1/4"), a simple heel lift should suffice. A leg length discrepancy greater than 6.3mm should warrant use of full-length insoles. When the lifts are greater than 12.7mm (1/2"), addition of the total height should be added to the shoe out-sole. The rationale for full-length-insole and out-sole use is the change in foot mechanics with pure heel elevation. Altering foot biomechanics may lead to other somatic dysfunction, tendonitis and tendonosis, or with chronic adaptation of posture, osteoarthritis:

Lift Recommendations:

- 4 - 6mm = heel lift
- 6.3mm - 12.7mm = full-length insoles
- >12.7mm = shoe out-sole addition

Included at the end of this chapter in Appendix B is a sample order for standing postural xrays.

Treatment Examples: Direct Procedures

L3 FRS$_R$ using ME

1. Place a fulcrum on L3 using the thumb and index finger of the right hand: This hand will also monitor the left facet pair closing: Place your left hand on the patient's left shoulder.

2. Create a lever by locking down (extending) the spine until motion is felt on the L3 segment above L4: It is ideal to place the dysfunctional segment (L3) at the apex of the curve created by extension.

3. Further reverse the components of the positional diagnosis by side-bending left and rotating left into the position ERS$_L$: Side-bending can be achieved by pushing the L3 right: Once at the closed pack position, slightly release tension to the "feather edge" of the barrier.

4. Create an isometric contraction by having the patient rotate toward the center of their body (as indicated by the red arrow) while the operator resists for 3-5 seconds: Have the patient relax: Reposition at the new barrier by taking up the slack in flexion, left side-bending, or left rotation: Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

L3 FRS$_R$ using HVLA (Lumbar Roll)

1. Place the patient in a right lateral recumbent position with the left side up: Locate L4 and monitor it.
2. Pull the patient's right arm cephlad and place it under their head, creating type II spinal motion until motion is felt just above L4: This locks down the spine to the L3 so that motion only occurs between L3 and L4: Further, this action side-bends the patient left and extends the entire spine, reversing the positional diagnosis into ERS$_L$.

3. Flex and extend the thigh until motion is felt to L4, localizing motion to this segment: This action creates a lever using L4, L5, and the pelvis.

4. Hold L3 with the left hand, and the L4/lever with the right: Have the patient breathe in and out to take up further slack: Feel for the closed-packed position and the direction of free motion into this locked position: Create a rotational impulse using elbow to rotate the lever anteriorly; by maintaining the fulcrum on L3, L4 will rotate right closing the facets:

5. Recheck the original diagnosis to ensure resolution of somatic dysfunction

Treatment Examples: Indirect Procedures

L3 FRSR using Functional Procedures

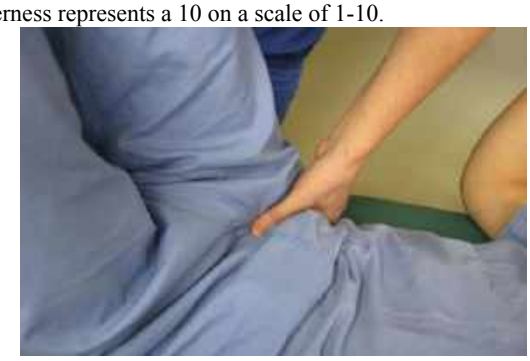
1. Place the thumb and index finger of the left hand on L3: This hand will monitor the L3 as it changes during treatment:

2. Place your left hand and forearm across the patient's shoulders to gain control of the trunk:
3. While sitting behind the patient; introduce flexion, right side-bending and right rotation until to position at L3 FRSR: As in the direct techniques, place L3 at the apex of the curve: Fine tune L3 until it is balanced in three-dimensional space by adjusting flexion-extension, side-bending, and rotation (i.e., find the dynamic balance point, DBP):

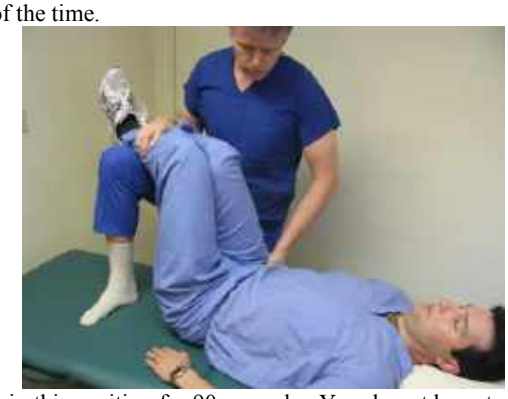
4. Add gentle compression from the left hand, through the thorax toward L3: Monitor the DBP as it changes using the left hand and adjust the flexion-extension, side-bending, and rotation to maintain balance: If the DPB reaches a still point wait for 10-20 seconds; if there is no change, challenge the tissues by either increasing or decreasing compression into L3: When the tissue relaxes the treatment is finished:
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

L3 FRSR using S/CS (aka AL3- Right Tenderpoint)

1. Place the patient in the supine position: Monitor AL3-right with one finger: Confirm that AL3 is tender; tell the patient that this tenderness represents a 10 on a scale of 1-10.



2. Flex the patient's hips until L3 is at the apex of the curve: Flexion will usually reduce the tenderness dramatically.
3. Side-bend and rotate the patient right until tenderness is reduced to at least 3 out of 10; often the patient will ask if you are pushing in the same location or as hard as before: Sometimes to achieve 70% comfort the patient will have to side-bend or rotate left for comfort: These points are called "rogue" points and occur 5 to 10% of the time.



4. Maintain this position for 90 seconds: You do not have to monitor the tender point during this time: If you choose to monitor, you may feel characteristic tissue changes.
5. SLOWLY, reverse side-bending and rotation, and SLOWLY reduce flexion until the patient is fully supine again:
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

References

1. "Evaluation of Low Back Pain in Adults." Accessed July 13, 2016: http:/ww.uptodate.com/contents/evaluation-of-low-back-pain-in-adults.
2. Chila, Anthony G. *Foundations of Osteopathic Medicine.* Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2011.
3. Waddell, Gordon. *The Back Pain Revolution*. Edinburg: Churchill Livingstone, 1998.
4. Bogduk, Nikolai and Lance T. Twomey. *Clinical Anatomy of the Lumbar Spine.* Melbourne: Churchill Livingstone, 1991.
5. Kuchera, Michael L. "Osteopathic Manipulative Medicine Consideration in Patients with Chronic Pain. *Journal of the American Osteopathic Association.* (105 2005).
6. Fritz, Jan, et al. "Management of Chronic Low Back Pain: Rationales, Principles, and Targets of Imaging-guided Spinal Injections." *Radio Graphics* 27, no 6. (11 2007):1751-771. doi:10.1148/rg.276065509.
7. Capobianco, John D., and M. Protopapas. Rivera-Martinez. "Understanding the Combined Motions of the C3/C4 Vertebral Unit: A Further Look at Fryette's Model of Cervical Biomechanics." *American Academy of Osteopathy Journal,* no. 3 (12 2002): 15-30.
8. Gibbons, P., and P. Tehan. "Muscle Energy Concepts and Coupled Motion of the Spine." *Manual Therapy* 3, no. 2 (05 1998): 95-101. doi:10.1016/s1356-689x(98)80025-8.
9. Fujii, Ryutaro, et al: "Kinematics of the Lumbar Spine in Trunk Rotation: In Vivo Three-dimensional Analysis Using Magnetic Resonance Imaging." *European Spine Journal Eur Spine J* 16, no. 11 (06, 2007): 1867-874. doi:10.1007/s00586-007-03730-3.
10. Harrison, Deed E., et al: "Lumbar Coupling During Lateral Translations of the Thoracic Cage Relative to a Fixed Pelvis." *Clinical Biomechanics* 14, no. 10 (12 1999): 704-09. doi:10.1016/s0268-0033(99)00030-3.
11. Greenman, Phillip E: "Syndromes of the Lumbar Spine, Pelvis, and Sacrum." *Physical Medicine Rehabilitation Clinics of North America.* (7 1996):73-85.
12. Page, Phillip, Clare C. Frank, and Robert Lardner: *Assessment and Treatment of Muscle Imbalance: The Janda Approach*: Champaign, IL: Human Kinetics, 2010.
13. Knutson, Gary A. "Anatomic and Functional Leg-length Inequality: A Review and Recommendation for Clinical Decision-making: Part I, Anatomic Leg-length Inequality: Prevalence, Magnitude, Effects and Clinical Significance." *Chiropractic & Osteopathy*, no. 1 (2005): 11: doi:10.1186/1746-1340-13-11.
14. Terry, Michael A., et al. "Measurement Variance in Limb Length Discrepancy." *Journal of Pediatric Orthopaedics* 25, no. 2 (2005): 197-201. doi:10.1097/01.bpo.0000148496.97556.9f.
15. Irvin, Robert E. "Reduction of Lumbar Scoliosis by use of Heel Lift to Level the Sacral Base. *Journal of the American Osteopathic Association*, no. 1 (91 1991): 37-44.
16. Greenman, Phillip E. "Lift Therapy: Use and Abuse." *Journal of the American Osteopathic Association*, no. 4 (79 1979): 238-50.
17. Ward, Robert C. *Foundations for Osteopathic Medicine*. Philadelphia: Lippincott Williams & Wilkins 1998.
18. Jones, Lawrence H., et al: *Strain-counterstrain*: Boise, ID: Jones Strain-Counterstrain, 1995.

Clinical Vignette

Patient Name: CL
June 13, 2020

CC: LBP

HxCC: Patient is a 45year-old female presenting with approximately two months of LBP and right leg weakness. She noted symptoms after a ten-mile bike ride on a road bike in which she hit a hard bump and had a vertical impact of her spine. She states she occasionally experiences pain into her right buttocks, posterior thigh, and posterior leg radiating into her posterior heel. She finds she has tripped due to dragging her foot several times and has modified her gait to avoid this. Currently she is performing yoga, Pilates, biking, and lifting with minimal restrictions. She was seeing a physical therapist that performs manual medicine with decent results; she is still performing exercises directed to her obliques and gluteus muscles. She says she feels stiffness in her midsection that leaves her feeling rigid and stif. Her pain overall is 2-3/10. She had an x-ray of her lumbar spine ten months ago that showed only disc-space narrowing at L5-S1.

ROS:
Constitutional: no mental status changes, no travel or sick contacts
Eyes: no change in vision
Endocrine: no fever, night sweats, weight change
Cardiovascular: no chest pain or palpitations
Respiratory: no dyspnea, cough, or sputum
Gastrointestinal: no nausea, vomiting diarrhea; no change in bowel habit, no blood in stool
Genitourinary: no change in urine, no dysuria, no blood in urine
Neurologic: no loss of consciousness, no seizures
Musculoskeletal: per HxCC, significant for weakness
Psychiatric: no homicidal or suicidal ideations, no signs of depression
Allergy: no angioedema, no urticaria, no anaphylaxis, no symptoms of seasonal allergies

PMHx/PSHx:
1. Cholecystectomy
2. Colonoscopy x 2 which were normal

Allergies:
-no known drug or food allergies

MEDs:
-none

Immunizations:
-managed per her primary care physician

Social:
-no smoking
-no alcohol
-no drugs
-work = nurse

FamHx:
Mom = hypothyroidism, colon cancer
Dad = coronary artery disease

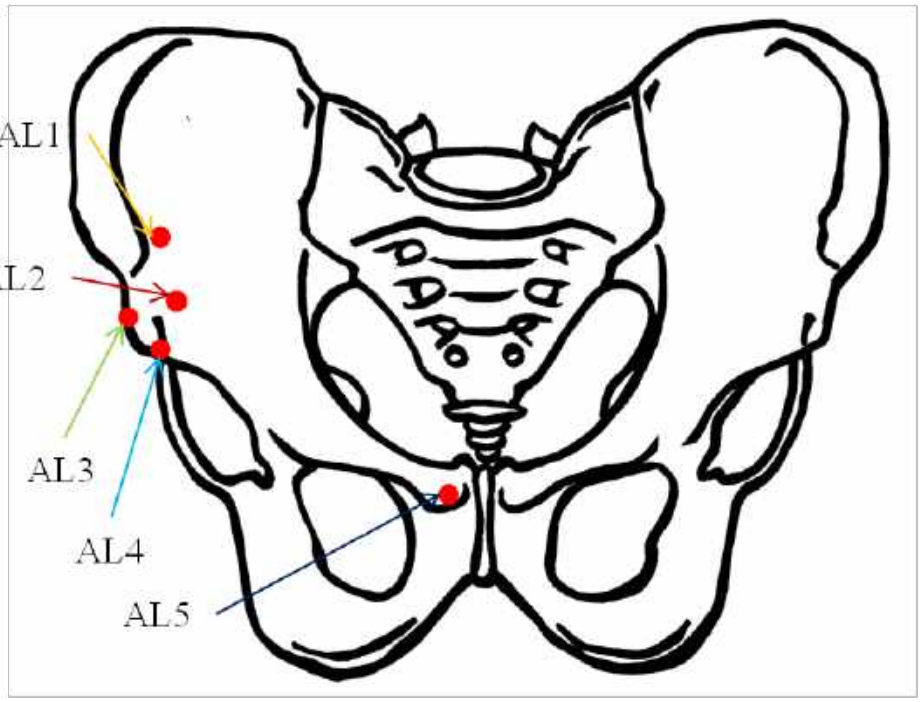
EXAM:
VITALS = 5"6", 134#, 100/66, 78 beats per minute
GEN = awake, alert, oriented to time, place, situation, self, in no acute distress
HEENT = normal cephalic, a-traumatic, extraocular muscles intact, no otic/nasal discharge
NECK = supple, thyroid w/o masses
HEART = regular rate, no clicks/rubs/murmurs
LUNGS = clear to auscultation, without crackles, wheeze, or ronchi
GI = soft, non-tender, no rebound tenderness, +bowel sounds x 4 quadrants, no hepato-spleenomegaly
CV = no jugular venous distention, no carotid bruits, mucus membrane moist, no edema, 2/4 peripheral pulses, no lymphadenopathy
N/M = cranial nerves 2-12 intact, 5/5 global strength except the **patient is unable to walk on right toes… she has weakness in her right calf on plantar flexion of 3/5**. 2/4 global reflexes except **she has 1/4 right achilles reflex**. Cerebellum is intact. Sensations are symmetric in upper and lower extremities. **She has a positive right straight leg raise exactly replicating her radiating pain.**
Biomechanical Exam = pertinent findings include - **Crest height on the right is elevated compared to the left by ~0.5cm in seated and standing position.**

A/P

1. Low Back Pain
2. Somatic Dysfunction of Lumbar and Pelvis
3. Radicular symptoms consistent with right S1 nerve root compression
    a. Red flags of weakness, diminished reflex, radicular symptoms below the knee, symptoms greater than one month, and back pain unresponsive to treatment are present; hence, manual medicine should be deferred at this time. As she is having weakness and her injury is 2months old I am concerned of permanent neurological injury if left untreated.
    b. Prescribe steroid taper to decrease inflammation surrounding the S1 nerve root.
    c. I have contacted a local neurosurgeon who has arranged magnetic resonance imaging of her lumbo-sacral area today and will immediately see her at its completion.

Jason Browder, DO

Appendix A: Anterior Lumbar (AL) S/CS Tenderpoints



AL1 = medial to the anterior superior iliac spine*

AL2 = medial to the anterior inferior iliac spine*

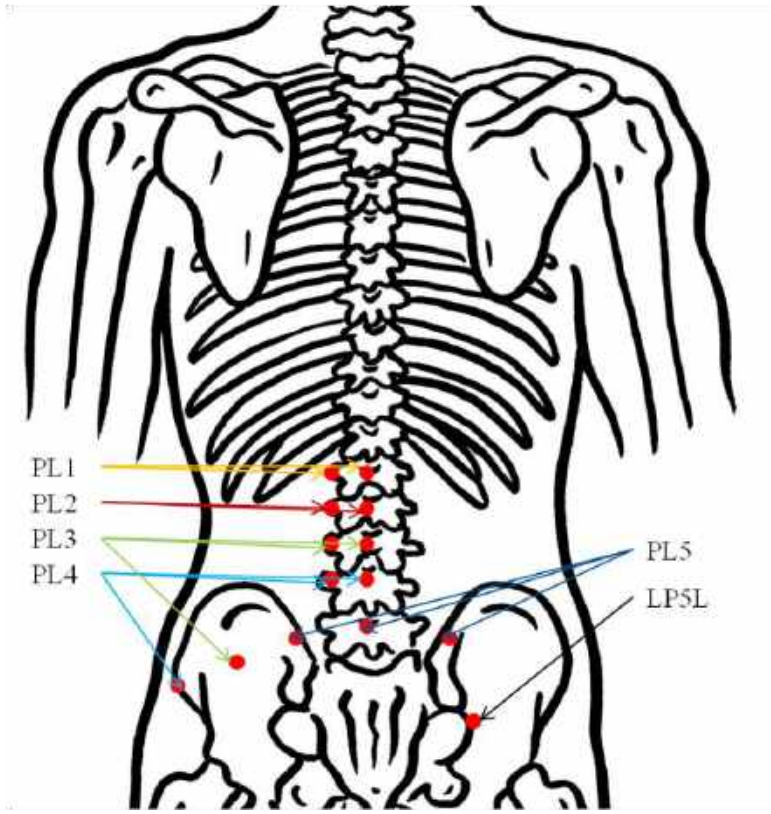
AL3 =lateral to the anterior inferior iliac spine*

AL4 = inferior to the anterior inferior iliac spine*

AL5 = superior aspect of pubic bone medial to symphysis*

*Jones[18(pp73)]

Appendix A: Posterior Lumbar (PL) S/CS Tender Points



PL1 = tip of spinous process of L1 on each side, and posterior transverse process of L1 bilaterally*

PL2 = tip of spinous process of L2 on each side, and posterior transverse process of L2 bilaterally*

PL3 = tip of spinous process of L3 on each side, posterior transverse process of L3 bilaterally, and half way between pelvic points of PL4 and PL5*

PL4 = tip of spinous process of L4 on each side, posterior transverse process of L4 bilaterally, and the posterior ilia behind the inferior edge of the anterior superior iliac crest*

PL5 = tip of spinous process of L5 on each side, and superior-medial edge of the posterior superior iliac spine bilaterally*

LP5L (Lower Pole 5th Lumbar) = approximately 1" below the posterior superior iliac spine*

*Jones[18(pp70)]

Appendix B: Standing Postural Study – Orders

Diagnosis:

- ☐ Back Pain (ICD-10 Code M54.5)
- ☐ Leg Length Discrepancy, congenital (ICD-10 Code M26.55)
- ☐ Leg Length Discrepancy, acquired (ICD-10 Code M21.759)
- ☐ Other_____

Protocol:

☐ Initial (Three Views) Please do not use a pelvic shield

X-rays are taken with the patient standing, knees locked, shoes off, feet places symmetrically, on a grid with heels directly under the femoral heads. A vertical plumb line (for measurements) is in front of the buck and behind the patient, centered midway between the feet. The patient stands with weight equally distributed on both feet and just in front of the wall bucky, being careful not to touch or distort the plumb line.

1. **A-P View**- Thoraco-Lumbar Spine including T-spine and upper Lumbar vertebrae to evaluate scoliosis. (The central beam is positioned at the xiphoid process or approximately T9)
2. **A-P View**- Pelvis and Lumbar spine, including symphysis, trochanters, and most of the lumbar vertebrae to evaluate leg length and sacral base level/un-leveling. (The central beam is positioned at the level of the iliac crest).
3. **Lateral View**- Pelvis including Sacrum, pubis symphysis, lumbo-sacral junction and lumbar vertebrae to include at least all of L3 for sacral index and to evaluate possible spondylolisthesis, arthritic changes, etc.

☐ Follow-up (single view) Please do not use a pelvic shield

X-ray is taken in the same position as the initial study, with the patient standing, knees locked, feet placed symmetrically on a grid with mid heel directly under the femoral heads, **EXCEPT with shoes on and heel lift/arch supports in place.**

1. **A-P View**- Pelvis and Lumbar spine, including symphysis, trochanters, and most of the lumbar vertebrae to evaluate status of scoliosis, leg length, sacral base level, and effectiveness of lift therapy. (The central beam is positioned at the level of the iliac crest).

Review Questions

1. While seated in neutral, a patient's L2-5 segments have posterior right transverse processes. When still in neutral, these segments bend easily left. The diagnosis is:
    a. Type I motion, L2-5 rotated right, sidebent left
    b. Ipsilaterally coupled motion, L2-5 rotated right, sidebent right
    c. Contralaterally coupled motion, L2-5 rotated right, sidebent left
    d. Type II Motion, L2-5 rotated right, sidebent right

2. The major motion of the lumbar spine occurs in which plane?
    a. Coronal
    b. Sagittal
    c. Transverse
    d. Midsagittal

3. The iliac crest is located at the same level as which lumbar segment?
    a. L1
    b. L2
    c. L3
    d. L4
    e. L5

4. While screening for scoliosis, a patient is noted to have left paraspinal prominence from T12-L4. Assuming integrity of the first and second principles of spinal motion, this most likely represents:
    a. Left sidebending with left rotation
    b. Left sidebending with right rotation
    c. Right sidebending with left rotation
    d. Right sidebending with right rotation

5. What components of a patient history would represent a red flag to treatment of the lumbar spine:
    a. Somatic symptoms from the sacroiliac joint to the thorax
    b. Weight-loss of 2# due to emesis
    c. Muscle atrophy of the thigh for uncertain etiology
    d. Edema overlying the lumbo-sacral junction

6. Which one of the following represents TART criteria:
    a. Subjective tenderness
    b. Congenital bony asymmetry
    c. Active range of motion
    d. Tissue edema

7. While performing HVLA on the lumbar spine, which of the following would be an acceptable lever/fulcrum pairing at L5-S1?
    a. Fulcrum at L5, lever using L5 spinous process

b. Fulcrum at L5, lever using sacrum-innominate-lower extremity
c. Fulcrum at L5, lever using the entire spine
d. Fulcrum at S1, lever using L4 transverse process

8. According to the dirty half dozen, as described by Phillip Greenman, DO, the most common cause of LBP is:
    a. Non-neutral lumbar dysfunctions
    b. Pubic shears
    c. Innominate shears
    d. Muscle imbalance
    e. Posterior sacral dysfunction

9. For determining anatomical leg length discrepancy, using standing postural studies, the ideal landmark used is for leveling the sacrum is:
    a. Stratum-of-eburnation
    b. Sacral base
    c. Iliac crest
    d. Superior aspect of femoral head

10. According to this chapter, a full-length shoe insole should be considered with an anatomical leg length discrepancy greater than:
    a. 4.2mm
    b. 6.3mm
    c. 9.6mm
    d. 12.8mm

Answers:

1. A
2. B
3. D
4. C
5. C
6. D
7. B
8. D
9. A
10. B

Chapter 4: The Cervical Spine

Principles Presented in this Chapter:

- Spinal mechanics
- Diagnosis using passive range of motion testing and spinal motion
- Treatment using spinal mechanics

Outline:

- Anatomy of the Cervical Spine
- The Lower Cervical Spine C2-C7
    - Biomechanics of C2-C7
    - Diagnosis of C2-C7
    - Treatment of C2-C7
- The Upper Cervical Spine C1-C2
    - Diagnosis of C1-C2
    - Treatment of C1-C2
- The Upper Cervical Spine C0-C1
    - Diagnosis of C0-C1
    - Treatment of C0-C1
- Treatment Pearls of the Cervical Spine
- Clinical Vignette
- Appendix A: Strain-counterstrain Tenderpoints
- Review Questions

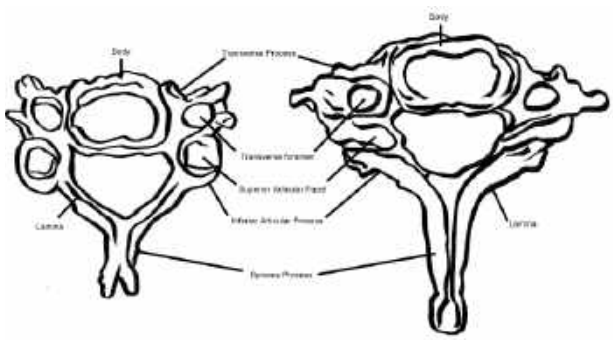
Chapter 4: The Cervical Spine

There are a number of notable similarities and differences between the anatomy and biomechanics of the cervical and thoracic spine. The significant similarities include: spinal mechanics, naming of somatic dysfunction, and many treatment elements. The differences include: rib attachments to thoracic vertebrae and unique upper cervical vertebrae configurations. The primary principle presented in this chapter is identifying the similarities thorough passive, rather than active, range of motion (ROM) testing. Although this chapter appears conceptually simple, it is often difficult for beginning students to appreciate the subtle differences of passive ROM in actual practice.

Anatomy of the Cervical Spine

Anatomically, the lower cervical vertebrae (C2-C7) are similar to the thoracic spine (Figure 1). Notable resemblances include spinous processes, vertebral bodies, and the superior and inferior articular processes. The convex shape of the body allows unlimited directional movement. The transverse process' anterior position limits palpation, and thus the articular facets and lateral condyles (aka articular pillars) offer more suitable surfaces for diagnosing cervical motion. The seventh cervical vertebrae (C7) is the most easily palpable spinous process of the cervical spine, hence its name, "vertebra prominens."

Figure 1



C3 Superior View C7 Superior View

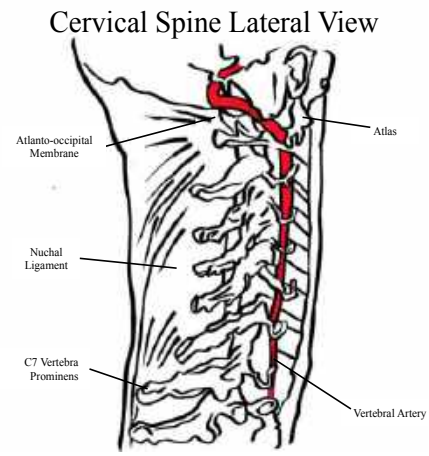


Figure 1: Anatomy of the lower cervical spine

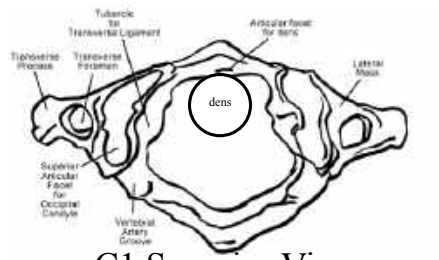
Third cervical vertebrae (C3), seventh cervical vertebrae (C7), and lateral view.

Note the path of the vertebral artery as mentioned in the basic principles chapter and its course through the transverse foramen. From the C1 to C6 is an area of potential stenosis (note, it will also traverse C7 in approximately 7% of the population).[1] With extension and rotation of segments C1-3, the vertebral artery may narrow significantly enough on the contralateral side to produce stroke symptoms.[2,3] Additionally, further compression can occur as it traverses the posterior atlanto-occipital membrane on its ascent to the foramen magnum. Figure 2 depicts C1 and C2, more commonly called the atlas and the axis, respectively. Rightly named, the motion between these segments is almost totally rotational around an imaginary vertical axis through the dens.

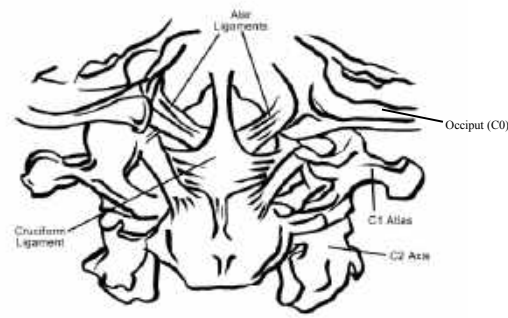
Figure 2

Figure 2: Anatomy of First (C1) and Second (C2) Cervical Vertebrae

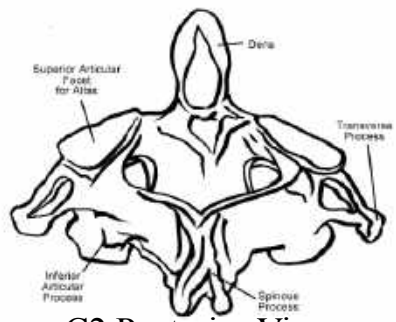
Note the dens is an anterior cervical structure.



C1 Superior View



C1 and C2 Posterior to Anterior (posterior section cut away)



C2 Posterior View

Due to the deep musculature of the cervical spine, the articular facets of C1 are not palpable from the posterior; however, the transverse processes are laterally appreciated behind the angle of the mandible. Notice the convex contour of the superior facets that articulate with the occiput (C0); their shape limits motion of C0 on C1 to anterior and posterior nodding. C2 is palpable from the posterior immediately beneath the occipital prominence. If the operator rotates the head right and left while holding the articular pillars of C2, they should feel C2 begin to rotate at about 45° of head rotation to the right or left. In total, rotation of C1 on C2 is approximately 90° and thus is 50% of total cervical spine rotation.[4,7(pp518)] The cruciform and alar ligaments provide protection for the spinal cord against posterior subluxation of the dens. Laxity in these ligaments, as in rheumatoid arthritis or trisomy 21, may cause permanent neurological injury with cervical flexion.

The most notable difference in the upper and lower cervical segments is the absence of the vertebral body in C1 and C2. Biomechanically, this translates

into motion loss between the occiput and the atlas and the atlas and the axis. Thus, we have separated the upper cervical spine (C0-C2) and the lower (C2-C7) when discussing biomechanics.

The Lower Cervical Spine C2-C7

The lower cervical spine may be diagnosed through active motion testing, as demonstrated in the thoracic spine chapter. Personal experience has taught, however, that passive testing is preferable for several reasons: first, the patient relaxes their cervical musculature more easily when laying, allowing for better palpation, second, during passive testing, the patient is in a direct treatment position when the diagnosis is made. Both of these factors allow for greater efficiency.

Note - diagnosis of the lower cervical spine may be done easily:

1. Through seated ACTIVE testing (extrapolate from prior thoracic and lumbar chapters)
2. Through laying PASSIVE testing (presented in this chapter)

Biomechanics of C2-C7

In the thoracic spine chapter, we discussed how Harrison H. Fryette, DO, predicted that ipsilateral spinal coupling occurred exclusively in the non-neutral positions of flexion and extension.[4] He also predicted that contralateral coupling motion occurred exclusively in neutral positions of the thoracic and lumbar spines, but that it did not occur in the cervical spine. This long-held belief was challenged by John Capobianco, DO, in a well-designed study.[5] In this clinical trial, experienced Osteopathic physicians independently tested sidebending and rotation of C3 in relation to C4. The results of this study concluded that cervical sidebending and rotation occur in ipsilateral as well as contralateral coupled motion, and paradoxically, more frequently in contralateral motion. Other studies have also observed contralateral coupling behavior of the lower cervical spine.[6] Greenman notes that anatomy limits the lower cervical spine to non-neutral somatic dysfunction,[7(pp176)] which we also observe, henceforward, cervical spine somatic dysfunction is discussed as flexed or extended. Using Capobianco's model for independent testing of cervical sidebending and rotation, we introduce passive spinal diagnosis for the lower cervical spine.

Note: in the lower cervical spine there is NO neutral somatic dysfunction. However, we do observe contralateral and ipsilateral coupled somatic dysfunction in flexion and extension,. Test rotation and sidebending independently.

Diagnosis of C2-C7

Supine patient positioning is optimal for passive evaluation of the lower cervical spine. As in Capobianco's study, we diagnose sidebending and rotation separately, a diagnosis most easily done by identifying the rotational component first and sidebending second. Our method of testing is initially performed with the patient in the following manner:

Patient and operator position for passive ROM testing.

Step 1. Test rotation

a. Rotation is evaluated in flexion and extension compartments from C2-C7; somatic dysfunction may or may not be present. In this instance, C3 is pathologically rotated left.

Step 2. If rotational somatic dysfunction is present, test sidebending. For instance, in *C3 rotated left*, the operator then tests sidebending in the non-neutral compartment where limited rotational ROM was identified.

a. In flexion, via LATERAL translation **OR**
b. In extension, through DIAGONAL translation

Throughout the remainder of this section, we demonstrate the diagnosis of somatic dysfunction of C3 in relation to C4 using this methodology.

Lower Cervical Spine – Passive Rotational Diagnosis

With patient in supine position, testing is initially performed by the operator placing the patient in flexion or extension. The operator passively tests the right and left articular pillars by introducing posterior-to-anterior glide of the lower cervical segments. In this example, we use the articular pillars of C3 as they approximate to C4 (Figure 3).

Figure 3

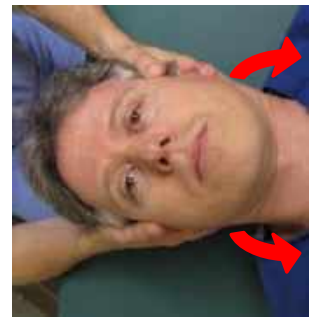


Figure 3: Testing Rotation in the Lower Cervical Spine

The patient is positioned passively in flexion or extension, and the operator translates the articular pillars anteriorly one side at a time.

A restriction in anterior movement of the left pillar of C3, for instance, would indicate left rotation of C3.

Lack of anterior motion on one side indicates restricted range of motion (ROM) and rotation toward the side of restriction.

As discussed in the lumbar spine chapter, flexed somatic dysfunction is noted while the patient is positioned in extension (Figure 6b, Lumbar Spine Chapter). Similarly, if a supine patient is passively moved into flexion and left anterior motion is restricted compared to right, the positional diagnosis would be *C3 Extended and Rotated Left (C3 ER$_L$).*

Lower Cervical Spine – Passive Sidebending Diagnosis

As in rotational testing, the supine position is best utilized for passive evaluation of lower cervical spine sidebending. Continuing with the previous example, while at the patient's head, the practitioner's fingertips are placed on the articular pillars of C3 laterally. While the entire cervical spine is placed in flexion, the practitioner translates medially on the right and then left, of C3. Each side should glide evenly indicating opening of the CONTRALATERAL facets (Figure 4a).

Figure 4a

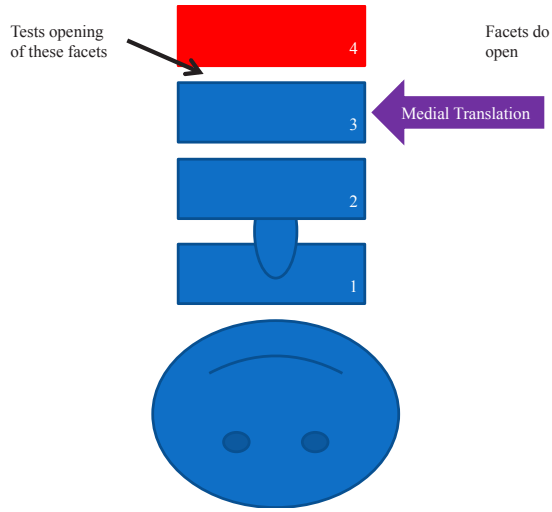
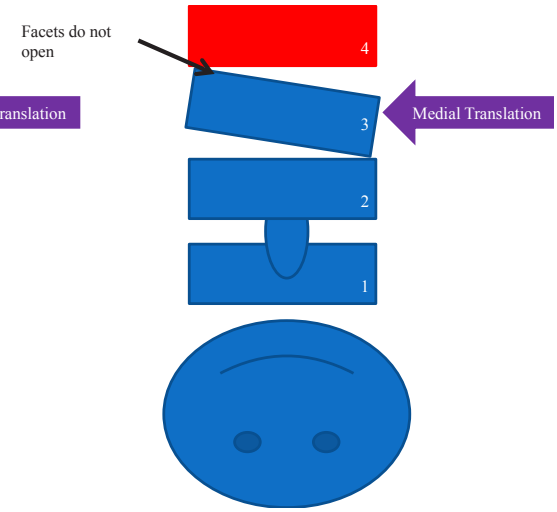


Figure 4b



If for example, there is decreased motion while translating right-to-left on C3, this would indicate that the left facets do not open; thus, the vertebral body is *side-bent left* (Figure 4b). By combining this finding with the previous knowledge that the patient is positioned in flexion and rotated left, a positional diagnosis of *C3 ERLSL* is obtained.

To test side bending of the C3, while the patient is placed in extension the same procedure is performed with one difference: in the coronal plane, translate inferomedially on the right and then left. Each side should glide evenly indicating closing of the IPSILATERAL facets (Figure 5a).

Figure 5a

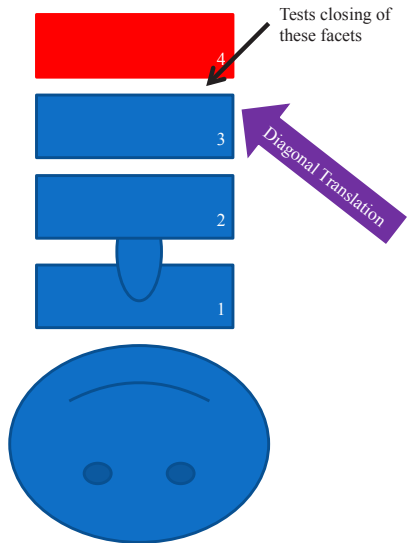
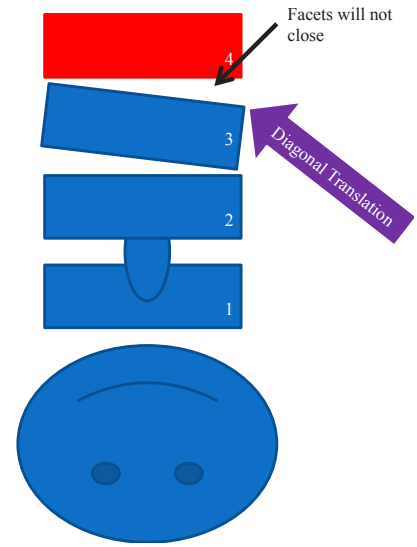


Figure 5b



If for instance, there is decreased motion while translating inferomedially right-to-left on C3 (Figure 5b), this would indicate that the right facets do not close; thus, the vertebral body is *flexed and sidebent left, C3 FSL*. Continuing with this

Figure 4a & 4b: Testing Sidebending - Supine Passive ROM while Patient is Positioned in Flexion

Note in Figure 4a, that joint play (translation) is horizontal along the coronal plane. This tests the contralateral facets for opening while the patient is in flexion. Figure 4b demonstrates translational restriction from right to left, indicating the left facets do not open and that C3 is sidebent left in relation to C4.

With the patient supine, C3 appears sidebent right in the diagram. However, if the patient is moved upright, it is indeed sidebent left (see picture below).

Figure 5a &5b: Testing Sidebending - Supine Passive ROM while Patient is Positioned in Extension

Note that the joint play (translation) is diagonal instead of lateral. This tests the ipsilateral facets for closure while the patient is in extension.

example, if rotational motion is restricted when gliding the left articular pillars anteriorly (also while the patient is passively positioned in extension), the positional diagnosis would be *C3 FRLSL* (Figure 6).

Figure 6

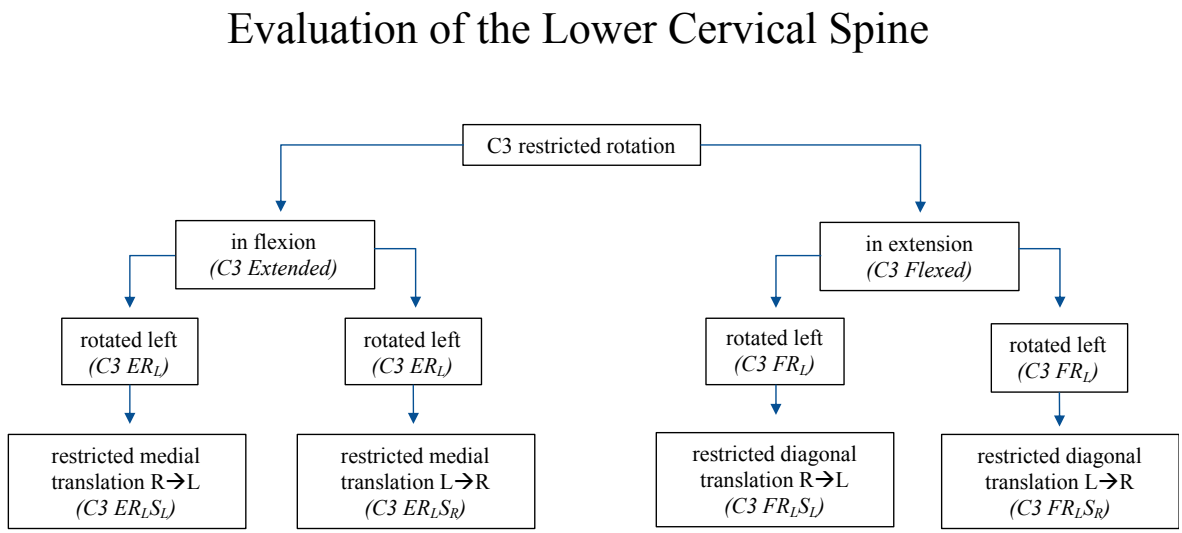


Figure 6: Algorithm for the Diagnosis of the Lower Cervical Spine

It is important to point out that a diagnosis cannot be formed by combining findings (i.e., a pathological left sidebending observed in extension with a pathological rotation found in flexion). Restricted ROM must be found in the same non-neutral compartment to form a diagnosis of somatic dysfunction.

Direct Treatment of C2-C7

The advantage to using passive ROM in diagnosis is that the patient is positioned for direct treatment when the diagnosis is made. Use the example *C3 ERSL* in which the left facet of C3 cannot open in relation to C4. During the diagnosis, the patient is supine, in *flexion, sidebent right, rotated right* with a fulcrum at C4; a lever is formed using C0-C3. Now, the operator need only introduce a force. In review, the steps for direct treatment *C3 ERSL* are:

1. Obtain the positional diagnosis *C3 ERSL.*
2. Place a fulcrum on C4.
3. Create a lever by locking down (flexing) the spine to C3.
4. Reverse the components of the positional diagnosis by placing the patient against the barrier in C3 FRSR (if using passive ROM testing, the patient will already be in this position).
5. Create a force by using an impulse or isometric contraction.

See the examples provided for high velocity low amplitude (HVLA) and muscle energy (ME) that reiterate the principles in this section.

Indirect Treatment of C2-C7

The advantage conferred when using passive ROM testing for direct procedures does not apply to indirect. When using the previous positional diagnosis *C3 ERS*$_L$ in an indirect treatment, just place the patient in *C3 ERS*$_L$. To review, the steps for indirect treatment *C3 ERS*$_L$ are:

1. Obtain the positional diagnosis *C3 ERS*$_L$.
2. Place the patient in the position *C3 ERS*$_L$.
3. Exaggerate the extension, left sidebending and left rotation to a position of three-dimensional balance or patient comfort.

Refer to the strain/counterstrain (S/CS) and Functional procedure examples for subtle differences of each indirect technique. Many times, the patient will have tenderness to palpation on the articular pillar. Reduction of the tenderness will also yield a positional diagnosis.

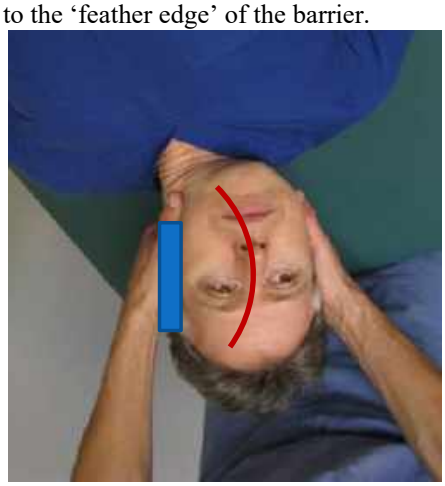
Note - reduction of tenderness will yield a positional diagnosis if treating with S/CS.

Treatment Examples: Direct Procedures

C3 ERS$_L$ using ME

1. If treating from the diagnostic position of passive ROM, the patient is already supine and in cervical spine flexion with right sidebending.
2. Place a fulcrum across the posterior portion of C4 using the index finger of the left hand.

3. Create a lever by flexing the cervical spine until motion is felt in C3.
4. Maintain the right sidebending and rotate the cervical spine right until to a closed pack position of C3-C4. The patient is now at the apex of the curve in C3 FRS$_R$. Slightly release tension to the 'feather edge' of the barrier.



5. Create an isometric contraction by instructing the patient to rotate toward the center of their body (left in this instance) while the operator resists with a blocking force (blue bar) for 3-5 seconds. Have the patient relax. Reposition at the new barrier by taking up the slack in flexion, right sidebending, or right rotation. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

C3 ERS$_L$ using HVLA

1. If treating from the diagnostic position of passive ROM, the patient is already supine and in cervical spine flexion with right sidebending.
2. Place a fulcrum across the posterior portion of C4 using the index finger of the left hand.

3. Create a lever by flexing the cervical spine until motion is felt in C3.
4. Maintain the right sidebending and rotate the cervical spine right until to a closed pack position of C3-C4. The patient is now at the apex of the curve in C3 FRS$_R$.

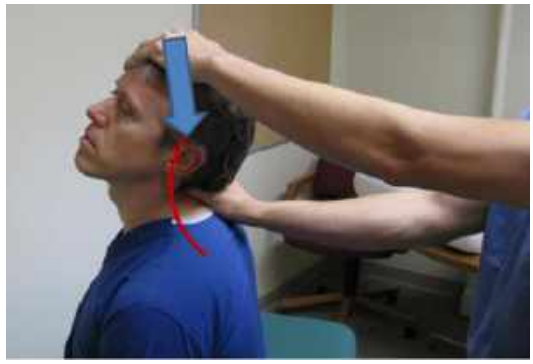
5. Create an impulse by thrusting on the C0-C3 component from the left to right, in an attempt to open the left C3-C4 facets. If the positional diagnosis had been *C3 FRS$_L$*, the patient would be positioned ERS$_R$, and the impulse would have been diagonally toward the C7 spinous process, to close the right C3-C4 facets.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Treatment Examples: Indirect Procedures

C3 ERSL using Functional Procedures

1. Place the thumb and index finger of the right hand on C3. This hand will monitor the C3 as it changes during treatment.

2. Place the left hand on the patient's head.
3. While standing behind the patient, introduce extension, left sidebending and left rotation to position the segment in *C3 ERSL*. As in the direct procedures, place the extension at the apex of the curve. Fine tune C3 until it is balanced in three-dimensional space by adjusting flexion-extension, sidebending, and rotation (the dynamic balance point).

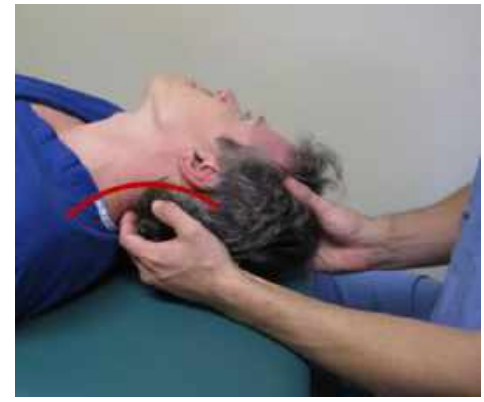


4. Add gentle compression from the left hand, through the head toward C3. Monitor the dynamic balance point (DBP) as it changes using the right hand and adjust the flexion-extension, sidebending, and rotation to maintain balance. If the DPB reaches a still point wait for a 10-20seconds; if there is no change, challenge the tissues by either increasing or decreasing compression into C3. When the tissue relaxes the treatment is finished.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

C3 ERSL using S/CS (aka PC3- left lateral)

1. Place the patient supine on a table. Monitor the C3 tender point on the left with one finger. Confirm that C3 is tender on the left articular pillars; tell the patient that this tenderness represents a 10 on a scale of 1-10.

2. Extend the patient's cervical spine until C3 is at the apex of the curve. Extension will usually reduce the tenderness dramatically.



3. Sidebend and rotate the patient left until tenderness is reduced to approximately 3 out of 10; often the patient will ask if you are pushing in the same location or as hard as before. Sometimes, to achieve 70% comfort, the patient will have to sidebend or rotate right for comfort. These points are called "rogue" points and occur 5 to 10% of the time. **Note that a "rogue point" may also represent a misdiagnosis.**
4. Maintain this position for 90 seconds. You do not have to monitor the tender point during this time. If you choose to monitor, you may feel characteristic tissue changes.
5. SLOWLY, reverse sidebending and rotation, and SLOWLY reduce extension until the patient is supine again.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

The Upper Cervical Spine C1-C2

Diagnosis of C1-C2

Flexing the head/neck to 45° prevents movement below C2, effectively 'locking out' C2, and only allows C1 to rotate on C2.

Absence of a vertebral body in the first and second cervical segments results in more predictable motion than in the lower cervical spine. As mentioned in the anatomy section, the dens limits motion of C1 to right and left rotation in relation to C2. To appreciate this motion, the operator places the pads of their index fingers on the articular pillars of C2; flexion is introduced to the cervical spine and the occiput raised to approximately 45° off the table, or until motion is felt in C2. The head is then rotated left until the operator feels the body of C2 begin to rotate on his index finger; this is the end of C1 rotation on C2. Left rotation is then compared with the right.

Pathology is present if, for instance, rotation to the left is decreased when compared to the right. The positional diagnosis is *C1 rotated right*, since C1 is rotated right in relation to C2, limiting left rotation (Figure 7).

Figure 7a **Figure 7b**

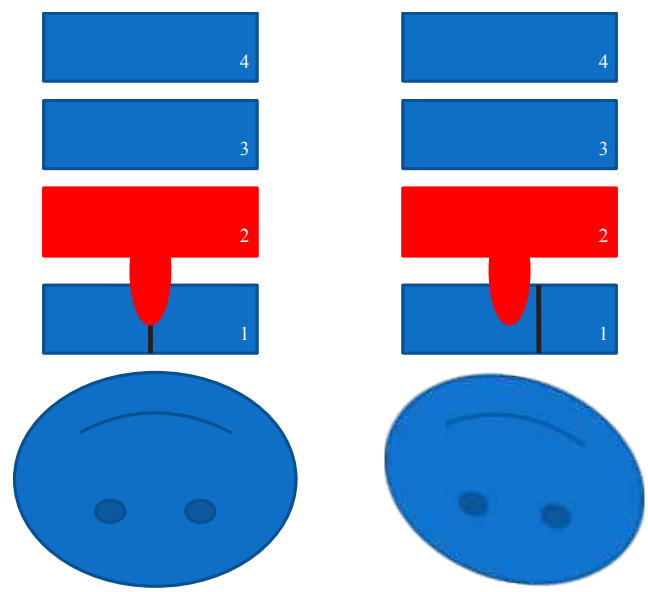


Figure 7a & 7b: C1 Rotated Right

The center of C1 is denoted by the black line and is in the center with the absence of pathology. Note, that when rotated right, the center of C1 is to the right of the dens. Also note how the occiput (C0) is rotated right.

Direct Treatment of C1-C2

Passive ROM used in the diagnosis of C1-C2 provides instant positioning for direct treatment. Again, use the example *C1 rotated right*; the patient is supine, in flexion, with a lever created by C0-C1flexion, and a fulcrum on C2 using both index fingers. The operator need only introduce a force. In review, the steps for direct treatment *C1 rotated right* are:

1. Obtain the positional diagnosis C1 rotated right.
2. Place a fulcrum on C2.
3. Create a lever by flexing the spine to C1, or approximately 45°.

4. Reverse the components of the positional diagnosis by placing the patient against the barrier in C1 rotated left (if using passive ROM testing, the patient will already be in this position).
5. Create a force by using an impulse or isometric contraction.

See the examples provided for HVLA and ME that allow the practitioner to quickly treat the patient.

Indirect Treatment of C1-C2

Indirect treatment of the atlas on the axis requires repositioning after diagnosis, but is not complicated since there is only a rotational component to consider. When using the previous example, *C1 rotated right*, in an indirect treatment, place C1 in right rotation.

1. Obtain the positional diagnosis C1 rotated right.
2. Place the patient in the position C1 rotated right.
3. Exaggerate the right rotation to a position of three-dimensional balance or patient comfort.

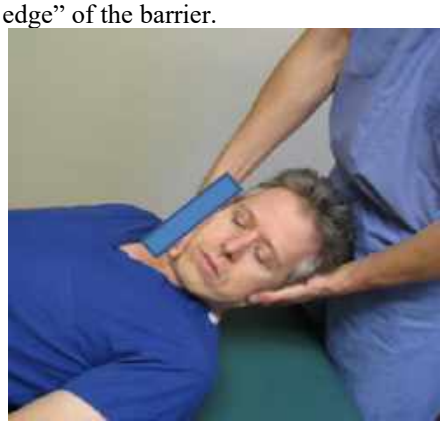
Refer to the S/CS and Functional Procedure examples for the subtle differences of each indirect procedure.

Treatment Examples: Direct Procedures

C1 rotated right using ME

1. If treating from the diagnostic position of passive ROM, the patient is already rotated left and at ~45° flexion.

2. A fulcrum is maintained by the index finger of the left hand on the articular pillar of C2.
3. A short lever is already formed with C0-C1 when the patient is placed to forward flexion. Slightly release tension to the "feather edge" of the barrier.

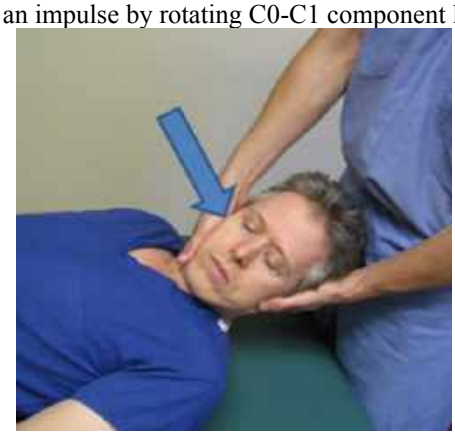


4. Create an isometric contraction by instructing the patient to rotate toward the center of their body (right in this instance) while the operator resists for 3-5 seconds. Have the patient relax. Reposition at the new barrier by taking up the slack in left rotation. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

C1 rotated right using HVLA

1. If treating from the diagnostic position of passive ROM, the patient is already rotated left and at ~45° flexion.

2. A fulcrum is maintained by the index finger of the left hand on the articular pillar of C2.
3. A short lever is already formed with C0-C1 when the patient is placed to forward flexion. Further rotate C0-C1 right to a closed pack position of C1-C2.
4. Create an impulse by rotating C0-C1 component left.



5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

WARNING: HVLA techniques to the upper cervical spine may produce irreversible damage to the vertebral artery and spinal cord. We recommend that HVLA be used as a last resort in C0-C2.

Treatment Examples: Indirect Procedures

C1 rotated right using Functional Procedure

1. With the patient supine, place one finger on the occipital protuberance and one on C2.
2. Place another hand on the patient's forehead.

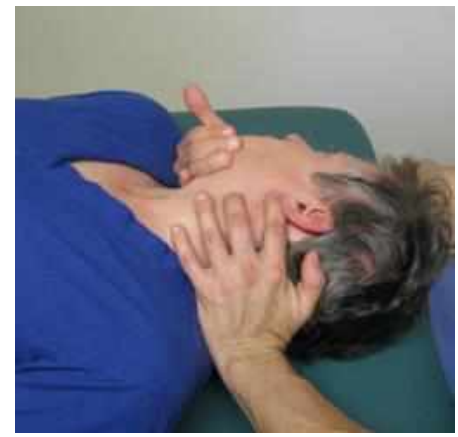
3. Instruct the patient to tuck his chin until the finger on the occiput slides off onto the upper cervical spine. This position overlies the posterior arch of C1 and places it at the apex of the curve.

4. Add gentle compression from the hand on the forehead toward C1-C2. Monitor the DBP at C1-C2 as it changes using the monitoring hand and adjust the flexion-extension, sidebending, and rotation to maintain balance. If the DPB reaches a still point wait for a 10-20 seconds; if there is no change, challenge the tissues by either increasing or decreasing compression into C1. When the tissue relaxes the treatment is finished.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

C1 rotated right using S/CS (Tender Point C1- left)

1. Place the patient supine on a table. Monitor the C1 tender point located at the angle of the mandible on the left transverse process. Confirm that C1 is tender on the left transverse process; tell the patient that this tenderness represents a 10 on a scale of 1-10.

2. Rotate the head right until the tender point is perpendicular to the table, or until tenderness is reduced to a 3 out of 10; often the patient will ask if you are pushing in the same location or as hard as before. Sometimes, to achieve 70% comfort, the operator will need to forcibly rotate the head to the right and hold it rotated right during the treatment.
3. Maintain this position for 90 seconds. You do not have to monitor the tender point during this time. If you choose to monitor, you may feel characteristic tissue changes.



4. SLOWLY, release rotation until the head is centered again.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

The Upper Cervical Spine C0-C1

Diagnosis of C0 on C1

The absence of a vertebral body restricts the motion of the occiput (C0) on C1 to anterior and posterior nodding. With C0 extension, the occiput should move anteriorly on the condyles and posteriorly with flexion (Figure 8).

Figure 8

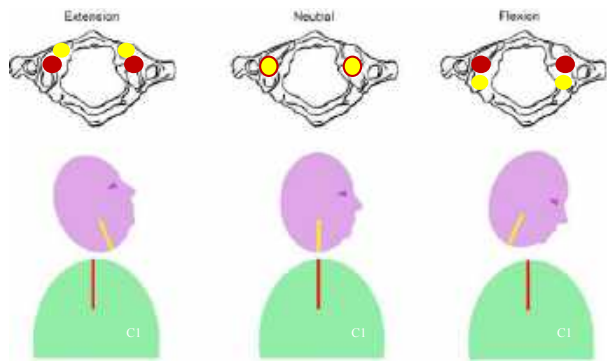


Figure 8: C0-C1

During flexion the occiput moves posteriorly on C1 and anteriorly during extension. The centers of the occipital condyles are marked by yellow and the center of C1 by red.

Although most observed movement is flexion and extension, C0-C1 displays slight contralateral coupling mechanics with sidebending and rotation to opposite sides when the condyles move anterior or posterior.[7(pp175),8(pp518)]

Note: There is NO neutral somatic dysfunction at the C0-C1 level.

Passive ROM testing is performed with the patient supine, hands wresting on the lateral sides of the head with the index fingers at the base of the occiput. Slight flexion is introduced until resistance is met, representing the limit of C0-C1 flexion. The occiput is then translated right and left in the coronal plane. C0-C1 is then placed in extension and translation is retested. Since the facets at C0-C1 do not open and close as in the lower cervical segments, horizontal translation tests CONTRALATERAL sidebending of the occiput in both flexion and extension (Figure 9).

Figure 9

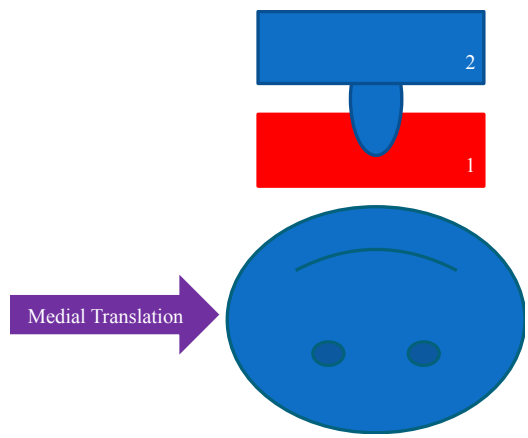


Figure 9: Motion Testing C0-C1

In both flexion and extension, translation from left to right tests sidebending of C0-C1 on the right side. The rotational component is inferred since only contralateral coupled motion occurs between these segments.

Manifested by reduced sidebending, somatic dysfunction is present when one occipital condyle is pathologically anterior or posterior. If for instance, the patient is placed in extension and translation from left to right is restricted, this would indicate left sidebending. This is due to restricted anterior motion of the right condyle during flexion and gives the diagnosis of *C0 FRRSL* (Figure 10).

Figure 10

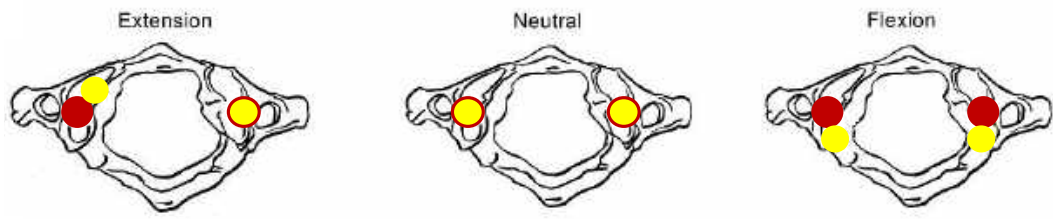


Figure 10: C0 FRRSL

The facets of C1 are denoted by the red circle and the facets of the occiput by the yellow. Note that the right occiput does not glide anteriorly during extension, thus causing right rotation. Since motion of the C0-C1 is contralaterally coupled, the sidebending is presumed left.

Similarly, restriction of the right condyle to posterior movement in flexion results in right occipital rotation with left sidebending. This diagnosis would be *C0 ERLSR*, and would be found with the patient in flexion with left to right translation (Figure 11).

Figure 11

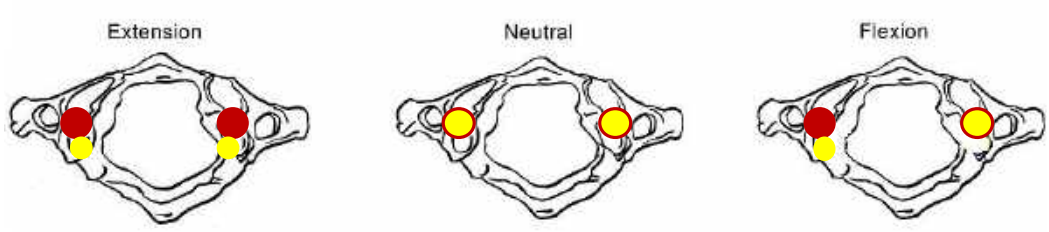


Figure 11: C0 ERLSR

The facets of C1 are denoted by the red circle and the facets of the occiput by the yellow. Note that the right occiput does not glide posteriorly during flexion, thus causing left rotation. Since motion of the C0-C1 is contralaterally coupled, the sidebending is presumed to be right.

Treatment of C0-C1

The treatment principle of the occiput on the atlas is identical to that of the lower cervical spine. Once restricted range of motion is identified, the patient is in a position for a direct procedure. Indirect procedures require treatment beginning in the location of their positional diagnosis. Due to the risk of vertebral artery and spinal cord damage, caution must be exercised when using HVLA techniques in the upper cervical spine. For a review of the principles, please see the lower cervical spine treatment section.

Cervical Spine Treatment Pearls

With regard to the cervical spine, clinical practice has provided treatment "pearls" to aid in determining key areas of somatic dysfunction. They are listed below:

- The cervical spine has a natural lordosis or extension curve. Flexed segments within this curve reduce lordosis and are highly pathologic.
- For best results, combine rotation and sidebending with flexion and extension to reverse all three components of the positional diagnosis.
- If possible, place the spinal segment treated at the apex of the sidebending, flexion, and extension curves for both direct and indirect treatments.
- If cervical spine treatment fails with multiple procedures, evaluate the thoracic spine or head for primary somatic dysfunction, as the cervical spine pathology may be due to secondary somatic dysfunction.
- Some patients fear HVLA of the cervical spine and guard voluntarily. Either choose a different treatment modality, or distract the patient with respiration or toe movement prior to impulse.
- Treat using direct methods first and then indirect, to integrate the dysfunctional segment with surrounding soft tissues.
- Spasm of the levator scapulae causes recurrence of C2-5 ERS pathology due to its anterior attachment on the transverse processes.
- C7 restrictions will cause chronic, recurrent restrictions of the first ribs.

References

1. Kajimoto Ben Hur J, et al. “Anatomical Study of the Vertebral Artery Path in Human Lower Cervical Spine*.” Acta Ortopédica* Brasileira, no. 2 (15 2007): 84-86
2. Mitchell, Jeanette. "Vertebral Artery Blood Flow Velocity Changes Associated with Cervical Spine Rotation: A Meta-Analysis of the Evidence with Implications for Professional Practice." Journal of Manual & Manipulative Therapy 17, no. 1 (2009): 46-57. doi:10.1179/106698109790818160.
3. Okawara, S., and D. Nibbelink. "Vertebral Artery Occlusion Following Hyperextension and Rotation of the Head." Stroke 5, no. 5 (1974): 640-42. doi:10.1161/01.str.5.5.640.
4. Fryette, Harrison H., and W. Fraser. Strachan. *Principles of Osteopathic Technic.* Carmel, CA: Academy of Applied Osteopathy, 1954.
5. Capobianco, John D., and M. Protopapas. Rivera-Martinez. “Understanding the Combined Motions of the C3/C4 Vertebral Unit: A Further Look at Fryette’s Model of Cervical Biomechanics.”*American Academy of Osteopathy Journal,* no. 3 (12 2002): 15-30.
6. Ishii, Takahiro, et al. “Kinematics of the Cervical Spine in Lateral Bending.” *Spine* 31, no. 2 (01 2006): 155-60. doi:10.1097/01.brs.0000195173.47334.1f.
7. Greenman, Philip E. *Principles of manual medicine.* Philadelphia: Williams and Wilkins, 2003.
8. Chila, Anthony G. *Foundations of Osteopathic Medicine*. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2011.
9. Jones, Lawrence H., et al. *Strain-counterstrain*. Boise, ID: Jones Strain-Counterstrain, 1995.

Clinical Vignette

Patient Name: CV
June 12, 2020

CC: headache (HA)

HxCC: Patient is a 30-year-old, white female who was referred by her primary care physician for chronic HA and migraines. The patient first noted her HA in 2005. The HA occurred de-novo and lasted one year. She was seen by several neurologists, had a normal magnetic resonance imaging (MRI) of the brain and cervical spine. She saw a cranio-sacral therapist in Boston, Massachusetts who treated her and after a few treatments saw almost complete resolution of her HA. She was receiving maintenance treatments until that time and was doing well. She had dental work two months ago that required her to leave her mouth open for two hours on two occasions; this caused dramatic pain and return of her HA. She denies any fillings done at the time of the dental procedure. She is here for evaluation of HA that has returned. Her pain is 7/10, begins at the base of her skull and radiates to her face.

ROS:
Constitutional: no mental status changes, no travel or sick contacts
Eyes: no change in vision
Endocrine: no fever, night sweats, weight change
Cardiovascular: no chest pain or palpitations
Respiratory: no dyspnea, cough, or sputum
Gastrointestinal: no nausea, vomiting diarrhea; no change in bowel movement, no blood in stool
Genitourinary: no change in urine, no dysuria, no blood in urine
Neurologic: no anesthesia, no paresthesias, no loss of consciousness, no seizures
Musculoskeletal: no weakness
Psychiatric: no homicidal or suicidal ideations, no signs of depression
Allergy: no angioedema, no urticaria, no anaphylaxis, no symptoms of seasonal allergies

PMHx/PSHx:
1. Migraine headaches beginning in 2005

Allergies:
-no known food or drug allergies

MEDs:
-Fiorcet once daily as needed for HA
-Drospirenone/ethinyl estradiol oral birth control daily for two months

Immunizations:
-per her primary care physician

Social:
-no smoking, no alcohol, no drugs, works as a pharmacist

FamHx:
Sister = multiple sclerosis

VITALS = 120/70, 196#, 72bpm, 5'4", 98.7F
GEN = awake, alert, oriented to time, place, situation, self, in no acute distress
HEENT = normal cephalic, a-traumatic, extraocular muscles intact, no otic/nasal discharge
NECK = supple, thyroid w/o masses
HEART = regular rate, no clicks/rubs/murmurs
LUNGS = clear to auscultation, without crackles, wheeze, or rhonchi
GI = soft, non-tender, no rebound tenderness, +bowel sounds x 4 quadrants
CV = no carotid bruit, no edema, 2/4 peripheral pulses, no lymphadenopathy
N/M = cranial nerves 2-12 intact, 5/5 global strength, 2/4 global reflexes, cerebellum intact, sensation symmetric, gait normal, on lateral view her standing posture is normal.
Biomechanical Exam = pertinent findings include - **tender to palpation on cervical spine C4 on right posteriorly, spinous processes C5-7; left occipital mastoid restriction. Left-levator scapulae spasm and tender to palpation (TTP) on the muscle belly.** No intra-oral restriction noted with gloved hand nor pain in the gum line associated with her teeth. There is no apparent tenderness of the mandible or other structures of the head.

Assessment/Plan

1. Migraine HA
2. Somatic Dysfunction of Head, Neck, Upper Extremity:
   -Uncertain of the etiology for her recurrent migraines. No red flags are present. I do note she is on birth control that may contribute to migraines and was begun approximately two months ago. Although her exam does not reveal pain of her jaw, another consideration is dental associated infection. Her pain may be purely biomechanical etiology as well, or due to unrealized trauma.
   -Somatic dysfunction is present so will treat with Osteopathic manual medicine (OMM); if she fails to respond to treatment, I will consider stopping her birth control or sending her back to her dentist for evaluation of dental infection. Consider treatment of interspinous ligaments C5-7 with prolotherapy as she may have hypermobility of her cervical spine.
   -The patient was counseled regarding a normal treatment reaction. A treatment reaction may include muscle soreness, pain, and mild edema that may last 2-3days. If the patient experiences symptoms that outlay a normal treatment reaction, she should contact a physician or go to the ER.
      -C4 TTP posterior-right treated with S/CS
      -L-occipital mastoid restriction treated with cranial
      -R-levator scapulae TTP treated with S/CS
   -Will have the patient begin levator scapulae stretches three times daily for 20-30 seconds until follow up.
   -Follow up in 7 days for reevaluation.

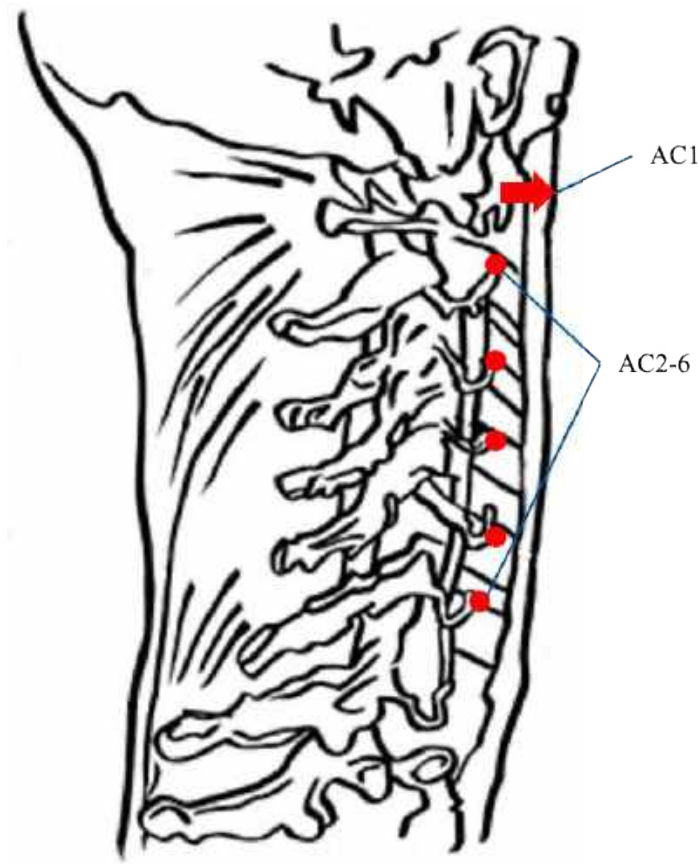
Jason Browder, DO

Appendix A: Anterior Cervical (AC) S/CS Tenderpoints

The anterior cervical vertebrae tenderpoints are on the anterior surface of the tips of the transverse processes. The first cervical vertebrae tenderpoint (AC1) is located on the posterior surface of ascending ramus of the mandible.

AC7 (not diagramed) is on the superior surface of the clavicle between the heads of the sternocleidomastoid muscle. AC8 (not diagramed) is located on the medial end of the clavicle.

There are additional tenderpoints on the anterior surface of the articular columns of the neck. Jones named them the "anterior lateral column"[9(pp39)] AC2-6.

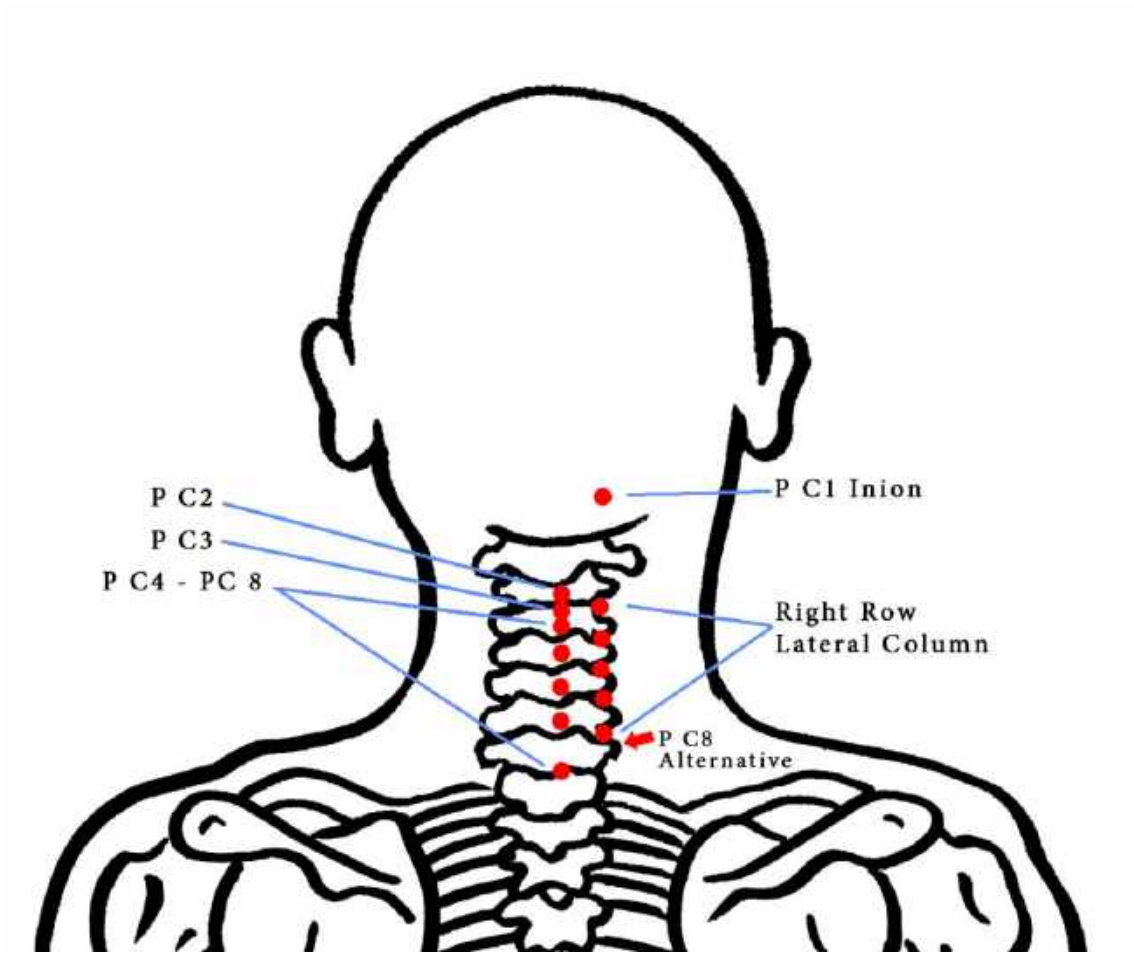


Appendix A: Posterior Cervical (PC) S/CS Tenderpoints

The posterior cervical tenderpoints are located on the right and left sides of the spinous processes (left sided points not diagramed). Note that the second cervical vertebrae have two tenderpoints: the PC2 on the superior aspect and the PC3 on the inferior aspect of the spinous process. The remaining vertebrae have solitary points on the inferior portion of the spinous processes; hence, PC4 tenderpoint is on the third cervical vertebrae on the inferior aspect of the spinous process.

Because the first cervical vertebrae cannot be palpated posteriorly, Jones named a PC1-inion tenderpoint for tenderness found 2cm below the inion lateral to the main muscle mass. There is an alternative PC8 tenderpoint anterior to the trapezius on posterior surface of the tip of the transverse process of the seventh cervical vertebrae.

There are additional posterior tenderpoints on the surface of the articular columns of the neck Jones named them the "right row lateral column."[9(pp38)]



Review Questions

1. The preferred anatomical landmarks for cervical spine palpation are:
    a. Articular pillars
    b. Lateral masses
    c. Pedicle
    d. Spinous processes
    e. Transverse processes

2. Palpation of C1 is most easily palpated at the
    a. Spinous process
    b. Lateral condyles
    c. Transverse processes
    d. Pedicle

3. The major motion of the occiput (C0) on the atlas (C1) is:
    a. Flexion-extension
    b. Rotation
    c. Sidebending

4. Approximately what percentage of rotation occurs at the atlanto (C1) axial (C2) junction?
    a. 5%
    b. 15%
    c. 30%
    d. 50%
    e. 85%

5. What type of spinal motion occurs between the occiput (C0) on the atlas (C1)?
    a. Type I motion
    b. Type II motion
    c. Contralaterally coupled motion
    d. Ipsilaterally coupled motion

6. Which of the following best describes the motion of C2-C7?
    a. While in flexion, translation of a segment from right to left tests right facets
    b. While in extension, translation of a segment from right to left tests left facets
    c. While in flexion, translation of a segment from left to right tests right facets
    d. While in extension, translation of a segment from left to right tests right facets

7. Which of the following adverse outcomes are associated with treatment modality or position of treatment?
    a. HVLA to C1-C2; carotid artery dissection
    b. Extension of the whole cervical spine during endotracheal tube intubation; dens subluxation in a patient with rheumatoid arthritis
    c. HVLA to C0-C1; spinal cord damage

d. Extension and rotation of C1-C3; stroke due to narrowing of the ipsilateral vertebral artery.

8. The "vertebra prominens" is at which level?
    a. C4
    b. C5
    c. C6
    d. C7
    e. T1

9. Severe complications of OMT increase in which treatment position:
    a. HVLA to C3 in extension
    b. HVLA to C3 in flexion
    c. HVLA to C4 in extension
    d. HVLA to C4 in flexion

10. Anatomically, the vertebral artery most often passes through the transverse foramen of:
    a. C1-C5
    b. C1-C6
    c. C2-C5
    d. C2-C6

11. Injury to the spinal cord in a patient with rheumatoid arthritis would most likely occur in which clinical scenario:
    a. The dens contacting the anterior spinal cord
    b. The spinous process of C2 contacting the dorsal columns
    c. The articular pillars contacting the anterior spinal artery
    d. The cruciform ligament obstructing blood flow through the vertebral artery.

12. While flexed, passive anterior translation of the C3 articular pillars with restriction of the left side indicates a positional diagnosis of:
    a. Extended, side bent left
    b. Flexed, rotated left
    c. Extended, rotated left
    d. Flexed, side bent left

13. While passively flexed, the operator translates the right lateral condyles of C3 medially with good ease; when the left condyles of C3 are translated medially, they are restricted indicating a positional diagnosis of:
    a. C3 Flexed, sidebent right
    b. C3 Extended, sidebent right
    c. C4 Flexed, sidebent left
    d. C4 Extended, sidebent left

14. While passively extended, the operator translates the right lateral condyles of C3 inferomedially with good ease; when the left condyles of C3 are translated inferomedially, they are restricted indicating a positional diagnosis of:
    a. C3 Extended, sidebent left
    b. C3 Flexed, sidebent right
    c. C3 Extended, sidebent right
    d. C3 Flexed, sidebent left

15. When the patient is placed in extension, C1 is rotated on C2; if there is restriction in rotation toward the right then the diagnosis is:
    a. C0 left rotated
    b. C0 right rotated
    c. C1 right rotated
    d. C1 left rotated

16. While extended, C0 is translated in relation to C1 and restriction is noted when translating C0 from the left to the right. The diagnosis is thus:
    a. C0 FR_LS_L
    b. C0 FR_RS_R
    c. C0 FR_LS_R
    d. C0 FR_RS_L

17. If treating C5 ERS_R with functional procedures, the position of treatment will be:
    a. C5 ERS_R
    b. C5 ERS_L
    c. C5 FRS_R
    d. C5 FRS_L

18. A patient presents to your office after a diving accident in which she hit her head on the bottom of the pool; she says her cervical spine was compressed by the injury. She has cervical pain and insists on treatment. She says she will leave your practice if you do not treat her immediately. You:
    a. Treat the patient on the initial day of the visit
    b. Evaluate the patient with passive ROM testing and decide based on the exam your next step
    c. Order an x-ray to rule out fracture
    d. Consult neurosurgery

Answers:

1. A
2. C
3. A
4. D
5. C
6. C
7. C
8. D
9. A
10. B
11. A
12. C
13. B
14. B
15. D
16. C
17. A
18. C

Chapter 5: The Ribcage

Principles Presented in this Chapter:

- Diagnosis of the ribcage somatic dysfunction
- Treatment of the ribcage
- Clinical significance of abdominal diaphragm
- "Thoracic outlet/inlet"

Outline:

- Anatomy of the Ribcage
- Biomechanics of the Ribcage
- Diagnosis of Ribcage Somatic Dysfunction
- Treatment of the Ribcage
    - Direct Treatment of Ribcage Somatic Dysfunction
    - Indirect Treatment of Ribcage Somatic Dysfunction
- Ribcage Treatment Pearls
- Clinical Vignette
- Appendix A: Ribcage Strain-counterstrain Tenderpoints
- Review Questions

Chapter 5: The Ribcage

The clinical significance of a normally functioning ribcage cannot be understated. Based on clinical experience, it remains the most common location where authors JB and PJ begin treatment. Structurally, the ribcage provides rigidity for protection of the heart and lungs, but it also serves as the mechanical driver for cellular exchange in aerobic respiration, providing the gradient for return of venous blood and lymph. Inability of the ribcage to function normally may result in heart failure, edema, ascites, thoracic outlet syndrome and a host of other pathology.

Anatomy of the Ribcage

The structure and function of the rib cage influence the biomechanical aspects of somatic dysfunction and respiration, and additionally have an important role in the lymphatic and circulatory systems of the body. We review some of these important interrelationships, starting with the bony anatomy of the thoracic cage:

Figure 1

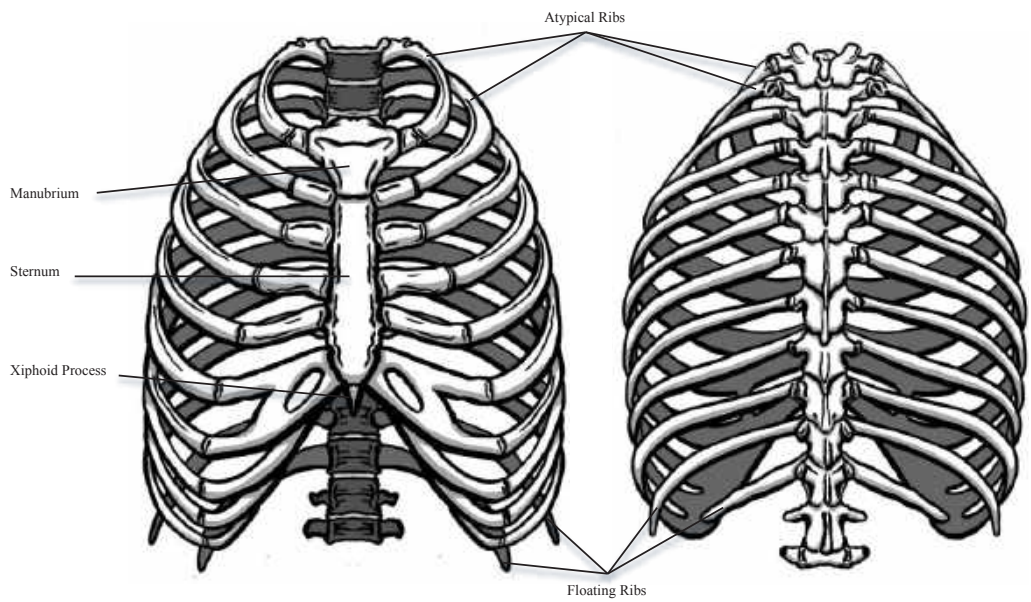


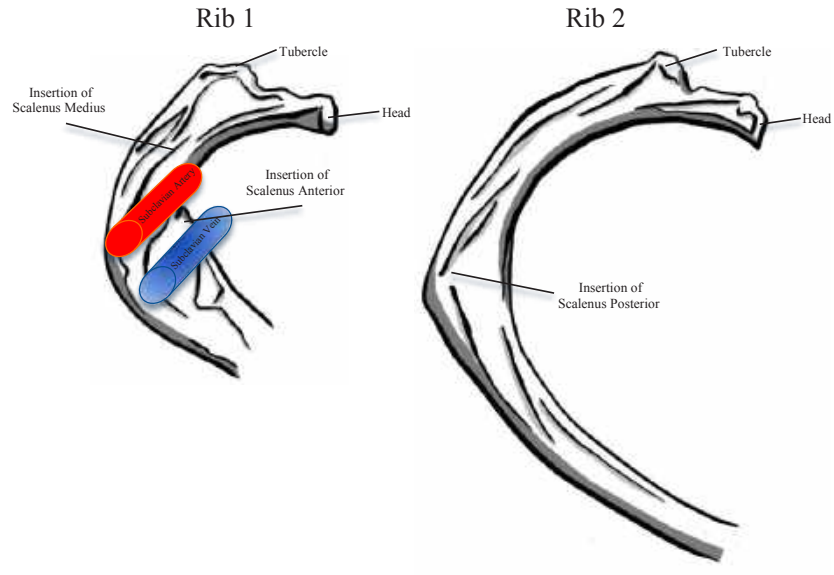
Figure 1: Anatomy of the Ribcage

Atypical ribs have a number one or two in them, thus include ribs 1, 2, 11, and 12.

The rib cage can be divided into "typical" and "atypical ribs" (Figure 1). Neither the first or second ribs have typical rib angles; the second rib has a large tuberosity for the insertion of the posterior scalene muscle (Figure 2).

Figure 2: Anatomy of Ribs 1-2

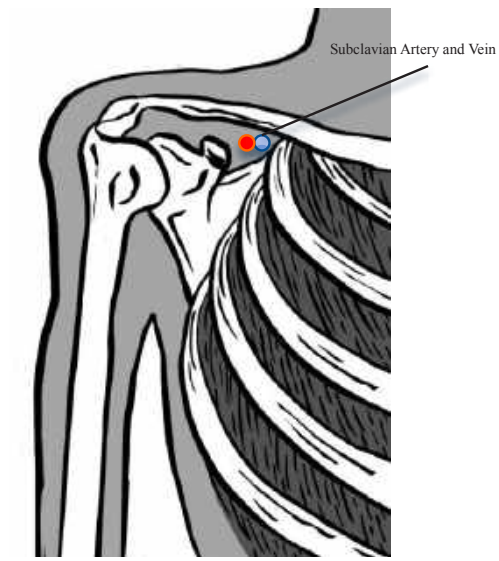
Figure 2



Both the anterior and medial scalenes attach to the first rib. The first rib is also unique in its articulation with the thoracic spine – it has a single facet forming the costotransverse joint, while typical ribs articulate with the thoracic vertebral body and the transverse process. Ribs one and two are important with respect to the thoracic inlet and neurovascular bundle (Figure 3).

Figure 3: Anatomy of the Thoracic Inlet

Figure 3



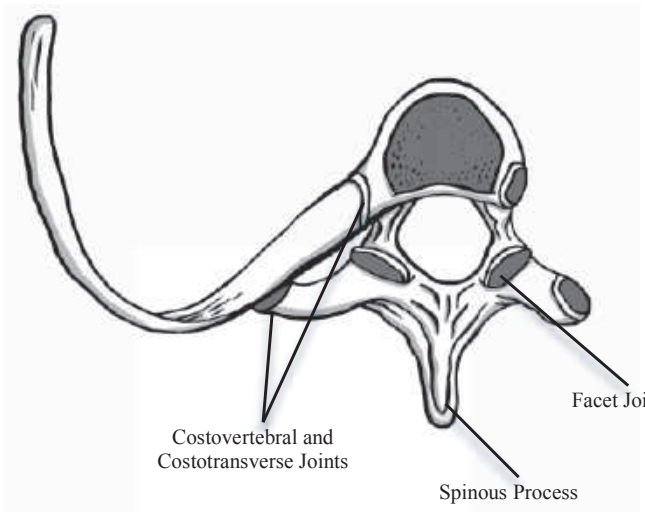
Thoracic Inlet - anatomical borders include:
-T1 posteriorly
-Ribs 1-2 laterally and anteriorly
-Superior border of the manubrium anteriorly

The thoracic inlet is made up of the first thoracic vertebra posteriorly, the first pair of ribs laterally, and the superior border of the manubrium anteriorly. Clinically, restriction of the ribcage, thoracic spine, scalene muscles, or the upper extremity, through the clavicle and pectoralis muscles, may lead to thoracic outlet syndrome (TOS). Clinical symptoms of TOS are the result of compression of the brachial plexus, the subclavian artery, or the subclavian

vein. Rarely, symptoms may be caused by a lung mass pushing superiorly on these neuro-vascular structures. These symptoms may include anesthesia, paresthesia, cold upper extremity, and absent or diminished pulse. Anecdotally, the most common clinical presentation of TOS involves first rib somatic dysfunction with ulnar distribution paresthesia, and a positive Roos test (see upper extremity chapter). Repeat testing of this provocative maneuver after treatment of the 1st rib often reveals relief in the paresthesias.

The typical ribs, ribs 3 through 10, have both costovertebral and costotransverse articulations (Figure 4) and attach to the sternum via the cartilage or costal arch. They have prominent angles to which the iliocostalis muscles also attach. Overlying the iliocostalis attachment to the rib angle, the serratus posterior-superior also inserts on the rib angles of ribs 2-5 from its origin at C7-T3; its role is to elevate the ribs. This is a common area of tenderness to palpation on physical exam and the location of posterior rib strain-counterstrain (S/CS) tender points.

Figure 4



Ribs 11 and 12, often referred to as "floating ribs" since they do not attach to the sternum anteriorly, are invested in the abdominal musculature, and have single costovertebral facets.

Especially in the muscle energy (ME) model, important musculature related to rib cage motion and treatment includes the anterior, medial and posterior scalenes, and the pectoralis minor and serratus anterior for "exhalation" rib dysfunction. Likewise, ribs trapped in "inhalation" utilize the internal intercostal and abdominal musculature for treatment (Figure 5).

Thoracic Outlet Syndrome: symptoms produced by compression of the components of the brachial plexus, the subclavian artery, or the subclavian vein.

Typical Ribs:

- Articulate with the thoracic vertebral body and the transverse process posteriorly
- Articulate with the sternum through costal cartilage
- Have prominent angles to which the iliocostalis attaches

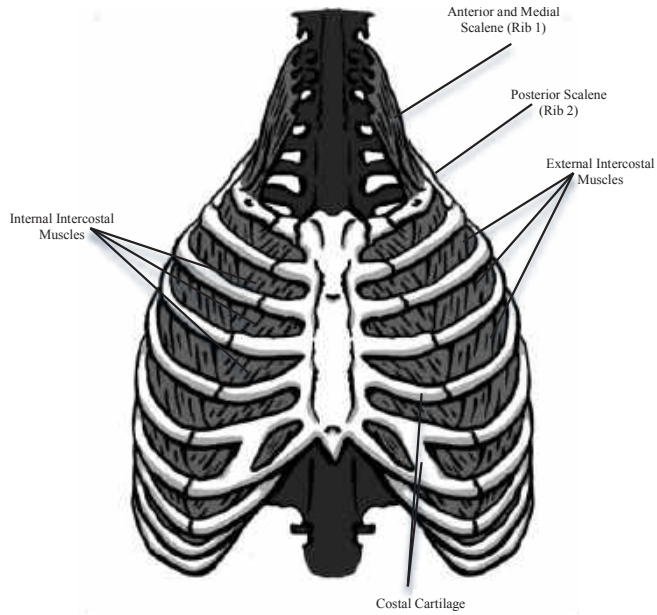
Figure 4: Anatomy of the typical ribs

Note how the ribs of thoracic vertebrae 3-10 actually have two articulations with the vertebral: on the body and the transverse process.

Figure 5: Musculature of the Ribcage.

Note the attachments:
Rib 1 – anterior and medial scalene
Rib 2 – posterior scalene
Rib 3-5 – pectoralis minor
Ribs 6-10 – serratus anterior
All ribs have intercostal muscles between them

Figure 5

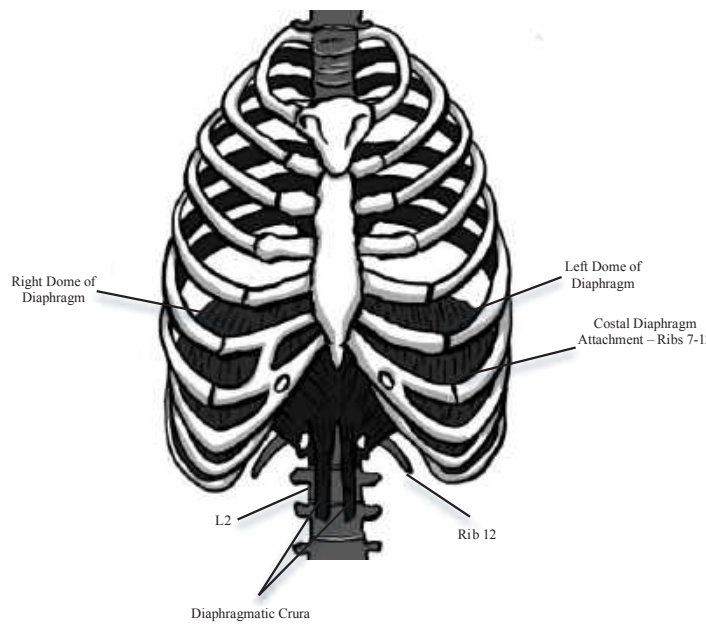


The abdominal diaphragm has an intimate relationship with all parts of the musculoskeletal system (Figure 6): innervation through the phrenic nerve, direct attachment to the lower six ribs, fascial attachments to the quadratus lumborum and psoas muscles, and attachment of its crura to L1-3. Because of its relationship with these structures, the diaphragm not only affects circulation and respiration, but has direct implications for the neuro-musculoskeletal system.

Figure 6: The Abdominal Diaphragm

Note the attachment of the diaphragm to ribs 7-12.

Figure 6



Biomechanics of the Ribcage

There are two patterns of rib cage motion: “pump handle” and “bucket handle” (Figure 7). Pump handle motion occurs in the sagittal, anteroposterior (A/P) plane and typically refers to ribs 1 through 4. During inhalation the sagittal diameter of the rib cage increases; this movement is best palpated in the anterior para-sternal portion of the rib cage.

Figure 7

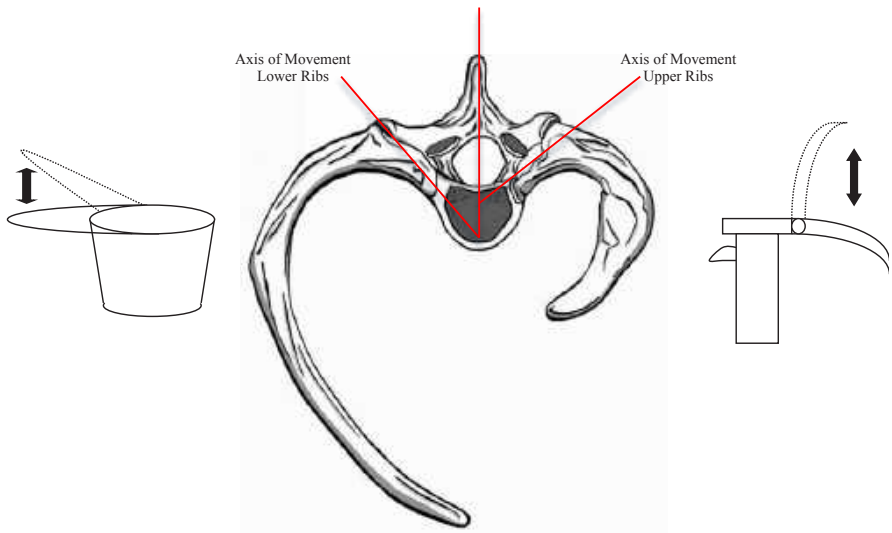


Figure 7: Rib Motion

- Ribs 1-4 demonstrate “pump handle” mechanics like that of an old-fashioned water pump.
- Ribs 5-6 have mixed motion of pump and bucket handle.
- Ribs 7-10 expand laterally in a “bucket handle” fashion.
- Ribs 11-12 have caliper motion.

Bucket handle motion occurs in ribs 7 through 10. This movement occurs in the coronal plane and increases the transverse diameter of the thoracic cage during inhalation. This motion is best appreciated with hand placement on the mid axillary line. Ribs 5 and 6 demonstrate a combination of both pump-handle and bucket-handle mechanics.

Ribs 11 and 12 demonstrate a type of caliper motion. Caliper ribs flare in or flare out during inhalation/exhalation. Anecdotally, caliper ribs rarely manifest somatic dysfunction and thus rarely require treatment. For this reason, we do not address diagnosis and treatment of caliper ribs in this text.

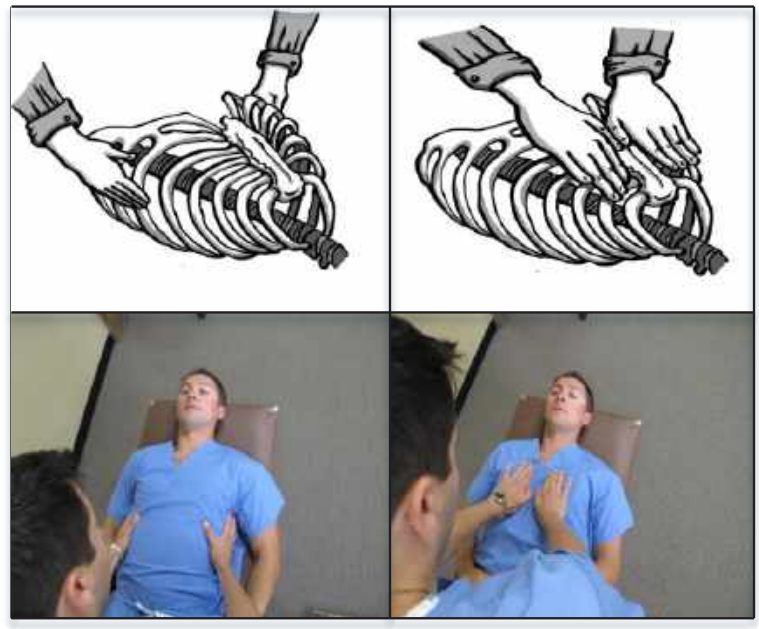
Diagnosis of Ribcage Somatic Dysfunction

Diagnostic evaluation of rib cage dysfunction is based on motion testing and thus lends itself to treatment through multiple methods. The operator examines the patient with their dominant eye over the center of the chest. To test “pump-handle” rib motion, with palms resting on ribs1-4, the operator places his finger pads anteriorly at the costosternal junction and observes for static symmetry on the right and left (Figure 8).

Figure 8: Evaluation for Rib Somatic Dysfunction

Note that the operator has his left dominant eye over the patient's midline.

Figure 8



The practitioner then instructs the patient to take a full inhalation and exhalation through the mouth. During inhalation and exhalation, the practitioner observes the right and left upper ribs for symmetry of motion. Likewise, when monitoring lower rib cage motion, or "bucket-handle" ribs, the operator palpates the lower rib cage at the mid-axillary line bilaterally to observe static symmetry. To test for motion symmetry in the coronal plane, the patient is then instructed to inhale and exhale through the mouth. Somatic dysfunction exists if asymmetry is noted in the static AND dynamic phase of testing.

One of three possible patterns emerge during the process of static and dynamic testing:

1. Normal symmetry and motion during inhalation (i.e. no somatic dysfunction).

2. One rib or a group of ribs will statically appear in an anterior or elevated position compared with the other ribs; yet during inhalation this same group will appear symmetric to the normally functioning ribs. This group of ribs or rib are termed: "inspired ribs," "inhalation ribs," "inhaled ribs," or as an "exhalation rib restriction."[1(pp52)]

3. The other pattern of rib cage biomechanical dysfunction occurs when a rib or group of ribs appear symmetric on static evaluation and incompletely rise during inhalation compared with the normally functioning ribcage. This rib, or group of ribs, is diagnosed as "exhaled ribs" and termed "expired ribs," "exhalation ribs," or as an "inhalation rib restriction."[1(pp52)]

To avoid confusion with diagnostic terminology, it is often helpful to think about the positional diagnosis of the ribs and then add the appropriate label.

Once an area of motion restriction is identified, then the operator palpates individual ribs for TART criteria. Using open mouth breathing and inhalation and exhalation, motion testing is then repeated to identify the "key rib" involved in the dysfunction. The key rib concept relates to the terminal rib when a group of ribs demonstrate somatic dysfunction - the top rib of the group for expired ribs and the bottom rib of the group for inspired ribs. Since restoring its motion will restore normal motion to the remaining ribs involved in the dysfunction, the key rib concept becomes the focus of treatment.

While the practitioner can make the diagnosis of rib cage restriction by motion testing as described above, they can also make the diagnosis by palpating rib tender points, or a hybrid of both; diagnose rib restriction by motion testing, then examine for correlating S/CS tender points (appendix A).

Key Rib Location:
Use the acronym BI|TE
Bottom = Inhaled Ribs
Top = Exhaled Ribs

Correlation of the static/dynamic motion testing model and S/CS tender points are as follows:

- If the positional diagnosis is "exhaled rib," the corresponding rib tender point is located anteriorly.
- If positional diagnosis is "inhaled rib," there is a corresponding posterior tender point at the rib angle of the key rib.

Often a group of ribs will have multiple anterior or posterior tender points (TPs). S/CS practitioners observe that the posterior rib TPs may occur in groups of three that are then treated individually, starting with the bottom first. However, treating all ribs in the group may be unnecessary in the context of the key rib concept as described above. When applied to S/CS and functional procedures, the key rib is the rib that brings the group of ribs to the position of ease (POE). For example, when treating *Left Rib 1-5 Inhaled*, the key rib is always the bottom rib, regardless of using direct or indirect treatments. Additionally, the key rib is almost always most tender to palpation.

Correlation of Tender-points with Dynamic Testing:
Inhaled-Ribs = posterior tenderpoints.
Exhaled-Ribs = anterior tenderpoints.

To Summarize ribcage diagnosis:

1. Grossly test the rib cage for static symmetry, TPs, or both.
2. Motion test in inhalation and exhalation.
3. Evaluate individual ribs for TART criteria to identify the "key-rib" of the inhalation or exhalation dysfunction. Remember the "BITE" acronym.

Using S/CS to diagnose, the key rib is almost always the rib most tender to palpation (TTP).

Treatment of Ribcage Somatic Dysfunction

Treatment of the ribcage follows the principles of previous chapters. Once a positional diagnosis has been established, either direct or indirect procedures may be utilized to treat the area depending on the type of barrier encountered. Please refer to principles chapter regarding procedures that match a given somatic dysfunction.

Direct Treatment of Ribcage Somatic Dysfunction

The steps for direct treatment *Right Ribs 1-5 Exhaled*:

Obtain the positional diagnosis *Right Ribs 1-5 Exhaled*.

1. Place a fulcrum on T1 adjacent to rib 1 (key rib). The fulcrum is generally created at the rib's thoracic attachment.
2. Create a lever using the angle of right rib 1 posteriorly.
3. Reverse the components of the positional diagnosis by placing the patient's rib in an inhaled position (i.e., cervical and thoracic extension).
4. Create a force by using an impulse or isometric contraction that moves the rib into inhalation.

Refer to the examples provided for high-velocity low amplitude (HVLA) and muscle energy (ME) that further extrapolate these concepts.

Indirect Treatment of Ribcage Somatic Dysfunction

Again, use the positional diagnosis *Right Ribs 1-5 Exhaled.* To treat these ribs using indirect procedures, just place the right ribs in an inhaled position with localization to the key rib.

To review, the steps for indirect treatment *of Right Ribs 1-5 exhaled* are:

1. Obtain the positional diagnosis *Ribs 1-5 exhaled.*
2. Place the patient in the position *Ribs 1-5 exhaled*, with a focus on rib 1 (key rib).
3. Exaggerate the exhaled position with the dynamic balance point or POE focusing on rib 1.

Follow these steps as a basis for indirect treatment of the ribcage. For understated differences of technique, examples are provided for strain-counterstrain (S/CS) and Functional procedures below.

Ribcage Treatment Pearls

We identify the ribcage as the most common cause of somatic dysfunction. Somatic dysfunction of the ribcage is, in our opinion, responsible for most causes of chest wall pain (so called costochondritis) and is often mistaken for acute coronary syndrome and other life-threatening causes of chest pain. On initial evaluation, do not overlook a life-threatening condition with a preference for somatic dysfunction since pain may present in the form of a visceral-somatic reflex (see neurophysiology chapter). Listed below are additional pearls for treatment of the ribcage:

- The left ribs usually have the positional diagnosis of inhaled, and the right ribs usually have the positional diagnosis of exhaled.
- Treat out-of-pattern ribs FIRST. For example, inhaled ribs on the right, or exhaled ribs on the left.
- Common Patterns of Rib Dysfunction[2]
  - Expired ribs (right side)
    - Ribs 1 through 10 expired
    - Ribs 3 through 5 expired
    - Ribs 6 through 10 expired
  - Inspired Ribs (left side)
    - Rib 1 inspired
    - Ribs 1 through 2 inspired
    - Ribs 1 through 5 inspired
    - Ribs 1 through 10 inspired

- Muscles Utilized During Muscle Energy Techniques

Exhaled Ribs	Muscle
1	Anterior and Medial Scalenes
2	Posterior Scalenes
3-5	Pectoralis minor
6-10	Serratus Anterior
Inhaled Ribs	
1-5	Internal Intercostals
6-10	Internal Intercostals and Abdominals

- As Larry Jones, DO, stressed, treat anterior TPs (in addition to posterior); the “anchor” of the dysfunction is often found in the anterior TPs.

Treatment Examples: Direct Procedures

Right Ribs 1-5 Exhaled using ME – Ant. Scalene

1. Place the patient supine with the forearm of the affected side on the forehead; this allows access to the first rib and automatically moves rib 1 (key rib) into an inhaled position.
2. A fulcrum between T1 and rib 1 will naturally form due to cervical rotation and extension. The lever is the rib itself and is moved by contraction of the scalene.
3. Have patient flex his head forward at different degrees of cervical spine rotation until the most motion is palpated at the first rib with contraction of the anterior scalene muscle.

4. Operator applies counterforce against head/neck flexion with right hand and left hand applies the lateral and caudad traction posteriorly at the angle of the first rib during patient's muscle effort.
5. The procedure is repeated 3-5 times until no further change in first rib motion is appreciated.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Right Ribs 1-5 Exhaled using HVLA

1. The patient is seated and the operator monitors the first rib with the right thumb at the rib angle while using the left hand to extend, side bend, and rotate the head, "locking down" to the associated first thoracic (T1) vertebrae.
2. A fulcrum between T1 and rib 1 will naturally form due to cervical rotation and extension. The lever is the angle of rib 1 itself.

3. Determine the vector that yields the most motion to move the rib inferiorly.
4. The operator provides an impulse in an inferior direction at the rib angle to bring the anterior part of the first rib into inhalation.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Treatment Examples: Indirect Procedures

Right Ribs 1-5 Exhaled using Functional Procedures

1. The patient is supine and the operator monitors the first rib (key rib) with both hands: anterior hand at costal cartilage and posterior hand at the rib angle.
2. The operator positions rib 1 moving through the cardinal planes of motion: anterior, posterior, medial, and lateral to find the dynamic balance point (DBP) with their hands.
3. Then the operator adds compression between both hands to activate the treatment.

4. Monitor the DBP as it changes using both hands while maintaining first rib balance. If the DPB reaches a still point, wait for 10-20 seconds; if there is no change, challenge the tissues by applying distraction or compression into the first rib.
5. When the tissue relaxes, the treatment is finished.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Right Ribs 1-5 Exhaled using S/CS (AR1)

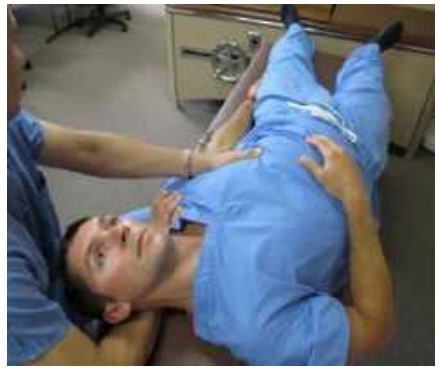
1. The tender point is located on the first costal cartilage beneath the clavicle, just lateral to the sternum.
2. The patient's head is flexed, side bent and rotated towards the tender point until at least 70% of tenderness is alleviated. The position is held for approximately 120 seconds, or until resolution.

3. The patient is then gently and slowly returned to the neutral position.
4. Reassess for tenderness at the first rib. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Treatment Examples: Direct Procedures

Left Ribs 1-5 Inhaled using ME

1. The patient is supine with operator cradling head and neck with right hand. The operator places left hand on the anterior chest at costochondral junction of rib 5. The fulcrum is formed by left sidebending and slight flexion of thoracic spine, effectively locking down the spine to T6. The lever is formed at the fifth rib.



2. The patient takes a shallow inhalation followed by a long exhalation (activating the internal intercostal muscles), as the operator glides rib 5 inferiorly and flexes and further side bends the patient's trunk to "take up the slack."
3. The practitioner maintains an unyielding counterforce while the patient repeats the short shallow inhalation, followed by a long exhalation to follow the rib to its new restrictive barrier. The procedure is repeated approximately 3-5 times or until no further change is achieved
4. Recheck the original diagnosis to ensure resolution of somatic dysfunction

Left Ribs 1-5 Inhaled using HVLA

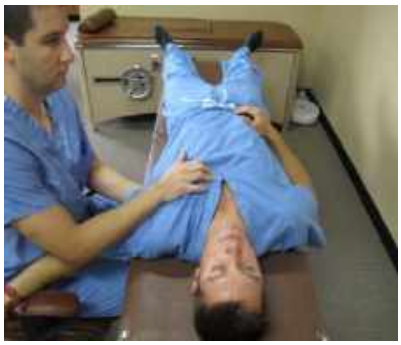
1. The patient is supine with arms crossed across the chest with the ipsilateral arm on top for torso/lever control.
2. Operator approaches patient from the right side and rolls patient toward him/her while supporting patients head and neck.

3. The operator creates a fulcrum by flexing patient one thoracic level below the key rib (T6) to "lock down" vertebral/rib segment. The lever is formed on the angle of rib 5 by the operator's thenar eminence.
4. The operator contacts the patient's elbows with their hypogastrum and gently springs to find the best vector that glides the rib angle superiorly.
5. The operator instructs the patient to take a small inhalation followed by full exhalation. At this point, the operator applies an impulse by dropping down toward the table via contact points of abdomen with patient's elbows, while maintaining the lever.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction

Treatment Examples: Indirect Procedures

Left Ribs 1-5 Inhaled using Functional Procedures

1. The patient is supine and the operator monitors the fifth rib (key rib) with both hands: anterior hand at costal cartilage and posterior hand at the rib angle.
2. The operator positions rib 5 moving through the cardinal planes of motion: anterior, posterior, medial, and lateral to find the dynamic balance point (DBP) with their hands.
3. Then the operator adds compression between both hands to activate the treatment.



4. Monitor the DBP as it changes using both hands and maintain balance on the fifth rib. If the DPB reaches a still point wait for a 10-20 seconds; if there is no change, challenge the tissues by applying distraction or compression into the fifth rib.
5. When the tissue relaxes the treatment is finished.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Left Ribs 1-5 Inhaled using S/CS (PR5)

1. The patient is supine. The operator uses their right hand to adduct patient's left shoulder to palpate posterior tender point at angle of rib 5 with left finger pads.
2. Operator then brings shoulder toward a position of scapular retraction using the arm to then apply traction/compression, internal rotation/external rotation, and abduction/adduction to find the position that reduces the tender point by at least 70%.

3. Position is held for approximately 120 seconds or until the point resolves.
4. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Additional S/CS position

References

1. Giusti, Rebecca, DO. *Glossary of Osteopathic Terminology*. Third ed. Chevy Chase, MD: American Association of Colleges of Osteopathic Medicine, 2017.
2. Edward G. Stiles lecture notes from KYCOM 1999.
3. Jones, Lawrence H., et al. *Strain-counterstrain*. Boise, ID: Jones Strain-Counterstrain, 1995.

Clinical Vignette

Patient Name: GG
July 6, 2016

CC: new patient visit, shortness of breath

HxCC: Patient is a 31year, white male presenting as a new patient. He has a long-standing history of asthma which he claims is uncontrolled. When well, his peak flow is 400 liters/minute. For the past week, he is using his albuterol metered dose inhaler (MDI) every 2-4 hours due to persistent symptoms of wheezing. He thinks his worsening symptoms are due to seasonal allergies for which he is sneezing and has post-nasal drip. He is a smoker of 10 cigarettes daily. GG notes no fevers, chills, cough or sputum.

ROS:
Constitutional: no mental status changes, no travel or sick contacts
Eyes: no change in vision
Endocrine: no fever/night sweats/weight change
Cardiovascular: no chest pain/palpitations
Respiratory: per history of chief complaint
Gastrointestinal: no nausea/vomiting/diarrhea, no change in bowel habit
Genitourinary: no change in urine, no dysuria, no blood in urine
Neurologic: no anesthesia/pararasthesia, no loss of consciousness/seizures
Musculoskeletal: no weakness, aches, or pains
Psychiatric: no homicidal/suicidal ideations, no signs of depression
Allergy: no angioedema, urticara, anaphylaxis, no symptoms of seasonal allergies

PMHx/PSHx:
1. Left-inguinal hernia status post repair twice
2. Gastro-esophageal reflux disease
3. Asthma diagnosed age 5

Allergies:
-no known food or drug allergies

MEDs:
-Albuterol metered dose inhaler

Immunizations:
-none

Social:
-Smoker 10cigs/day for 10 years
-1-2 alcoholic beverages per day
-No drugs
-Work as a restaurant owner

FamHx:
Mom = none
Dad = died in motor vehicle accident

VITALS = 6", 178#, 108/80, 74 beats per minute
GEN = awake, alert, oriented to time, place, situation, self, in no acute distress
HEENT = normal cephalic, a-traumatic, extraocular muscles intact, no otic/nasal discharge
NECK = supple, thyroid w/o masses
HEART = regular rate, no clicks/rubs/murmurs
LUNGS = soft wheezing in all lung fields, without crackles or ronchi; peak flow is 350 liters/minute
GI = soft, non-tender, no rebound tenderness, +bowel sounds x 4 quadrants, no hepato-spleenomegaly
CV = no jugular venous distention, no carotid bruit, mucus membrane moist, no edema, 2/4 peripheral pulses, no lymphadenopathy
N/M = cranial nerves 2-12 intact, 5/5 global strength, 2/4 global reflexes, cerebellum intact, sensation symmetric, gait normal, observation of lateral standing posture is normal
Biomechanical Exam = significant findings include: **right ribs 6-10 inhaled with right hemi-diaphragm restricted in range of motion**

A/P

1. Asthma

   -Uncontrolled with his albuterol MDI by the "rule-of-twos," but does not require hospitalization. Suspect there are environmental triggers for his worsening asthma, as well as somatic dysfunction, which may be contributing.

   -Begin montelukast 10mg po qday which will treat asthma and seasonal allergic rhinitis.

   -His in-office peak flow was 350 liters/min so will not prescribe steroids at this time.

   Asthma action plan done today: based on his previous peak expiratory flow (PEF) of 400 liters/min on his home peak flow meter, he should call me for a same day appointment if he has symptoms and scores of 200-300 liters/min. He should go to the emergency room for a score <200 liters/min.

   -OMM done to right inhaled ribs 6-10 using muscle energy; treatment of the right hemi-diaphragm with direct myofascial release.

   -Follow up in 2 days or call if worse.

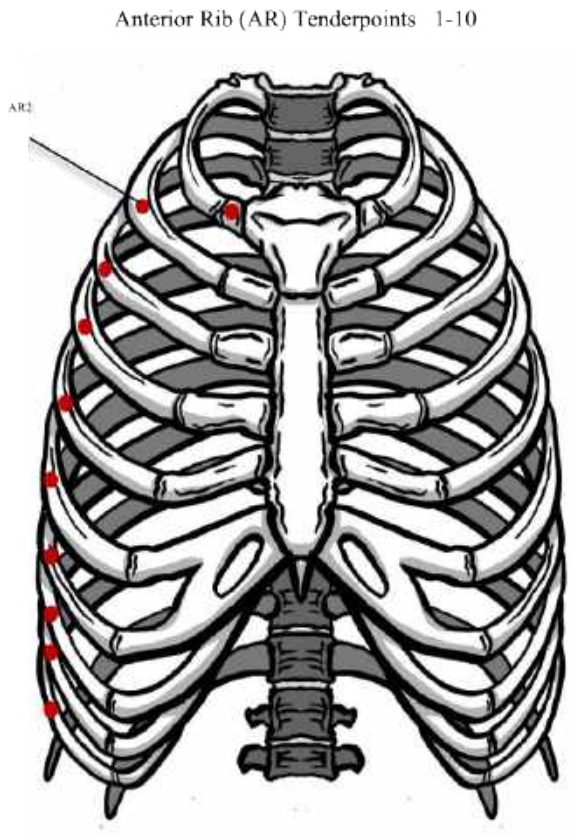
2. Tobacco Use

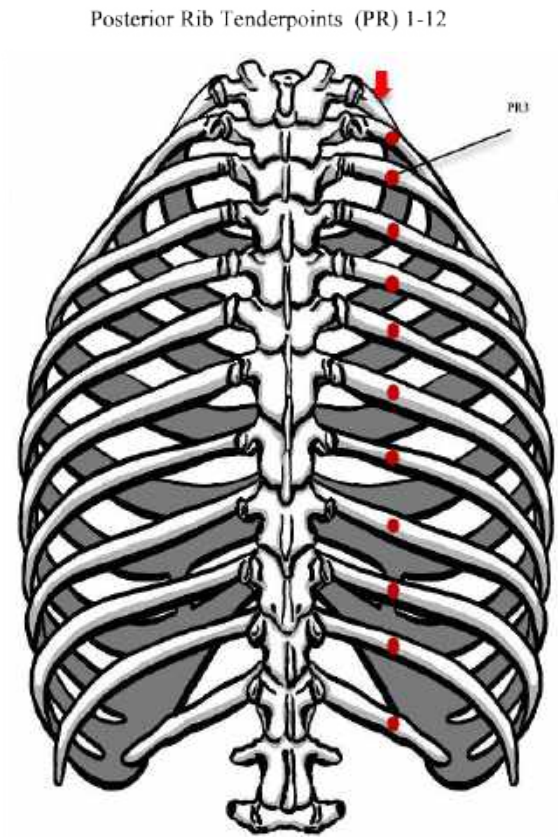
   -He is advised to quit smoking as leads to chronic obstructive pulmonary disease, multiple forms of cancer, and gastro-esophageal reflux disease… he will consider.

Paul Johnson, DO

Appendix A: Rib Cage S/CS Tenderpoints

Clinically, we encounter rib tender points daily. We find that S/CS is a very effective and elegant method of treatment. Anterior rib (AR) tender points are typically located on the anterior axillary line, with the exception of the first rib that is located on the costo-cartilage just lateral to the manubrium and inferior to the clavicle.[3(pp62-65)] The AR tenderpoints correlate with exhaled ribs.





The posterior rib (PR) tenderpoints are located on the rib angles[3(pp50,66-67)] with the exception of the first rib that is superior-to-anterior in nature (designated by arrow). The posterior rib tenderpoints correlate with inhaled ribs.

Review Questions

1. A patient presents with right upper extremity paresthesias, and you perform the necessary neurological examination as well as a biomechanical structural exam. Essentially, you find a non-localizing or "non-focal" neurological exam, tenderness to palpation in the upper cervical region, and a right 1st rib restriction. Which provocative test would lead you to have thoracic outlet/inlet syndrome high on your list of differential diagnoses?
    A. Roos test
    B. Spurling's test
    C. McMurry's test
    D. Brudzinski's test

2. Relating to the same patient in the question above, what is the pertinent muscular anatomy relating to a diagnosis of thoracic outlet/inlet syndrome?
    A. Anterior and medial scalenes
    B. Posterior and medial scalene
    C. Serratus anterior and anterior scalene
    D. Serratus posterior superior and anterior scalene

3. A patient is found to have rib cage dysfunction with the following diagnosis: ribs 3-5 exhaled on the right. You choose to use muscle energy to restore normal rib cage mechanics. Which muscle is utilized to perform the muscle effort?
    A. Anterior scalene
    B. Medial scalene
    C. Pectoralis minor
    D. Pectoralis major

4. A patient of yours with long standing congestive heart failure due to ischemic causes has been stable on medications with an angiotensin converting enzyme inhibitor, beta blocker, and a thiazide diuretic until recently, when he was released from the hospital with a "negative" cardiac workup. You question the patient about changes in salt intake, medication compliance and a host of other factors that could lead to his chief complaint of leg swelling and shortness of breath. After you eliminate all of these other potential causes and perform a biomechanical physical exam, what finding could potentially help alleviate this patient's symptoms?
    A. Ribs 6-9 inhaled on the right
    B. C1-2 restricted in rotation to the left
    C. Pronated radial head
    D. Anterior talus

5. A patient recently suffered an injury in a motor vehicle accident and presents with neck and peri-scapular pain on the right side. You find the most restricted area to be the first rib on the right which is exhaled. Which muscles do you utilize to return the rib to a more normal range of motion with muscle energy?
    A. Anterior scalene
    B. Pectoralis minor
    C. Pectoralis major
    D. Serratus anterior

6. The same patient in question comes back in a week for follow up evaluation saying he has improvement in his symptoms with the prior treatment. You evaluate him again and find right ribs 6-9 exhaled on the right. Which muscle treats this area using muscle energy?
    A. Anterior scalene
    B. Serratus posterior
    C. Serratus anterior
    D. Medial scalene

7. Trigger points and tender points are manifestations of musculoskeletal injury found on physical examination. What are the differentiating features of a trigger point and a tender point?
    A. A tender point sends referred pain when palpated
    B. A trigger point is painful, but sends referred pain only when a trigger point injection is performed
    C. A tender point doesn't send referred pain when palpated
    D. A trigger point is not painful and sends referred pain only when palpated

8. A posterior rib 3 tender point on the left is associated with what positional diagnosis?
    A. Exhaled 3rd rib on the right
    B. Exhaled 3rd rib on the left
    C. Inhaled 3rd rib on the right
    D. Inhaled 3rd rib on the left

9. An anterior 6th rib tender point on the right is associated with what positional diagnosis?
    A. Exhaled 6th rib on the left
    B. Exhaled 6th rib on the right
    C. Inhaled 6th rib on the left
    D. Inhaled 6th rib on the right

10. Rib 1 and 2 demonstrate which type of mechanics?
    A. Bucket handle mechanics
    B. Pump handle mechanics
    C. Caliper rib mechanics
    D. Mixed pump handle/bucket handle mechanics

Answers:

1. A
2. A
3. C
4. A
5. A
6. C
7. C
8. D
9. B
10. B

Chapter 6: The Sacrum and Pelvis

Principles Presented in this Chapter:

- Pelvic Axes
- Pelvic Diagnosis using Pelvic Axes
- Treatment using Pelvic Axes

Outline:

- Anatomy of the Sacrum and Innominate
- Sacrum and Innominate Anatomy
- The Pelvic Axes
- Diagnosis of Innominate Somatic Dysfunction
    - Innominate Shears
    - Innominate Rotations
    - Pubic Shears
- Treatment of Innominate Somatic Dysfunction
    - Direct Treatment of Innominate Somatic Dysfunction
    - Indirect Treatment of Innominate Somatic Dysfunction
- Treatment Examples
- Diagnosis of Sacral Somatic Dysfunction
    - Left Sacral Flexion
    - Right Extension
    - Left on Left Sacral Torsion
    - Left of Right Sacral Torsion
- Treatment of sacral somatic dysfunction
    - Direct Treatment of Sacral Somatic Dysfunction
    - Indirect Treatment of Sacral Somatic Dysfunction
- Treatment Pearls of the Sacrum and Innominates
- Clinical Vignette
- Appendix A: Strain-counterstrain Tenderpoints
- Appendix B: Evidence Based Medicine of Sacral and Innominate Testing
- Appendix C: Chart of Sacral and Innominate Somatic Dysfunction
- Review Questions

Chapter 6: The Sacrum and Pelvis

Pelvic somatic dysfunction is the topic beginning students struggle with most. Often challenging, this topic has many similarities to spinal mechanics, yet it has its own principles that guide diagnosis and ultimately treatment. The content in this chapter is a launching point for advanced understanding and is not intended to be comprehensive; nevertheless, the principles presented are complete within the biomechanical framework.

Sacrum and Innominate Anatomy

Pelvic anatomy provides the framework for understanding normal anatomical motion. Boney structures include the sacrum, coccyx, and innominate. Three bones embryologically form the innominate: the ilium, the ischium and the pubis. These intersect to form the acetabulum where the femur articulates with the pelvis. Three joints form a ring structure of the pelvis; anteriorly, the pubes articulate on the pubic symphysis, and posteriorly the innominates articulate with the sacrum to form the sacroiliac (SI) joints. The fifth lumbar segment sits above the sacrum that forms the foundation of the spinal column. Pelvic landmarks useful for the remainder of this chapter are depicted in figures 1a, 1b, and 2.

Figure 1a

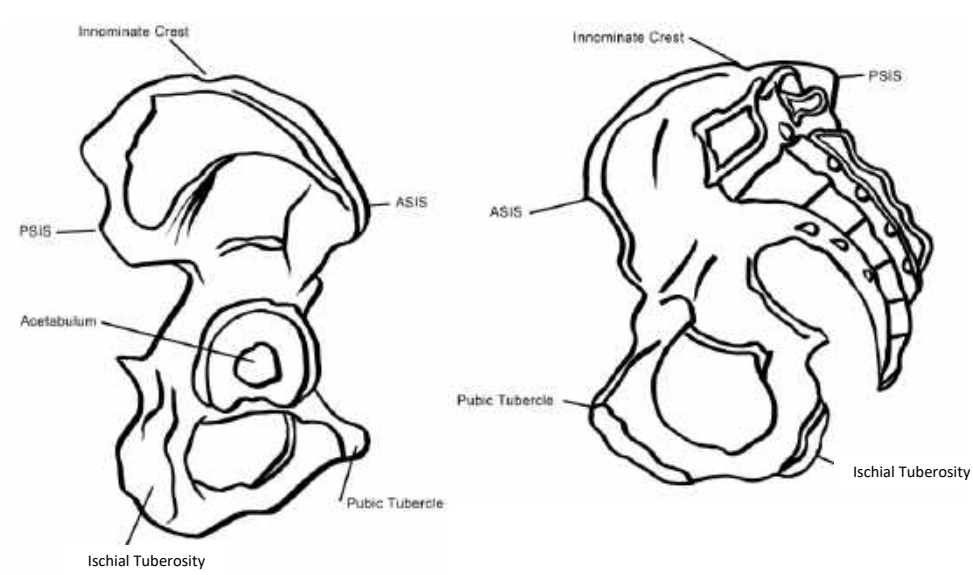


Figure 1a: Innominate Lateral and Medial Views

Notice how the sacrum sits inside the innominates and how the pubes articulate at the pubic symphysis.

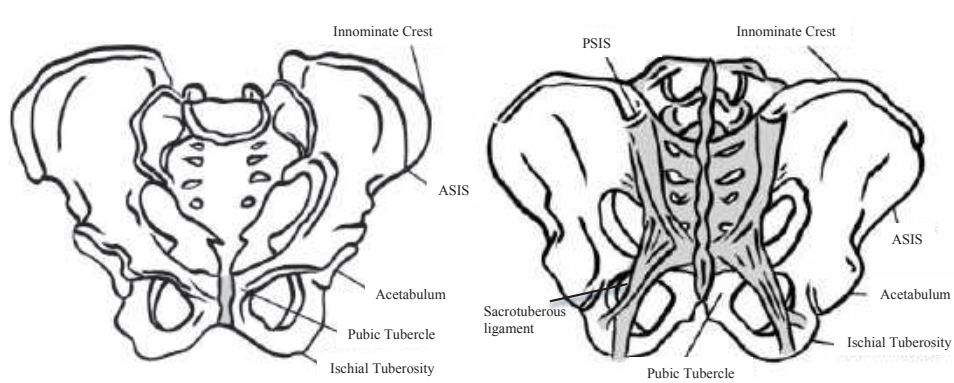
ASIS = Anterior Superior Iliac Spine.
PSIS = Posterior Superior Iliac Spine.

Figure 1b: Pelvis Anterior and Posterior Views

Note the thick posterior suspensory ligaments overlying bilateral SI joints.

ASIS = Anterior Superior Iliac Spine.
PSIS = Posterior Superior Iliac Spine.

Figure 1b



Few muscular structures support the sacrum: the piriformis and gluteus maximus horizontally cross the SI joint, while the longissimus and spinalis attach posterosuperior on the sacral body. The sacrum has a bilateral "L-shaped" concavity that articulates with innominate convexity in a tongue-and-groove manner; this joint surface relationship is altered in segments of the population and predisposes certain patients to somatic dysfunction. Short and long posterior SI ligaments suspend the sacrum between the innominates (Figure 1b); there are relatively few anteriorly supporting SI ligaments.

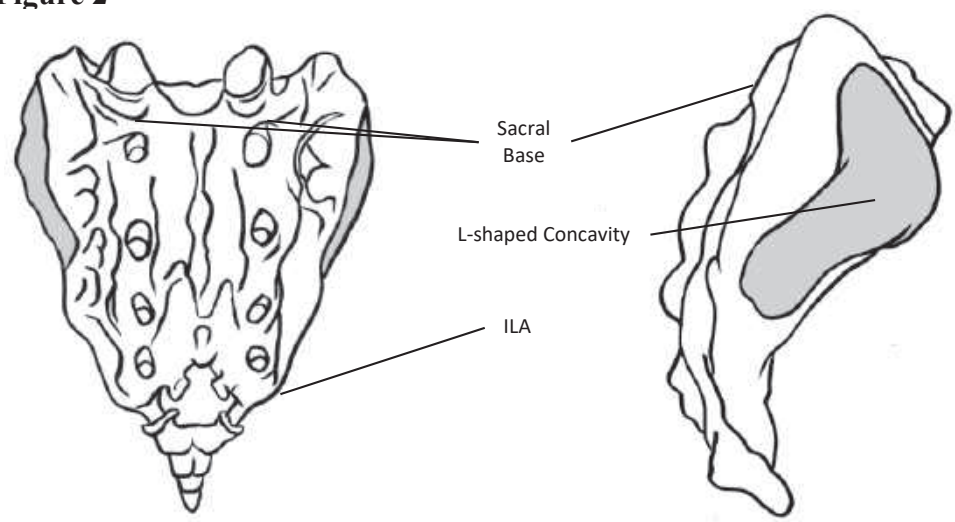
The inherent structures of the SI joint provide a small amount of flexibility and a floating compression that allows subtle motion during gait and spinal flexion and extension. Edward Stiles, DO, calls the sacrum the "great adapter" due to its ability to support change in the spine, pelvis, and lower extremities while maintaining normal pelvic motion. Consequently, the sacrum may demonstrate secondary somatic dysfunction to compensate for the primary somatic dysfunction of the innominate, L5, or the remainder of the body.

Figure 2: The Sacrum

Note how the L-shape concavity of the sacrum will articulate with the innominate in a tongue-and-groove manner.

ILA = Inferior Lateral Angle of Sacrum.

Figure 2



The Pelvic Axes

Pelvic motion remained enigmatic until 1958, when Fred Mitchell Sr., DO, published "Structural Pelvic Function."[1] Through his careful clinical observations, Mitchell introduced six theoretical pelvic axes that define its movement. It is best to conceptualize movement in the pelvis around one axis at a time, although with ambulation, motion occurs on more than one axis.

The first axis occurs horizontally along the pubic symphysis (Figure 3). This axis permits a rotational motion between the pubes during gait,[2(pp308)] where the non-weight baring pube rotates clockwise and counterclockwise. Disruption of this axis may result in unusual sacral mechanics due to the compensatory motion of the iliosacral axes.

Figure 3

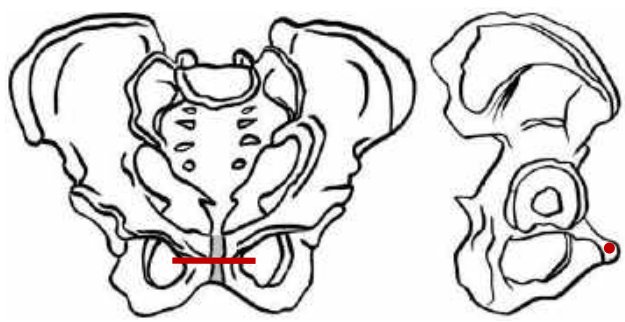


Figure 3: Horizontal Pubic Axis

The Middle Transverse Axis (MTA), also known as the postural axis (Figure 4), nearly passes through the body of the second sacral segment (S_2). While sitting, the sacrum is functionally "floating" between the innominates since it is supported primarily by ligaments. Its motion is limited inferiorly and laterally by the bony anatomy of the innominates.

Figure 4

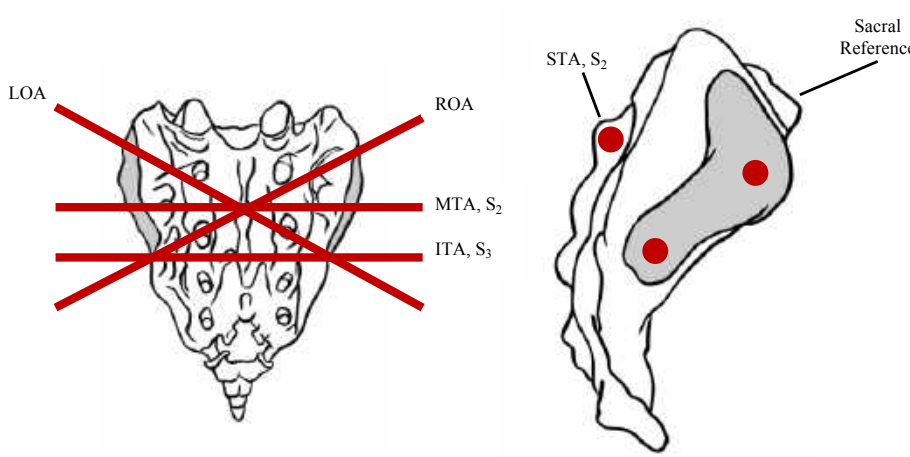


Figure 4: Sacral Axes

5 of the 6 SI axes are depicted looking at the posterior aspect of the sacrum. The axes exist between the sacrum and the innominate.

LOA = Left Oblique Axis
ROA = Right Oblique Axis
STA = Superior Transverse Axis
MTA = Middle Transverse Axis
ITA = Inferior Transverse Axis

Note: Some authorities name a 7th pelvic axis through the sacrum which may create a right or left pathological rotation about the sacrum. We do not observe this in clinical practice.[3(pp1103)]

The reference for naming sacral motion is the anterior-superior sacral base. As in spinal mechanics, the anterior motion of the base is termed flexion and the posterior motion extension. Less commonly used terms for sacral flexion and extension are nutation (the Latin word to "nod") and counternutation, respectively. While seated, sacral flexion and extension occur along the MTA that overlies S2. Notice how the lumbar spine increases its lordotic curve with sacral flexion and straightens with extension (Figure 5). During a static exam, loss or exaggeration of this curve may indicate somatic dysfunction.

Figure 5

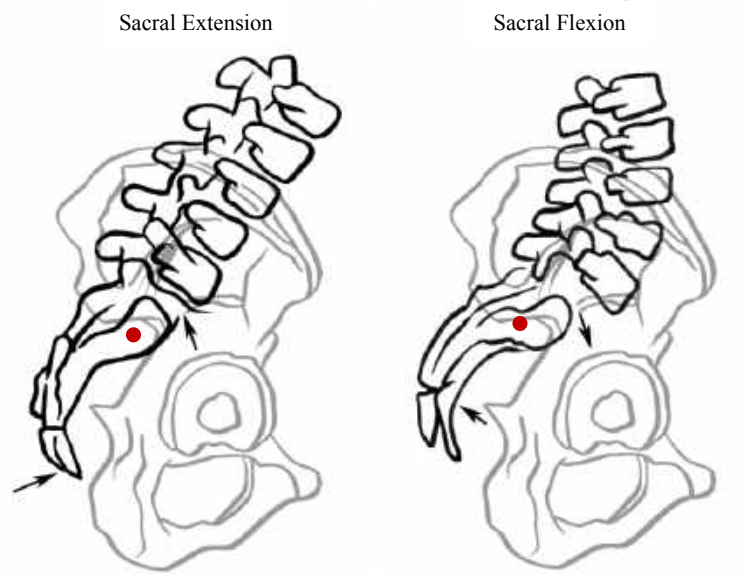


Figure 5: Sacral Flexion and Extension

The anterior-superior sacral base is the reference for naming sacral position in three-dimensional space. Note the increased curvature of the spine in flexion, and loss during extension with rotation around the MTA.

Nutation: anterior sacral movement.

Counternutation: posterior sacral movement.

Superficial palpation of the MTA is performed by locating the posterior superior iliac spines (PSIS) bilaterally and moving the thumbs medially onto the sacral base; in this position the hands fall naturally to S2 (Figure17). Location of the MTA is important for treatment purposes as well as location of the oblique axes.

The right oblique axis (ROA) and the left oblique axis (LOA) are conceptual axes responsible for sacral movement during gait (Figure 4). Clinical observations[3(pp586), 11(pp54)] have shown that during ambulation, when weight is transferred to the right foot, the ROA is the principle axis engaged, and that SI rotation occurs around this axis. Likewise, SI motion occurs around the LOA when weight is transferred to the left. Stiles made the observation that he could localize the MTA and convert it to either the ROA or the LOA by shifting the patient's weight and position; this biomechanical principle is utilized during treatment.

The Superior Transverse Axis (STA) roughly passes through the spinous process of S2, posterior to the dural attachment. Since sacral motion initiated by the primary respiratory mechanism is thought to occur here (see cranial

chapter), this axis is known as the "respiratory axis."[3(pp1103), 11(pp54)] Motion of the sacrum around the STA is palpated by first locating the MTA at S2 and moving one fingerbreadth superiorly to S1; in this position, though not actually on the axis, sacral flexion and extension initiated by the primary respiratory mechanism is appreciated.

The Inferior Transverse Axis (ITA) passes approximately at the level of S3. Clinical observation has shown that anterior and posterior innominate rotation, in relation to the sacrum, occurs about the ITA. Palpating the MTA at S2 and moving one fingerbreadth inferiorly localizes the ITA. Understanding the six pelvic axes is the key to understanding sacral and innominate movement. The principles taught throughout the remainder of this chapter are diagnosing somatic dysfunction of the pelvis and restoration of normal range of motion (ROM) using Mitchell's axes.

Diagnosis of Innominate Somatic Dysfunction

Recall that abnormal pelvic movement due to somatic dysfunction of the sacrum is named sacroiliac dysfunction. Conversely, somatic dysfunction of the innominates that cause changes in the sacrum is called iliosacral dysfunction. Two active and one passive range of motion (ROM) tests define the presence or absence of biomechanical pelvic dysfunction: the ASIS compression test, the standing forward bending test (SFBT), and the stork test. Used alone, or in a corroborative manner, these tests identify restricted ROM of the innominates in relation to the sacrum; for all three tests, a positive test identifies ipsilateral somatic dysfunction of the innominate.

The SFBT, ASIS compression test, and the stork test identify the side of somatic dysfunction.

Utilizing the ASIS compression test,[3(pp588), 4(pp210)] a provider can test the STA, MTA, and ITA for motion restriction. This test is performed on a supine patient; the operator, places the thenar eminence of both hands on the patient's ASIS (Figure 6).

Figure 6

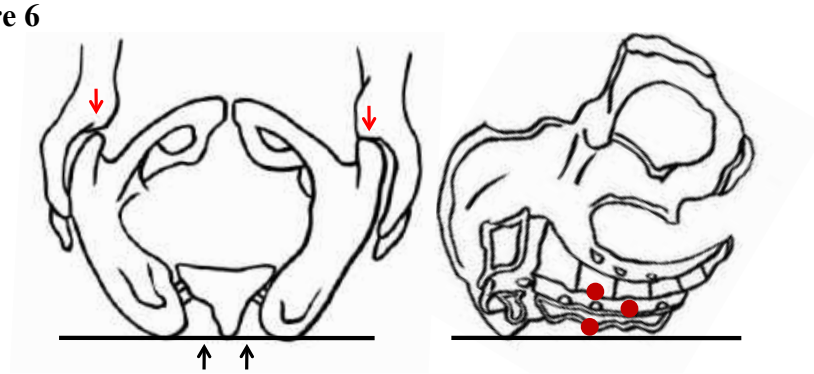


Figure 6: ASIS Compression Test

Each innominate is tested separately in a rocking motion to identify restriction in the STA, MTA, or ITA. Note, the operator can face the head or the feet of the patient when performing this test. The table provides stability to the sacrum as indicated by the arrows. This ASIS compression test evaluates iliosacral dysfunction.

To test the right hemipelvis, the left side is stabilized, and the operator compresses down toward the table; to test the left, the process is repeated with the right hemipelvis stabilized. Motion directed perpendicular to the table

tests the ITA. Motion directed superiorly, at angles of approximately 15° and 30°, evaluate the MTA and STA, respectively. Somatic dysfunction is present when restricted motion is present on one side compared with the other and is termed the *positive ASIS compression test*. This passive motion test is useful in confirming restricted ROM in the stork test and the SFBT, and as we will see, positioning the patient for treatment.

The SFBT[2(pp312)] evaluates motion of the innominates around the ITA (Figure 7). The SFBT is performed with patient standing, feet about 15-30 centimeters apart. The physician stands behind the patient with thumbs palpating both PSIS inferiorly. As the patient bends forward, the physician maintains contact with the posterior superior iliac spines (PSISs).

Figure 7

Figure 7: The SFBT

This test assesses ilio-sacral somatic dysfunction.

With normal motion, the PSISs should move symmetrically superior. When somatic dysfunction is present, one PSIS will move a fingerbreadth further superior than the other, indicating pathology on that side. For instance, if the PSIS on the left moves more superior than the right, this would be regarded as a *positive left SFBT* and indicate pathology in the left innominate.

The stork test (aka Gillet test)[2(pp313), 4(pp207-8)] is performed with the patient standing, feet apart ~ 15-30 centimeters. The physician stands behind the patient with one thumb palpating the PSIS inferiorly and the other in the middle of the sacral base to serve as a reference point (Figure 8). The patient flexes the hip 90° while the physician maintains contact with the PSIS and the center of the sacral base. The test is then repeated with the opposite extremity. With normal motion, the PSIS should move symmetrically inferior. When somatic dysfunction is present, the PSIS will fail to move inferiorly and may in fact move superiorly, indicating dysfunction on that side. For instance, if the PSIS on the right moves less inferior than the left, this would be regarded as a *positive right stork test* and indicates right innominate somatic dysfunction.

Figure 8

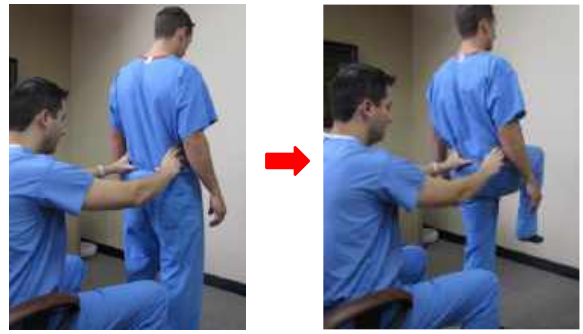


Figure 8: The Stork Test

This test evaluates iliosacral somatic dysfunction. Note, that while performing the stork test, we simultaneously perform the Trendelenburg test (see Lower Extremity chapter).

The pelvis has twelve commonly recognized somatic dysfunctions: innominate superior and inferior shears, anterior and posterior innominate rotations, and pubic superior and inferior shears. We present six of the twelve in Table 1 with their characteristic physical exam findings. The remaining six dysfunctions have similar characteristics of their counterpart on the opposite side and are listed in Appendix C.

Table 1

	SFBT	ASIS	PSIS	Crest Height	Pubes	Leg Length
L-sup. innominate shear	+ Left	L-superior	L-superior	L-superior	+/- L-superior	L-short
R-inf. innominate shear	+ Right	R-inferior	R-inferior	R-inferior	+/- R-inferior	R-long
R-anterior inn. rotation	+ Right	R-inferior	R-superior	Symmetrical	Symmetrical	R-long
L-posterior inn. rotation	+ Left	L-superior	L-inferior	Symmetrical	Symmetrical	L-short
L-sup. pubic shear	+ Left	L-lateral	Symmetrical	Symmetrical/high	L-Superior	L-long
R-inf. pubic shear	+ Right	R-medial	Symmetrical	Symmetrical/low	R-inferior	R-short

Table 1: Common Patterns of Iliosacral dysfunction[5]

Note that the side of the SFBT indicates the pathological side. With regard to this chart, SFBT, Stork, or ASIS compression test could be used interchangeably, to indicate the side of pathology. Leg length is functional, not anatomical.

The SFBT correlates with the side of somatic dysfunction. Confirmation of a positive SFBT is made with the stork test, or by localizing the ITA with the ASIS compression test, which, should exhibit decreased "spring" on the pathological side.

Innominate Shears

A *left superior innominate shear* commonly occurs when someone walking on level ground unknowingly steps off of a curb; the absence of left gluteal firing during a misstep may cause disruption of the left pelvic ligaments allowing pathological innominate movement superiorly (Figure 9).

Figure 9

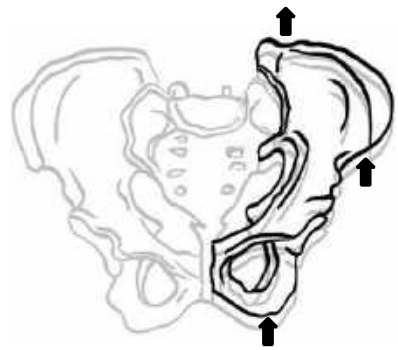


Figure 9: Left Superior Innominate Shear

Notice that the crest height, ASIS, PSIS are all superior when compared to the opposite side. The pube may or may not be involved.

Functionally short leg: short leg due to somatic dysfunction.

Anatomically short leg: short leg due to unequal structural length of the lower extremity.

- If the patient has an anatomically short or long leg, measuring leg length may be misleading. Hence, we do not commonly check leg length during diagnosis.
- Simultaneous pubic superior shear with innominate superior shear may result in apparent leg length equality.

Usually, the patient recognizes the moment of injury as pain immediately ensues. During a hemipelvic shear, the STA, MTA, ITA and potentially the pubic axis, are disrupted, resulting in severe gait debilitation. A left superior innominate shear will have the following findings: positive left SFBT, superior left PSIS and ASIS, an elevated left crest height while standing, sitting, or lying, and a functionally short left leg. The left pubic tubercle may be superior or remain level compared with the right.

One important diagnostic distinction is the difference between a functionally short leg and an anatomically short leg: a functionally short leg appears so due to somatic dysfunction but resolves after treatment, whereas, an anatomically short leg is due to unequal length of the lower extremity. Comparison of leg length is performed on a supine patient by comparing the medial aspect of the right and left distal malleolus.

A commonly overlooked diagnostic criterion in an innominate superior-shear is that the PSIS, ASIS, and crest height remain superior in lying, sitting, and standing positions. As discussed in the lumbar spine chapter, if these landmarks resolve when the patient is in a sitting or lying position, the patient may have an anatomically short leg.

A *right inferior innominate shear* is usually the result of sudden, downward traction on the lower extremity (Figure 10).

Figure 10

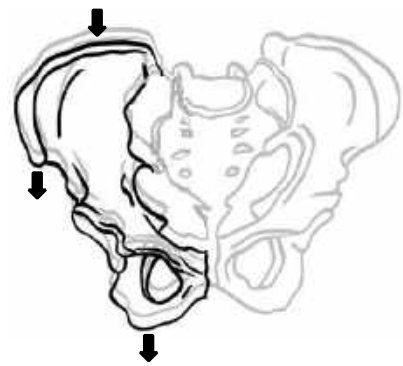


Figure 10: Right Inferior Innominate Shear

Notice that the crest height, ASIS, PSIS are all inferior when compared to the opposite side. Pubic height asymmetry may or may not be involved.

For example, this pathology is seen when an equestrian's lower extremity is trapped in a stir-up, and they are dragged along the ground by their horse. Right inferior innominate shears have the following findings: positive right SFBT, inferior right PSIS, ASIS, a low right crest height with standing, sitting, or lying, and a functionally long right leg.

Innominate Rotations

Two types of hemipelvic rotations occur: anterior and posterior. These rotations occur about the ITA and can be confirmed by using the ASIS compression test or the stork test to localize the side of somatic dysfunction.

A *right anterior innominate rotation* (Figure 11) commonly occurs as compensation for a short right anatomical leg or is the result of a hamstring muscle spasm.

Figure 11

Figure 11: Right Anterior Innominate Rotation:

Notice that the crest height is level, ASIS inferior, and PSIS superior when compared to the opposite side. Pubic asymmetry is usually not observed.

Right anterior innominate rotations have the following findings: right positive SFBT, right superior PSIS, right inferior ASIS, symmetrical crest height, symmetrical pubic tubercle height, and a functionally long right leg.

A *left posterior innominate rotation* (Figure 12) is usually caused by bio-mechanical compensation for an anatomically long left leg or a taut left psoas (muscle imbalance).

Figure 12

Figure 12: Left Posterior Innominate Rotation

Notice that the crest height is level, ASIS superior, and PSIS inferior when compared to the opposite side. Pubic asymmetry is usually not be observed.

Left posterior innominate rotations have positive left SFBT, inferior left PSIS, superior left ASIS, symmetrical crest height, symmetrical pubic tubercle height, and a functionally short left leg. This dysfunction is very common but is usually compensatory due to another underlying process.

Pubic Shears

Pubic shears are classified as either superior or inferior. Superior and inferior shears occur frequently after vaginal childbirth that disrupts the pubic axis. *Left superior pubic shears* (Figure 13) have the following left-sided findings:

positive left SFBT, superior left pube, symmetrical PSIS height, symmetrical to lateral ASIS, symmetrical to low iliac crest height, and a functionally long left leg.

Figure 13

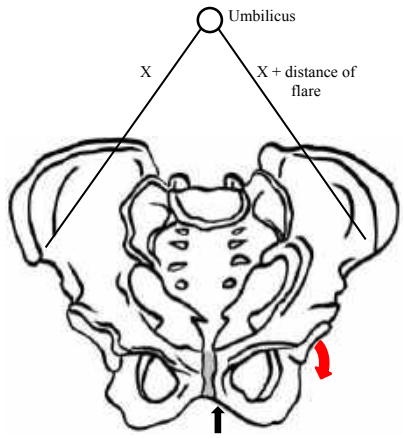


Figure 13: Left Superior Pubic Shear

Notice that on the side of the superior shear, the pelvis flares laterally, and the acetabulum moves inferiorly (red arrow) resulting in a functionally long leg.

Notice that the left ASIS, and therefore innominate, is more lateral to the umbilicus than the right. This is denoted as "X + distance of flare."

Counter to intuition, note that the leg is functionally longer on the side of superior shear. Functional leg lengthening occurs mechanically; as the pubic tubercle moves up, the pelvis flares laterally, and the acetabulum moves inferior carrying with it the femur.

Figure 13 also demonstrates lateral flare of the left innominate consistent with a *left superior pubic shear*. Pelvic flare is measured using the umbilicus as a center reference point and the anterior superior iliac spines (ASISs) as lateral landmarks. Greater distance from the center indicates lateral flare and less distance medial flare.

Right inferior pubic shears occur when unilateral downward traction is placed on the right leg; for instance, riding on a ski lift with a snowboard dangling from the right leg. They have the following right-sided findings: positive right SFBT, right inferior pube, normal PSIS, normal to medial ASISs, normal to high crest height, and a functionally short right leg. Essentially, all findings are opposite those of the superior pubic shear (Figure 14).

Figure 14

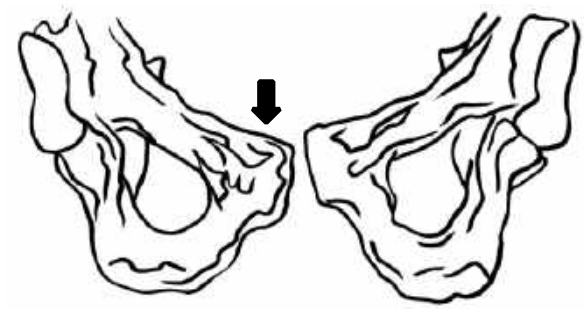


Figure 14: Right Inferior Pubic Shear

Note that on the side of the inferior shear, the pelvis flares medially, and the acetabulum moves superiorly resulting in a functionally short leg.

Many practitioners claim that medial and lateral pelvic flares occur independent of pubic shears. They observe that medial flares arise from pubic symphysis compression and are lateral with distraction. The pubic symphysis is composed of rigid cartilage and although slight compression may occur, it is doubtful that clinical findings would be evident. We believe these are misdiagnosed pubic shears. Fortunately, if pelvic flares do occur, they are managed in the same manner as pubic shears with distraction and re-approximation.

Treatment of Innominate Somatic Dysfunction

When using pelvic mechanics to treat innominate pathology directly or indirectly, knowledge of the positional diagnosis is all that is necessary. Treatment modalities, while numerous, are dictated by simple and easy to understand principles based on anatomy, use of the pelvic axis, and positional diagnosis.

Direct Treatment of Innominate Somatic Dysfunction

Use the example diagnosis of a *right anterior innominate rotation* due to hamstring spasm. Direct treatment involves reversal of the positional diagnosis by placing the right innominate in posterior rotation. The practitioner then utilizes a fulcrum, lever, and force as is done in any direct procedure. An important element, in treating the pelvis, is use of a rotational-fulcrum formed by localizing motion to the ITA. To summarize, the principles in direct treatment of a right anterior innominate rotation are:

1. Obtain the positional diagnosis *right anterior innominate rotation.*
2. Localize the fulcrum to the ITA.
3. Reverse the components of the positional diagnosis by placing the patient against the barrier in right posterior innominate rotation.
4. Create a force by using an impulse or isometric contraction.

The principles used in direct treatment remain the same regardless of the body region treated. For further details using lateral and supine positions, please see the examples provided for high velocity low amplitude (HVLA) and muscle energy (ME).

Indirect Treatment of Innominate Somatic Dysfunction

Conceptually, indirect procedures are much simpler than direct. They are however, more difficult for students beginning practice. When using the positional diagnosis of *right anterior innominate rotation,* exaggerate the position while using the ITA. To summarize, the steps:

1. Obtain the positional diagnosis *right anterior innominate rotation.*
2. Localize motion to the ITA.
3. Place the right innominate in anterior rotation.
4. Exaggerate the anterior rotation to a position of three-dimensional balance or patient comfort.

As we will see, direct and indirect treatments of the sacrum are very similar to the innominates. To appreciate this similarity, the student must first understand the biomechanics behind the four common sacral dysfunctions.

Treatment Examples: Direct Procedures

Right Anterior Innominate Rotation using ME

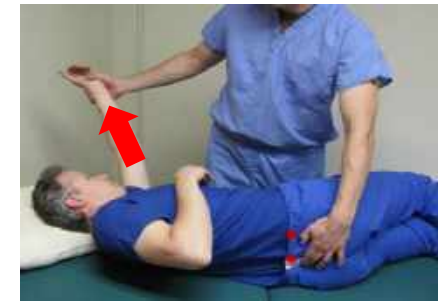
1. With the patient supine, localize the ITA (fulcrum) using the ASIS compression test as you abduct and adduct the right leg. The ITA will be perpendicular to the table.

2. Once motion is felt on the ITA, create a lever by flexing the right thigh toward the patient's chest. This also reverses the right anterior innominate into a posterior position.

3. Create an isometric contraction by having the patient straighten their leg while the operator resists and lifts anteriorly on the ischial tuberosity for 3-5 seconds. Instruct the patient to relax. Reposition at the new barrier by posteriorly rotating the innominate. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
4. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Right Anterior Innominate Rotation using HVLA

1. Place the patient in a left lateral recumbent position with the right innominate up. Locate the ITA posteriorly (MTA is labeled with red dots).
2. Pull the patient's left arm, creating type I spinal mechanics until motion is felt on the ITA. This locks down the spine to the sacrum.



3. Create a lever with the thigh, by flexing and extending the spine until motion is felt on the ITA. Place the patient's right foot behind their left leg to lock in this position.

4. Take hold of the posterior innominate with the left hand. The operator controls the trunk with the patient's right arm. Have the patient breathe in and out to take up further slack. Feel for the closed packed position and the direction of free motion into this locked position. Create a posterior rotational impulse along the direction of free motion as shown by the arrows.

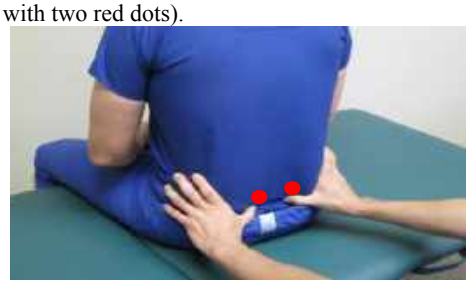
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

The principle in HVLA treatment of innominate somatic dysfunction is to place the pathological side up.

Treatment Examples: Indirect Procedures

Right Anterior Innominate Rotation using Functional Procedure

1. With the patient sitting on the table, locate the ITA (the MTA is labeled with two red dots).



2. Place the right hand and forearm across the patient's shoulders to gain control of the trunk. Flex and extend the patient until motion is felt on the ITA.

3. To find the Dynamic Balance Point (DPB), loose pack the right SI joint by gently leaning the patient to the right. This further anteriorly rotates the right innominate along the ITA.
4. Add gentle compression from the patient's left shoulder toward the left ischial tuberosity. Monitor the right innominate along the ITA as it changes using the left hand and adjust the flexion-extension, sidebending, and rotation to maintain balance. If the DPB reaches a still point, wait for 10-20 seconds. If there is no change, challenge the tissues by either increasing or decreasing compression. When the tissue relaxes the treatment is finished.

5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

The principle taught in this functional procedure is to loose-pack the restricted SI joint (the side of the SFBT) and add compression into the SI on the side of sacral rotation (the left). To treat a left posterior innominate, loose pack and add compression on the left side.

Right Anterior Innominate Rotation using S/CS (aka Posterior Ischial Tuberosity/Hamstring TP)

1. The tender point for the anteriorly rotated innominate will be at the hamstring attachment to the ischial tuberosity.
2. With the patient supine, find the ITA using the ASIS compression test as you abduct and adduct the right leg. The ITA will be perpendicular to the table.

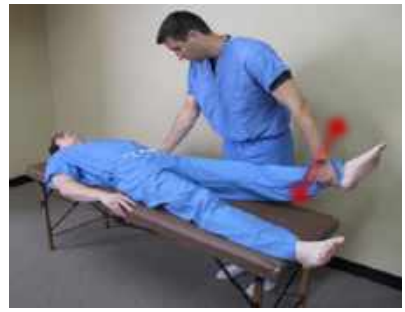
3. Monitor the ischial tuberosity TP with one finger; tell the patient that this tenderness represents a 10 on a scale of 1-10. Further anterior rotate the innominate and introduce compression, distraction, and internal and external rotation of the thigh until tenderness is a 3 out of 10.

4. Maintain this position for 90 seconds. You do not have to monitor the tender point during this time. If you choose to monitor, you may feel characteristic tissue changes.
5. SLOWLY reduce anterior rotation and return the patient's leg to the table
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

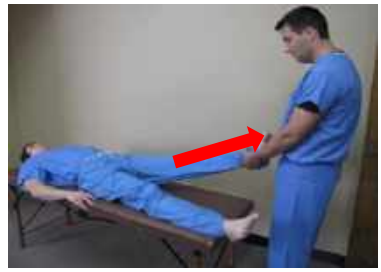
Treatment Examples: Direct/Indirect Procedures

Left Superior Innominate Shear using HVLA

1. With the patient supine, locate the ITA (fulcrum) using the ASIS compression test as you abduct and adduct the right leg. The ITA will be perpendicular to the table.



2. Once motion is felt on the ITA, create a lever by straightening the lower extremity.



3. Have the patient hold the table firmly to prevent sliding on the table during the impulse.
4. The operator internally rotates the lower extremity to lock the femoral head into the glenoid fossa, further lengthening the lever. Using distraction, localize motion to the SI joint with joint play.
5. Instruct the patient to breathe in and out to take up further slack. Create a distractive impulse as shown above.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Left Superior Innominate Shear using Functional Procedures

1. With the patient sitting on the table, locate the ITA (the MTA is labeled with two red dots.

2. Place the right hand and forearm across the patient's shoulders to gain control of the trunk. Flex and extend the patient until motion is felt on the ITA.

3. To find the DBP, loose pack the left SI joint by gently leaning the patient to the right.
4. Add gentle compression from the patient's right shoulder toward the right ischial tuberosity. Monitor the right SI through the ITA as it changes using the left hand and adjust the flexion-extension, sidebending, and rotation to maintain balance. If the DPB reaches a still point, wait for a 10-20 seconds; if there is no change, challenge the tissues by either increasing or decreasing compression. When the tissue relaxes the treatment is finished.

5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

The principle taught in this functional procedure is to loose-pack the restricted SI joint (the side of the SFBT) and add compression into the SI on the side of sacral rotation (the right). To treat a L-innominate inferior-shear, loose pack and add compression on the left side.

Treatment Examples: Direct/Indirect Procedures

Left Superior Pubic Shear using ME

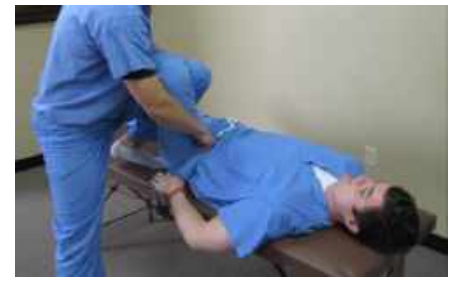
1. With the patient supine, locate the ITA (fulcrum) using the ASIS spring test as you abduct and adduct the right leg. The ITA will be perpendicular to the table.

2. Once motion is felt on the ITA, create a lever by lowering the thigh off the table.

3. The operator places his right hand on innominate to create a stable fulcrum.
4. Create an isometric contraction by instructing the patient adduct their thigh for 3-5 seconds. This should cause inferior movement of the pubic bone due to reversal of the origin and insertion of the adductor complex. Instruct the patient to relax. Reposition at the new barrier by further abducting the thigh. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Left Superior Pubic Shear using S/CS (Low Ilium)

1. With the patient supine, find the ITA using the ASIS spring test as you abduct and adduct the right leg. The ITA will be perpendicular to the table.
2. Monitor the left pubic tubercle TP with one finger; tell the patient that this tenderness represents a 10 on a scale of 1-10. Further posteriorly, rotate the innominate and introduce compression, distraction, internal and external rotation of the thigh until tenderness is 3 out of 10.

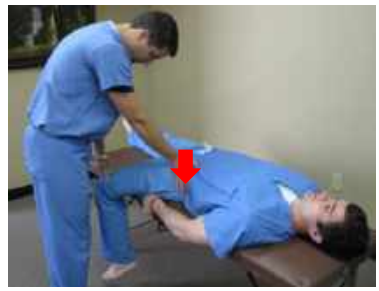


3. Maintain this position for 90 seconds. You do not have to monitor the tender point during this time. If you choose to monitor, you may feel characteristic tissue changes.
4. SLOWLY return the patient's leg to the table
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

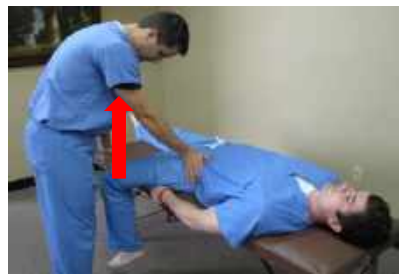
Treatment Examples: Direct Procedure

Left Posterior Innominate Rotation using ME

1. With the patient supine, localize the ITA (fulcrum) using the ASIS spring test as you abduct and adduct the left leg. The ITA will be perpendicular to the table.



2. The operator stabilizes the left ASIS with their right hand and holds the patient's thigh with his left. A lever is already formed by the lower extremity that also reverses the left posterior innominate into an anterior position.



3. Create an isometric contraction by instructing the patient to flex his thigh for 3-5 seconds. Instruct the patient to relax. Reposition at the new barrier by anteriorly rotating the innominate. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
4. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Left Posterior Innominate Rotation using HVLA

1. Place the patient in a right lateral recumbent position with the left innominate up. Locate the ITA posteriorly (MTA is labeled with red dots).
2. Pull the patient's left arm, creating type I spinal mechanics until motion is felt on the ITA. This locks down the spine to the sacrum.

3. Create a lever with the lower extremity/pelvis by flexing and extending the spine until motion is felt on the ITA. Place the patient's left foot behind their left leg to lock-in this position.

4. Take hold of the innominate with the right hand, the other hand controls the trunk with the patient's left arm. Instruct the patient to breathe in and out to take up further slack. Feel for the closed packed position and the direction of free motion into this locked position. Create an anterior rotational impulse along the direction of free motion as shown by the arrows.

5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

The principle in HVLA treatment of innominate somatic dysfunction is to place the pathological side up.

Treatment Examples: Indirect Procedures

Left Posterior Innominate Rotation using Functional Procedures

1. With the patient sitting on the table, locate the ITA (the MTA is labeled with two red dots.

2. Place the right hand and forearm across the patient's shoulders to gain control of the trunk. Flex and extend the patient until motion is felt on the ITA.

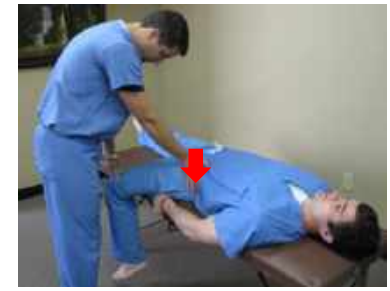
3. To find the DBP, loose pack the left SI joint by gently leaning the patient to the right.
4. Add gentle compression from the patient's left shoulder, toward the left ischial tuberosity. Monitor the left SI as it changes using the left hand and adjust the flexion-extension, sidebending, and rotation to maintain balance. If the DPB reaches a still point wait for a 10-20 seconds; if there is no change, challenge the tissues by either increasing or decreasing compression. When the tissue relaxes the treatment is finished.

5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

The principle taught in this functional procedure is to loose-pack the restricted SI joint (the side of the SFBT) and add compression toward the ischial tuberosity on the side of sacral rotation (the left).

Left Posterior Innominate Rotation using S/CS (aka Iliacus TP)

1. Left posterior innominate rotations are commonly caused by spasm of the iliopsoas muscle that is the primary hip flexor.
2. With the patient supine, localize the ITA using the ASIS spring test as you abduct and adduct the right leg. The ITA will be perpendicular to the table.



3. Tenderness is located in the psoas major muscle superior to the inguinal ligament. Monitor the TP with one finger; tell the patient that this tenderness represents a 10 on a scale of 1-10. Posteriorly rotate the innominate and introduce compression, distraction, internal, and external rotation of the thigh until tenderness is 3 out of 10.

4. Maintain this position for 90 seconds. You do not have to monitor the tender point during this time. If you choose to monitor, you may feel characteristic tissue changes.
5. SLOWLY reduce anterior rotation and return the patient's leg to the table.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Diagnosis of Sacral Somatic Dysfunction

NOTE: Sacral somatic dysfunction is present if the ILA is inferior/posterior compared to the contralateral side.

Diagnosis of sacral somatic dysfunction remains one of the most challenging concepts in manual medicine. Among the major schools in Europe and the USA, a single naming system has yet to be formulated. In osteopathic education there are two separate naming systems: the Chicago model developed at the Chicago College of Osteopathic Medicine and the Mitchell model developed by Fred G. Mitchell, Sr., DO, FAAO. Both describe the sacrum according to its positional diagnosis and are named in relation to the innominate. This section attempts to clarify sacral biomechanics through the use of anatomy and the pelvic axes according to the Mitchell Model. For historical preservation, the corresponding Chicago diagnoses are also mentioned.

There are four common sacral pathologies: unilateral sacral flexion, unilateral sacral extension, anterior sacral torsion, and posterior sacral torsion. Each of them occurs on the right or left side, totaling eight sacral diagnoses. Sacral somatic dysfunction is present if the inferior lateral angle of the sacrum (ILA) is un-level compared to the contralateral side (i.e., inferior and posterior). This bears repeating, sacral somatic dysfunction is present if the patient has an un-level ILA that will be inferior and posterior compared with its contralateral side. We present four common sacral somatic dysfunctions based on an inferior/posterior left ILA (Table 2); appendix C summarizes all eight diagnoses.

Table 2: Common Diagnoses of the Sacrum

Note that all are based on an inferior and posterior ILA on the left.

Only the ILA, SitFBT, and the BB-Test are needed to diagnose the eight sacral dysfunctions. These tests are differentiated from other supporting findings by the vertical black line in the table. The other positional landmarks serve to confirm the diagnosis.

- L-Flexion = Left Unilateral Sacral Flexion
- R-Extension = Right Unilateral Sacral Extension
- L/L Torsion = Left on Left Sacral Torsion
- L/R Torsion = Left on Right Sacral Torsion

ILA = Inferior Lateral Angle
SitFBT = Sitting Forward Bending Test
BB-Test = Backward Bending Test

Table 2

	ILA	SitFBT	BB-Test	Base Depth	Rotation L5	Leg Length
L-Flexion	Inf/Post Left	+ Left	Negative	Ant. Left	Left	Long Left
R-Extension	Inf/Post Left	+ Right	Positive	Post. Right	Left	Short Right
L/L Torsion	Inf/Post Left	+ Right	Negative	Ant. Right	Right	Long Right
L/R Torsion	Inf/Post Left	+ Left	Positive	Post. Left	Right	Short Left

The remaining four sacral dysfunctions include R-flexion, L-extension, R/R torsion, and R/L torsion, all of which exhibit an inferior and posterior right ILA. Once a student understands the clinical findings of the four dysfunctions presented in this chapter, those with a right posterior and inferior ILA follow logically and are omitted for brevity sake. Since there are eight total sacral diagnoses, knowledge of three key clinical findings are needed for diagnosis: ILA position, the Sitting forward bending test (SitFBT), and the Backward Bending test (BB-Test). Mathematically, the equation $2^3=8$ describes the eight diagnoses that are confirmed by two anatomical sides - right and left, and three diagnostic tests.

The authors acknowledge the potential diagnoses bilateral sacral flexion and bilateral sacral extension that are listed in osteopathic literature.[2(pp329), 3(pp594), 4(pp220)] Clinically, we rarely encounter these pathologies and more often observe them in patients with bilateral osteoarthritis, bilateral sacroiliac ligament laxity, spondylolisthesis of the lumbar spine, and bilateral sacroiliitis as seen in Crohn's disease, ulcerative colitis, ankylosing spondylitis, and psoriatic arthritis (HLA-B27 pathology). Additionally, it is challenging for advanced practitioners of manual medicine to correctly diagnose bilateral sacral flexion or extension, as patients exhibit bilaterally symmetrical ILA, level/symmetrical sacral base, negative SitFBT, and symmetrical leg length.

The SitFBT[2(pp316), 4(pp209)] evaluates sacroiliac motion. It therefore tests motion of the sacrum around the MTA in relation to the innominate. The SitFBT is performed with the patient sitting, feet resting on a level surface. The physician is behind the patient with thumbs palpating both PSISs inferiorly (Figure 15). As the patient bends forward, the physician maintains contact with the PSISs. With normal motion, the PSISs should move symmetrically superior. Pathology is present if one PSIS, for instance the left, glides a fingerbreadth more superior than the right, indicating a restriction in motion around the MTA.

Figure 15

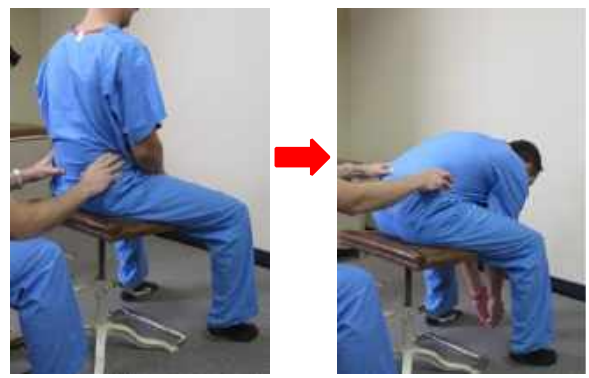


Figure 15: The SitFBT

Notice that the patient's feet are flat on the floor and that the operator's thumbs are inferior to the PSIS. The SitFBT evaluates sacroiliac somatic dysfunction.

The side of the Superior PSIS correlates with the side of the somatic dysfunction.

The side of the superior PSIS correlates with the side of the somatic dysfunction, and in this instance, is named *positive left SitFBT*. It is imperative that the patient's feet remain on the floor since the legs and thighs prevent anterior rotation of the pelvis around the acetabulum and allow the sacrum to move on the MTA independent of the innominate.

Evaluate the ILA position[2(pp321), 4(pp212)] on the prone patient and palpate the inferior-lateral sacral angle by comparing the right to the left (Figure 16). If the left ILA, for instance, is found to be inferior and posterior, it is named *left inferior and posterior ILA*. If the ILAs are symmetrical bilaterally, it is

Figure 16:
ILA Examination

Thumbs are used to evaluate the ILA. Note that the sacrotuberous ligament will also be taught on the side of the inferior and posterior ILA.

unlikely that unilateral sacral dysfunction is present. Concomitantly, the sacrotuberous ligament will be taut on the side of the inferior and posterior ILA.

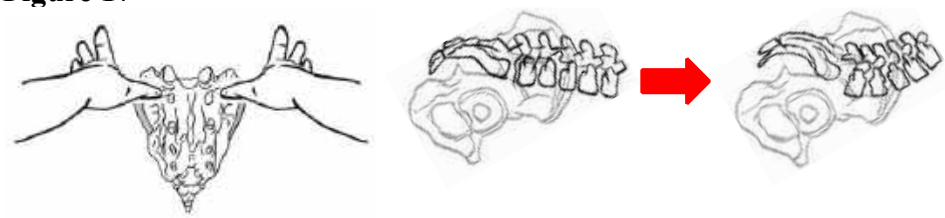
Figure 16

The backward bending test (BB-test) aka sphinx test[2(pp323), 3(pp690)] compares the right sacral base to the left and ultimately establishes the position of the sacrum with respect to the innominates. This test is typically performed on a prone patient (Figure 17).

Figure 17: Backward Bending Test

Thumb placement on the sacral base is demonstrated. Note that the sacrum should move from neutral to flexion as the patient extends their spine.

Figure 17



The operator palpates the sacral base bilaterally and compares the left and right depth while the patient is in a neutral position. The operator then directs the patient to move to their elbows, inducing lumbar spinal extension; this moves the sacral base anteriorly into flexion. In a non-pathological sacrum, the right and left base appear symmetrical in neutral and flexed positions.

A positive test is noted when the base is initially un-level, for instance the right more posterior than the left, and the un-leveling of the base worsens during lumbar extension. The previous example would be named a *positive right BB-test*. If, however, the initial un-level base becomes level during lumbar extension, the test is regarded as a *negative BB-test*. Ultimately, this test measures the ability of the sacral base to glide into flexion around the MTA, as is normal during gait and lumbar extension.

For instance, a *left unilateral sacral flexion* should initially appear un-level and improve during lumbar extension yielding a *negative BB-test* (Figure 18).

Figure 18

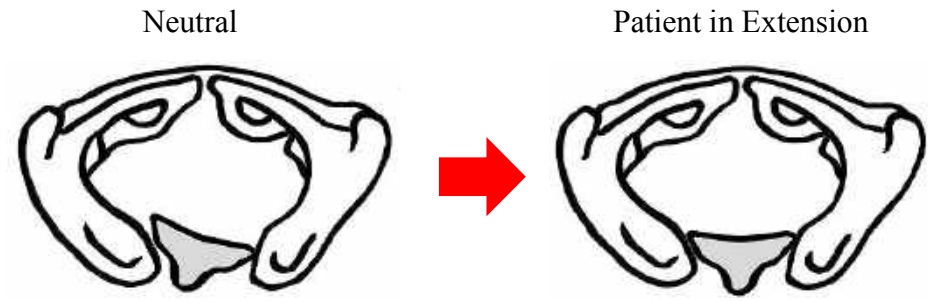


Figure 18: Negative BB-test of a Left Sacral Flexion

Note that the sacral base appears anterior on the left and posterior on the right in the prone position. During lumbar extension, the right sacral base moves anteriorly, and hence, the sacral bases are level indicating a negative BB-test.

This occurs because the left pathological base is already anterior, and the right is in neutral relative to the innominates. During spinal extension, the right sacral base glides into flexion and becomes level with the left.

A *right unilateral sacral extension* will initially appear exactly like a *left unilateral sacral flexion* with a left anterior base (Figure 19). However, an un-level base becomes more apparent during lumbar extension resulting in a *positive BB- test*.

Figure 19

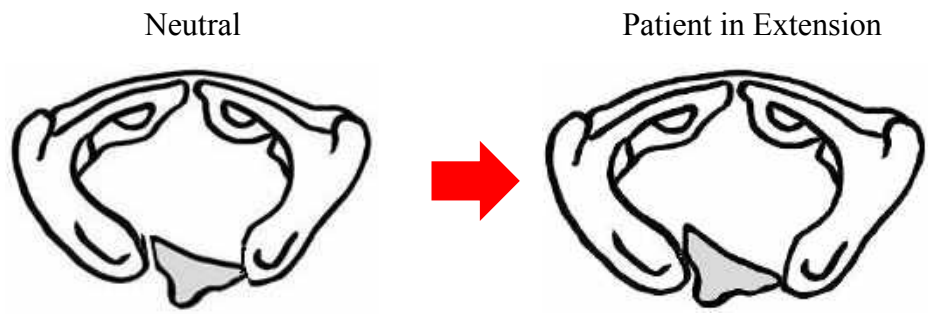


Figure 19: Positive BB-Test of a Right Sacral Extension

Notice that the sacral base appears anterior on the left and posterior on the right while in the prone position. During lumbar extension, the sacral bases become more un-level indicating a positive BB-test.

In this instance, the left base is actually in a neutral position and the right posterior in relation to the innominates. During spinal extension, the left sacral base glides into flexion; the right sacral base, however, is unable to move anteriorly and clinically appears as if it is further posterior.

To review, a pathologically anterior sacral base will have a negative BB-test, while a pathologically posterior sacral base will be positive. Many practitioners use a spring test[2(pp323),4(pp212)] to supplement the BB-test. It is performed on a prone patient by pressing anteriorly on the center of the sacral base to assess for a soft or hard-end feel. The test is negative if spring is felt and is positive with a hard end feel.

A *positive spring test* indicates a pathologically posterior sacral base, and thus is identical to a positive BB test. Likewise, a *negative spring test* yields to springing and is identical to a negative BB-test.

NOTE: The Spring Test will always yield the same result as a BB-test but does not identify the side (right or left) of the dysfunctional sacral base.

NOTE: If somatic dysfunction is present, L5 and the sacrum should always rotate oppositely.

Unless there is sacralization of L5 or lumbarization of a S1 when sacral somatic dysfunction is present, the fifth lumbar segment will rotate **opposite** the sacrum.[3(pp594-6)] This opposite rotation allows the sacrum to adapt to the somatic dysfunction above and below and maintain postural balance and normal spinal and pelvic mechanics - hence its nickname "the great adapter." The next section reviews four common sacral dysfunctions in detail.

Left Sacral Flexion

Left sacral flexion is clinically common; this dysfunction is due to anterior shear of the left sacral base with disruption of the MTA (Figure 20). On exam, while in neutral the left base will appear anterior compared to the right.

Figure 20

Figure 20: Left Sacral Flexion

Note that the left base is anterior to its normal position resulting in a positive left SitFBT. Since this is an anterior sacral dysfunction, there is no abnormality in gait.

A = Anterior
P = Posterior/Inferior ILA

The left anterior shear causes decreased movement between the left base and innominate, resulting in a positive left SitFBT. Because the left base is pathologically anterior, the BB-test is negative (Figure 18). Anterior movement of the left base causes anterior rotation of the left innominate, functionally lengthening the left leg. The left ILA is shifted posteriorly and inferiorly. Due to right rotation of the sacral base, L5 is rotated left. For obvious reasons, some manual medicine/manual therapy training programs name this dysfunction *left inferior sacral shear*.

Right Sacral Extension

On initial inspection of the sacral base, a left sacral flexion and a right sacral extension appear very similar. With both, the left ILA is posterior and inferior, and the left sacral base appears anterior compared with the right (Figures 18 & 19). However, this pathology is due to posterior shear of the right sacral base causing disruption of the MTA (Figure 21).

Figure 21

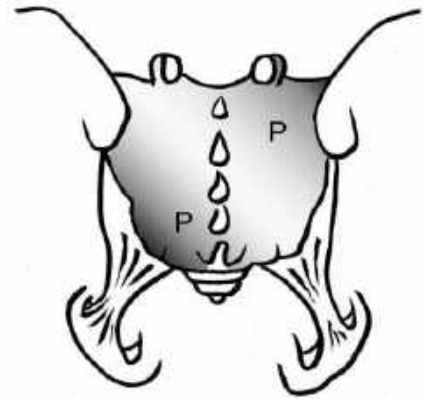


Figure 21: Right Sacral Extension

Note that the right base is posterior to its normal position resulting in a positive right SitFBT. On initial palpation this will appear exactly like a left sacral flexion. Since this is a posterior sacral dysfunction there is a gait abnormality.

P = Posterior (note the ILA is posterior and inferior)

The right posterior shear causes decreased movement between the base and innominate resulting in a positive right SitFBT. Because the right base is pathologically posterior, the BB-test is positive (Figure 19). Posterior movement of the right base causes posterior rotation of the innominate, functionally shortening the right leg. The left ILA shifts posteriorly and inferiorly. L5 is rotated left due to right rotation of the base. This dysfunction is extremely debilitating since the right base cannot move anteriorly during gait. Some manual medicine/manual therapy training programs name this dysfunction *right superior sacral shear*, however, this term is not preferred since it is does not follow normal anatomical naming practices.

Left on Left Sacral Torsion

Sacral torsions have many similarities to unilateral flexions and extensions; however, the fulcrum of movement occurs around the oblique axis. In naming a *left on left (L/L) sacral torsion*, the first "left" refers to the rotational direction of the sacrum and the second "left" refers to the oblique axis about which it rotates (Figure 22).

Figure 22

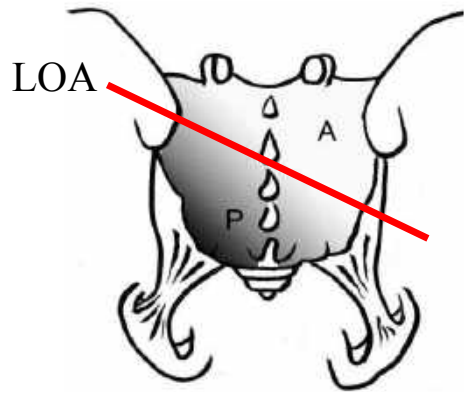


Figure 22: Left on Left Sacral Torsion

Note that the right base rotates anterior around the LOA to its normal position resulting in a positive right SitFBT. Since this is an anterior sacral dysfunction there is no abnormality in gait.

A = Anterior
P = Posterior/Inferior ILA

L/L Sacral Torsion: the first 'left' identifies rotational direction, the second 'left' identifies the oblique axis about which it rotates.

L/L torsions typically occur while the patient is in a squatting position that places the sacrum on the MTA; pelvic twisting motions convert the MTA to

Recurrent anterior sacral torsions occur with an anatomically short leg, muscle imbalance, and sacroiliac ligament instability.

the LOA. Spinal extension rotates the right base anteriorly. If the patient stands without moving the sacrum back to the MTA/neutral, the right sacral base remains pathologically anterior on the LOA.

The anteriorly rotated right base causes decreased right sacroiliac movement and thus a positive right SitFBT. The right base's anterior rotation likewise rotates the right innominate anteriorly, producing a functionally long right leg. Forward rotation of the right sacral base positions the left ILA posteriorly and inferiorly and L5 rotates right. Since the right base is anterior the BB-test is also negative. L/L torsions do not interfere with gait and the patient is often asymptomatic. The Chicago model names L/L torsions *anterior sacrum right*;[3(pp592)] conversely, we believe that knowledge of the LOA allows the much simpler description of L/L sacral torsion.

Left on Right Sacral Torsion

L/R sacral torsions arise similarly to L/L torsions. Posterior motion of the left sacral base is caused by spinal flexion - pelvic twisting motions convert the MTA to the ROA. Should the patient stand without moving the sacrum back to neutral, the left sacral base remains posterior producing left sacral rotation on the ROA (Figure 23).

Figure 23

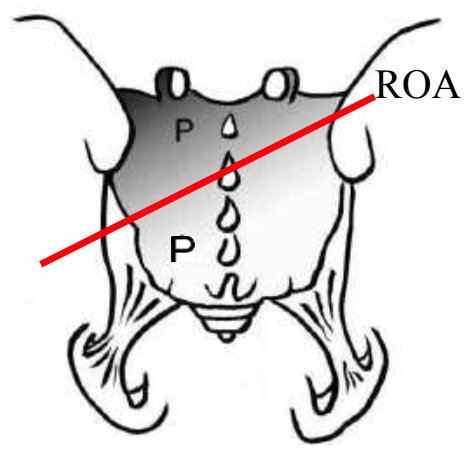


Figure 23: Left on Right Sacral Torsion

Note that the left base rotates posteriorly around the ROA resulting in a positive left SitFBT. Since this is a posterior sacral dysfunction there is gait abnormality.

P = Posterior sacral base and Posterior/Inferior ILA

Once the left base is posterior, the patient immediately notices that they cannot stand straight; this is due to decreased lordosis of the lumbar spine. The left posterior sacral base causes decreased movement across the sacroiliac joint, and thus a positive left SitFBT. The base's posterior movement rotates the left innominate posteriorly producing a functionally short left leg. The right ILA rotates anteriorly around the ROA, making the left ILA appear posterior and inferior by comparison and L5 rotates right. Since the left base is posterior, L/R torsions have a positive BB-test and interfere with gait. The Chicago model names this position *posterior sacrum left*, as it is left sacral sidebending with left rotation.[3(pp592)]

Note: a principle of sacral treatment is use of the sacral axes for direct and indirect treatments.

Treatment of Sacral Somatic Dysfunction

Indirect and direct treatments of the pelvis share one principle - the use of the pelvic axes. In this section, treatment concepts will be applied to a *L/R sacral torsion*, a very common and debilitating problem. In this instance, both direct and indirect techniques are easily managed as long as the patient is positioned on the ROA. This is executed by first locating the MTA with two fingers on the right and left sacral base. The operator then localizes motion under those fingers. The MTA is then converted to the ROA by several methods:

1. If sitting, the spine is commonly used as the lever and the patient is flexed and extended until motion is felt on the MTA. The patient is then instructed to cross their right leg over the left. The MTA has converted to the ROA when the finger over the right base moves superiorly, and the finger over the left base moves inferiorly. If the patient crosses their legs and the LOA is engaged, ask the patient to cross their left leg over the right.
2. If in a lateral position, the thigh may be used as the lever and the lower extremity flexed and extended until motion is felt on the MTA. The lower extremity is then lowered off the table to a point of tension between the sacrum and innominate while localized on the MTA. The thigh is then moved inferiorly; the finger over the right base should move superiorly, while simultaneously, the finger over the left base inferiorly, indicating motion is localized to the ROA. If motion is on the LOA, move the thigh into flexion until motion is felt on the ROA.

Direct Treatment of Sacral Somatic Dysfunction

To treat *L/R sacral torsion* using direct technique, utilize the ROA as the fulcrum. After the fulcrum is engaged, the positional diagnosis is reversed. Finally, as is done in any direct treatment, a force is generated using the lever. In summary:

1. Obtain the positional diagnosis *L/R sacral torsion.*
2. Localize the fulcrum to the MTA.
3. Convert the MTA to the ROA.
4. Reverse the components of the positional diagnosis by placing the patient against the motion barrier in right rotation on the ROA.
5. Implement a force using isometric contraction with relaxation or an impulse.

Indirect Treatment of Sacral Somatic Dysfunction

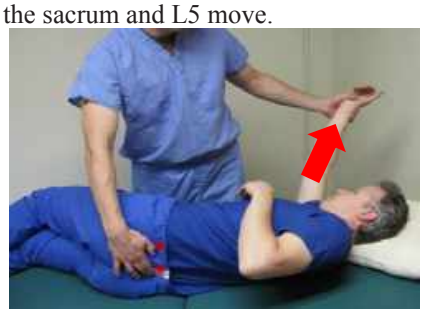
L/R sacral torsions are treated indirectly on the ROA by utilizing the positional diagnosis and exaggerating this position. To summarize:

1. Obtain the positional diagnosis *L/R sacral torsion.*
2. Localize motion to the MTA and convert it to the ROA
3. Exaggerate the posterior rotation of the left base to a position of three-dimensional balance or patient comfort.

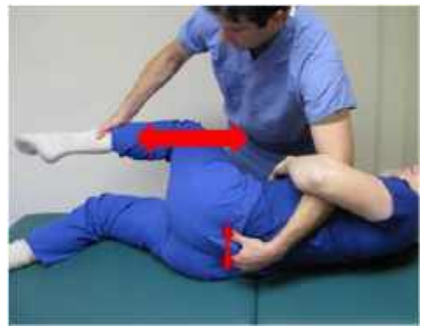
Treatment Examples: Direct Procedures

L/R Sacral Torsion using ME

1. Place the patient in a right lateral recumbent position with the left base up. Locate the MTA posteriorly (MTA identified by red dots).
2. Pull the patient's right arm, creating type I spinal mechanics until motion is felt on L5. This locks down the spine to L5 so that only the sacrum and L5 move.



3. Create a lever with the thigh by flexing and extending until motion is felt on the MTA.



4. Lower the left lower extremity off the table while on the MTA. Move the thigh inferiorly – the operator should observe the MTA covert to the ROA as previously described. The ROA is the fulcrum.

5. Create an isometric contraction by instructing the patient to push their leg toward the ceiling. Have the patient relax. Reposition at the new barrier by adducting the lower extremity. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.

6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

L/R Sacral Torsion with L5 and HVLA

1. Place the patient in a right lateral recumbent position with the left base up. Locate the MTA posteriorly (MTA identified by red dots).
2. Pull the patient's right arm, creating type I spinal mechanics until motion is felt on L5. This locks down the spine to L5 so that only the sacrum and L5 move.

3. Create a lever with the thigh by flexing and extending until motion is felt on the MTA.

4. Lower the left lower extremity off the table while on the MTA. Move the thigh inferiorly – the operator should observe the MTA covert to the ROA as previously described. The ROA is the fulcrum. Place the patient's left foot behind the thigh as pictured below.
5. Maintain one hand on L5 and one on the ROA. Hold the ROA firmly maintain the fulcrum. Thrust downward with rotation moving L5 further left; this causes right rotation of the sacrum on the ROA.

6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Treatment Examples: Indirect Procedures

L/R Sacral Torsion using Functional Still Techniques

1. With the patient sitting on the table, locate the MTA (labeled with two red dots).

2. Place the right hand and forearm across the patient's shoulders to gain control of the trunk. Flex and extend the patient until motion is felt on the MTA. Have the patient cross their legs converting the MTA to the ROA.

3. To find the dynamic balance point (DBP), loose pack the left SI joint by gently leaning the patient to the right. This further posteriorly rotates the left sacral base on the ROA.
4. Add gentle compression from the patient's left shoulder into their left ischial tuberosity. Using the left hand, monitor the left SI along ROA as it changes and adjust the flexion-extension, sidebending, and rotation to maintain balance. If the DPB reaches a still point, wait for 10-20seconds; if there is no change, challenge the tissues by either increasing or decreasing compression. When the tissue relaxes the treatment is finished.

5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

The principle taught in this functional treatment is to loose-pack the restricted SI joint (the side of the SitFBT), and add compression into the SI on the side of sacral rotation (the left). To treat a L/L torsion, use the LOA, loose pack the right SI and add compression on the left side.

L/R Sacral Torsion using S/CS (aka PS1)

1. With the patient prone, adduct and abduct the right lower extremity until motion is felt under the MTA. Lay the lower extremity on the table.

2. Convert the MTA to the ROA by posteriorly rotating the left innominate.

3. The tender point is on the left sacral base; tell the patient that this tenderness represents a 10 on a scale of 1-10. Further posteriorly, rotate the left base by pushing anteriorly on the right ILA until tenderness is a 3 out of 10.

4. Maintain this position for 90 seconds. You do not have to monitor the tender point during this time. If you choose to monitor, you may feel characteristic tissue changes.
5. SLOWLY reduce pressure on the right ILA and return the patient's leg on the table.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Treatment Sequencing of Pelvic Somatic Dysfunction:
1. Innominate shears
2. Pubic shears
3. All sacral dysfunctions
4. Innominate rotations

Treatment Pearls for the Sacrum and Innominates

Often the patient will have simultaneous innominate and sacral dysfunctions causing confusion during pelvic treatment. Often, the provider is unsure what dysfunction to treat first. Edward Stiles, DO, FAAO, advocates treating the pelvis in the following order:

1. Innominate shears
2. Pubic shears
3. All sacral dysfunctions
4. Innominate rotations

At first glance, this may not appear to have any inherent logic, but it does when in the context of the pelvic axes. Treatment of innominate shears allows restoration of all pelvic axes. In other words, a sacral torsion or pubic shear may be the result of an innominate shear that will correct with reduction of the shear.

As mentioned previously, pubic shears cause abnormal mechanics of the sacrum. This is because the pelvis is a ring structure with three joints; pubic shears impair movement of the sacroiliac joints. Restoration of the pubic axis will often correct sacral dysfunction; hence, pubic dysfunction should always be corrected before sacral.

Due to severe gait debilitation caused by posterior sacral dysfunction, the sacrum should be treated before innominate restrictions. Inherently this makes sense because innominate rotations are often compensatory to muscle imbalance and anatomical leg length dysfunctions.

It is the authors' hope that students understand pelvic diagnosis and treatments rather than memorize diagnoses and treatment techniques. Understanding the principles provided in this chapter allows students to individualize treatments based on their capabilities and the patient's treatment tolerance.

References:

1. Mitchell, Fred L., Sr. "Structural Pelvic Function." American Academy of Osteopathy Yearbook, 1958, 71-90.
2. Greenman, Philip E. *Principles of manual medicine.* Philadelphia: Williams and Wilkins, 2003.
3. Chila, Anthony G. *Foundations of Osteopathic Medicine.* Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2011. (aka FOM3)
4. DiGiovanna, Eileen L., Stanley Schiowitz, and Dennis J. Dowling. An Osteopathic Approach to Diagnosis and Treatment. Philadelphia: Lippincott Williams and Wilkins, 2005.
5. Stiles, Edward G. Lecture notes from Osteopathic Principles and Practice, Kentucky College of Osteopathic Medicine, 1998-1999.
6. Principles of Osteopathic Medicine, Pelvis. Performed by Phillip E. Greenman. 1993. VHS.
7. Myers, Harmon L., et al. *Clinical Application of Counterstrain*. Tucson, AZ: Osteopathic Press, a Division of Tucson Osteopathic Medical Foundation, 2012.
8. Jones, Lawrence H., et al. *Strain-counterstrain*. Boise, ID: Jones Strain-Counterstrain, 1995.
9. Levangie, Pamela K. "Four clinical tests of sacroiliac joint dysfunction: the association of test results with innominate torsion among patients with and without low back pain." *Physical Therapy* 79, no. 11 (1999): 1043-1057.
10. Potter, Nancy A., and Jules M. Rothstein. "Intertester reliability for selected clinical tests of the sacroiliac joint." *Physical therapy* 65, no. 11 (1985): 1671-1675.
11. Giusti, Rebecca, DO. *Glossary of Osteopathic Terminology*. Third ed. Chevy Chase, MD: American Association of Colleges of Osteopathic Medicine, 2017.

Clinical Vignette

Patient Name: CD
November 10, 2013

CC: new patient visit, pain

HxCC: Patient is 53year old, female presenting for evaluation of a head and pelvic injury from approximately three weeks ago. The patient was gardening and turned her head while on a patio, striking her L-frontal/parietal area; this whipped her head forward and she lost her balance. She suddenly stepped off the patio approximately one foot down causing further pain and injury to her R-hip. She now has R-neck and R-hip pain. The patient is taking ibuprofen with little relief.

She was involved a motor vehicle accident at age 26 that resulted in whiplash; she has no permanent injury from this event.

ROS:
Constitutional: no mental status changes, no travel or sick contacts
Eyes: no change in vision
Endocrine: no fever, night sweats, weight change
Cardiovascular: no chest pain or palpitations
Respiratory: no dyspnea, cough, or sputum
Gastrointestinal: no nausea, vomiting diarrhea; no change in BM, no blood in stool
Genitourinary: no change in urine, no dysuria, no blood in urine
Neurologic: no anesthesia, no paresthesias, no loss of consciousness, no seizures
Musculoskeletal: no weakness
Psychiatric: no homicidal or suicidal ideations, no signs of depression
Allergy: no angioedema, no urticaria, no anaphylaxis, no symptoms of seasonal allergies

PMHx/PSHx:
1. Allergic Rhinitis

Allergies:
-sulfa, naproxen, cyclobenzaprine

MEDs:
-none

Immunizations:
-per her PCM

Social:
-no smoking, + alcohol 2 per week, no drugs, work = architect

FamHx:
-coronary artery disease in mother

VITALS = 5"6", 140 pounds, 110/80, 83 beats per minute
GEN = awake, alert, oriented to time, place, situation, self, in no acute distress
HEENT = normal cephalic, a-traumatic, extraocular muscles intact, no otic/nasal discharge
NECK = supple, thyroid w/o masses
HEART = regular rate, no clicks/rubs/murmurs
LUNGS = clear to auscultation, without crackles, wheeze, or ronchi
GI = soft, non-tender, no rebound tenderness, +bowel sounds x 4 quadrants, no hepato-spleenomegaly
CV = no jugular venous distention, no carotid bruit, mucus membrane moist, no edema, 2/4 peripheral pulses, no lymphadenopathy
N/M = cranial nerves 2-12 intact, 5/5 global strength, 2/4 global reflexes, cerebellum intact, sensation symmetric, gait with limited right hip movement.
Biomechanical Exam = positive findings include: **very TTP right occipital-mastoid suture; no pain with internal and external rotation of hip. +SFBT right with elevated ASIS, elevated crest, elevated PSIS compared to the left, + stork test right.**

Assessment/Plan

1. Head Trauma with anterior whiplash injury
   -her injury has produced no visible scar; I believe when hitting the metal bracket, she has caused somatic dysfunction to her head and cervical spine. I will attempt OMT to see if she has relief of symptoms. Currently, she has no red flags to prevent treatment.
   -patient counseled of a normal treatment reaction. A treatment reaction may include muscle soreness, pain, and mild edema that may last 2-3days. If patient experiences symptoms that outlay a normal treatment reaction, she should contact a physician or go to the emergency room.
2. Right-hip pain
   -this is caused by a unilateral pelvic up-shear – it occurred by her sudden step off the porch. It was successfully treated today with restoration of function and 100% pain relief immediately after treatment.
3. Somatic Dysfunction
   a. Head – left parietal-frontal restriction with cranial; right occipital-mastoid restriction with cranial
   b. Neck – C1 L-rotated with S/CS; C3 ERS$_L$ with ME
   c. Thorax – T1 L-rotated with ME
   d. Ribs – L-rib #1 inhaled with functional procedure
   e. Pelvis – R-innominate superior shear with HVLA and MFR

   -follow up in 1 week

Jason Browder, DO

Appendix A: Anterior S/CS Tenderpoints

Tenderpoints of the anterior pelvis typically correlate with musculature attachments at that site or the muscle belly. Clinical correlations to Mitchell model diagnoses have not been made.

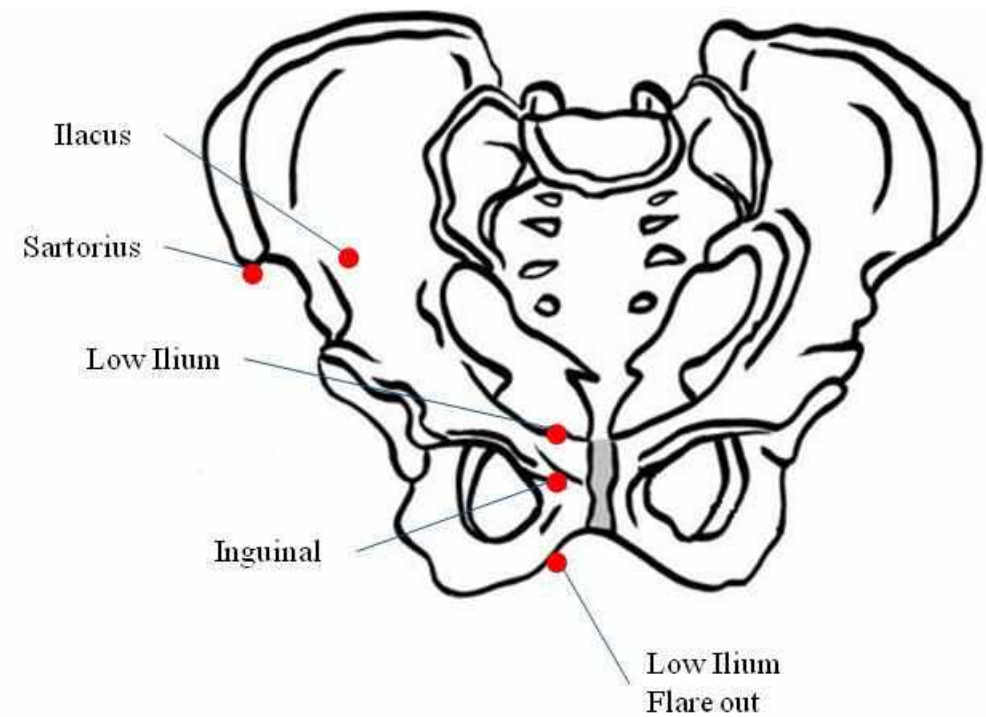
Iliacus = correlates to the belly of the iliacus portion of the iliopsoas muscle.[7(pp123),8(pp87)]

Sartorius = found an inch inferior to the ASIS; it correlates with its named muscle.[7(pp161), 8(pp88)]

Low Ilium = may correlate with the attachment of psoas minor on the lateral ramus of the pubic tubercule.[7(pp158),8(pp81)]

Inguinal = appears to correlate with adductor muscles and in the musculature just inferior to the surface of the pubic tubercle.[7(pp160),8(pp89)]

Low Ilium Flare Out = this tenderpoint is on the inferomedial surface of the descending ramus of the ilium. Its muscular correlate may be the obturator externus muscle.[7(pp159), 8(pp82)]

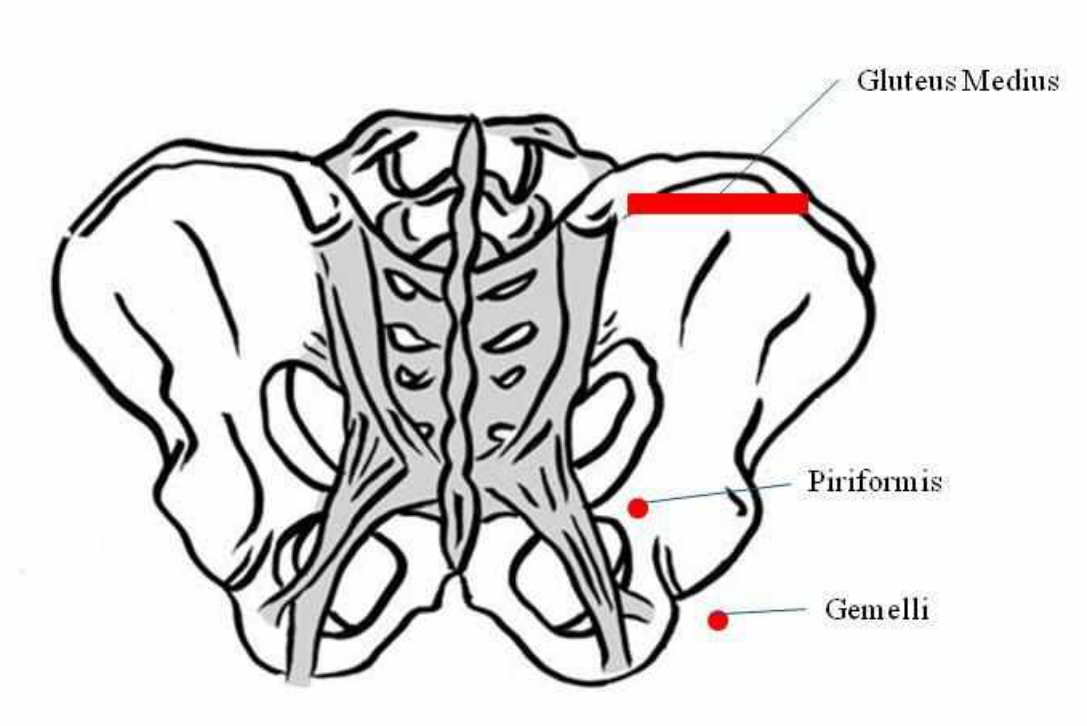


Appendix A: Posterior S/CS Tenderpoints

Gluteus medius = tenderpoint is on a line along the crest of the ilium.[7(pp176),8(pp91)]

Piriformis = this muscle originates on the anterior side of the sacrum and inserts on the superior surface of the greater trochanter. Tenderpoints can be found along the muscle's course on the belly, approximately 7cm medial to the greater trochanter.[7(pp175&177),8(pp92)]

Gemelli = tenderpoint is found from the inferior lateral surface of the ischial tuberosity to its attachment on the posterior femur and correlates to the muscle's origin and insertion.[7(pp178),8(pp93)]

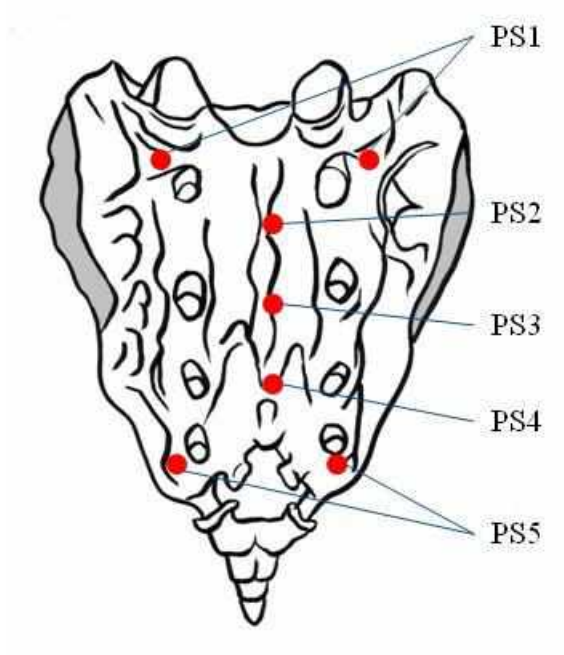


Appendix A: Posterior Sacral (PS) S/CS Tenderpoints

There is no known anatomical correlation to posterior sacral Counter-Strain tenderpoints. We see PS1 and PS5 almost ubiquitously with sacral somatic dysfunction. There is thought that PS1 correlates to attachment of the long-paraspinal musculature such as iliocostalis, multifidi, or longissimus.[7(pp182-185)] Anatomically, we know that the gluteus maximus crosses that SI joint in the lower half of the sacrum, and that there may be tenderness from ligamentous structures on strain such as the sacrotuberous ligament that attach near PS5 bilaterally, as well as, the thick ligaments of the sacroiliac joint that attach at PS1.

Treatment with counterstrain has classically been to identify tenderness and reduce it with pressure on the opposite side.[8(pp84)] For instance, if tenderness is found at right-PS1, posterior to anterior pressure on left-PS5 (or on the left ILA) will often reduce the tenderness.

Alternatively, we propose that diagnosing the sacrum using spinal mechanics, such as L/R torsion, and exaggerating that positional diagnosis will commonly reduce tenderpoints found on exam.



Appendix B: Evidence Based Medicine of Sacral and Innominate Testing

There are insufficient diagnostic trials for sacral and pelvic somatic dysfunction (SD); after literature review, we present two. Tests lacking evaluation are listed as "no data." Levangie's well-powered trial for anterior or posterior innominate rotation indicates that classically taught tests lack positive and negative predictive value. We point out that there is no "gold standard" to diagnose SD, no tests such as MRI, CT or radiographs to confirm the diagnoses we palpate on exam. Additionally, Levangie, did not consider sacral axes and treatment order in the evaluation of pelvic SD. In the practice of authors JB and PJ, and as in Styles', innominate rotations are the last pelvic somatic dysfunction we treat because they are most often compensatory to muscle imbalance and anatomical asymmetry, and in essence, pathology other than innominate rotations is usually present.

Test	Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value
ASIS Compression test	no data	no data	no data	no data
SFBT	17%	79%	61%	34%
Stork test (Gillet test)	8%	93%	67%	35%
SitFBT	9%	93%	78%	28%
BB- test/Spring test	no data	no data	no data	no data
ILA symmetry	no data	no data	no data	no data
Sacral base symmetry	no data	no data	no data	no data
L5 rotational position	no data	no data	no data	no data

**study performed on 144 subjects with low back pain and 137 subjects without low back pain to determine presence of anterior or posterior innominate rotation.[9]

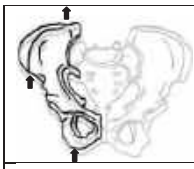
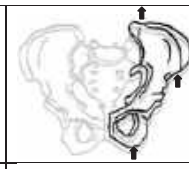
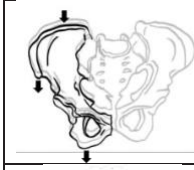
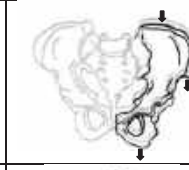
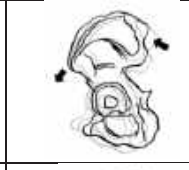
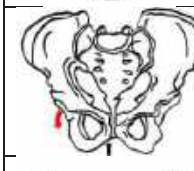
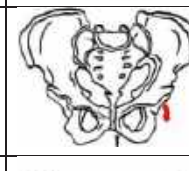
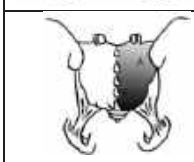
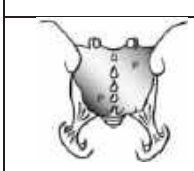
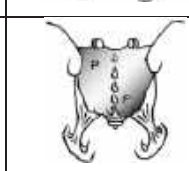
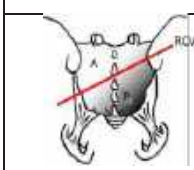
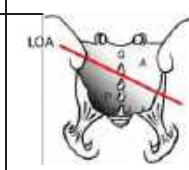
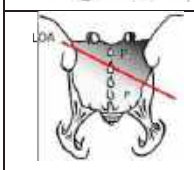
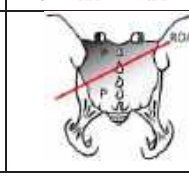
Interestingly, the most inter-tester reliability[10] was found with the supine iliac gapping test, a test that closely resembles the ASIS compression test, but is not commonly taught in osteopathic clinical education. The supine iliac gapping test is performed on a supine patient with the examiner facing the patient's head. The examiner's hands are crisscrossed over the ASISs with the operator's right hand on patient's right ASIS and operator's left hand on left ASIS. The examiner then applies an inferior lateral force through both ASISs, effectively gapping the anterior sacroiliac joint and compressing the posterior sacroiliac joint. Pain implicates pathology of the anterior SI ligament, but does not indicate what, if any, SD is present.

Unfortunately, this study[10] with seventeen patients (N=17) lacks sufficient power to convince this reviewer of its validity.

Test	Agreement %
Palpation in standing of iliac crest levels	35.29
Palpation in sitting of iliac crest levels	41.18
Palpation in standing of PSIS levels	35.29
Palpation in sitting of PSIS levels	35.29
Palpation in standing of ASIS levels	37.50
Palpation in sitting of ASIS levels	43.75
ASIS Compression test	no data
SFBT	43.75
Stork test (aka – Gillet test)	46.67
SitFBT	50
Supine iliac gapping	94.12

**study performed on 17 patients with back pain.[10] "Agreement %" indicates inter-tester physical exam findings that are the same.

Appendix C: Sacrum and Innominate Somatic Dysfunction

	Right Innominate Superior Shear + right SFBT right crest cephalad right ASIS cephalad right PSIS cephalad functionally short right leg		Left Innominate Superior Shear + left SFBT left crest cephalad left ASIS cephalad left PSIS cephalad functionally short left leg
	Right Innominate Inferior Shear + right SFBT right crest caudad right ASIS caudad right PSIS caudad functionally long right leg		Left Innominate Inferior Shear + left SFBT left crest caudad left ASIS caudad left PSIS caudad functionally long left leg
	Right Innominate Ant. Rotation + right SFBT crest heights symmetrical right ASIS caudad right PSIS cephalad functionally long right leg		Left Innominate Ant. Rotation + left SFBT crest heights symmetrical left ASIS caudad left PSIS cephalad functionally long left leg
	Right Innominate Post. Rotation + right SFBT crest heights symmetrical right ASIS cephalad right PSIS caudad functionally short right leg		Left Innominate Post. Rotation + left SFBT crest heights symmetrical left ASIS cephalad left PSIS caudad functionally short left leg
	Right Pubic Superior Shear + right SFBT crest heights symmetrical or right cephalad right ASIS lateral PSIS' symmetrical functionally long right leg		Left Pubic Superior Shear + left SFBT crest heights symmetrical or left cephalad left ASIS lateral PSIS' symmetrical functionally long left leg
	Right Pubic Inferior Shear + right SFBT crest heights symmetrical or right caudad right ASIS medial PSIS' symmetrical functionally short right leg		Left Pubic Inferior Shear + left SFBT crest heights symmetrical or left caudad left ASIS medial PSIS' symmetrical functionally short left leg
	Right Sacral Flexion (aka right inferior sacral shear) Right ILA inf./post. + right SitFBT - BB-test/spring functionally long right leg L5 rotated right		Left Sacral Flexion (aka left inferior sacral shear) Left ILA inf./post. + left SitFBT - BB-test/spring functionally long left leg L5 rotated left
	Right Sacral Extension (aka right superior sacral shear) Left ILA inf./post. + right SitFBT + BB-test/spring functionally short right leg L5 rotated left		Left Sacral Extension (aka left superior sacral shear) Right ILA inf./post. + left SitFBT + BB-test/spring functionally short left leg L5 rotated right
	Right on Right Sacral Torsion (aka anterior sacrum left) Right ILA inf./post. + left SitFBT - BB-test/spring functionally long left leg L5 rotated left		Left on Left Sacral Torsion (aka anterior sacrum right) Left ILA inf./post. + right SitFBT - BB-test/spring functionally long right leg L5 rotated right
	Right on Left Sacral Torsion (aka posterior sacrum right) Right ILA inf./post. + right SitFBT + BB-test/spring functionally short right leg L5 rotated left		Left on Right Sacral Torsion (aka posterior sacrum left) Left ILA inf./post. + left SitFBT + BB-test/spring functionally short left leg L5 rotated right

ASIS = anterior superior iliac spine; BB-test = backward bending test; ILA = inferior lateral angle; inf./post. = inferior and posterior; PSIS = posterior superior iliac spine; SitFBT = sitting forward bending test; SFBT = standing forward bending test

Review Questions

1. A patient presents with complaints of low back pain. On exam, the left sacral base is easily mobile while the right is immobile. The left ILA is inferior and posterior. The spring test is positive. The diagnosis is:
    a. L/L torsion
    b. L/R torsion
    c. R/R torsion
    d. R/L torsion

2. A patient has a positive left SFBT. The iliac crest is more inferior on the right than the left. The likely diagnosis is:
    a. Left anterior innominate
    b. Left posterior innominate
    c. Left innominate superior shear
    d. Left innominate inferior shear

3. On a prone patient with deep left sacral sulcus, right posterior and inferior ILA, and negative BB- test, the most likely diagnosis is:
    a. Right unilateral flexion
    b. Left unilateral flexion
    c. R/R torsion
    d. R/L torsion
    e. L/L torsion

4. You evaluate a patient for low back pain. On exam your findings include left posterior and inferior ILA, anterior left sacral base, positive spring test, and L5 rotated left. This is most consistent with:
    a. Left unilateral flexion
    b. Right unilateral flexion
    c. Left unilateral extension
    d. Right unilateral extension

5. The reference point for the sacrum is:
    a. Anterior superior sacral base
    b. Anterior apex of sacrum
    c. 1st posterior spinous process of the sacrum
    d. Posterior superior sacral base

6. The preferred treatment sequence of the pelvis is:
    a. Innominate rotation, pubic shear, sacrum, innominate shear
    b. Innominate rotation, sacrum, pubic shear, innominate shear
    c. Innominate shear, pubic shear, sacrum, innominate rotation
    d. Pubic shear, innominate shear, sacrum, innominate rotation
    e. Sacrum, innominate shear, sacrum, innominate rotation

7. When compressing perpendicular to the table, the ASIS compression test measures somatic dysfunction along which axes:
    a. The STA
    b. The MTA
    c. The ITA
    d. The ROA
    e. The LOA

8. When sitting forward, a positive bending test (SitFBT) demonstrates:
    a. Cranio-sacral dysfunction
    b. Iliosacral dysfunction
    c. Sacroiliac dysfunction
    d. Iliopsoas spasm
    e. Leg length discrepancy

9. When standing forward, a bending test (SFBT) potentially demonstrates:
    a. Cranio-sacral dysfunction
    b. Iliosacral dysfunction
    c. Sacroiliac dysfunction
    d. Iliopsoas spasm
    e. Leg length discrepancy

10. A confirmatory test to the SFBT is the:
    a. SitFBT
    b. Stork Test
    c. BB-Test
    d. ILA position

11. The ideal treatment axis for a unilateral sacral flexion is the:
    a. STA
    b. MTA
    c. ITA
    d. ROA
    e. LOA

12. The ideal treatment axis for a pubic shear is the:
    a. STA
    b. MTA
    c. ITA
    d. ROA
    e. LOA

13. A primary sacral dysfunction is present as long as:
    a. There is a positive sitting forward bending test
    b. There is a positive standing forward bending test
    c. There is an inferior and posterior ILA
    d. There is a positive BB-test

14. Your 29-year-old patient presents with right hip pain for two weeks. Her history reveals stepping off a curb unexpectedly with instant pain in her pelvis. According to history, she most likely has:
    a. An anterior sacral torsion
    b. An anterior rotation of the innominate
    c. A unilateral pubic inferior shear
    d. A unilateral innominate superior shear

15. A patient presents with a positive standing forward bending test on the right, a long right leg, and level innominate crest. Plausible diagnoses from the given information would include:
    a. Pubic superior shear
    b. Innominate inferior shear
    c. Posterior innominate rotation
    d. R/L sacral torsion

16. Superior pubic shear is treated with muscle energy using which muscle group:
    a. Hamstrings
    b. Adductors
    c. Quadriceps
    d. Abductors

17. Counter-strain treatment of a L/R torsion would include:
    a. Localization on the LOA with anterior compression of the right ILA
    b. Localization on the ROA with anterior compression of the right ILA
    c. Localization on the LOA with anterior compression of the left ILA
    d. Localization on the ROA with anterior compression of the left ILA

18. A patient falls from the tail-gait of her truck three feet to the cement below. Exam reveals a positive SitFBT on the right, L-inferior and posterior ILA, and a positive BB-test on the left. Neurological exam of the lower extremities is normal. To treat this dysfunction, you would:
    a. Localize to the MTA
    b. Localize to the ROA
    c. Localize to the ILA
    d. Send for radiographic studies

Answers:

1. B
2. D
3. C
4. D
5. A
6. C
7. C
8. C
9. B
10. B
11. B
12. C
13. C
14. D
15. A
16. B
17. B
18. D

Chapter 7: The Upper Extremity

Principles Presented in this Chapter:

- Orthopedic Model of Upper Extremity Evaluation
- Evidenced Based Evaluation of the Upper Extremity using the Orthopedic Model
- Origin and Insertion of Upper Extremity Musculature and its use in Diagnosis and Treatment

Outline:

- Shoulder Anatomy
- Orthopedic Evaluation of the Shoulder
- Evaluation of Shoulder Somatic Dysfunction
- Treatment of Shoulder Somatic Dysfunction

- Elbow Anatomy
- Orthopedic Evaluation of the Elbow
- Evaluation of Elbow Somatic Dysfunction
- Treatment of Elbow Somatic Dysfunction

- Wrist/Hand Anatomy
- Orthopedic Evaluation of the Wrist/Hand
- Evaluation of Wrist/Hand Somatic Dysfunction
- Treatment of Wrist/Hand Somatic Dysfunction

- Upper Extremity Treatment Pearls
- Appendix A: Shoulder Strain-counterstrain Tenderpoints
- Appendix B: Elbow Strain-counterstrain Tenderpoints
- Appendix C: Wrist/Hand Strain-counterstrain Tenderpoints
- Clinical Vignette
- Review questions

Chapter 7: The Upper Extremity

The complexity of the shoulder girdle region with respect to the neurovascular bundle, the musculoskeletal anatomy, and myofascial pain syndromes, make identifying primary somatic dysfunction challenging. Commonly, patients with upper extremity symptoms have somatic dysfunction in the upper rib cage, cervical spine, scapulothoracic joint, and sternoclavicular (SC) joint. Complaints often include anterolateral shoulder pain, upper extremity paresthesias, and periscapular pain. Anecdotally, when addressing somatic dysfunction of the cervical spine, thoracic spine and rib cage, many patients presenting with these symptoms get relief due to the affect these regions play on scapular motion. In an attempt to introduce students to complimentary schools of thought, this chapter provides traditional Osteopathic as well as orthopedic evaluation of the upper extremity

Shoulder Anatomy

The shoulder girdle bony structures consist of the scapula, humerus, and clavicle (Figures 1 & 2). Note, that the scapula normally extends from the 2nd to 7th ribs and typically lays 30° to the body's coronal plane.

Figure 1

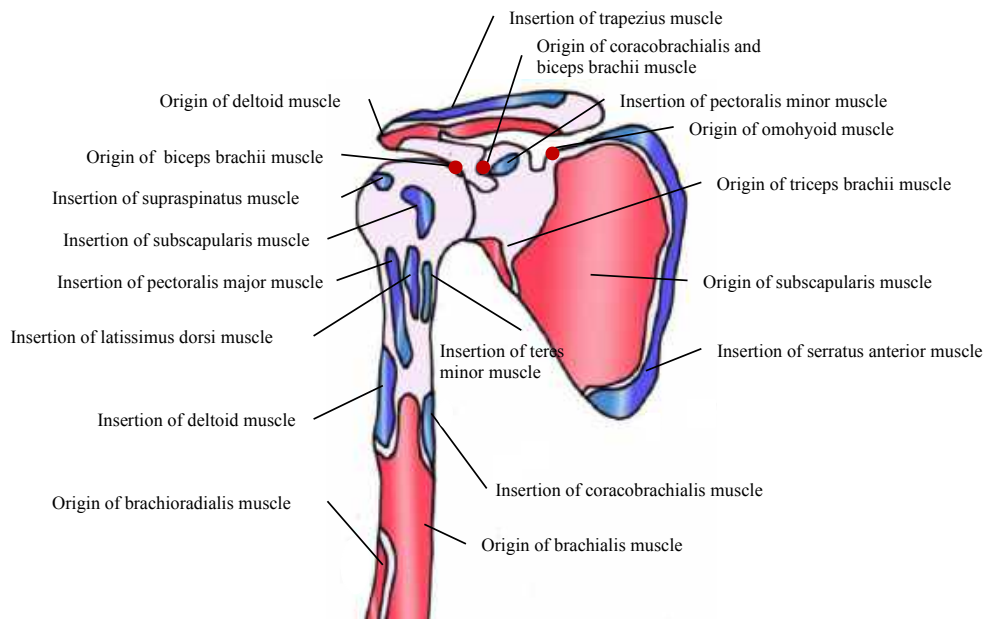
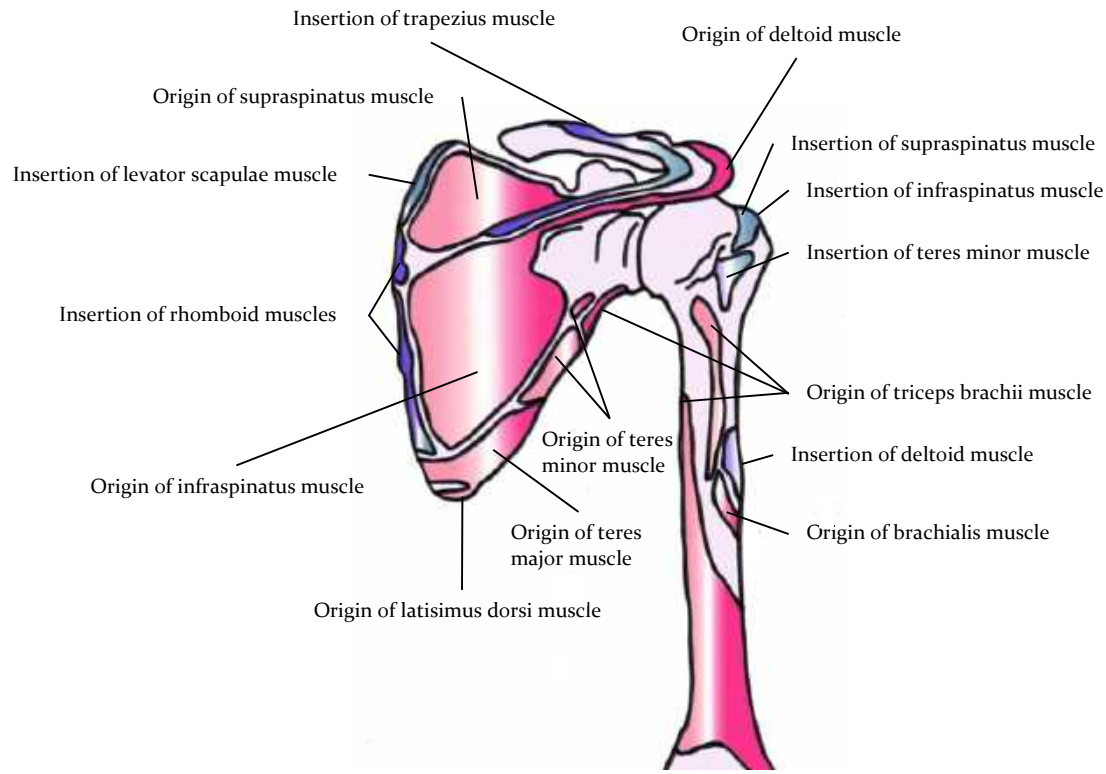


Figure 1: Anterior View of the Shoulder Bony Anatomy with Origins and Insertions

Students should have a good appreciation for the general bony anatomy of the upper extremity (UE). As we will see, the origin and insertion of the muscles play a large role in the evaluation and treatment of UE somatic dysfunction and helps us better understand the orthopedic model for shoulder testing.

Figure 2: Posterior View of the Shoulder Bony Anatomy with Origins and Insertions

Figure 2



The only bony attachment of the upper extremity to the thorax is the clavicle through the SC joint. The remainder of the upper extremity attaches to the thoracic cage via muscular attachment. The ligaments of the SC joint are very strong and have attachments to the 1st rib. The clavicle is often thought of as a strut that guides the shoulder during abduction.

Rotator Cuff Muscles
- Subscapularis
- Supraspinatus
- Infraspinatus
- Teres Minor

When compared with the hip, the glenohumeral joint is often described as a "golf ball on a tee" versus a true "ball and socket" joint. Its shallow socket makes it inherently unstable, yet it provides the greatest range of motion (ROM) of any joint in the human body. The glenoid labrum, a cartilage lining of the glenoid, deepens the cavity of the joint and further supports the joint with other "static stabilizers," including the capsule and glenohumeral ligaments.

The subscapularis, supraspinatus, infraspinatus, and teres minor make up the muscles of the rotator cuff. The rotator cuff musculature acts as a "dynamic stabilizer" of the glenohumeral joint by retracting the humeral head when the shoulder moves into abduction. In conjunction with the long head of the tricep, they keep the humeral head in the glenohumeral joint.

Table 1 summarizes the actions of the shoulder musculature; where the origin and insertions are less apparent, they have been included.

Table 1: Shoulder Muscles

TABLE 1[1,2]	
Muscle	Action
Subscapularis	Internal rotation of humerus.
Supraspinatus	Initiates abduction of humerus.
Infraspinatus	External rotation and "centering" of the humeral head.
Teres minor	External rotator of humerus.
Teres major	Strong adductor of the humerus.
Coracobrachialis	Abducts and flexes the humerus; assists in retaining head of humerus in contact with glenoid cavity trunk.
Serratus anterior	Arises from ribs 1-9 anteriorly, inserting on the medial border of the scapula. It protracts and stabilizes scapula and assists in upward rotation. When the upper and lower fibers contract together, the scapula stays pressed to the rib cage. When the upper fibers contract, the scapula moves upwards.
Levator scapula	Elevates the scapula.
Rhomboid Major & Minor	Arises from the spine at T4-T5 and inserts on the medial scapular border. Acts to retract and elevate the scapula.
Trapezius	Upper half - arises from the occiput, midline from T1-3, and nuchal ligament. Inserts on the upper edge of the spine of the scapula, acromion and lateral 1/3 of the clavicle. Elevates the scapula bilaterally. Lower half - arises from the midline of T4-12 and inserts on the spine of the scapula. Acting together with the upper trapezius, retracts the scapula bilaterally.
Pectoralis minor	Depresses the scapula.
Omohyoid	Depresses the hyoid bone, larynx.
Pectoralis major	Adductor of the humerus; internal rotator when other muscles oppose it.
Latissimus dorsi	Originates from T7 to the sacrum in the midline. Also originates from the posterior iliac crest, lower 4 ribs, and tip of the scapula. It travels around teres major to insert on the posterior edge of the bicipital groove. Action: adductor of the humerus, depression of the scapula.
Deltoid	Anterior portion flexes, middle portion abducts, and posterior portion extends humerus.

Orthopedic Evaluation of the Shoulder

Evaluation of the shoulder girdle region should involve a systematic approach with the following sequence of evaluation:

1. Inspection
2. Range of motion
3. Palpation
4. Motor strength testing
5. Neurovascular testing
6. Orthopedic Special Tests
7. Evaluation for Somatic Dysfunction

After taking a thorough history of the patient's presenting complaint, it is important to make sure that the problem is not a "musculoskeletal masquerader" representing underlying medical/visceral illness such as: gallbladder disease, cardiac ischemia, cervical radiculopathy, etc. Other considerations include history of trauma, sport related activity (the patient's position on their team), occupation, onset, and activity of the problem.

In the context of identifying biomechanical factors in the trunk and axial skeleton that predispose the patient to shoulder pain, it is often clinically important to evaluate the cervical/thoracic spine and rib cage prior to specific evaluation of the shoulder; this is further extrapolated in other chapters and detailed in the sequencing chapter.

Inspection

Important information regarding the patient's condition can be gleaned from watching the patient put on or take off their shirt or jacket and observing the skin of the shoulder girdle region for skin lesions (e.g., herpes zoster) or bony asymmetries (e.g., cephalad scapular spine with protracted scapula).

Range of Motion Testing of the Shoulder

Evaluate the patient's active and passive ROM during arm flexion, abduction, external rotation, and internal rotation. During ROM evaluation, watch the patient from behind to closely observe scapular motion. Because the clavicle acts as a strut, guiding upper extremity movement in relation to the thorax, it is also important to test the motion of the SC and AC joints as demonstrated later in this chapter.

Palpation of the Shoulder

In this section, students should refer to the origin and insertion diagrams to aid in anatomical location. Starting medially, palpate the SC joint then proceed laterally along the clavicle to the acromioclavicular (AC) joint. While palpating the humerus, internally rotate the shoulder to move the supraspinatus anteriorly and palpate its insertion on the greater tuberosity. To palpate the subscapularis tendonous insertion, place the arm in the anatomical position with the elbow bent at 90°; then, externally rotate the arm to bring the lesser tuberosity more anterior. Returning to the neutral position, palpate the bicipital groove between the lesser and greater tuberosities to locate the biceps tendon. Locate the infraspinatus tendon by placing the hand of the affected side on the opposite shoulder; it is localized at the midpoint of the lateral humerus with the teres minor tendon just posterior to it.

Motor Strength Testing of the Shoulder

On strength testing, full resistance or 5/5 strength is normal.

When testing the rotator cuff musculature, it is difficult to completely isolate a particular muscle. Normal patient resistance is graded and documented as 5/5 in strength; during this testing, the patient should provide maximal effort. In general, the supraspinatus is tested *en scaption*, i.e., in the plane of the scapula that is approximately 45° from the coronal plane (Figure 3A). Infraspinatus is tested with the arms adducted and the elbow flexed to 90° (Figure 3B); the patient then externally rotates their arm. For teres minor, the shoulder is abducted to 90° with the elbow bent to 90° (Figure 3C); the patient then externally rotates the arm. The belly press is used to test subscapularis muscle strength (Figure 3D), and is especially useful for patients with an inability to fully internally rotate the arm (e.g., they cannot place the dorsum of their hand on their back). This test is performed sitting or standing. The patient holds the palm of their hand on their belly and presses against the abdomen while the examiner maintains maximal resistance.

Figure 3

Figure 3: Motor Testing of the Shoulder Girdle Muscles

A. Supraspinatus testing
B. Infraspinatus testing
C. Teres minor testing
D. Subscapularis

The test is positive in the absence of a 5/5 strength measurement or if wrist flexion is necessary to maintain arm position.

Neurovascular Tests of the Shoulder

Thoracic outlet syndrome, as mentioned in the ribcage chapter, represents clinical symptomatology from compression of the subclavian artery, vein, lymphatics, or brachial plexus. Roos test[3] (Figure 4) attempts to reproduce these symptoms; both shoulders are abducted and externally rotated to 90° and the elbows flexed to 90°. The patient is asked to repeatedly clench their fist for 3 minutes. If the patient cannot maintain the position for the duration of the test or develops paresthesias, then the test is positive for thoracic outlet syndrome.

Figure 4

Figure 4: Roos Test for Thoracic Outlet Syndrome

Allen's test[4] (Figure 5) is another test for thoracic outlet syndrome. The patient is seated with the shoulder abducted and externally rotated at 90° with the elbow flexed to 90°. The patient rotates their head toward the unaffected side and the clinician palpates to determine if the radial pulse is present, absent or diminished.

Figure 5

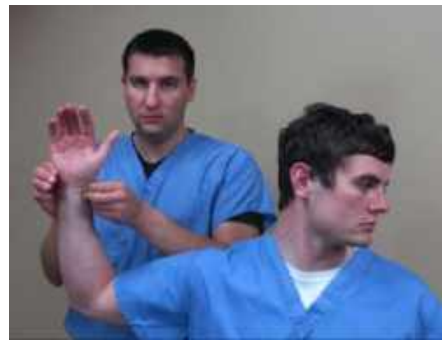


Figure 5: Allen's Test

Neuroforaminal stenosis involves compression of the nerve root as it exits the intervertebral foramen. This may occur with pathologies such as intervertebral disc herniation, degenerative disc disease, vertebral body spondylosis, bony overgrowth, or foreign bodies such as cancer. Spurling's test[5] (Figure 6) attempts to reproduce symptoms of nerve root compression in the cervical

spine. The test is performed on a seated patient with their head extended and side-bent toward the symptomatic side. The clinician applies a downward, compressive force of approximately 7 lbs. through the head, effectively further narrowing the intervertebral foramen. The test is positive if pain or paresthesias are reproduced down the arm, neck, head, or shoulder.

Figure 6

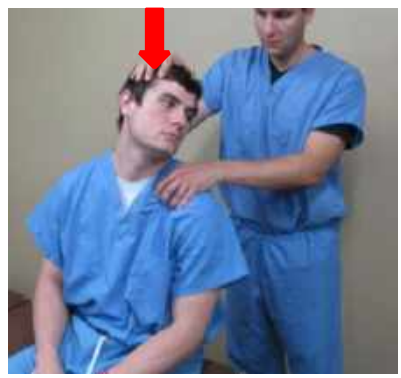


Figure 6: Spurling's Neuroforaminal Compression Test

It is worth mentioning that full neurovascular evaluation of the upper extremity should include testing of radial and ulnar pulses, and sensory as well as reflex testing of the biceps (C5), brachioradialis (C6), and tricep (C7) muscles. As clinicians, we are always evaluating to obtain a unifying diagnosis that will explain patients' complaints; in this way we are identifying and treating the underlying cause - NOT symptoms.

Orthopedic Special Tests for the Shoulder

This section reviews clinical tests for identifying specific pathology of the shoulder. Diagnosis of shoulder pathology is especially helpful for practitioners of manual medicine: we can correctly isolate the problem and later retest to determine if treatment is efficacious. These tests also aid in diagnosing hypermobility or instability and allow us to create a treatment plan that may include exercise, surgery, or prolotherapy.

Shoulder impingement syndrome refers to irritation of the supraspinatus tendon as it passes under the acromion and attaches to the humerus. Causes of this include bony projections from the acromion or deformity of the acromion that physically compress the tendon. During shoulder abduction, muscle imbalance of the rotator cuff muscles may lead to joint malfunction and pathological upward movement (instead of inward retraction) of the humeral head, ultimately compressing the tendon. The Hawkins–Kennedy test[6,7] (Figure 7) evaluates for shoulder impingement syndrome; the shoulder is positioned to 90° of flexion and then internally rotated. The test is positive if it reproduces pain.

Figure 7

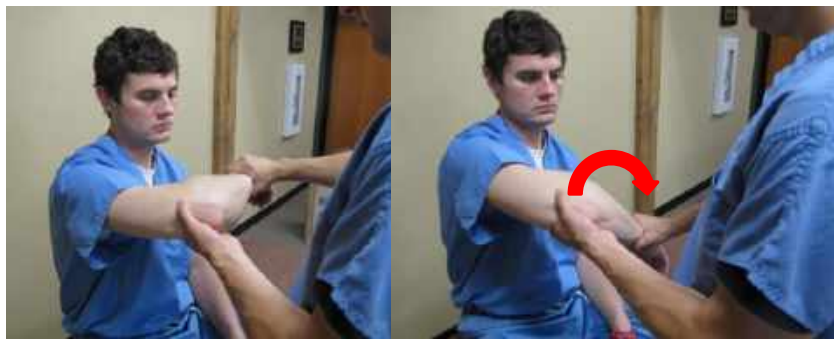


Figure 7: Hawkin's-Kennedy Test for Shoulder Impingement

Neer Test[7,9] (Figure 8) also assesses for shoulder impingement syndrome. This test is performed by forced, passive flexion/elevation of the humerus with internal rotation, while stabilizing the shoulder girdle. If symptoms are reproduced the test is positive.

Figure 8

Figure 8: Neer Test for Shoulder Impingement Syndrome

Note that the operator is stabilizing the scapula against the thorax with his left hand.

Supraspinatus tendon rupture usually occurs with repeated irritation of the tendon from impingement or trauma. The drop arm test[8] (Figure 9) evaluates for supraspinatus tendon rupture. The shoulder is passively abducted to ~170° and patient lowers their arm down to the side. Inability to lower the arm can indicate a significant rotator cuff tear. If pain is thought to limit the patient's ability to lower their arm, the test can be repeated after injection of an anesthetic into the subacromial space.

Figure 9

Figure 9: Drop Arm Test for Supraspinatus Tendon Tear

Recall from anatomy that the bicep attaches to the coracoid (short head) as well as inside the glenohumeral joint (long head). Yergason's test[9] evaluates for the pathology of the long head as it passes through the bicipital groove. This test is performed with the elbow flexed to 90° while the forearm is pronated (Figure 10). The clinician palpates the bicipital groove with one hand and provides resistance to active supination at the wrist. The test is positive if pain is perceived in the bicipital groove.

Figure 10

Figure 10: Yergason's Test for Pathology of the Long Head of the Bicep

Speed's test[10] also tests for pathology of the long head of the bicep in the bicipital groove. The elbow is maintained in extension and supination while the patient flexes the arm against resistance (Figure 11). Pain reproduced in the bicipital groove is a positive test.

Figure 11

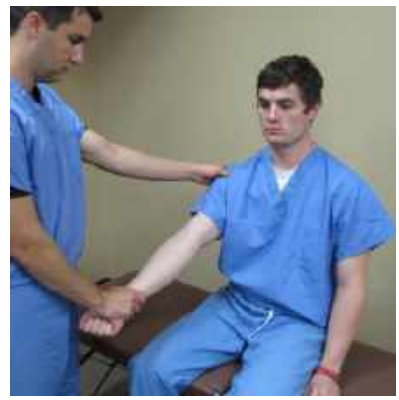
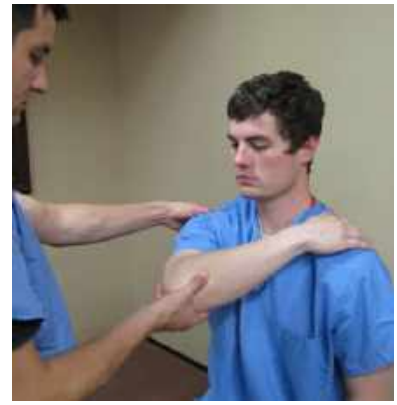


Figure 11: Speed's Test for Pathology of the Long Head of the Bicep

Acromioclavicular (AC) joint pathology may occur from osteoarthritis and AC-ligament disruption (commonly called shoulder separation). The cross-arm adduction test (aka, Apley-Scarf test)[7,16] assesses for pathology at the AC joint. The hand of the affected side is placed on the patient's contralateral shoulder and the clinician raises the elbow to 90°, effectively loading the AC joint (Figure 12). A positive test is present if pain localizes to the AC joint while the examiner further adducts the patient's arm.

Figure 12: Cross-arm Adduction Test for AC Joint Pathology

Figure 12

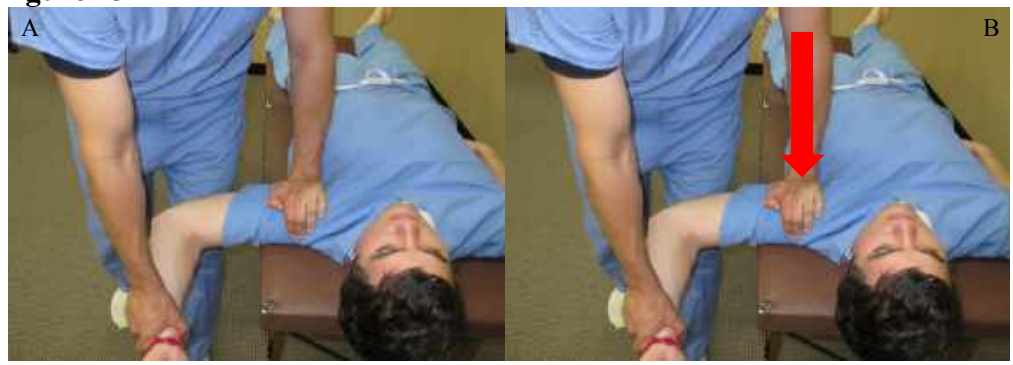


True shoulder dislocation occurs at the glenohumeral joint. Due to the shallow seating of the joint (i.e., golf ball on a tee) the joint is inherently unstable. The anterior and posterior apprehension tests and Jobe's relocation test evaluate for shoulder instability. To perform the anterior apprehension test[11] the patient is supine with a shoulder abducted and externally rotated to 90° (Figure 13). The arm is then further externally rotated; if the patient perceives their shoulder will dislocate, the test is positive. During the Jobe's relocation test,[12] the clinician repeats the anterior apprehension test with the exception of placing an anterior-to-posterior stress over the humeral head, stabilizing the joint. The test is positive if the operator can externally rotate the arm further before the patient feels apprehension. Note, to evaluate for anterior shoulder instability, the anterior apprehension test and Jobe's relocation test are used in conjunction.

Figure 13: Anterior Apprehension Test (A) and Jobe's Relocation Test (B).

Note that during the relocation test, an anterior to posterior force is placed on the humeral head.

Figure 13



The posterior apprehension test[13] (Figure 14) evaluates for posterior shoulder instability. To perform this test, the patient is supine with the humerus adducted ~20°. The clinician provides an axial compression from the elbow towards the ground. If the patient perceives their shoulder dislocating, it is a positive test.

Figure 14

Figure 14: Posterior Apprehension Test

Labral pathology is evaluated in a two-step method using Obrien's test[14] (aka active compression test); the shoulder is flexed to 90°, adducted ~20°, and internally rotated thumb down (Figure 15). The patient resists the clinician's downward force (red arrow in the picture). This is then repeated in the externally rotated position with the thumb up. If the patient feels pain "deep inside" the shoulder in the first step and pain is relieved during the second, there is suspicion of labral pathology.

Figure 15

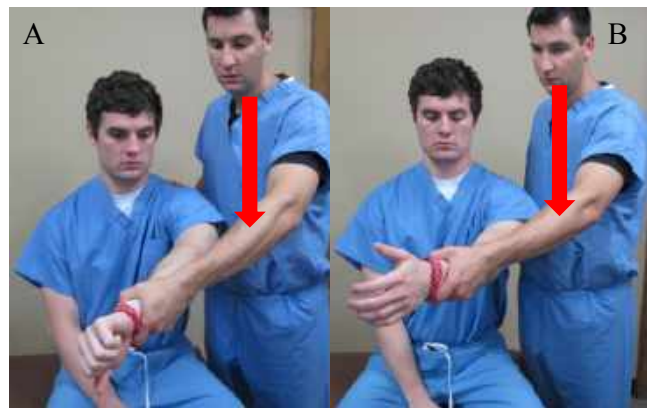


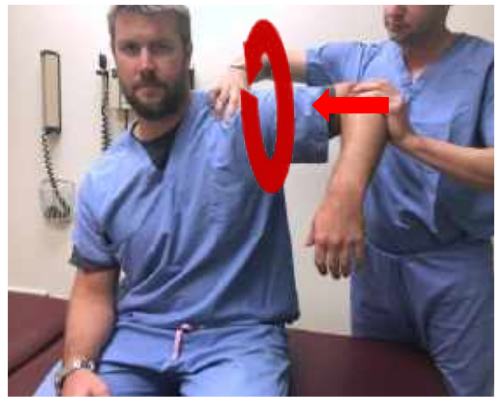
Figure 15: Obrien's Test for Labral Pathology

Note how the test is a two-step process in which the patient's thumb is initially pointed down in the first step and then up in the second step. The red arrow denotes the clinician's downward force while the patient is resisting.

The crank test[15] (Figure 16) also evaluates for labral pathology. During this test, the shoulder is abducted to ~160° in the scapular plane. While externally rotating the shoulder, an axial compression from the elbow toward the glenohumeral joint is applied. If the patient feels a clunk or pain deep inside the shoulder, it is a positive test.

Figure 16: The Crank Test for Labral Pathology

Figure 16



Understanding the orthopedic shoulder tests, while important, is not wholly comprehensive in the evaluation of the patient. These tests have both limitations in reproducibility between practitioners **and** in their ability to diagnose shoulder pathology. When compared to arthroscopy, the diagnosis gold standard, they have limitations of sensitivity and specificity that are included in Table 2.

Table 2: Statistical Analysis of Orthopedic Shoulder Tests

Table 2

Test	Sensitivity	Specificity	Positive Predictive value	Negative Predictive value
O'Brien[15]	67%	41%		
Crank[11] (combined with sulcus, apprehension, and relocation tests)	90%	85%		
Neer[9]	88.7%	30.5%		
Hawkins-Kennedy[6,7] (Rotator Cuff Pathology)[6] (Impingement)[7]	87.5% 91.7%	42.6% 44.3%	N/A 42.6%	
Drop Arm[8]	10%		100%	
Yergason's[9]	37%	86.1%		
Speed's[10]	90%	13%		
Cross-arm adduction (Apley Scarf)[7,16]	Not studied			
Apprehension test[11] (Combined Tests)	90%	85%		
Relocation (Fowler's)[12]	68%	100%		

Evaluation of Shoulder Somatic Dysfunction[19,20,21]

Osteopathic evaluation of the shoulder begins with understanding normal anatomy: specifically, origin and insertion of the muscles, the interplay of the spine and ribcage, neurovascular anatomy, and fascia. Many fail to recognize that distal structures influence shoulder mechanics. For instance, the latissimus dorsi or the quadratus lumborum may spasm, produce somatic dysfunction of the pelvis or ribcage, and ultimately alter the scapula tracking. After many years or even a few weeks this can cause clinical pathology of osteoarthritis or tendonosis/itis. It is not enough to diagnose shoulder impingement syndrome; the question is, *"Why did impingement develop*? *Was there bony overgrowth? Is there muscle imbalance or somatic dysfunction?"*

To truly master shoulder diagnosis and treatment, consider structures outside of the traditional orthopedic model such as fascia, lymphatics, and the diaphragm, as diagnosis and treatment of these structures are beyond the scope of this book. We begin by evaluating somatic dysfunction of the SC joint, AC joint, and the subacromial joint.

Subacromial Joint

Evaluation of the scapula begins with the scapular-clavicle motion test.[19] This two-step test is performed by placing the thenar eminence of both hands across the spine of the scapula (Figure 17). Digits 2-4 are placed comfortably across the medial to lateral clavicle. Passive motion is added using a posterior-medial spring with the thenar eminence and the fingers, almost as if unscrewing a lid from a large jar. If somatic dysfunction is present, one side will have restricted ROM compared with the other. Next, observe the position of the scapular spines; the abnormal side is compared with respect to the position of the normal side, inferior or superior.

Figure 17

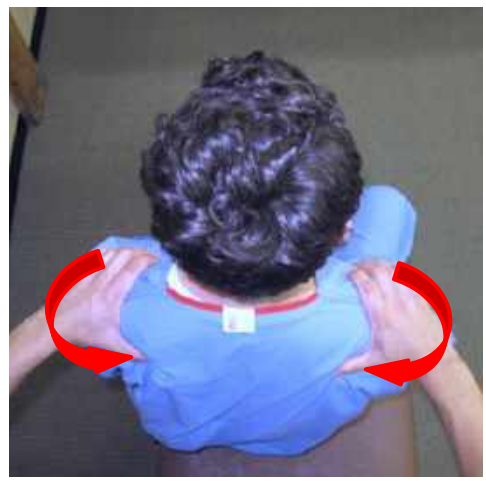


Figure 17: The Scapular-Clavicle Motion Test

Ideally, test both sides at the same time for symmetry.

To evaluate for SC joint restriction, we perform the sternoclavicular shrug test.[19] While standing in front the patient, the operator places the finger pads of their index fingers on the superior-medial edge of both clavicles, observing the position of the clavicle for symmetry. The patient then shrugs his shoulder by contracting the levator scapula (Figure 18).

Figure 18

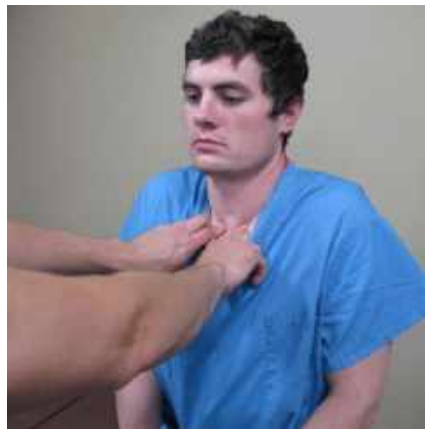


Figure 18: Sternoclavicular Shrug Test

Ideally, test both sides at the same time for symmetry.

A normal test is evident by symmetrical inferior movement during the shoulder shrug. The SC joint is diagnosed as "*inferior*" when it is initially inferior (compared to the opposing clavicle), and if during the shoulder shrug it does not glide further inferiorly. The SC joint is diagnosed as "*superior*" when it is initially superior (compared to the opposing clavicle) and if during the shoulder shrug it does not glide inferiorly.

SC joint restriction can result in first rib somatic dysfunction: the SC joint in a cephalad position results in an *inhaled first rib*, while a caudad position results in an *exhaled first rib*. Because of the first rib's close proximity to the sternal attachment, should first rib treatment become challenging, it is important to check for SC joint dysfunction, or unresponsiveness to manual medicine. Clinically, SC joint somatic dysfunction is frequently associated with thoracic outlet syndrome via first rib dysfunction.

Treatment of the Shoulder Somatic Dysfunction

In this section, we reviewed the anatomy of the upper extremity to emphasize the origin and insertion of the muscles. Additionally, to provide students a method of diagnosis, orthopedic tests were highlighted to emphasize abnormal functioning of these structures. As in all medicine, all treatments stem from correct diagnosis.

Once the diagnosis is obtained, treatment of the upper extremity involves the same basic principles taught throughout this text - specifically, during direct treatments the utilization of a fulcrum, lever, and force. Likewise, during

position and then exaggerate to a point of three-dimensional balance or patient comfort.

Specifically in the shoulder, we find that for direct treatments, muscle energy (ME) procedures produce excellent efficacy with the least risk of injury. Using strain-counterstrain (S/CS), indirect treatments may be utilized with minimal discomfort or risk. Both methods rely on knowledge of the muscles and their origin and insertion. For example, subscapularis originates from the anterior scapula and inserts medially to the lesser tubercle of the humerus; its function is to internally rotate the humerus. This knowledge allows us to find tender points at the insertion, origin, or belly of the subscapularis muscle and reduce its tenderness by internally rotating the humerus. With this same anatomical knowledge, treatment using ME involves stabilizing the scapula as a fulcrum, externally rotating the humerus to use the forearm as a lever, and employing an isometric contraction of the upper extremity; all while the operator maintains an unyielding counter force to the patient's internal rotation.

Application of these basic principles provides the launch point for further development in manual medicine.

Treatment Examples:[19,20, 21]

Superior Scapula:

Left Superior Scapula with ME

1. Stabilize the scapula to create a fulcrum so it remains immobile during treatment.
2. Create a lever by abducting the arm to ~85° in the plane of the scapula (*en scaption*) as shown by grasping the left arm.
3. Reverse the components of the positional diagnosis by turning the upper extremity thumb up; the operator will feel the scapula move inferiorly.
4. Once at the closed pack position, slightly release tension to the "feather edge" of the barrier.

5. Create an isometric contraction by instructing the patient to adduct their upper extremity while the operator maintains an unyielding counter force for 3-5 seconds. Instruct the patient to relax. Reposition at the new barrier by taking up the slack in further abduction. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times. By the end of treatment most patients will passively abduct to ~180°.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Left Superior Scapula with S/CS

1. Check for tender points of the teres major, and subscapularis muscles. Tender points may be at the musculo-tendonous junction or muscle belly.
2. The clinician maintains contact with the tender point, extends, abducts, and internally rotates the patient's arm in a position that alleviates at least 70% of the patient's tenderness.

3. Maintain this position for 90 seconds.
4. SLOWLY return the patient to the neutral position.
5. Recheck the original diagnosis to ensure resolution of tender point.

Inferior Scapula:

Left Inferior Scapula with ME

1. Stabilize the scapula to create a fulcrum so it remains immobile during treatment.
2. Create a lever by abducting the arm to ~85° in the plane of the scapula (*en scaption)* as shown by grasping the left arm.
3. Reverse the components of the positional diagnosis by turning the upper extremity thumb DOWN; the operator will feel the scapula move superiorly.
4. Once at the closed pack position, slightly release tension to the "feather edge" of the barrier.

5. Create an isometric contraction by instructing the patient to adduct their upper extremity while the operator maintains an unyielding counter force for 3-5 seconds. Have the patient relax. Reposition at the new barrier by taking up the slack in further abduction. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times. By the end of treatment most patients will passively abduct to ~180°.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Left Inferior Scapula with S/CS

1. Check for tender points of the teres minor and supraspinatus muscles. Demonstrated here is the teres minor tenderpoint (see Appendix A).
2. The clinician flexes, abducts, and externally rotates the patient's shoulder and elbow on the affected side until at least 70% of the tenderness is relieved

3. Maintain this position for 90 seconds.
4. SLOWLY return the patient to the neutral position.
5. Recheck the original diagnosis to ensure resolution of tender point.

Superior SC Joint:

Left Superior SC Joint with ME

1. Place a fulcrum on the SC joint using the thumb and index finger of the right hand. This hand will also monitor the SC joint for motion.
2. Create a lever by extending the patient's left arm as shown while grasping the left forearm.
3. As the arm is extended posteriorly, the components of the positional diagnosis will reverse, and the operator will feel the clavicle move inferiorly. Once at the closed pack position, slightly release tension to the "feather edge" of the barrier.

4. Create an isometric contraction by instructing the patient to flex their arm toward the while the operator maintains an unyielding counter force for 3-5 seconds. Have the patient relax. Reposition at the new barrier by taking up the slack in further extension. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Left Superior SC Joint with S/CS[32(pp46),20(pp46)]

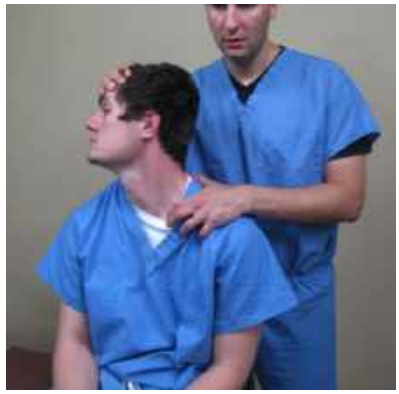
1. Check for anterior tender point C8 (Jones) or sternal attachment of the SCM (see Appendix A, Myers).
2. The operator uses marked flexion of the cervical spine with slight sidebending and rotation away from the tenderpoint until at least 70% of the tenderness is relieved.

3. Maintain this position for 90 seconds.
4. SLOWLY return the patient to the neutral position.
5. Recheck the original diagnosis to ensure resolution of tender point.

Inferior SC Joint:

Left Inferior SC Joint with ME

1. Place a fulcrum on the scapula by stabilizing it with the right hand. This index finger monitors the SC joint for motion.
2. Create a lever by extending/rotating the patient's neck as shown. The best vector to maximally activate the sternocleidomastoid muscle is determined by having the patient flex his head a few times to feel which motion produced upward movement of the medial clavicle. This effectively reverses the components of the positional diagnosis.
3. Once at the closed pack position, slightly release tension to the "feather edge" of the barrier.



4. Create an isometric contraction by instructing the patient to flex their head while the operator maintains an unyielding counter force for 3-5 seconds. Have the patient relax. Reposition at the new barrier by taking up the slack in flexion, sidebending, or rotation. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Left Inferior SC Joint with S/CS[32(pp97)]

1. Check for a tender point on the subclavius muscle (see Appendix A). It is located just medial to the coracoid process.
2. With patient seated, rotate the arm internally. Push the elbow anteriorly to further internally rotate the shoulder until at least 70% of the tenderness is relieved.

3. Maintain this position for 90 seconds.
4. SLOWLY return the patient to the neutral position.
5. Recheck the original diagnosis to ensure resolution of tender point

Elbow Anatomy

Located in the middle of the upper extremity, the shoulder has the ability to flex and extend to different lengths in time and space, thereby enabling the various functions of the hand. Often thought of as one joint, the elbow is actually two distinct joints: the humeroulnar joint that acts like a hinge for flexion and extension of the forearm and the radioulnar joint that allows forearm pronation and supination.

Figure 19

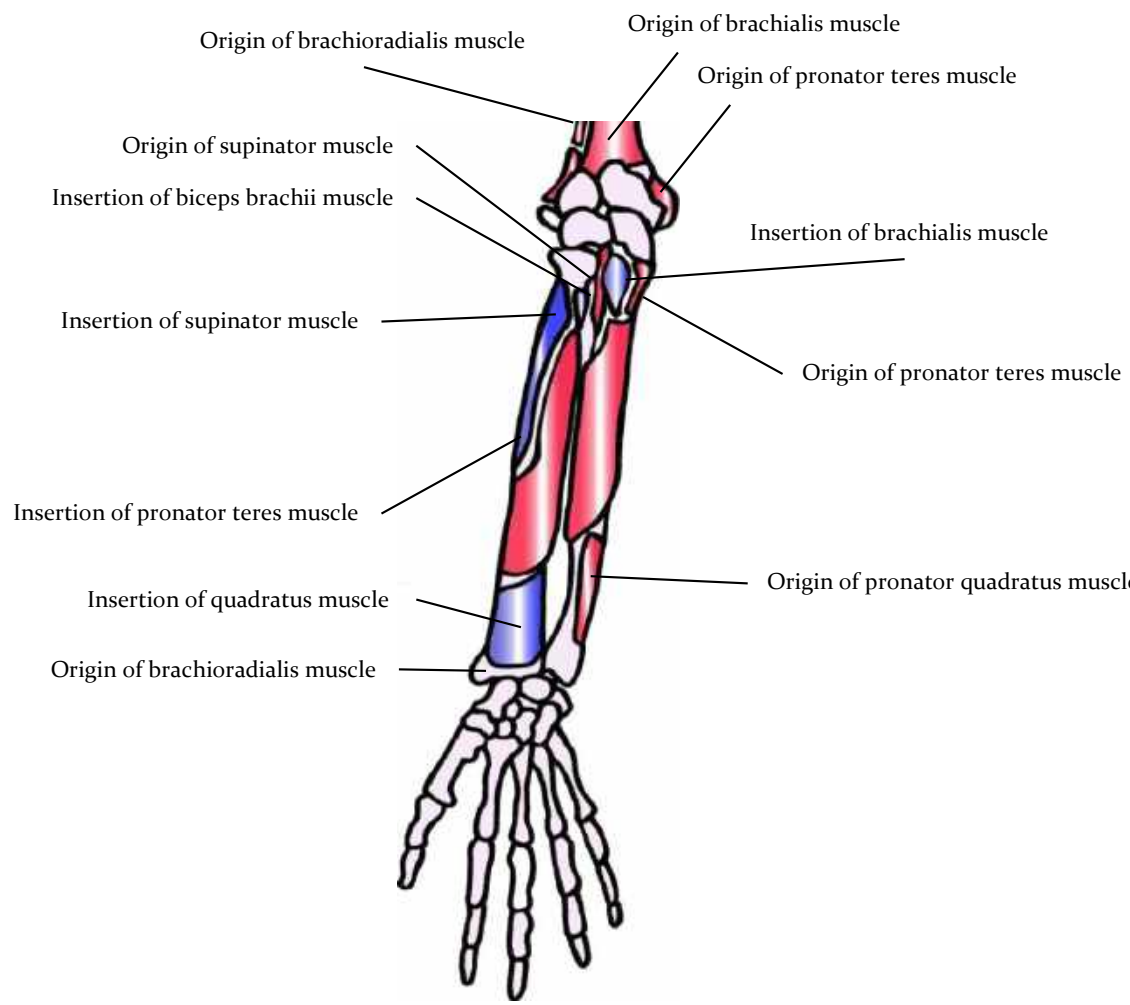


Figure 19: The origin and insertion of the forearm muscles

The radius and the ulna are connected by the interosseous membrane that prevents sliding of one bone on the other. On the lateral side of the ulna is a notch for articulation of the radial head. Muscles of the elbow and forearm are listed in Table 3. As in the shoulder, the origin and insertion point of muscles dictates diagnosis and treatment (Figure 19).

TABLE 3	
Muscle	Action
Brachialis	Flexion of the forearm
Biceps	Flexion of the forearm
Brachioradialis	Flexion of the forearm
Triceps	Extension of the forearm
Anconeus (not pictured)	Extension (minor extensor) of the forearm
Pronator teres	Pronates the forearm
Pronator quadrates	Pronates the forearm
Supinator	Supinates the forearm (note radial nerve travels through this muscle)

Table 3: Major muscle groups of the forearm

Orthopedic Evaluation of the Elbow

Since the elbow is an intermediary between the hand and shoulder, when considering dysfunction in the elbow it is important to examine proximally as well as distally. As with any patient interview, ask about the mechanism of injury, onset, duration, alleviating/exacerbating factors, occupational/repetitive tasks, sport specific tasks, referred pain, other joint involvement, and mechanical symptoms. For instance, in the overhead-throwing athlete, the elbow can often be the victim of abnormal motion patterns of the shoulder/scapula or disruptions in the kinetic chain.

Evaluation of the forearm should involve a systematic approach with the following sequence of evaluation:

1. Inspection
2. Range of motion
3. Palpation
4. Motor strength testing
5. Neurovascular testing
6. Orthopedic Special Tests
7. Evaluation for Somatic Dysfunction

Normal carrying angle
- Men 5-10°
- Women 10-15°

Inspection

Observe the patient in the anatomic position and observe the carrying angle that is the valgus angle between the shaft of the humerus and the centerline of the forearm. Normal is between 5-10° in men and 10-15° in women. Also, note any soft tissue swelling or bony deformities/asymmetries.

Range of Motion

Check active and passive ROM of flexion (135-150°) and extension (0-5°) that are the primary function of the humeroulnar joint. Pronation (90°) and supination (90°) are the primary function of the radioulnar joint.

Palpation

Starting at either the medial or lateral epicondyle, palpate both the bony and soft tissue structures of the elbow. The medial elbow palpatory exam consists of the medial epicondyle, common flexor, and the ulnar collateral ligament that is an important stabilizer of the elbow. The ulnar nerve can also be palpated passing through the ulnar groove of the distal humerus. On the lateral elbow, examine the lateral epicondyle, common extensor origin, radial head and annular ligament. Note any tenderness, or masses.

Strength
- 5/5 = normal strength

Motor strength

As with ROM, motor strength is tested in flexion/extension and pronation/ supination. The biceps are best tested with the wrist/forearm supinated, while the brachialis is tested more specifically with the forearm/wrist pronated.

Upper Extremity Reflexes
- Biceps = C5
- Brachioradialis = C6
- Triceps = C7

Neurovascular

The biceps reflex (C5) is tested by tapping the distal insertion of the biceps tendon in the antecubital fossa. The brachioradialis reflex (C6) is evaluated by tapping the tendon as it travels along the lateral forearm/wrist. The triceps' reflex (C7) is tested by tapping the triceps tendon at the posterior elbow towards its insertion on the olecranon process.

Nerve entrapment at the radial tunnel is evaluated using Tinel's test. The radial tunnel is approximately 4 fingerbreadths distal from the lateral epicondyle on the dorsal forearm. For the ulnar nerve, Tinel's test is also performed at the cubital tunnel. Perform sensation testing along the dermatomes (Figure 20): C5 lateral arm and elbow, C6 lateral forearm and thumb, C7 middle finger, C8 little finger and medial forearm, T1 medial elbow and arm. Palpate for pulse of the brachial artery in the cubital fossa.

Figure 20

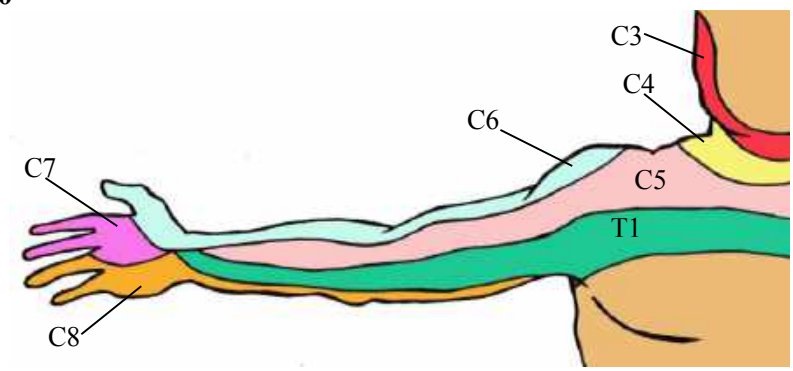


Figure 20: Upper Extremity Dermatomes

Clinicians combine motor strength testing with sensory and reflexes to aid in clinical diagnosis of radicular symptoms. For example, a patient who falls on her neck and exhibits diminished lateral arm sensation, decreased biceps reflex, and weakness of her bicep likely has cervical compression of the C5 nerve root and should be considered for imaging prior to initiation of manual medicine.

Orthopedic Special Tests

The third digit resisted extension test[22] is used to detect lateral epicondylosis (tennis elbow). The elbow is extended and the clinician resists as the patient extends the middle finger (Figure 21). The test is positive if the patient experiences a reproduction of lateral elbow pain with resisted 3rd digit extension, indicating pathology of the extensor carpi radialis brevis.

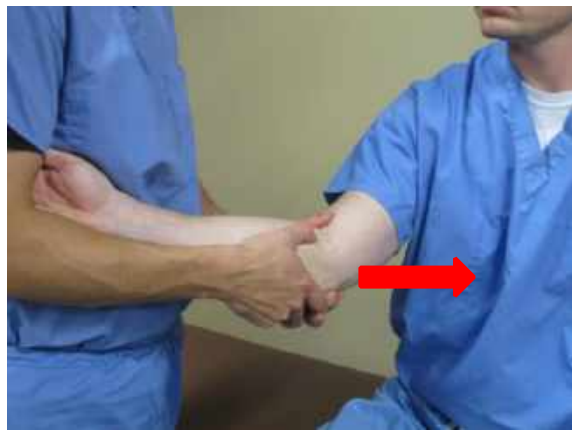
Figure 21

Figure 21: Third Digit Resisted Extension Test

The Ulnar collateral ligament (aka medial collateral ligament) valgus stress test[23] is performed to detect laxity. The clinician places one hand on the lateral elbow and the other hand on the distal forearm and applies a valgus load at 0 and 30° of flexion (Figure 22). The test is performed on the contra-lateral side for comparison. The test is positive when the joint opens up compared to the opposite side, or the patient experiences a reproduction of painful symptoms.

Figure 22: Ulnar Collateral Ligament Valgus Stress Test

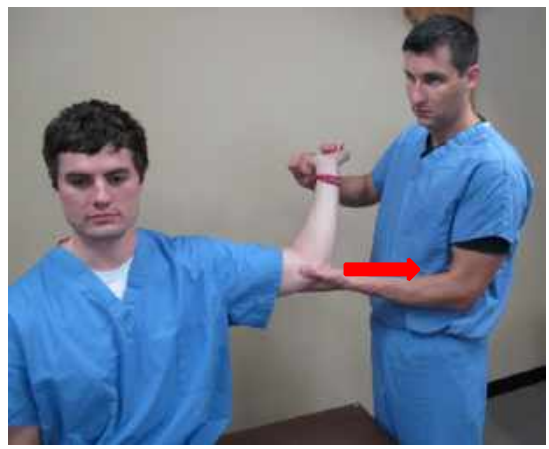
Figure 22



The dynamic milking maneuver[24] is more of a functional test for overhead athletes with medial elbow pain as it tests for ulnar collateral ligament instability/pain. The shoulder is positioned at 90° of abduction and at varying degrees of elbow flexion (usually from 30 to 60°). The examiner holds the thumb of the affected extremity with one hand and stabilizes the elbow on the lateral side with the other hand; they then externally rotate the elbow while applying a valgus load to the elbow (Figure 23). The test is positive if the patient has a reproduction of pain symptoms.

Figure 23: Dynamic Milking Maneuver

Figure 23



The Radial head subluxation - lateral pivot shift test[25] evaluates posterolateral elbow instability and is best performed with the patient under anesthesia. With the patient supine and the forearm supinated, the clinician holds the elbow in full extension while maintaining a valgus load on the elbow (Figure 24). While supinating and flexing the elbow to approximately 40°, from the extended position the clinician introduces an axial load to the elbow. During this maneuver there is a palpable subluxation then reduction of the radial head.

Figure 24

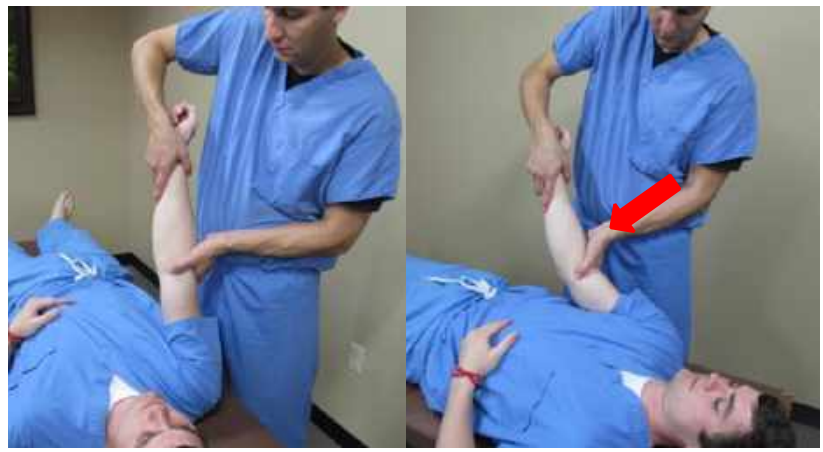


Figure 24: Radial Head Subluxation – Lateral Pivot Shift Test

Radial collateral ligament (aka lateral collateral ligament) stability - varus stress test:[26] Place the patient at 20° of elbow flexion (Figure 25) and apply varus force to assess laxity of lateral collateral ligament.

Figure 25

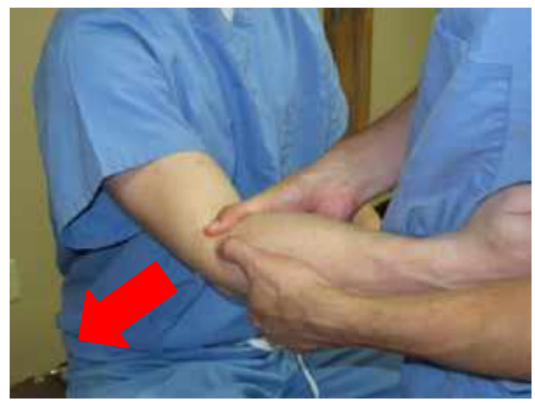


Figure 25: Radial Collateral Ligament Stability - Varus Stress Test

Due to practitioners' inability to reproducibly diagnose pathology, elbow special tests have limitations. The evaluation of sensitivity and specificity compared to the gold standard of diagnosis with arthroscopy or MRI arthrogram are included in Table 4.

Table 4: Statistical Analysis of Elbow Orthopedic Tests

Table 4				
Test	Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value
3rd digit resisted extension[22,34]	88%			85%
Valgus stress[23,35]	66%	60%		
Dynamic milking[24,36]	100%	75%		
Lateral pivot shift[25,37]	100% (anesthesia) 38% (awake)			
Varus stress[26]	Not studied			

Evaluation of Elbow Somatic Dysfunction

Clinically, radial head dysfunction can be related to biomechanical restriction and pain complaints at the elbow. In the case of lateral epicondylosis or overuse tendinopathy, it can also be related to pain syndromes in the shoulder and wrist (i.e., carpal tunnel syndrome).

Diagnosis of Radial Head Somatic Dysfunction

The patient is sitting with arms to the side and elbows at 90° of flexion. The operator holds both forearms proximal to the wrist so that the retinaculum of the wrist can also be monitored by palpation. The operator passively tests for both pronation, and supination.

Figure 26

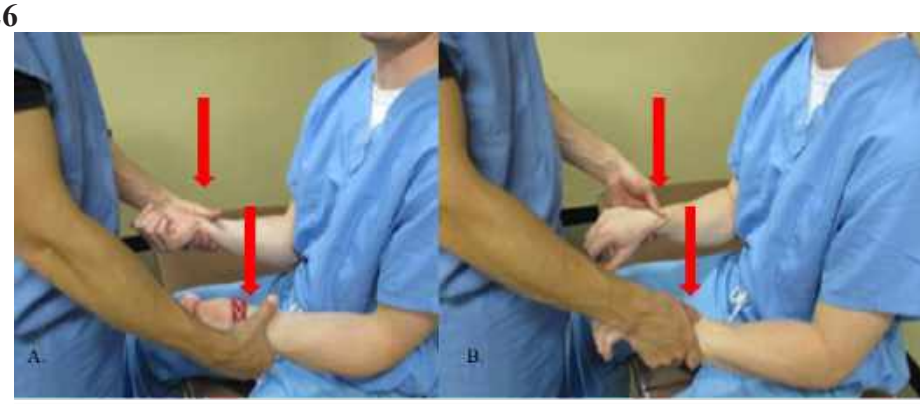


Figure 26: Biomechanical Testing of Radial Head Somatic Dysfunction

Note that the operator "springs" inferiorly into the elastic barrier in both supination (A.) and pronation (B.) comparing the right and left sides.

Treatment of Elbow Somatic Dysfunction

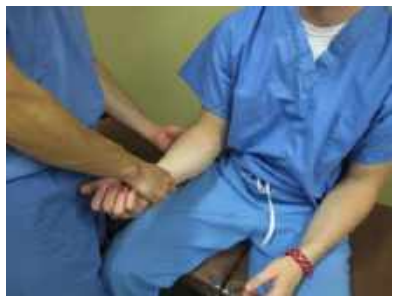
Again, we emphasize that there are numerous tender points in this area, often on the opposite side of the patient's complaint. Recalling the origin and insertion of the muscles will allow a practitioner to easily treat with indirect methods.

Treatment Examples:[19,20,21]

Pronated Radial Head

Pronated Radial Head: ME

1. Treat in the position the diagnosis was made in (i.e., supination). To treat a pronated radial head, supinate to the restrictive barrier.
2. Create a fulcrum at the ulna by holding the olecranon process.
3. Create a lever with the radius.



4. Create an isometric contraction by instructing the patient to pronate the forearm and wrist for 3-5 seconds. Note, the clinician should be able to monitor the rotation of the radial head. Instruct the patient to relax. Reposition at the new barrier by taking up the slack in supination. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Pronated Radial Head: HVLA

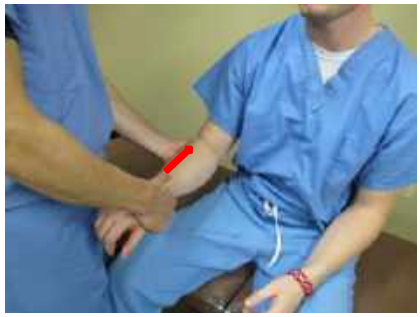
1. As in prior treatments, the clinician is facing the patient, while the patient holds the elbow flexed at 90◦. The clinician places one hand on the elbow while monitoring the radial head and holds the patient's wrist with the other hand.
2. Create a fulcrum at the ulna by holding the olecranon process.
3. Create a lever with the radius.

4. The clinician supinates the radial head to the restrictive barrier, and to find the best vector, springs the radial head posteriorly (in this instance with his left thumb) from different positions of external and internal rotation of the elbow. Once this vector is identified, the clinician provides the impulse while simultaneously supinating the distal radius.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Pronated Radial Head

Pronated radial head: Functional Procedures

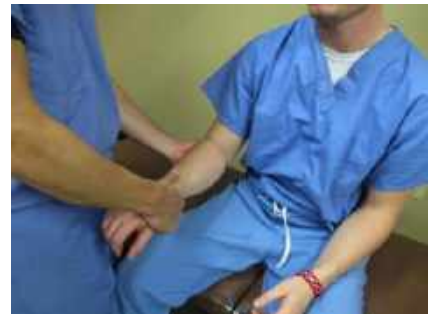
1. The clinician holds the patient's elbow at approximately 90° of flexion while supporting the elbow and simultaneously monitoring the radial head with their left hand. The other hand holds the patient's wrist.



2. The operator then places the forearm in the positional diagnosis by introducing pronation.
3. Effort is made to find the dynamic balance point
4. An axial compression is added toward the radial head initiating treatment.
5. The clinician rechecks motion of the radial head with supination and pronation.

Pronated radial head: S/CS

1. The clinician palpates for the tender point of pronator teres (or pronator quadratus).
2. While maintaining contact with the tender point, the clinician places the forearm in the positional diagnosis by introducing pronation.
3. Tenderness is reduced to a 3 out of 10.

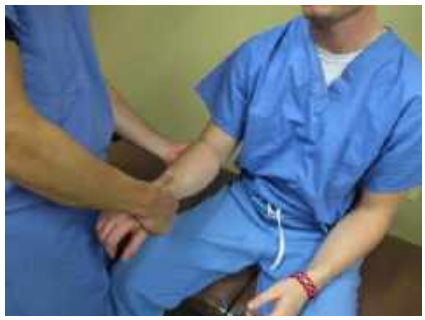


4. Maintain this position for approximately 90 seconds.
5. SLOWLY, return the elbow to the neutral position.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Supinated Radial Head

Supinated Radial Head: ME

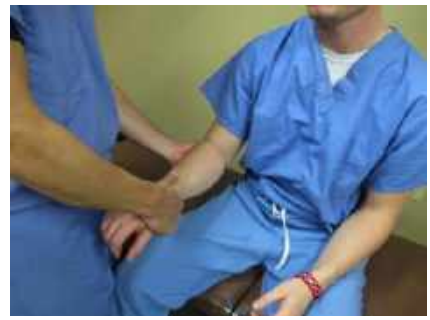
1. Treat in the position the diagnosis is made (i.e., pronation).
2. Create a fulcrum at the ulna by holding the olecranon process.
3. Create a lever with the radius.



4. Create an isometric contraction by instructing the patient to supinate the forearm and wrist for 3-5 seconds. Note, the clinician should be able to monitor the rotation of the radial head. Have the patient relax. Reposition at the new barrier by further pronating. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Supinated Radial Head: HVLA

1. As in prior treatments, the clinician is facing the patient, while the patient holds the elbow flexed at 90°. The clinician places one hand on the elbow while monitoring the radial head and holding the patient's wrist with the other.

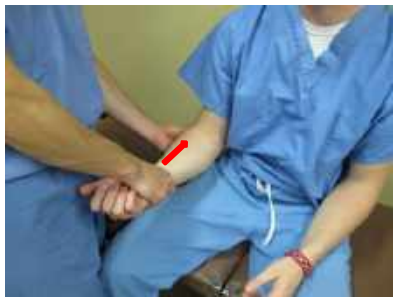


2. Create a fulcrum at the ulna by holding the olecranon process.
3. Create a lever with the radius.
4. The clinician pronates the radial head to the restrictive barrier. Then, to find the best vector for pronating the radial head, springs the radial head anteriorly (in this instance, with his left thumb) from different positions of external and internal rotation. Once this vector is found, the clinician provides the impulse while simultaneously pronating the distal radius.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Supinated Radial Head

Supinated radial head: Functional Procedures

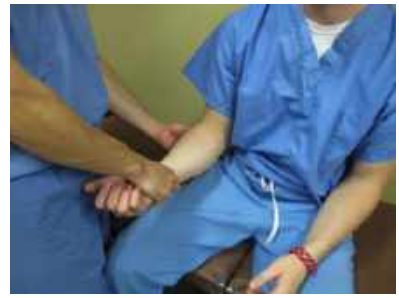
1. The clinician holds the patient's elbow at approximately 90° of flexion while supporting the elbow and simultaneously monitoring the radial head with his left hand. The other hand holds the patient's wrist.



2. The operator then places the patient in the positional diagnosis by supinating the forearm.
3. Effort is made to find the dynamic balance point
4. An axial compression is added toward the elbow to initiate treatment.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Supinated radial head: S/CS

1. The clinician palpates for the tender point of supinator muscle.
2. While maintaining contact with the tender point, the operator then places the patient in the positional diagnosis by supinating the forearm.
3. Tenderness is reduced to a 3 out of 10.



4. Maintain this position for 90 seconds.
5. SLOWLY return the elbow to the neutral position.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Wrist/Hand Anatomy

As mentioned in the elbow section, the radius and ulna form the distal radial ulnar joint (DRUJ). The distal ulna has a head and medial projection named the ulnar styloid. The head has an articular surface for the radius. The radius has two articular surfaces: a broad concave surface for the proximal carpal row and another for the ulna. It also has a distal projection named the radial styloid. The triangular fibrocartilage complex (TFCC) is formed by the triangular fibrocartilage disc, the ulnocarpal ligaments, and the radioulnar ligaments (Figure 27).

Figure 27

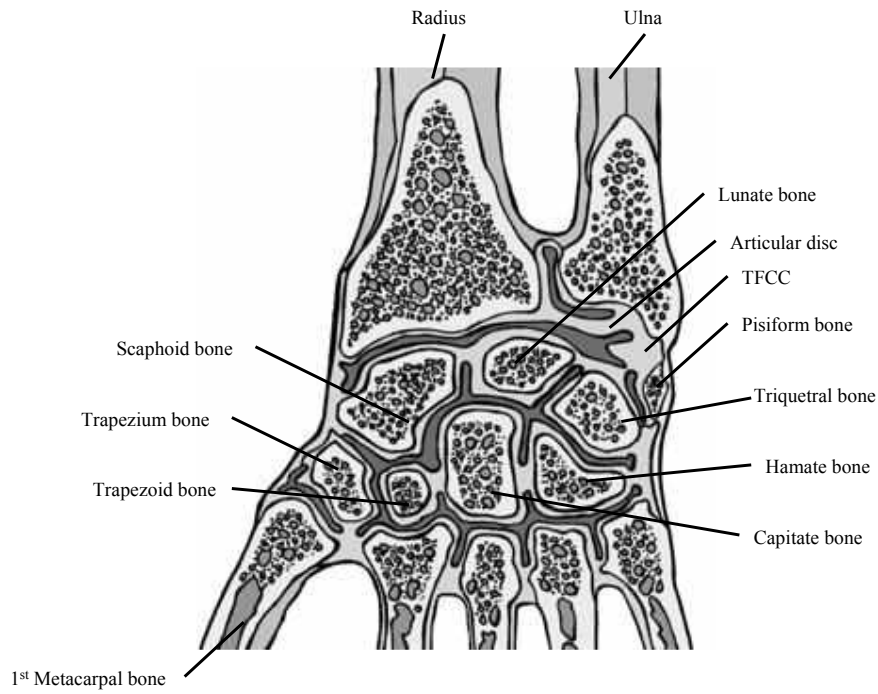


Figure 27: Bones and Ligaments of the Hand and Wrist

TFCC = Triangular fibrocartilage complex.

The wrist joint is made up of the radiocarpal and midcarpal joints. The distal radius articulates with three of the four bones of the proximal carpal row: scaphoid, lunate and triquetrum (pisiform does not articulate) to form the radiocarpal joint. The midcarpal joint is composed of the proximal and distal carpal row: trapezium, trapezoid, capitate and hamate. The proximal carpal row forms a concave surface for articulation with the distal carpal bones. The distal carpals then articulate with the metacarpal bases.

Wrist flexion and extension both occur at the radiocarpal and midcarpal joints, while ulnar and radial deviation occurs at the radiocarpal joint. In contrast to the elbow that relies on the ulna's bony interlocking features for stability, most wrist stability is attributable to its strong surrounding ligaments. The main stabilizing ligaments involved in wrist flexion and extension are the palmar and dorsal radiocarpal ligaments. The radial and ulnar collateral ligaments stabilize the wrist in radial and ulnar deviation.

Figure 28

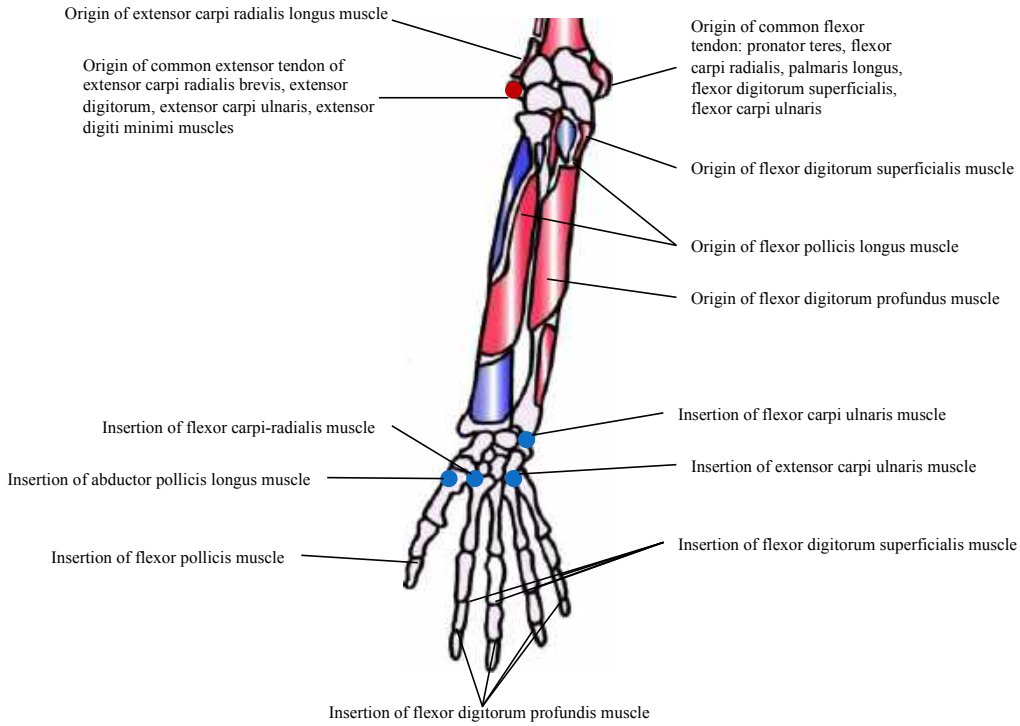


Figure 28: Origin and Insertion of the Hand and Wrist Muscles

TABLE 5	
Muscle	Action
Flexor carpi radialis (FCR)	Wrist Flexion with the FCU; radial deviation if it contracts alone. Note: the ulnar nerve passes between the heads of the FCU and FCR.
Flexor carpi ulnaris (FCU)	Wrist Flexion with the FCR; ulnar deviation if it contracts alone.
Palmaris longus	Wrist Flexion (minor flexor)
Extensor carpi radialis longus (ECRL)	Wrist Extension with the ECRB and ECU; radial deviation if it contracts alone.
Extensor carpi radialis brevis (ECRB)	Wrist Extension with the ECRL and ECU; radial deviation if it contracts alone.
Extensor carpi ulnaris (ECU)	Wrist Extension with the ECRL and ECRB; ulnar deviation if it contracts alone.

Table 5: Major muscle groups of the hand and wrist

Orthopedic Evaluation of the Wrist/Hand

Evaluation of the hand and wrist should also involve a systematic approach using the following sequence of evaluation:

1. Inspection
2. Range of motion
3. Palpation
4. Motor strength testing
5. Neurovascular testing
6. Orthopedic Special Tests
7. Evaluation for Somatic Dysfunction

Inspection

After an acute injury, observe for any deformities such as swelling or break in the skin; also note how the patient holds their hand or if they are able to shake the examiner's hand.

Range of Motion

Test flexion/extension (80/90°), supination/pronation (90/90°), and radial/ulnar deviation (20/30°) actively, as well as passively. Also, observe opposition and abduction of the thumb. Note finger flexion/extension and MCP abduction/adduction.

Palpation

Palpate the distal radius, DRUJ, distal ulna, proximal carpal row including carpal joints (e.g., scapholunate joint), TFCC, distal carpal row, proximal metacarpals, and anatomic snuffbox.

Motor strength testing

Test grip strength and resisted flexion and extension of the wrist. Test the thumb using resisted thumb flexion/extension/abduction/adduction/opposition, MCP flexion/extension, PIP and DIP flexion/extension, and finger abduction/adduction.

Figure 29

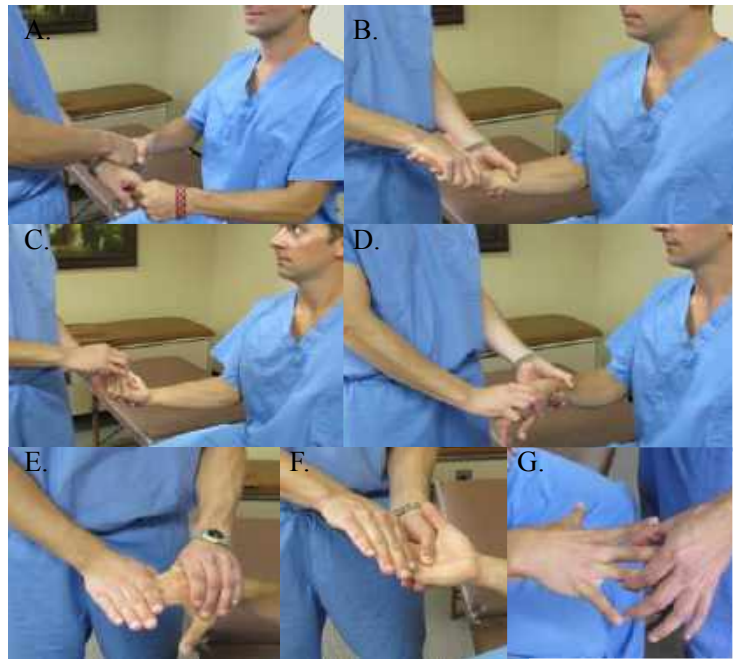


Figure 29: Strength Testing of the Hand

A. Hand grip
B. Resisted wrist flexion
C. Resisted MCP flexion
D. Resisted wrist extension
E. Resisted MCP extension
F. Resisted PIP flexion
G. Resisted finger abduction

Neurovascular

If clinically relevant, perform the Tinel's test for median nerve entrapment at the wrist or ulnar nerve compression at Guyon's canal.

Orthopedic Special Tests

Finkelstein's Test[27] (Figure 30) evaluates for De Quervain's tenosynovitis that is actually tendonosis of the abductor pollicis longus and extensor pollicis brevis tendons. The patient makes a fist with the fingers wrapped around the thumb. While moving the wrist into a position of ulnar deviation to reproduce the radial-sided symptoms, the clinician braces the forearm with one hand and grasps the patient's fist with the other hand,

Figure 30

Figure 30: Finkelstein's Test

Phalen's Test[28,29] (Figure 31) assesses for median nerve entrapment and what is commonly referred to as carpal tunnel syndrome. The patient is seated with the dorsal aspect of both hands in contact and both wrists flexed; the position is held for one minute. A positive test is evident if the patient experiences paresthesia in the median nerve distribution of the affected hand.

Figure 31

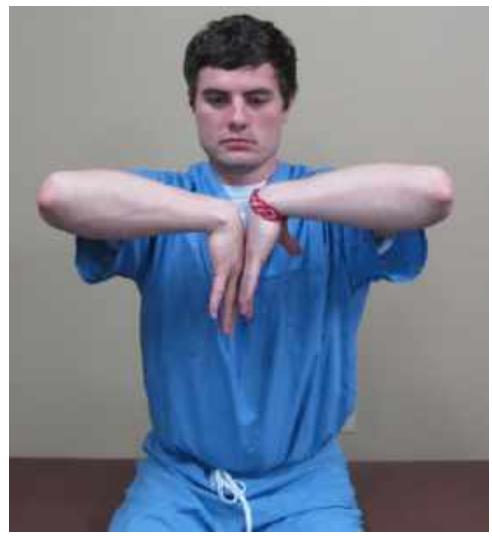


Figure 31: Phalen's Test

Tests for carpal tunnel syndrome (paresthesia of digits 1-3, and lateral half of 4).

Watson's Test[30] (Figure 32) evaluates for scapholunate subluxation/instability secondary to ligament injury. The patient is seated with the elbow flexed at 90° while the clinician palpates the scaphoid tubercle on the volar side with his thumb while holding the wrist in ulnar deviation. The patient is then instructed to move the wrist into radial deviation as the clinician discerns a palpable subluxation and reduction of the scaphoid (the scaphoid will move dorsally) - this indicates scapholunate dissociation. If the patient only experiences pain with this maneuver, then there is suspicion for scapholunate enthesopathy.

Figure 32

Figure 32: Watson's test

The Ulno-carpal stress test (TFCC grind test)[31] (Figure 33) assesses for TFCC injury; it can be thought of as a McMurray's test for the wrist. The clinician

braces the DRUJ with one hand and grasps patient's hand with the other. The patient's wrist is placed in maximal ulnar deviation while adding a rotational axial load. If painful clicking or a reproduction of typical pain symptoms are present, suspect TFCC injury or disruption.

Figure 33

Figure 33: TFCC Grind Test

During ulnar deviation, an axial load is introduced while rotation is performed.

Table 6 lists statistical data regarding the orthopedic special tests. Note the relative limitations compared to the gold standard of MRI arthrography and arthroscopy for TFCC injury.

Table 6

Test	Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value
Finkelstein's[27]	Not studied			
Phalen's[8,29]	71%	80%		
Watson's[30]	69%	64-68%		
Ulno-carpal stress (TFCC grind)[31]	Not studied			

Table 6: Statistical Analysis of the Orthopedic Special Test for the Hand and Wrist

Evaluation of Wrist/Hand Somatic Dysfunction[19,21]

Carpal bone restriction often occurs in the proximal row, likely because the distal row has greater ligamentous density and hence less motion. Somatic dysfunction of the carpal bones most commonly results from restricted ROM in the anterior-to-posterior (AP) plane. During the examination, the operator places both thumbs on the dorsal side and their index fingers on the volar side of the patient's pronated wrist (Figure 34). Diagnosis is made as the examiner palpates individual carpal bones and introduces an AP springing motion to detect restriction of motion; the examination is repeated across the proximal and distal carpal rows. Once somatic dysfunction is detected, the operator initiates direct or indirect procedures to treat the restriction.

Figure 34

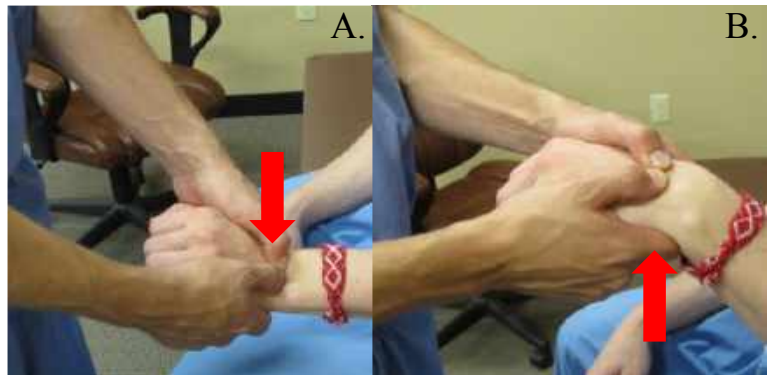


Figure 34: Manual Medicine Evaluation of the Carpal Bone Somatic Dysfunction

Note that the operator introduces wrist extension while compressing down on the carpal bones (A.) and then introduces wrist flexion while compressing up from the volar side (B.)

Theoretically, a carpal bone can have somatic dysfunction in a position that is anterior, posterior, rotated, flexed, or extended. Palpation of the exact position requires experience beyond the scope of beginners. At this educational level, we focus on freedom of movement in flexion or extension. If somatic dysfunction is noted during wrist flexion, the bone is extended and vice versa.

Treatment of Wrist/Hand Somatic Dysfunction

For direct treatments, use a fulcrum and lever; force is applied at the restriction barrier. Indirect treatments require placement in the positional diagnosis at the position of ease (functional procedures), or in a position that yields a reduction in maximal tenderness (S/CS).

Treatment Examples:
Wrist somatic dysfunction[19,20,21]

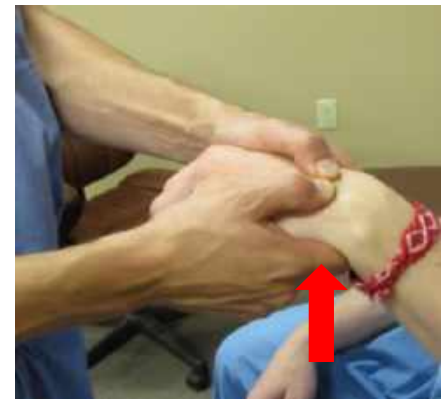
Extended Carpal Bone: ME

1. As with prior procedures, the clinician stands facing the patient, with the patient's elbow flexed at 90° in the hand and wrist is in a pronated position. The clinician places thumbs on the dorsal surface of the carpal bones, and fingers on the volar surface of the involved carpal.
2. Placing the wrist in flexion reverses the positional diagnosis. A fulcrum is naturally placed under the wrist when grasped. The hand acts as the lever.

3. After motion testing and determining which specific carpal bone is extended, the clinician finds the closed packed position of the restrictive barrier by moving the joint through all planes of motion: abduction/adduction, flexion /extension, pronation/supination. The clinician then releases the closed packed position to the "feather-edge."
4. Create an isometric contraction by instructing the patient to extend the hand for 3-5 seconds. Have the patient relax. Reposition at the new barrier by further flexing the wrist. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Extended Carpal Bone: HVLA

1. As with prior procedures, the clinician stands facing the patient with the patient's elbow flexed at 90° and the hand and wrist in a pronated position. The clinician places thumbs on the dorsal surface of the carpal bones and fingers on the volar surface of the involved carpal.
2. Placing the wrist in flexion reverses the positional diagnosis. A fulcrum is naturally placed under the wrist when grasped. The hand acts as the lever.



3. After motion testing and determining which specific carpal bone is extended, the clinician finds the closed packed position of the restrictive barrier by moving the joint through all planes of motion: abduction/adduction, flexion /extension, pronation/supination.
4. If the carpal bone is extended, the activating thrust will be directed in an anterior-to-posterior direction, or towards the ceiling (if extended, the activating thrust will be directed in a posterior-to-anterior direction, towards the floor).
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Wrist somatic dysfunction[19,20,21]

Extended Carpal Bone: Functional Procedures

1. The clinician identifies restricted carpal bones and places a finger and thumb of one hand on either side of the affected bone. The other hand holds the patient's hand as if "shaking hands."

2. If the carpal bone is "extended," the clinician extends the wrist to introduce the positional diagnosis.
3. The wrist is fine-tuned to the dynamic balance point, and the clinician then adds an axial compression or distraction to initiate treatment.
4. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Extended Carpal Bone: S/CS

1. Tender points are typically on the dorsal side.
2. The clinician checks for tender points related to the tendons of extensor digitorum superficialis, extensor carpi radialis longus and brevis, and extensor carpi ulnaris.
3. Once the tender point is identified, the clinician places the wrist in the positional diagnosis by extending the wrist and then fine-tunes with side bending toward the tender point, or until at least 70% of the tenderness is relieved.

4. The position of comfort is held for 90 seconds, before returning the patient's wrist and elbow to the neutral position.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Upper Extremity Treatment Pearls

Presuming somatic dysfunction is present in patients presenting with upper extremity pain, manual medicine provides a simple and practical diagnostic test. This method affords two advantages: confirmation of primary somatic dysfunction since the patient's symptoms will dramatically improve or resolve prior to leaving the office, and identification of secondary somatic dysfunction that is often related to underlying ligamentous hypermobility or tendinopathy. Also, as the patient improves from treatment, having a skillset to examine, diagnose, and treat somatic dysfunction often eliminates the need for radiographs, MRIs, physical therapy, bracing, or medication.

We find that many times, especially in cases of suspected thoracic outlet syndrome or cervical radiculopathy, treatment complements standard orthopedic testing in this same way; either the patient's complaints improve, or they do not.

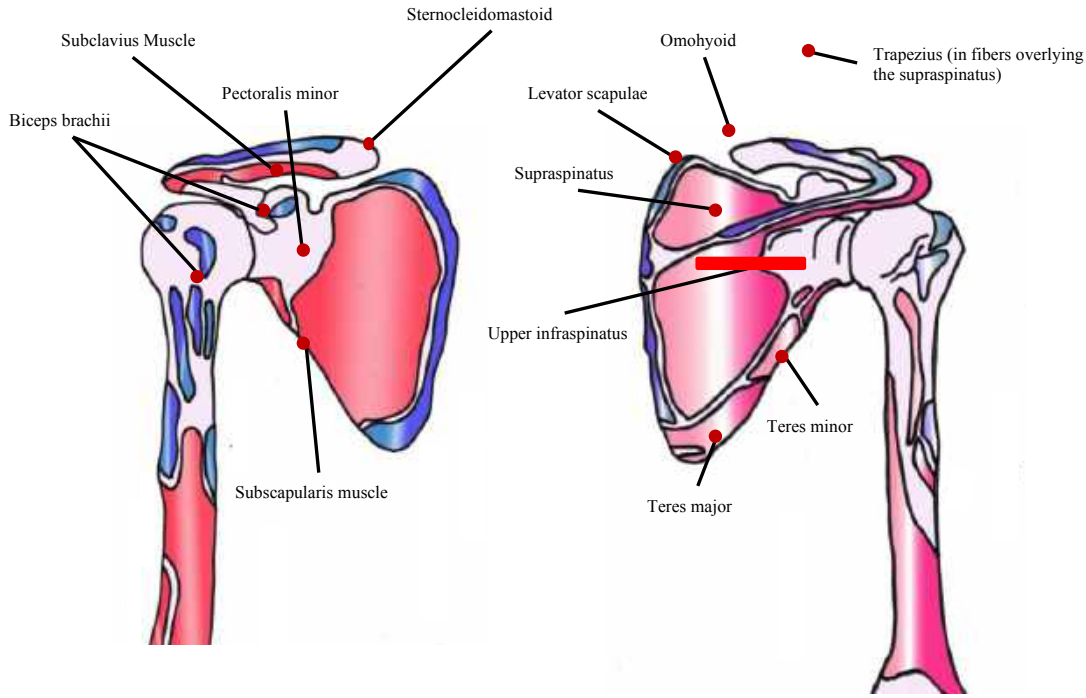
Anatomical knowledge of the insertion and origin of the upper extremity musculature is imperative when utilizing S/CS procedures. Shortening the involved muscle is usually accomplished by internal or external rotation plus-or-minus compression or distraction. Clinically, determining the anatomical structure with surface palpation is challenging; the position of ease often helps delineate between two structures in close proximity, such as internal rotation with the teres major and external rotation with the teres minor.

Always evaluate for and exclude red flags. When in doubt, avoid HVLA to reduce the probability of worsening of the patient's pathology.

Appendix A: Shoulder S/CS Tenderpoints[20]

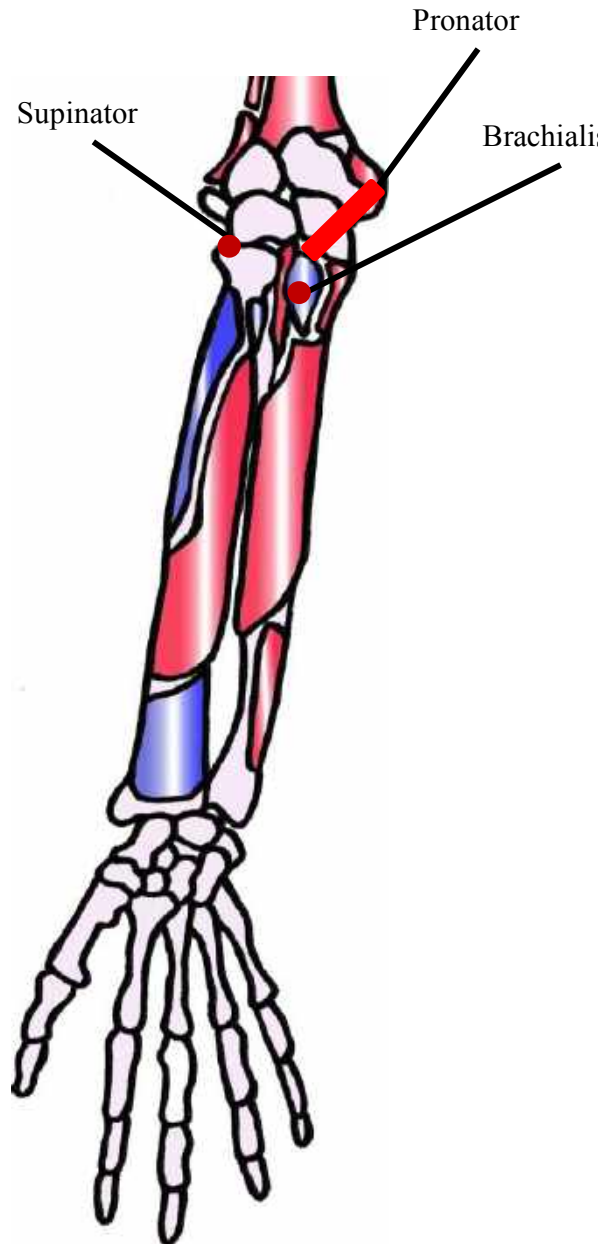
The Biceps brachii has tenderpoints in the bicipital groove (long head tendon) and on the coracoid process (short head). Meyers[32(pp94)] notes the pectoralis minor has a tenderpoint 3 to 4 cm inferior to, and 1 to 2 cm medial to the coracoid process at the musculo-tendon junction. He further correlates Jones' 1st-2nd rib tenderpoints as the those of the pectoralis major.[32(pp95)] The subscapularis is an often overlooked as the cause of rotator cuff pathology; its tenderpoint is on the anterolateral aspect of the scapula. Treatment is as expected, with exaggerated adduction and internal rotation on a supine patient. Author JB has encountered left subscapularis tenderpoints in patients admitted through the emergency room for chest pain evaluation. Ultimately, these patients are evaluated for chest pain, and when ruled-out for acute life-threatening causes, treated with manual medicine.

Posterior tenderpoints include the supraspinatus, infraspinatus, teres minor, and teres major. Above the scapular spine, the supraspinatus tenderpoint resides in the muscle belly. The tenderpoint designated upper infraspinatus is located in a band-like distribution below the scapular spine, while tenderpoint lower infraspinatus is located anywhere in the belly of the muscle. On the lateral border of the posterior scapula lays the teres minor tenderpoint and just below that the teres minor. Recall that the teres major attaches to the humerus anteriorly and so its treatment position is very similar to the subscapularis. The trapezius and levator scapulae are tonic muscles that often remain spasmed as a backup mechanism to nuchal/interspinous ligament disruption (as in whiplash injury) or poor posture resultant from chronic flexed neck position. In practice, we observe these tenderpoints frequently and recognize them as secondary somatic dysfunction rather than primary. Lastly, the omohyoid is a strap muscle with two bellies that originates medial to the suprascapular notch. The omohyoid tenderpoint is in the belly of the inferior portion and located anterior to the first rib's posterior tenderpoint. Many times, it is implicated in TMJ disorder as well as myofascial pain of the arm, shoulder, hand, neck scapular, supraclavicular, and mandibular areas.[33]



Appendix B: Elbow S/CS Tenderpoints[20]

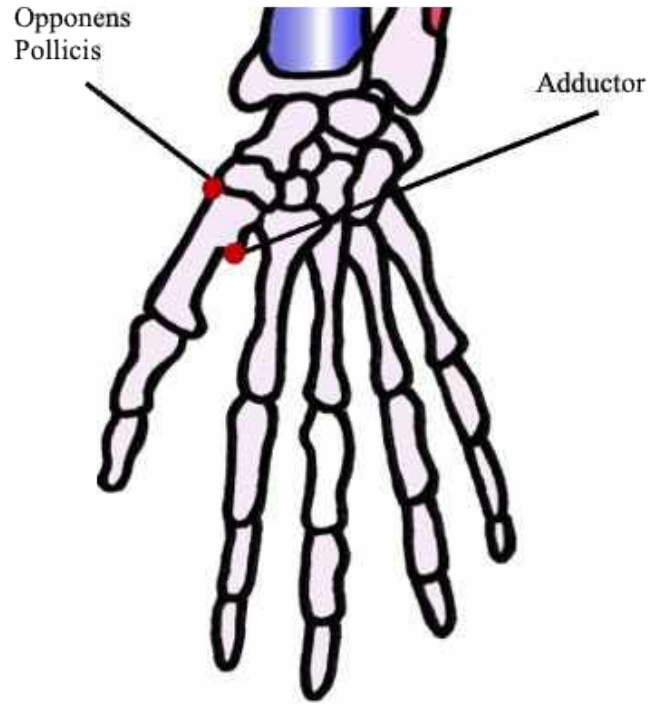
Triceps tenderpoint (not pictured) is located in the bellies of the triceps muscle or just superior to the olecranon process in the triceps common tendon. Dr. Myers notes that the supinator tenderpoint correlates to Jones' "radial head" tenderpoint[32(pp141)] located on the anterior lateral radial head. Likewise, the brachialis tenderpoint correlates to Jones' "flexion elbow" tenderpoint[32(pp143)] and is located on the brachialis' tendonous insertion. The pronator tenderpoint extends in a band from the medial epicondyle to the cubital fossa.[32(pp133)]



Appendix C: Wrist/Hand S/CS Tenderpoints

When evaluating the wrist tenderpoints, they occur either on the dorsal or flexor surfaces. Flexural tender points correlate with flexed somatic dysfunction and are treated with flexion and slight rotation.[20(pp135)] Likewise, dorsal tender points are found on the extensor surface of the wrist and are treated with extension and slight rotation.[20(pp136)]

With regard to the hand as a whole, we have omitted a thoroughly labeled diagram here due to the numerous tender points (including those of the carpal bones on both surfaces and the flexor and extensor tendons) that create diagrammatic clutter. Of note, there are tenderpoints that both Jones and Myer identify in the belly of the adductor pollicis[20(pp135),32(pp147)] that are treated by adducting the muscle. The opponens pollicis, which Jones named "flexion wrist and thumb,"[20(pp137)] is on the volar surface at the carpo-metacarpal joint at the base of the first metacarpal. The treatment is done with wrist flexion and slight thumb abduction using the thumb as a lever to induce further wrist flexion.



References

1. Netter, Frank H. *Atlas of Human Anatomy*. Philadelphia, PA: Saunders/Elsevier, 2011.
2. Agur, Anne M.R., and Ming J. Lee. *Grants Atlas of Anatomy*. Philadelphia: Lippincott Williams, 1999.
3. Roos, David B. “Congenital Anomalies Associated with Thoracic Outlet Syndrome.” *The American Journal of Surgery*132, no. 6 (1976): 771–78. https://doi.org/10.1016/0002-9610(76)90456-6.
4. Adson, Alfred W., and Jay R. Coffey. “Cervical Rib.” *Annals of Surgery*85, no. 6 (1927): 839–57. https://doi.org/10.1097/00000658-192785060-00005.
5. Vitkari-Juntura, E. "Interrater Reliability of Observations in Physical Examinations of the Neck." *Springer Reference* 67 (1987): 1526-532. doi:10.1007/springerreference_61548
6. Hawkins, R. J., J. P. Schutte, G. H. Huckell, and J. Abrams. "The assessment of glenohumeral translation using manual and fluoroscopic techniques." *Orthop Trans* 12 (1988): 727.
7. Macdonald, Peter B., Peter Clark, and Kelly Sutherland. “An Analysis of the Diagnostic Accuracy of the Hawkins and Neer Subacromial Impingement Signs.” *Journal of Shoulder and Elbow Surgery*9, no. 4 (2000): 299–301. https://doi.org/10.1067/mse.2000.106918.
8. Bryant, Lawrence, Ron Shnier, Carl Bryant, and George A.C. Murrell. “A Comparison of Clinical Estimation, Ultrasonography, Magnetic Resonance Imaging, and Arthroscopy in Determining the Size of Rotator Cuff Tears.” *Journal of Shoulder and Elbow Surgery*11, no. 3 (2002): 219–24. https://doi.org/10.1067/mse.2002.121923.
9. Calis, M. “Diagnostic Values of Clinical Diagnostic Tests in Subacromial Impingement Syndrome.” *Annals of the Rheumatic Diseases*59, no. 1 (January 2000): 44–47. https://doi.org/10.1136/ard.59.1.44.
10. Bennett, WF. “Specificity of the Speeds Test: Arthroscopic Technique for Evaluating the Biceps Tendon at the Level of the Bicipital Groove.” *Arthroscopy: The Journal of Arthroscopic & Related Surgery*14, no. 8 (1998): 789–96. https://doi.org/10.1016/s0749-8063(98)70012-x.
11. Liu, H., and M. Henry. "A Prospective Evaluation of a New Physical Examination in Predicting Glenoid Labral Tears." *American Journal of Sports Medicine24* (1996): 721-25. doi:10.1177/34.1.145-a.
12. Speer, K. P., J. A. Hannafin, D. W. Altchek, and R. F. Warren. "An Evaluation of the Shoulder Relocation Test." *The American Journal of Sports Medicine22*, no. 2 (1994): 177-83. doi:10.1177/036354659402200205.
13. O’Driscoll SW. A reliable and simple test for posterior instability of the shoulder. *J Bone Joint Surg [Br]* 1991;73(suppl. 1) 50
14. O’Brien, Stephen J., Michael J. Pagnani, Stephen Fealy, Scott R. McGlynn, and Joseph B. Wilson. "The active compression test: a new and effective test for diagnosing labral tears and acromioclavicular joint abnormality." *The American journal of sports medicine* 26, no. 5 (1998): 610-613.
15. Stetson, WB, and K. Templin. "The Crank Test, the O’Brien Test, and Routine Magnetic Resonance Imaging Scans in the Diagnosis of Labral Tears." *American Journal of Sports Medicine* 30 (2002): 806-09.

16. Chronopoulos, Efstathios, Tae Kyun Kim, Hyung Bin Park, Diane Ashenbrenner, and Edward G. McFarland. "Diagnostic value of physical tests for isolated chronic acromioclavicular lesions." *The American journal of sports medicine* 32, no. 3 (2004): 655-661.
17. Liu, Stephen H., Mark H. Henry, Steven Nuccion, Matthew S. Shapiro, and Fred Dorey. "Diagnosis of glenoid labral tears: a comparison between magnetic resonance imaging and clinical examinations." *The American journal of sports medicine* 24, no. 2 (1996): 149-154.
18. Stetson, WB, Templin, K. The Crank Test vs Obrien's Test in detecting SLAP Lesions of the shoulder. Poster exhibit. AAOS 67th Annual Meeting. 2000.
19. Stiles, EG Lecture notes from Osteopathic Principles and Practice, Kentucky College of Osteopathic Medicine, 1998-1999.
20. Jones, L, Kusnose, R, Goering, E. Jones Strain-Counterstrain 1995.
21. Ward, Richard. *Foundations for Osteopathic Medicine*. 2nd ed. USA: Lippincott Williams & Wilkins, 2002.
22. Lister, G. D., R. B. Belsole, and H. E. Kleinert. "The radial tunnel syndrome." *Journal of Hand Surgery* 4, no. 1 (1979): 52-59.
23. Jobe, F., H. Stark, and S. Lombardo. "Reconstruction of the ulnar collateral ligament in athletes." *The Journal of Bone & Joint Surgery* 68, no. 8 (1986): 1158-1163.
24. Sallis, Robert E., Bernard R. Bach JR, and Robert A. Creighton. "A More Useful Milking Maneuver." *The Physician and Sports medicine* 33, no. 7 (2005): 17-17.
25. O'Driscoll SW, Bell DF, Morrey BF. Posterolateral rotatory instability of the elbow. J Bone Joint Surg Am. 1991;73(3):440–446
26. Lee, Michael L., and Melvin P. Rosenwasser. "Chronic Elbow Instability." *Orthopedic Clinics of North America*30, no. 1 (1999): 81–89. https://doi.org/10.1016/s0030-5898(05)70062-6.
27. Finkelstein, SL. "Stenosing Tenosynovitis at the Radial Styloid Process." *The Journal of Bone & Joint Surgery*. No. 12 (1939): 509-540.
28. Gerr, F., and R. Letz. "The Sensitivity and Specificity of Tests for Carpal Tunnel Syndrome Vary with the Comparison Subjects." *Journal of Hand Surgery*23, no. 2 (1998): 151–55. https://doi.org/10.1016/s0266-7681(98)80163-0.
29. Gellman, H. R. Gelberman, A. Tan, and M. Botte. "Carpal tunnel syndrome. An evaluation of the provocative diagnostic tests." *The Journal of Bone & Joint Surgery* 68, no. 5 (1986): 735-737.
30. Marx, Robert G., Claire Bombardier, and James G. Wright. "What Do We Know about the Reliability and Validity of Physical Examination Tests Used to Examine the Upper Extremity?" *The Journal of Hand Surgery*24, no. 1 (1999): 185–93. https://doi.org/10.1053/jhsu.1999.jhsu24a0185.
31. Palmer, Andrew K. "Triangular fibrocartilage disorders: injury patterns and treatment." *Arthroscopy: The Journal of Arthroscopic & Related Surgery* 6, no. 2 (1990): 125-132.
32. Myers, Harmon L., et al. *Clinical Application of Counterstrain*. Tucson, AZ: Osteopathic Press, a Division of Tucson Osteopathic Medical Foundation, 2012.
33. Rask, Michael R. "The Omohyoideus Myofascial Pain Syndrome: Report of Four Patients." *Journal of Craniomandibular Practice*2, no. 3 (1984): 256–62. https://doi.org/10.1080/07345410.1984.11677872.

34. Saroja, G., Aseer P, A. and P M, V. AND (2014) and Diagnostic Accuracy of provocative Tests in Lateral Epicondylitis. International Journal of Physiotherapy and Research, 2 and (6) and, pp.815-823.
35. Karbach LE, Elfar J. Elbow Instability: Anatomy, Biomechanics, Diagnostic Maneuvers, and Testing. *J Hand Surg Am*. 2017;42(2):118–126. doi:10.1016/j.jhsa.2016.11.025
36. O'Driscoll SW, Lawton RL, Smith AM. The "moving valgus stress test" for medial collateral ligament tears of the elbow. Am J Sports Med. 2005;33(2):231–239.

Clinical Vignette

CC: Shoulder pain
HxCC: 52-year-old, right hand dominant, male mechanic presents with left anterior lateral shoulder pain for the past 9 months not related to any particular trauma or known mechanism of injury. The pain, which he describes as a "pinching pain," began gradually at a 5/10 in severity. It is worsened when reaching for a jug of milk from the refrigerator or performing overhead activities and interrupts his sleep at night. He denies any referred pain from the neck or thorax region, but has associated intermittent periscapular pain. He denies any weakness, urinary, bowel symptoms, but has occasional paresthesias in the left hand of ulnar nerve distribution. He denies any remote injury and hasn't participated in formalized physical therapy or other treatment, except for periodic ibuprofen that minimally alleviates his symptoms.

ROS:
Constitutional: no mental status changes, no travel or sick contacts
Eyes: no change in vision
Endocrine: no fever, night sweats, weight change
Cardiovascular: no chest pain or palpitations
Respiratory: no dyspnea, cough, or sputum
Gastrointestinal: no nausea, vomiting diarrhea; no change in BM, no blood in stool
Genitourinary: no change in urine, no dysuria, no blood in urine
Neurologic: no anesthesia, no loss of consciousness, no seizures
Musculoskeletal: no weakness
Psychiatric: no homicidal or suicidal ideations, no signs of depression
Allergy: no angioedema, no urticaria, no anaphylaxis, no symptoms of seasonal allergies

PMHx/PSHx:
-Vasectomy
-Colonoscopy

Allergies:
-none

MEDs:
-ibuprofen over the counter every 6 hours as needed

Immunizations:
-TDaP in 2017

Social:
-no smoking; no alcohol; no drugs; work = mechanic

FamHx:
-patient is adopted and family history is unknown

VITALS = 120/80, 190#, 72beats per minute, 6", 98.6F
GEN = awake, alert, oriented to time, place, situation, self, in no acute distress
HEENT = normal cephalic, a-traumatic, extraocular muscles intact, no otic/nasal discharge
NECK = supple, thyroid w/o masses
HEART = regular rate, no clicks/rubs/murmurs
LUNGS = clear to auscultation, without crackles, wheeze, or rhonchi
GI = soft, non-tender, no rebound tenderness, +bowel sounds x 4 quadrants, no hepato-splenomegaly
CV = no jugular venous distention, no carotid bruits, mucus membrane moist, no edema, 2/4 peripheral pulses, no lymphadenopathy
N/M = cranial nerves 2-12 intact, 5/5 global strength (except as below), 2/4 global reflexes, cerebellum intact, sensation symmetric, gait normal, Spurling's maneuver negative, Roos test positive, negative Tinel's at the cubital tunnel, and Guyon's canal
Manual medicine exam = pertinent findings include **obvious scapulothoracic dyskinesia, with 5/5 rotator cuff strength. He has tenderness to palpation on the supraspinatus tendon insertion, capsular insertion on the humerus, as well as along the posterior ribs 1-3, and associated hypertonic scalene. There is associated inhibition of the lower trapezius and serratus muscles on the left side. Additionally, he has positive subacromial impingement testing, without signs of AC joint pathology, labral pathology, biceps pathology or gross instability.**

Assessment/Plan:
→In the orthopedic paradigm, the patient would likely be offered a cortisone injection, sent to physical therapy, and barring an abnormal neurological examination, an MRI of the neck or shoulder would be deferred pending outcome of physical therapy.
→The manual medicine paradigm offers restored mechanics of the cervical spine, thoracic cage and upper extremity as it relates to the combined diagnosis of subacromial impingement and thoracic outlet syndrome vs cervical radiculopathy. Still, home exercises or formal physical therapy would likely be beneficial to correct muscle inhibition of scapular muscles.

Paul Johnson, DO

Review Questions

1. What orthopedic test is used in conjunction with a thorough history to diagnose subacromial impingement?
    a. Hawkins-Kennedy
    b. Obrien's test
    c. Kim's test
    d. Load and shift

2. What structure is most commonly involved in subacromial impingement?
    a. Glenoid labrum
    b. Biceps tendon
    c. Infraspinatus tendon
    d. Supraspinatus tendon

3. What are the two structures commonly implicated in scapular dyskinesis with regard to subacromial impingement?
    a. Serratus anterior and trapezius
    b. Infraspinatus and supraspinatus
    c. Biceps and coracobrachialis
    d. Latissimus and trapezius

4. What is the most accurate test in assessing glenoid labrum injury/pathology?
    a. Crank test
    b. Apprehension test
    c. Hawkin's–Kennedy test
    d. Obrien's test

5. What structure(s) does the Finkelstein's test assess?
    a. Extensor carpi radialis brevis and longus tendons
    b. Extensor carpi radialis and extensor digiti minimi
    c. Abductor pollicis longus and extensor pollicis brevis
    d. Extensor carpi ulnaris and extensor pollicis brevis

6. When performing the Watson's test for scapholunate instability, the findings on examination include?
    a. Volar displacement of the scaphoid bone with ulnar deviation
    b. Volar displacement of the scaphoid bone with radial deviation
    c. Dorsal displacement of the scaphoid bone with ulnar deviation
    d. Dorsal displacement of the scaphoid bone with radial deviation

7. What is the involved tendon in tennis elbow when the 3rd digit resisted extension test is positive?
    a. Palmaris longus
    b. Extensor carpi radialis brevis
    c. Flexor carpi radialis
    d. Extensor carpi radialis longus

8. Varus stress test of the elbow at 20^0 of flexion tests for laxity/instability of what structure?
    a. Radial collateral ligament
    b. Annular ligament
    c. Common extensor tendon
    d. Ulnar collateral ligament

9. The "dynamic milking maneuver" tests which structure?
    a. Ulnar collateral ligament
    b. Radial collateral ligament
    c. Annular ligament
    d. Common extensor tendon

10. Which test is positive in patients with suspected biceps tendonitis?
    a. Hawkins-Kennedy test
    b. Kim's test
    c. Yergason's test
    d. Load and shift test

11. The "belly press" test relates to which rotator cuff muscle?
    a. Supraspinatus
    b. Infraspinatus
    c. Teres Minor
    d. Subscapularis

12. The Cross arm adduction test indicates suspected?
    a. Subacromial impingement
    b. Acromioclavicular joint pathology
    c. Labral pathology
    d. Glenohumeral instability

13. Anterior apprehension is performed by?
    a. Internal rotation of the humerus at 90 degrees of shoulder abduction with patient in supine position.
    b. External rotation of the humerus at 90 degrees of shoulder abduction with patient in the supine position.
    c. Internal rotation of the humerus at 30 degrees of shoulder abduction with the patient in the supine position.
    d. External rotation of the humerus at 30 degrees of shoulder abduction with the patient in the supine position.

14. Roos test is performed for suspected
    a. Carpal tunnel syndrome
    b. Thoracic outlet syndrome
    c. Radial nerve entrapment
    d. Posterior interosseous nerve entrapment

15. Allen's test, evaluates
    a. Ulnar nerve entrapment at the elbow
    b. Ulnar artery patency
    c. Thoracic outlet syndrome
    d. Carpal tunnel syndrome

16. Phalen's test, assesses for suspected
    a. Ulnar nerve entrapment at the elbow
    b. Thoracic outlet syndrome
    c. Median nerve entrapment
    d. Ulnar nerve entrapment at the wrist

17. What two soft tissue structures are typically involved in thoracic outlet syndrome?
    a. Posterior and medial scalenes
    b. Anterior and medial scalenes
    c. Anterior and posterior scalene
    d. Anterior scalene and levator scapulae

18. What does the Jobe's "relocation" test indicate?
    a. Posterior instability/dislocation
    b. Anterior instability/dislocation
    c. Sub-acromial impingement
    d. Labral pathology

Answers:

1. A
2. D
3. A
4. A
5. C
6. D
7. B
8. A
9. A
10. C
11. D
12. B
13. B
14. B
15. C
16. C
17. B
18. B

Chapter 8: The Lower Extremity

Principles Presented in this Chapter:

- Orthopedic Model of Lower Extremity
- Evidenced Based Evaluation of the Lower Extremity using the Orthopedic Model
- Origin and Insertion of Lower Extremity Musculature and its use in Diagnosis and Treatment

Outline:

- Anatomy of the Hip
- Orthopedic Evaluation of the Hip
- Evaluation of Hip Somatic Dysfunction
- Treatment of Hip Somatic Dysfunction

- Anatomy of the Knee
- Orthopedic Evaluation of the Knee
- Evaluation of Knee Somatic Dysfunction
- Treatment of Knee Somatic Dysfunction

- Anatomy of the Foot & Ankle
- Orthopedic Evaluation of the Foot & Ankle
- Evaluation of Foot & Ankle Somatic Dysfunction
- Treatment of Foot & Ankle Somatic Dysfunction

- Lower Extremity Treatment Pearls
- Appendix A: Hip Strain-counterstrain Tenderpoints
- Appendix B: Knee Strain-counterstrain Tenderpoints
- Appendix C: Foot & Ankle Strain-counterstrain Tenderpoints
- Clinical Vignette
- Review questions

Chapter 8: The Lower Extremity

Anatomy of the Hip[1,2]

The pelvis is the foundation of the spine and one of the most confounding structures encountered by beginning students. In this section, we revisit the anatomical structures of the pelvis in the context of the orthopedic model. Also, we incorporate the origin and insertion of muscles that aid in manual medicine diagnosis and treatment.

Considered by many in the medical field to be a "fused" structure, clinical experience supports a dynamic pelvis with slight movement during gait; a pelvis with potential for adaption to structural changes, and hence a pelvis susceptible to pathological somatic dysfunction. Anatomically, the bony pelvis is made of the ilium and the ischium that are fused with the pubis to form the innominate. The right and left innominate bones articulate anteriorly at the pubic symphysis, and posteriorly the sacrum closes the "pelvic ring." The hip joint is a "ball and socket" joint formed by the femur and acetabulum, respectively. In contrast to the "golf ball on a tee" analogy used to describe the glenohumeral joint, the femoral head sits deeper within the acetabulum, providing increased stability for support of the upper body, and hence, has inherently less motion. The acetabulum also has a c-shaped articular surface with a fibro-cartilage rim called the acetabular labrum that adds joint depth.

As discussed in the sacrum and pelvis chapter, the SI joint and pubic symphysis play a significant role in movement, and stability of the lumbo-pelvic region as a "floating compression system" in the tensegrity model and in evaluation of somatic dysfunction.

The origin and insertion of pelvic muscles are shown in Figure 1; their role in motion is described in Table 1.

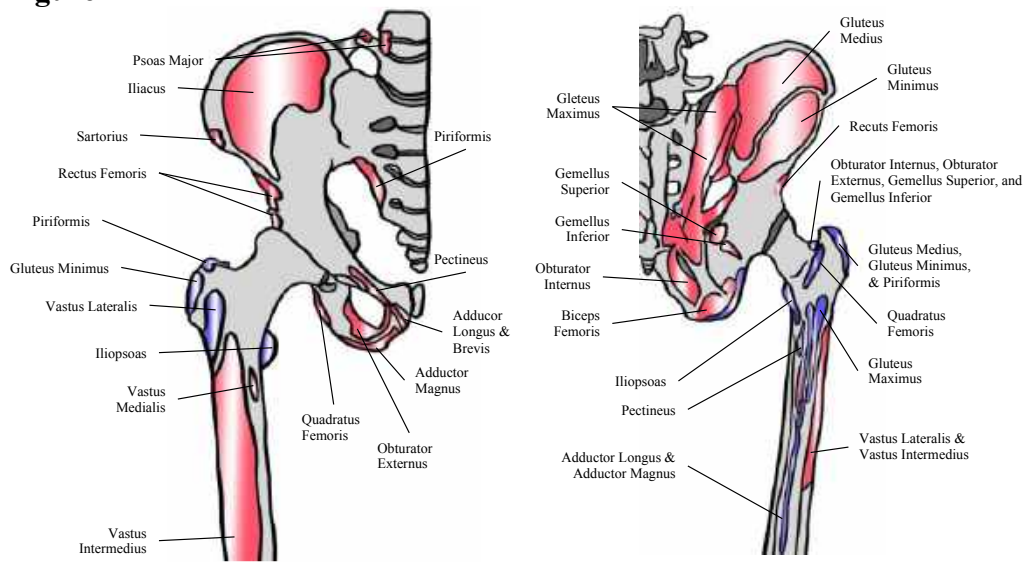
The Hip Joint

The hip capsule is a sleeve of ligaments that attaches along the acetabular labrum, with femoral-sided attachments along the inter-trochanteric line anteriorly, and part way along the femoral neck posteriorly. The underside portion of the ligamentous capsule is the weakest; in contrast, the strongest, and thickest is the anterior portion of the iliofemoral ligament. The posterior ligament is called the ischiofemoral ligament.

Figure 1: The Bony Pelvis with Origin and Insertions of Muscles

Muscle origins are colored red, insertions are colored blue.

Figure 1



Motions & Muscles of the Hip

Table 1: Major Muscle Groups of the Hip

TABLE 1	
Muscle	Action
Piriformis, Obturator Externus, Obturator Internus, Superior/Inferior Gemellus, Quadratus Femoris	Short External Rotators of the Thigh. Note that the Piriformis attaches anteriorly on the sacrum, while the other muscles attach posteriorly.
Adductor Magnus, Adductor Brevis, Adductor Longus (main), Pectineus, and Gracilis	Adductors of the Thigh
Gluteus Minimus/Medius, Tensor Fascia Lata	Abductors of the Thigh. Gluteus medius - the anterior portion is involved in medial rotation, but its main function is to abduct the hip.
Iliacus/Psoas	Flexor of the Thigh. Joins with the psoas muscle to insert on the medial thigh.
Rectus femoris	Flexor of the Thigh. Also blends with other quadriceps muscles to become the quadriceps tendon and extensor mechanism of the knee.
Sartorius	Flexor of the Thigh. Originates from the ASIS and inserts on the medial tibia to form the Pes Anserinus.
Hamstrings: Semimembranosus, Semitendinosus, and Biceps Femoris	Extensors of the Hip. Also functions to flex the knee
Gluteus maximus	Extensor of the Hip. Crosses the sacroiliac (SI) joint and stabilizes the bony pelvis.

Orthopedic Evaluation of the Hip

Evaluation of the hip should involve a systematic approach with the following sequence of evaluation

1. Inspection
2. Range of motion
3. Palpation
4. Motor strength testing
5. Neurovascular testing
6. Orthopedic Special Tests
7. Evaluation for Somatic Dysfunction

Remember the edict "do no harm" in your initial evaluation and take a thorough history of the patient's presenting complaint. Rule out so called red flags and leave your mind open to the possibility of pathology other than musculoskeletal injury. Do not lose sight of the possibility that on initial presentation, medical illness such as, appendicitis, intra-abdominal abscess, bowel perforation, nephrolithiasis, visceral hernias, and testicular and ovarian torsions may appear as musculoskeletal. Consider the mechanism of injury.

The identification of biomechanical factors in the trunk and axial skeleton that predispose the patient to pelvic pain requires clinical evaluation of the thoracic and lumbar spine, leg length inequality, and pathology of lower extremity.

Red Flags:[26]

- LBP with Presentation <20 years
- LBP with onset of symptoms >55 years
- Trauma
- History of cancer
- History of osteoporosis or its risk factors
- B-type symptoms including fevers, night sweats, weight change, pruritis
- Non-mechanical or radicular pain
- Cauda Equina Syndrome
- Filum Terminal Syndrome
- Pain out of proportion to exam

Inspection

As with other aspects of the physical exam, inspect for antalgic gait, asymmetries, areas of swelling, muscle contractures, and ecchymosis. Also, observe the patient standing and at rest. The bony landmarks of the anterior hip and pelvis are easily identified in most patients. At the anterior pelvis, the patient can be observed supine on the examination table; depending on their body habitus, the most prominent features include the anterior superior iliac spine (ASIS), the inguinal region between the ASIS and pubic tubercle, the area of the inguinal ligament, and the proximal thigh (quadriceps and adductors). On the lateral side, the greater trochanter, iliac crest, gluteal musculature and iliotibial tract can be observed and assessed. While prone, observe the posterior iliac crest, posterior superior iliac spine (PSIS), lumbopelvic region, gluteal region, and the patient's hamstrings.

Range of Motion

There are three different axes of motion of the hip: flexion/extension, abduction/adduction, and external rotation/internal rotation. These three axes relate to the 5 muscle groups involved in hip motion: short rotators, flexors, extensors, adductors, and abductors. Figure 2 displays passive range of motion (ROM) testing for the hip.

With the patient in the supine position, hip flexion, abduction, and adduction are easily assessed with passive range of motion (ROM) testing. Normal abduction

is approximately 45° with the hip and knee extended, 70-75° with the hip and knee flexed, and adduction is 20-30° with either position. Passive hip flexion is approximately 120-135° with the knee flexed and usually less than 90° when the knee is extended. Hip extension is best evaluated on a prone patient; normal range of extension is approximately 30°. With a flexed hip and knee, external rotation is 45° and internal rotation is 35°.

Figure 2

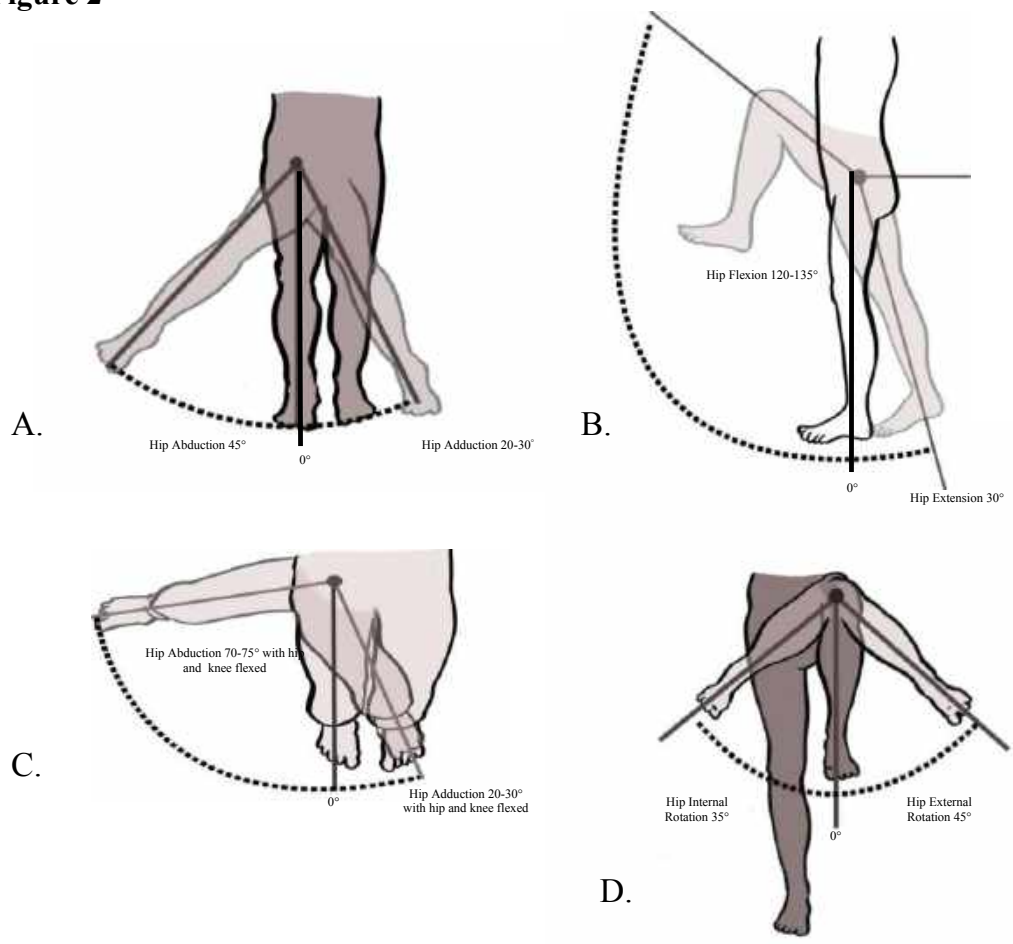


Figure 2: Passive Hip ROM Testing

Note how the normal ROM for the hip changes depending on patient position.

- In figure A-D the patient should be laying supine during testing, with the exception of hip extension, as in figure B, where the patient would ideally be prone.
- In figure C, note that hip abduction increases in ROM to 70-75° with flexion of the hip and knee.

Palpation

When leg length inequality is present, the pelvic crest height is unlevel when standing and level when sitting.

To evaluate pelvic obliquity or leg length inequality, with the patient standing, the examiner places their hands on the iliac crests. Simultaneously, the PSIS, the gluteal region, and the insertion of the short hip restrictors on the posterior greater trochanter are assessed for tenderness. With the patient supine, bony landmarks of the anterior pelvis such as the ASIS, anterior inferior iliac spine (AIIS), pubic symphysis, inguinal ligament, as well as the adductor and quadriceps muscles are appreciated. In the inguinal region, note the pulse of the femoral artery, and on deep palpation of ala (aka, wing of the ilium), check for tenderness of the iliopsoas muscle.

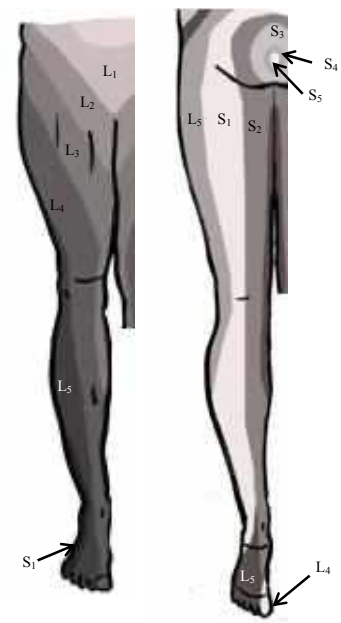
Motor Strength Testing

Strength testing is performed for each of the following groups: flexors, extensors, adductors, abductors, internal, and external rotators of the hip.

Neurovascular Testing

In the presence of pathology, neuromuscular testing will ideally correlate with muscle strength, reflexes, and dermatomal sensation with the affected nerve root. Test for sensation to soft touch and pinprick along the dermatomes seen in Figure 3. Consider the use of Tinel's test to elicit numbness/tingling for the peroneal nerve as it passes below the head of the fibula; this may help differentiate between peroneal nerve compression and L4 nerve root compression. Check the patellar and Achilles tendon reflexes.

Figure 3



Palpate the femoral pulse in the inguinal region, the popliteal artery behind the knee and the dorsalis pedis on the dorsum of the foot; in the absence of peripheral vascular disease or arterial pathology, all of these should be bilaterally symmetrical.

Orthopedic Special Tests

The Thomas test[3] assesses for restriction or hypertonicity of the iliopsoas muscle. Place the patient supine, legs hanging off the table (Figure 4). The patient then grabs the knee of one lower extremity and reclines to the supine position. The other thigh should remain flat on the table; with iliopsoas hypertonicity, the thigh will not lay flat on the table. The test is repeated for the contralateral side.

Strength testing:
5/5 = normal
4/5 = movement possible against some resistance by the examiner
3/5 = movement possible against gravity, but not against resistance by the examiner
2/5 = movement present, but not against gravity
1/5 = muscle contraction, but does not produce movement
0/5 = no contraction

Figure 3: Dermatomes of the Lower Extremities

Patellar Reflex = L4 nerve root
Achilles Reflex = S1 nerve root

Figure 4: Thomas Test

Depicted is a normal Thomas test.

Figure 4

Scour maneuver (aka quadrant test):[4] This test evaluates femoral acetabular impingement or labral pathology. The patient lays supine on the table, the hip and knee flexed to 90° and adducted. A compressive force is applied along the length of the femur toward the acetabulum, and the examiner moves the femur through a circular arc of motion. Reproduction of pain or "catching" indicates anterior hip subluxation, or labral pathology.

Figure 5: Scour Maneuver

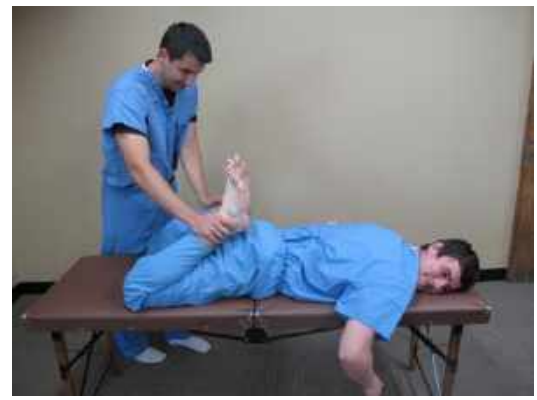
Note the leg and hip are first flexed to 90°. A compressive force is added along the femur into the acetabulum. A circular motion is finally introduced at the hip joint.

Figure 5

Ely's test[5] assesses rectus femoris spasticity. The patient is placed prone on the table with heels passively flexed toward the buttocks. The test is considered positive if the heels cannot touch the buttocks.

Figure 6: Ely's Test

Figure 6



The Trendelenburg test[6] evaluates weakness of the gluteus medius on the weight-bearing side. Similar to the stork test, the patient is standing on one leg with the examiner observing from behind. Ideally, the pelvis should remain level. The test is positive when the hip of the non-weightbearing leg drops, as seen in Figure 7.

Figure 7

Figure 7: Trendelenburg Test

In this instance, there is pathology of the right gluteus medius.

Ober's test[7] examines the contracture or tightness of the iliotibial band. The patient is side-lying on the treatment table with the affected leg flexed (Figure 8). The pelvis is stabilized by the examiner, and the lower extremity is abducted by the operator and then slowly adducted. If there is lateral knee pain or the lower extremity remains abducted, it indicates a contracture of the iliotibial band.

Figure 8

Figure 8: Ober's Test

Table 2: Statistical Analysis of Orthopedic Special Tests of the Hip

Table 2				
Test	Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value
Thomas Test	Not reported			
Scour Maneuver	Not reported			
Ely's Test	Not reported			
Trendelenburg Test[6]	72. 7	76. 9		
Ober's Test	Not reported			

Evaluation of Hip Somatic Dysfunction

Treatment of hip restrictors occurs along the ITA, except for the Piriformis that occurs along the MTA.

Osteopathic musculoskeletal evaluation of the hip begins with understanding the origin and insertion of the lower extremity musculature. This knowledge allows a practitioner to establish the structures involved, and by using muscle energy (ME) and counter-strain (S/CS), quickly determine a position of treatment. Do not lose sight of the interplay among structures above and below the hip, and the fact that somatic dysfunction of the pelvis will affect hip mechanics. Specifically, immobility of the SI joint, as in a unilateral sacral extension, will cause greater rotational motion of the ipsilateral hip to maintain gait length. This places undue stress on the hip capsule and the supporting structures. In the short term, this may lead to tendonitis or bursitis as is seen with trochanteric bursitis. Long term, this may lead to early osteoarthritis in the hip.

In the hip, there are six restrictors: flexors, extensors, abductors, adductors, internal rotators, and external rotators. In principle, all hip restrictors are treated by utilizing the inferior transverse axis (ITA) of the pelvis, with the exception of the piriformis muscle that uses the middle transverse axis (MTA).

Hamstrings

Hamstrings usually spasm and produce a restriction in hip flexion (i.e., extended hip). Hamstring evaluation is performed with the patient laying supine on the exam table. When monitoring the ipsilateral ASIS, the hip is flexed with the knee extended. When the ipsilateral innominate begins upward rotation, the hamstrings are on stretch. Compare to the opposite side for the length and end feel. See the treatment box below for treatment details.

Treatment Examples Extended Hip:[8,9,10]

Extended Hip - Tight Hamstrings with ME

1. The patient remains supine after diagnosis as described above.
2. Use the ASIS compression test to localize motion to the ITA by abducting and adducting the lower extremity.
3. Immobilize the right hemi-pelvis to create a fulcrum; in this case, compress the ASIS posteriorly so the pelvis remains immobile during treatment (this is essentially the position of diagnosis, and treatment is performed moments after the diagnosis is made).
4. Create a lever by placing the heel of the affected side on the operator's shoulder; in this instance the whole lower extremity is the lever.
5. Moving to the previously detected barrier of hip flexion reverses the components of the positional diagnosis.
6. Once at the closed pack position, slightly release tension to the "feather edge" of the barrier.

7. Create an isometric contraction by instructing the patient to extend their lower extremity while the operator maintains an unyielding counter force for 3-5 seconds. Have the patient relax. Reposition at the new barrier by taking up the slack in further flexion. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times. By the end of treatment, most patients will passively flex to ~90°.
8. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Extended Hip - Tight Hamstrings with S/CS utilizing the Biceps Femoris Tender point

1. The patient is prone on the treatment table and identifies the biceps femoris tender point.
2. Motion is localized to the ITA, which is 1 fingerbreadth inferior to the PSIS, by abducting and adducting the lower extremity.
3. The clinician maintains contact with the tender point and with the other hand flexes the knee to ~90°. The hip is extended, adding internal rotation with slight abduction to a position that alleviates at least 70% of the patient's tenderness.

4. Maintain this position for 90 seconds.
5. SLOWLY return the patient to the neutral position.
6. Recheck the original diagnosis to ensure resolution of tender point.

Hip Adductors

Adductors are evaluated with the patient supine, knee extended. The clinician tests hip adductors by bringing one leg at a time into abduction while supporting the ankle. Note that the ipsilateral ASIS is stabilized to prevent movement from the pelvis or spine. Tight adductors (i.e., adducted hip) are noted when a comparison is made between the opposite sides in terms of length and end feel.

Treatment Examples of Adducted Hip:

Adducted Hip - Tight Adductors with ME

1. The patient remains supine after diagnosis as described above.
2. Use the ASIS compression test to localize motion to the ITA by abducting and adducting the lower extremity.
3. Immobilize the left hemi-pelvis to create a fulcrum; in this case, compress the ASIS posteriorly so the pelvis remains immobile during treatment (this is essentially the position of diagnosis, and treatment is performed moments after the diagnosis is made).

4. Create a lever with the lower extremity and provide support by holding the ankle.
5. Moving to the previously detected barrier of hip abduction reverses the components of the positional diagnosis.
6. Once at the closed pack position, slightly release tension to the "feather edge" of the barrier.
7. Create an isometric contraction by instructing the patient to adduct their lower extremity while the operator maintains an unyielding counter force for 3-5 seconds. Have the patient relax. Reposition at the new barrier by taking up the slack in further abduction. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
8. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

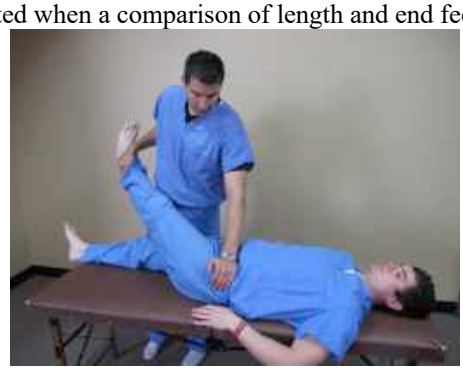
Adduced Hip - Tight Adductors with S/CS utilizing the Adductor Longus or Brevis Tender point

1. Patient is supine on the treatment table. The tender point is commonly located along the inguinal ligament or on the origin of the adductors on the pubic rami.
2. Use the ASIS compression test with abduction and adduction to localize the ITA.
3. The clinician maintains contact with the tender point and with the other hand adducts the hip, adding internal rotation to reach a position that alleviates at least 70% of the patient's tenderness.

4. Maintain this position for 90 seconds.
5. SLOWLY return the patient to the neutral position.
6. Recheck the original diagnosis to ensure resolution of tender point.

Hip Abductors

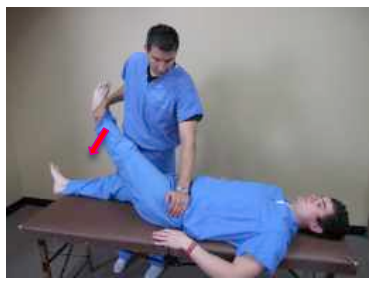
To test the hip abductors, the patient remains in the supine position and the clinician grasps the patient at the ankle with an extended knee; the lower extremity is moved into adduction. Comparison is made using both sides. Tight abductors (i.e., abducted hip) are noted when a comparison of length and end feel is made between opposite sides.



Treatment Examples of Abducted Hip:

Abducted Hip - Tight Abductors with ME

1. The patient remains supine after diagnosis as described above.
2. Use the ASIS compression test to localize motion to the ITA by abducting and adducting the lower extremity.
3. Immobilize the left hemi-pelvis to create a fulcrum; in this case, compress the ASIS posteriorly so the pelvis remains immobile during treatment (this is essentially the position of diagnosis, and treatment is performed moments after the diagnosis is made).
4. Create a lever with the lower extremity and provide support by grasping the ankle.
5. Reverse the components of the positional diagnosis by moving to the previously detected barrier of hip adduction.
6. Once at the closed pack position, slightly release tension to the "feather edge" of the barrier.



7. Create an isometric contraction by instructing the patient to abduct their lower extremity while the operator maintains an unyielding counter force for 3-5 seconds. Have the patient relax. Reposition at the new barrier by taking up the slack in further adduction. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
8. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

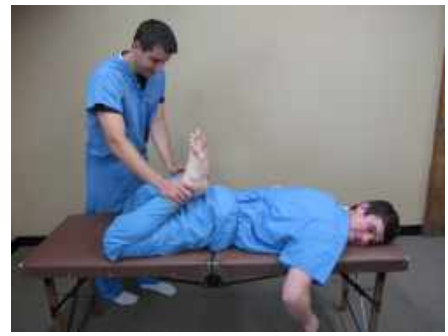
Abducted Hip - Tight Abductors with S/CS utilizing the Gluteus Medius Tender point

1. Patient is prone on the treatment table and gluteus medius tender point is identified behind the tensor fasciae latae approximately 2 inches inferior to the iliac crest.
2. Localize movement to the ITA that is 1 fingerbreadth below the PSIS.
3. The clinician maintains contact with the tender point and with the other hand extends the hip, adding internal rotation with slight abduction to a position that alleviates at least 70% of the patient's tenderness.

4. Maintain this position for 90 seconds.
5. SLOWLY return the patient to the neutral position.
6. Recheck the original diagnosis to ensure resolution of tender point.

Hip Flexors

To evaluate tight hip flexors, the patient is prone and the operator passively brings the heels of both feet to the buttock (Ely's Test), evaluating for asymmetry or hypertonicity of the quadriceps including the rectus femoris. Comparison is made using both sides. Tight flexors (i.e., flexed hip) are noted when a comparison of length and end feel is made between opposite sides.



Treatment Examples of Flexed Hip:

Flexed Hip - Tight Flexors with ME

1. The patient remains prone after diagnosis as described above.
2. Use the ASIS compression test to localize motion to the ITA by abducting and adducting the lower extremity.
3. Immobilize the left hemi-pelvis to create a fulcrum; in this case, compress the ASIS anteriorly so the pelvis remains immobile during treatment (this is essentially the position of diagnosis, and treatment is performed moments after the diagnosis is made).
4. Create a lever with the lower extremity while supporting the knee as depicted.
5. Reverse the components of the positional diagnosis by moving to the previously detected barrier.
6. Once at the closed pack position, slightly release tension to the "feather edge" of the barrier.

7. Create an isometric contraction by instructing the patient to flex their lower extremity while the operator maintains an unyielding counter force for 3-5 seconds. Have the patient relax. Reposition at the new barrier by taking up the slack in further extension. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
8. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Flexed Hip - Tight Flexors with S/CS utilizing the Rectus Femoris Tender point

1. The patient is supine on the treatment table. The rectus femoris tender point is inferior to the AIIS overlying the femoral neck/head.
2. Use the ASIS compression test with abduction and adduction to localize the ITA.
3. The clinician maintains contact with the tender point and with the other hand flexes the hip, adding internal rotation, to a position that alleviates at least 70% of the patient's tenderness

4. Maintain this position for 90 seconds.
5. SLOWLY return the patient to the neutral position.
6. Recheck the original diagnosis to ensure resolution of tender point

Internal Rotators

To test internal rotators (i.e., externally rotated hip), the patient is again placed in the prone position. While using the lever inherently created by the leg and thigh, test each hip by flexing the knee and externally rotating the thigh. Assess for end feel and asymmetry of motion to determine if the internal rotators are shortened on one side compared to the other.

Treatment Examples of Externally Rotated Hip:

Externally Rotated Hip - Tight Internal Rotators with ME

1. The patient remains prone after diagnosis as described above.
2. Localize movement to the ITA via abduction and adduction that is 1 fingerbreadth below the PSIS.
3. Immobilize the left hemi-pelvis to create a fulcrum; in this case, compress the ASIS anteriorly so the pelvis remains immobile during treatment (this is essentially the position of diagnosis, and treatment is performed moments after the diagnosis is made).
4. Create a lever with the leg flexed and thigh in a neutral comfortable position.
5. Reverse the components of the positional diagnosis by moving to the previously detected barrier.
6. Once at the closed pack position, slightly release tension to the "feather edge" of the barrier.

7. Create an isometric contraction by instructing the patient to internally rotate their lower extremity while the operator maintains an unyielding counter force for 3-5 seconds. Have the patient relax. Reposition at the new barrier by taking up the slack in further external rotation. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
8. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Externally Rotated Hip - Tight Internal Rotators with S/CS utilizing the Glut. Minimus Tender point

1. The gluteus minimus tender point is commonly located about 2. 5 inches inferior to the ASIS.
2. Use the ASIS compression test with abduction and adduction to localize the ITA.
3. The operator monitors the tender point with one hand and with the other hand flexes the hip to approximated 90°, adding slight abduction and internal rotation of the femur until tenderness is reduced by 70%.

4. Maintain this position for 90 seconds.
5. SLOWLY return the patient to the neutral position.
6. Recheck the original diagnosis to ensure resolution of tender point.

External Hip Rotators

Tight external rotators (i.e., internally rotated hip) are diagnosed with the patient prone, knees held tightly together while flexed to approximately 90°. Both hips are internally rotated as the clinician passively moves the ankles away from the midline. Note in the figure below that the patient's right hip exhibits somatic dysfunction. During this procedure, the clinician assesses for asymmetry that indicates restriction of the external rotators.

Treatment Examples of Internally Rotated Hip:

Internally Rotated Hip - Tight External Rotators (Piriformis) with ME

1. The patient remains prone after diagnosis as described above. Localize movement to the **MTA** via abduction and adduction that is medial to the PSIS.
2. Place a fulcrum on the ipsilateral hemi pelvis so the pelvis remains immobile during treatment.
3. Create a lever with the leg flexed and thigh in a neutral comfortable position.
4. Reverse the components of the positional diagnosis by moving to the previously detected barrier.
5. Once at the closed pack position, slightly release tension to the "feather edge" of the barrier.

6. Create an isometric contraction by instructing the patient to externally rotate their lower extremity while the operator maintains an unyielding counter force for 3-5 seconds. Have the patient relax. Reposition at the new barrier by taking up the slack in further internal rotation. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
7. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Internally Rotated Hip - Tight External Rotators with S/CS utilizing the Piriformis Tender point

1. To locate the piriformis tender point, palpate at the upper posterior edge of the greater trochanter along the tendon. Moving the palpating hand medially and slightly inferiorly (see Appendix A in the sacrum/pelvis chapter for tender point location).
2. Localize movement to the **MTA** via abduction and adduction that is medial to the PSIS.
3. The patient's leg is brought off the table with the knee flexed approximately 135° and the hip is externally rotated until the tender point is relieved by 70%.

4. Maintain this position for 90 seconds.
5. SLOWLY return the patient to the neutral position.
6. Recheck the original diagnosis to ensure resolution of tender point.

Anatomy of the Knee[1,2]

The knee is the largest joint in the human body; it is responsible for locomotion and sustaining heavy loads and is therefore susceptible to injury (Figure 9).
The distal femur has two smoothly curved surfaces known as the medial and lateral femoral condyles. The deep notch that separates them is called the intercondylar notch. On the sides of the medial and lateral femoral condyles are the medial and lateral epicondyles. The medial epicondyle has a sharp corner called the adductor tubercle, and owing to the attachment of the medial patellofemoral ligament, has importance related to patellofemoral joint stability.

Figure 9

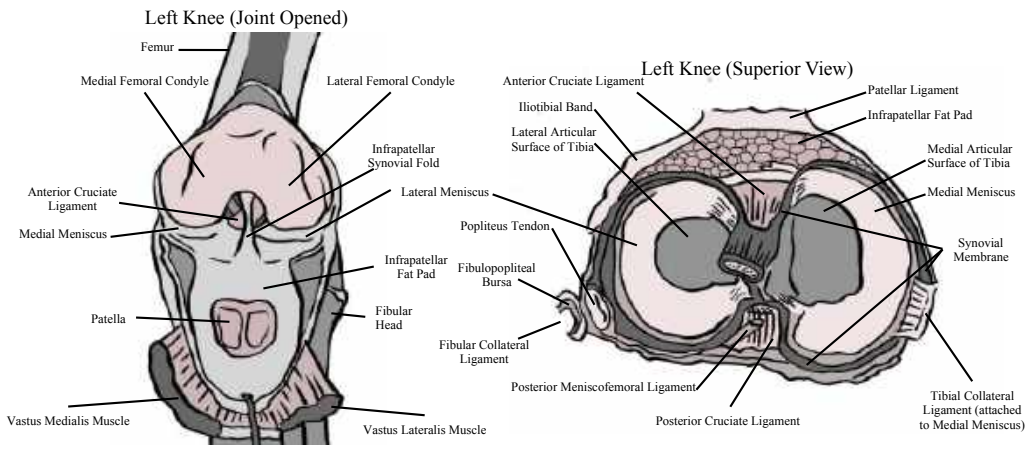


Figure 9: Anatomy of the Knee

The tibia and the fibula comprise the other bones of the joint; they join at the proximal and distal tibiofibular joints. Somatic dysfunction commonly occurs at the proximal tibiofibular joint affecting the fibular head and is frequently due to ankle inversion injuries.

The proximal tibia has medial and lateral articular surfaces; in between these lie the intraarticular area. Anteriorly, the patellar tendon attaches to the tibial tuberosity. Laterally, there is a facet for the proximal tibiofibular joint. On top of the tibial articular surfaces sit the crescent shaped fibrocartilage and the medial and lateral meniscus.

The roll and slide motion of the femur on the tibia during flexion and extension is stabilized by the medial and lateral menisci. The medial meniscus is c-shaped, whereas the lateral meniscus is circular. Both menisci have a thick outer edge that tapers and becomes thinner on the inner edge. The lateral meniscus is more mobile than the medial meniscus because its ends have closer attachments at the intraarticular area. Additionally, the capsular attachments on the lateral meniscus are looser and more mobile than those on the medial meniscus, making it more susceptible to injury. Both menisci attach to the capsule around the edge and are stabilized to the tibia by the coronary ligaments.

The remaining bone of the knee, the patella (Latin for dish or plate), is the largest sesamoid bone in the body. The patella is enmeshed within the quadriceps tendon that becomes the patellar tendon upon insertion to the tibial tuberosity; these structures are collectively referred to as the "extensor mechanism." On the posterior surface of the patella is the articular surface.

As the femur rolls and slides on the tibia, the cruciate ligaments provide stability for the knee joint. The anterior cruciate ligament (ACL) arises from the inner portion of the lateral femoral condyle and inserts on the anterior tibia, preventing the tibia from sliding out in front of the femur, as well as providing stability in rotation and hyperextension of the knee.

The posterior cruciate ligament (PCL) arises from the posterior tibia midline. It passes approximately 15° toward the insertion on the inner side of the medial femoral condyle and prevents the femur from moving anteriorly on the tibia. The collateral ligaments approximate the condyles to prevent varus and valgus displacement. The medial collateral ligament (MCL) arises from the medial epicondyle of the femur and inserts on the ant/med proximal tibial, blending with the meniscus at the deep layer of the MCL and the joint capsule.

In contrast to the MCL, the lateral collateral ligament (LCL) is extra-capsular and does not attach to the meniscus. It arises from the lateral femoral condyle proximal to the groove for the popliteus insertion. Both collateral ligaments become taught in knee extension and have more laxity in flexion. Muscles supporting and surrounding the knee are listed in Table 3 and depicted in Figure 10.

Figure 10

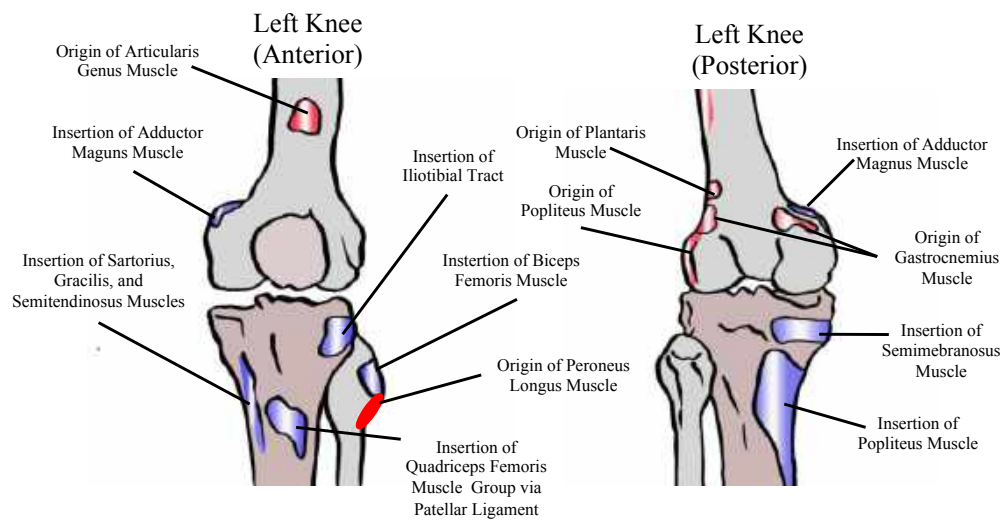


Figure 10: Muscular Origins and Insertions of the Knee

Motions & Muscles of the Knee

TABLE 3	
Muscle	Action
Quadriceps Femoris Group (Rectus Femoris, Vastus Medialis, Vastus Lateralis, Vastus Intermedius)	Extend the leg; during the gait cycle, maintain leg extension while the hamstrings extend the hip. In conjunction with gluteus maximus, the quadriceps contract when rising from a seated position and walking up and downhill.
Hamstrings (semimembranosus, semitendinosus, biceps femoris)	Flex the knee and extend the hip. The semimembranosus and semitendinosus insert on the medial tibia (semi-m post/med, semi-t ant/med) and biceps femoris inserts on the fibular head. Based on the location of these insertions, when the knee is flexed with the foot off the ground, semi-m/semi-t medially rotate the tibia, while biceps femoris laterally rotates the knee.
Sartorius and Gracilis	Minor flexors of the knee.
Popliteus	Medial rotator of the tibia, initiating the unlocking of the "screw home" mechanism of the knee.

Table 3: The Major Muscle Groups of the Knee

Orthopedic Evaluation of the Knee

Evaluation of the knee should involve a systematic approach with the following sequence of evaluation:

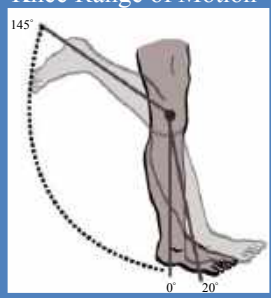
1. Inspection
2. Range of motion
3. Palpation
4. Motor strength testing
5. Neurovascular testing
6. Orthopedic Special Tests
7. Evaluation for Somatic Dysfunction

As an intermediary between the hip and ankle, it is important to examine proximally as well as distally when considering dysfunction in the knee. Also, with any patient history, ask about the mechanism of injury, onset, duration, alleviating/exacerbating factors, occupational/repetitive tasks, sport specific tasks, referred pain, other joint involvement, and mechanical symptoms. When considering the knee, presence of an effusion significantly narrows the differential diagnosis.

Causes of Knee Effusion:

- Traumatic - Meniscal Injury, MCL/ACL/PCL Injury, osteochondral fracture, capsular tear, patellar dislocation
- Infection
- Inflammation – Gout, Pseudogout, RA, SLE, etc.
- Osteoarthritis

Knee Range of Motion



Inspection

Especially when a patient presents after a traumatic injury, assess for antalgic gait, gross effusion, breaks in the skin, and ecchymosis. In patients who have longer standing pain, it is also important to assess for femoral anteversion as well as various biomechanical asymmetries relating to pes planus and any other pathology that can affect biomechanics of the knee.

Range of Motion

With the patient in the supine position, actively and passively assess flexion and extension. Normal flexion is approximately 145° and normal extension is 20°.

Palpation

There are various superficial soft tissue structures that are easily palpated by the bony landmarks about the knee. Starting from the suprapatellar region, palpate the quadriceps tendon and its various insertions on the medial and lateral side of the patella: the patellar tendon at the inferior pole of the patella, midsubstance, and at its insertion on the tibial tuberosity. The insertion of the pes anserine tendons (sartorius, gracilis, and semitendinosus) at the anterior medial tibia. The medial collateral ligament at the proximal medial femoral condyle just distal the adductor tubercle and medial proximal tibia. The iliotibial band at Gerdy's tubercle. The lateral collateral ligament (anterior) and biceps femoris(posterior) at fibular head. It is also important to feel motion of the patella and assess for presence of prepatellar bursal fluid. Posteriorly, feel for any sense of fullness such as a Baker's cyst, which will rise between the medial head of the gastrocnemius tendon and the semimembranosus tendon. Also, palpate for the popliteal pulse.

Motor strength testing

With the patient seated, legs unloaded, test quadriceps and hamstring strength against resistance. To test the gastrocnemius musculature, place the patient supine and actively plantar flex the foot against the resistance.

Orthopedic Special Tests

There are several tests that determine meniscal pathology. McMurray's test[11] (active axial compression, circumduction maneuver) is performed with the patient in a supine position and the hip flexed to 60° and the knee flexed to 90° (Figure 11). The examiner palpates the medial and lateral joint line with one hand and applies axial compression with the other hand. From the sole of the patient's foot, the examiner then applies the axial load with a circumduction maneuver to load each knee compartment. A palpable clunk is a positive test, and frequently patients experience pain with this maneuver.

Figure 11

Figure 11: McMurray's Test

The Childress test[12] also tests for meniscal pathology (Figure 12). During this test the patient is asked to perform a "duck walk." A test is positive when the patient reproduces their typical symptoms along the medial or lateral joint line.

Figure 12

Figure 12: Childress Test

Thessaly's test[13] evaluates meniscal pathology (Figure 13). The patient is standing on the affected knee with approximately 20° of joint flexion; the examiner introduces internal and external rotation of the femur on the tibia three times, in an attempt to grind the meniscus and reproduce joint pain (the examiner assists by holding the patient). The test is positive should the patient have reproduction of the symptoms on the medial or lateral side of the loaded extremity.

Figure 13

Figure 13: Thessaly's Test

The anterior drawer test[14] evaluates integrity of the ACL. The patient is supine on the table with the knee flexed at 90° and their foot on the table. The examiner stabilizes the leg by partially sitting on the dorsum of the foot and grasps the patient's lower leg at the tibial plateau. The examiner then provides an anterior translation to the tibia to test for laxity or increased movement. ACL injury is suspected if increased motion is noted when compared with the opposite side.

Figure 14

Figure 14: The Anterior Drawer Test

The Posterior drawer test[16] evaluates the PCL and is performed from the same position as the anterior test but with the translation of the tibia on the femur in a posterior direction. Again, the test is considered positive when there is laxity compared to the contralateral side.

Lachman's test[15,30] also evaluates for ACL injury; the patient is supine with the knee flexed to approximately 20-30°. The operator braces the thigh with one hand, and the other hand is placed on the posterior proximal tibia. The operator then applies anterior translation of the tibia on the femur to determine if there is a firm endpoint to the ACL. If laxity or absence of firm endpoint is noted (compared with the normal side), then ACL injury is suspected.

Figure 15

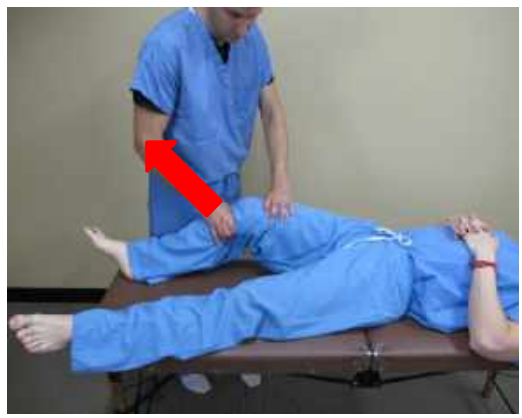


Figure 15: Lachman's Test

The reverse Lachman's test is performed by posterior translation of the knee.

The Reverse Lachman's test is performed from the same position but tests the integrity of the PCL. The tibia is translated posteriorly to test for laxity or firm endpoint of motion.

To test the medial and lateral collateral ligaments, varus and valgus loads are introduced to the knee at 0° and at 20-30°, thereby assessing the LCL and MCL respectively. Laxity noted with a varus or valgus load at 0° pathognomonically indicates multi-ligament injury. When using a varus and valgus stress test at 20-30° of flexion, increased laxity or the lack of a firm endpoint indicates injury to one of the collateral ligaments.

Figure 16

Figure 16: The Varus and Valgus Stress Test

Note that these tests are performed at 0o and 20-30o of flexion.

Patellar apprehension test[17] evaluates for patellar subluxation/dislocation. The test is performed with the patient is supine, the knee in full extension. The examiner grasps the patella and translates it laterally to medially. Should the patient feel reproduced symptoms, the test is positive.

Figure 17

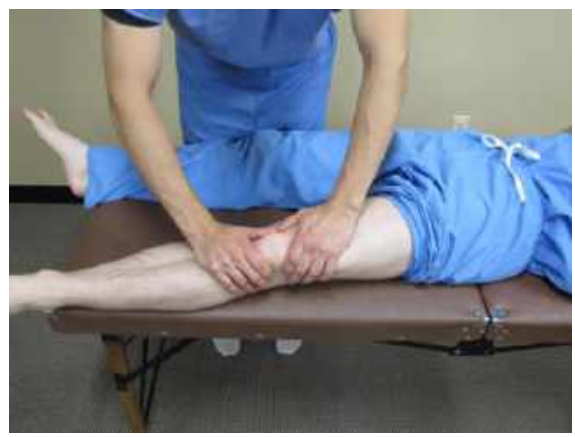


Figure 17: The Patellar Apprehension Test

The patellar grind test[18] evaluates patellofemoral syndrome (PFS) pathology caused by lateral traction of the patella due to quadriceps muscle imbalance or medial retinaculum laxity; abnormal tracking may cause chondromalacia patella. The patient is positioned supine with the knee extended. The examiner grasps the patella and pushes it posteriorly; the patient is directed to contract their quadriceps muscle. The patient may experience discomfort, as the pain is usually severe in the PFS.

Figure 18: The Patellar Grind Test

Figure 18

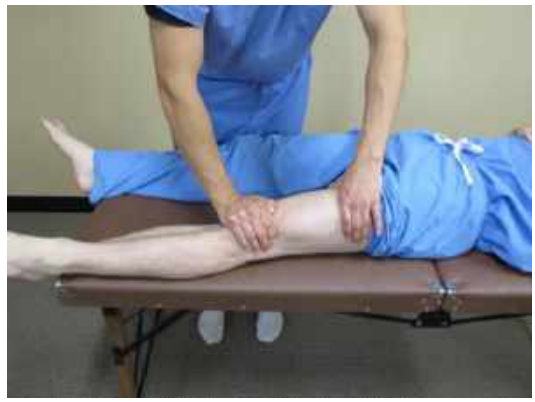


Table 4: Evidence Based Evaluation of Knee Orthopedic Special Tests

Table 4				
Test	Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value
McMurray's test	37	77		
Childress test	Not reported			
Thessaly's test: Medial Lateral	 89 92	 97 96		
Anterior Drawer Test (under anesthesia)	41	86		
Lachman's test	95	Not reported		
Posterior Drawer Test	90	99		
Varus Stress	25	Not reported		
Valgus Stress	86	Not reported		
Patellar apprehension test	39	Not reported		
Patellar grind test	Not reported			

Neurovascular

In the cause of trauma, it is especially important to check all the arterial pulses of the lower extremity starting from the groin: the femoral artery, the popliteal artery at the posterior knee, the tibialis posterior, and dorsalis pedis pulses of the foot and ankle region.

Evaluation of Knee Somatic Dysfunction

Osteopathic musculoskeletal evaluation of the knee begins with understanding the origin and insertion of the lower extremity musculature. The use of muscle energy (ME) and strain-counterstrain (S/CS) involves these muscles and the principles of direct and indirect treatment procedures: respectively, a lever, fulcrum, and force at the restrictive barrier, or at a point of ease in the diagnosis position. Knee pathology will often produce pain in the hip and pelvis as a result of compensatory gait. Clinically, we see somatic dysfunction between the tibia and fibula that is either anterior or posterior and between the tibia and femur that is either internally or externally rotated.

Anterior fibular head

Fibular head pathology is named in relation to the tibia. Hence, an anterior fibular head is pathologically anterior to its corresponding tibia. To diagnose this dysfunction, the patient is seated at the edge of the treatment table, legs hanging. The examiner palpates the fibular heads to assess if one is anterior or posterior compared with the contralateral side. The fibular heads are glided anteriorly and posteriorly to determine a restricted side. When the fibular head is anterior it will easily glide in that direction, however, it will resist posterior gliding.

Knee Treatment Examples of the Anterior Fibular Head:

Anterior Fibular Head with ME

1. The patient remains seated after diagnosis.
2. Create a lever by inverting the ankle and grasping the proximal portion of the fibula as seen.

3. Reverse the components of the positional diagnosis by moving the fibula posteriorly and rotating the tibia externally.
4. The fulcrum is actually established after the fibula is moved posteriorly by immobilizing the tibia (i.e., make sure all movement is between the tibia and fibula and that the tibia is immobile).
5. Once at the closed pack position, slightly release tension to the "feather edge" of the barrier.
6. Create an isometric contraction by instructing the patient to straighten their foot while the operator maintains an unyielding counter force for 3-5 seconds. Have the patient relax. Reposition at the new barrier by taking up the slack into further posterior movement. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
7. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Anterior Fibular Head with HVLA

1. The patient remains seated after diagnosis.
2. Create a lever by inverting the ankle and grasping the proximal portion of the fibula as seen.

3. Reverse the components of the positional diagnosis by moving the fibula posteriorly and rotating the tibia externally.
4. The fulcrum is actually established after the fibula is moved posteriorly by immobilizing the tibia (i.e., make sure all movement is between the tibia and fibula and that the tibia is immobile).
5. The operator "loose packs" the joint by introducing slight internal/external rotation of the tibia, and flexion/extension of the knee, prior to applying an anterior-to-posterior impulse on the fibular head with the thumb.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Knee Treatment Examples of the Anterior Fibular Head:

Anterior Fibular Head with Functional Procedures

1. The patient remains seated after diagnosis.
2. The clinician inverts the foot to "float" the fibular head.

3. The operator then moves the fibula into the positional diagnosis by internally rotating the tibia.
4. Effort is made to find the dynamic balance point.
5. An axial compression or distraction is added at the tibiofibular joint to initiate treatment.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Anterior Fibular Head with S/CS (aka Peroneus Longus Tender point)

1. The tender point is located on the anterior aspect of the proximal fibular head as seen below. This correlates with Myers' peroneus longus tender point.[27(pp209)]

2. The patient is seated with the operator seated on the same side of the affected leg. The patient's knee is flexed to ~90°and the foot is inverted/plantar flexed, while the leg and foot are internally rotated to bring the fibular head anterior until the tender point is relieved by 70%.
3. Maintain this position for 90 seconds.
4. SLOWLY return the patient to the neutral position.
5. Recheck the original diagnosis to ensure resolution of tender point

Posterior fibular head

To diagnose this dysfunction, the patient sits at the edge of the treatment table, legs hanging. The examiner palpates the anterior fibular head to assess if one is anterior or posterior compared with the contralateral side. The fibular heads are glided anteriorly and posteriorly to determine a restricted side. When a fibular head is "posterior," it will easily glide in that direction, however, it will resist anterior gliding.

Knee Treatment Examples of the Posterior Fibular Head:

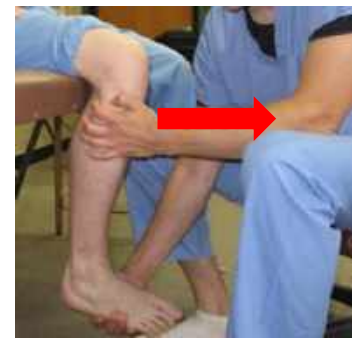
Posterior Fibular Head with ME

1. The patient remains seated after diagnosis.
2. Create a lever by inverting the ankle and grasping the proximal portion of the fibula as seen.

3. Reverse the components of the positional diagnosis by moving the fibula anteriorly and rotating the tibia internally.
4. The fulcrum is actually established after the fibula is moved anteriorly by immobilizing the tibia (i.e., make sure all movement is between the tibia and fibula and that the tibia is immobile).
5. Once at the closed pack position, slightly release tension to the "feather edge" of the barrier.
6. Create an isometric contraction by instructing the patient to straighten their foot while the operator maintains an unyielding counter force for 3-5 seconds. Have the patient relax. Reposition at the new barrier by taking up the slack into further anterior movement. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
7. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Posterior Fibular Head with HVLA

1. The patient remains seated after diagnosis.
2. Create a lever by inverting the ankle and grasping the proximal portion of the fibula as seen.
3. Reverse the components of the positional diagnosis by moving the fibula anteriorly and rotating the tibia internally.
4. The fulcrum is actually established after the fibula is moved posteriorly by immobilizing the tibia (i.e., make sure all movement is between the tibia and fibula and that the tibia is immobile).
5. The clinician "loose packs" the joint by introducing slight internal/external rotation of the tibia and flexion/extension of the knee, prior to applying posterior-to-anterior impulse on the fibular head with the index finger.



6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Knee Treatment Examples of the Posterior Fibular Head:

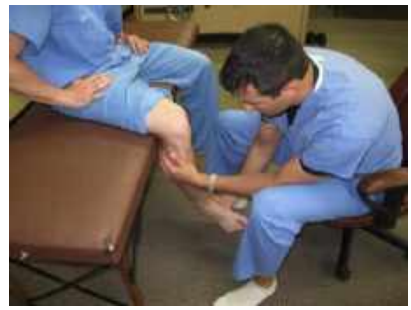
Posterior Fibular Head with Functional Procedure

1. The patient remains seated after diagnosis.
2. The clinician inverts the foot to "float" the fibular head.

3. The operator then places the fibula in the positional diagnosis by rotating the tibia externally.
4. Effort is made to find the dynamic balance point.
5. An axial compression or distraction is added at the tibiofibular joint to initiate treatment.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Posterior Fibular Head with S/CS (aka Lateral Hamstring Tender point)

1. The tender point is located on the posterior aspect of the proximal fibular head. Meyers names this the lateral hamstring tenderpoint.[27(pp204)]



2. The patient is seated with the operator seated on the same side of the affected leg. The patient's knee is flexed to ~90° and the foot is inverted/plantar flexed, while the leg and foot are externally rotated to bring the fibular head posteriorly until the tender point is relieved by 70%.
3. Maintain this position for 90 seconds.
4. SLOWLY return the patient to the neutral position.
5. Recheck the original diagnosis to ensure resolution of tender point.

.

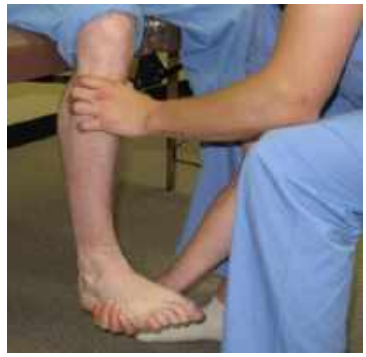
Externally Rotated Tibia

To diagnose this dysfunction, the patient is seated with legs hanging, knees bent to 90°. The ankle is held at 90° while the clinician internally and externally rotates both legs with a handhold on the dorsum of the foot. An "externally rotated tibia" is diagnosed when on motion testing, resistance or decreased internal rotation of the tibia is observed.

Knee Treatment Examples of Externally Rotated Tibia:

Externally Rotated Tibia with ME

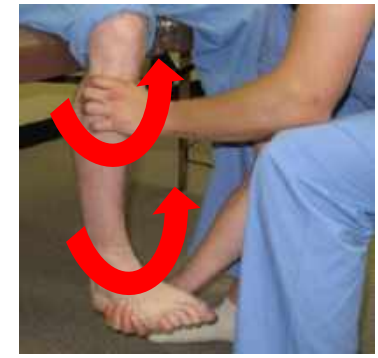
1. The patient remains seated after diagnosis.
2. The fulcrum is established by immobilizing the femur (i.e., in the seated position at the end of the table, the femur does not move).
3. A lever is created using the leg and foot as seen.



4. Reverse the components of the positional diagnosis by rotating the tibia internally.
5. Once at the closed pack position, slightly release tension to the '"feather edge" of the barrier.
6. Create an isometric contraction by instructing the patient to externally rotate their leg while the operator maintains an unyielding counter force for 3-5 seconds. Have the patient relax. Reposition at the new barrier by taking up the slack into further internal movement. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
7. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Externally Rotated Tibia with HVLA

1. The patient remains seated after diagnosis.
2. The fulcrum is established by immobilizing the femur (i.e., in the seated position at the end of the table, the femur does not move).
3. A lever is created using the leg and foot as seen.

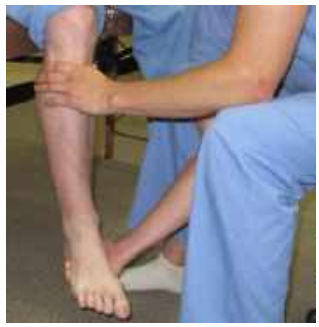


4. Reverse the components of the positional diagnosis by rotating the tibia internally.
5. The clinician "loose packs" the joint by introducing slight internal/external rotation of the hip and flexion/extension of the knee, prior to applying an internally directed rotational thrust of the leg with BOTH hands.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Knee Treatment Examples of Externally Rotated Tibia:

Externally Rotated Tibia with Functional Procedures

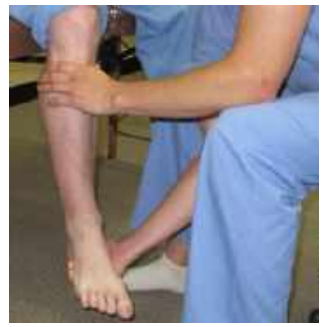
1. The patient remains seated after diagnosis.
2. The operator then places the tibia in the positional diagnosis by rotating the tibia externally.



3. Effort is made to find the dynamic balance point.
4. An axial compression or distraction is added at the knee to initiate treatment.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Externally Rotated Tibia with S/CS (aka Medial Hamstring Tender point)

1. The tender point is located on the posterior aspect of the medial tibial surface. Meyers names this the medial hamstring tenderpoint.[27(pp203)]



2. The patient is seated, knee is flexed to ~90° and tibia is externally rotated on the femur until the tender point is relieved by 70%. Jones[9(pp101)] and Meyers[27(pp203)] perform this treatment in a supine position.
3. Maintain this position for 90 seconds.
4. SLOWLY return the patient to the neutral position.
5. Recheck the original diagnosis to ensure resolution of the tender point.

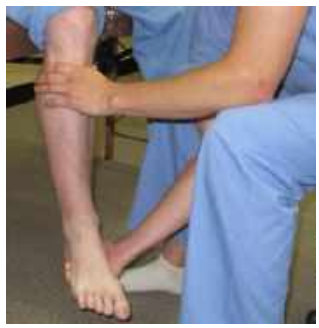
Internally Rotated Tibia

To diagnose this dysfunction, the patient is seated with legs hanging, knees bent to 90°. The ankle is held at 90°, while the clinician internally and externally rotates both legs with a handhold on the dorsum of the foot. An "internally rotated tibia" is diagnosed with resistance or decreased external rotation of the tibia on motion testing.

Knee Treatment Examples of Internally Rotated Tibia:

Internally Rotated Tibia with ME

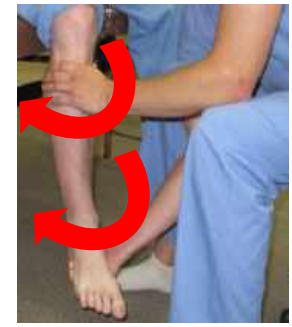
1. The patient remains seated after diagnosis.
2. The fulcrum is established by immobilizing the femur (i.e., in the seated position at the end of the table, the femur does not move).
3. A lever is created using the leg and foot as seen.



4. Reverse the components of the positional diagnosis by rotating the tibia externally.
5. Once at the closed pack position, slightly release tension to the "feather edge" of the barrier.
6. Create an isometric contraction by instructing the patient to internally rotate their leg, while the operator maintains an unyielding counter force for 3-5 seconds. Have the patient relax. Reposition at the new barrier by taking up the slack into further external movement. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
7. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Internally Rotated Tibia with HVLA

1. The patient remains seated after diagnosis.
2. The fulcrum is established by immobilizing the femur (i.e., in the seated position at the end of the table, the femur does not move).
3. A lever is created using the leg and foot as seen.

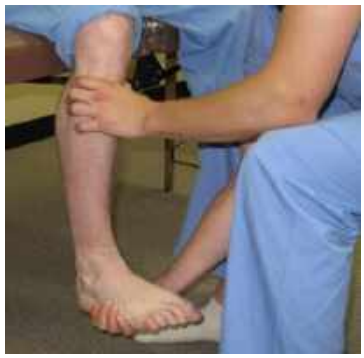


4. Reverse the components of the positional diagnosis by rotating the tibia externally.
5. The clinician "loose packs" the joint by introducing slight internal/external rotation of the hip, and flexion/extension of the knee, prior to applying an externally directed rotational thrust of the leg with BOTH hands.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Knee Treatment Examples of Internally Rotated Tibia:

Internally Rotated Tibia with Functional Procedure

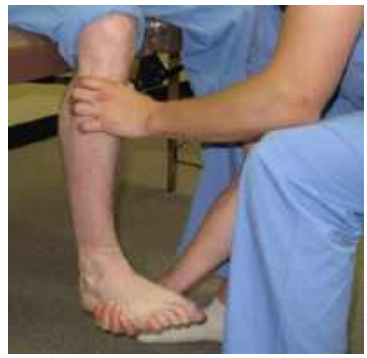
1. The patient remains seated after diagnosis.
2. The operator then places the tibia in the positional diagnosis by rotating the tibia internally.



3. Effort is made to find the dynamic balance point.
4. An axial compression or distraction is added at the knee to initiate treatment.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Internally Rotated Tibia with S/CS (aka MCL Tender point)

1. The tender point is located on the medial aspect of the medial tibial surface. Meyers names this the medial collateral ligament tender point.[27(pp193)]



2. The patient is seated with the operator seated on the same side of the affected leg. The patient's knee is flexed to ~90° and the tibia is internally rotated on the femur until the tender point is relieved by 70%.
3. Maintain this position for 90 seconds.
4. SLOWLY return the patient to the neutral position.
5. Recheck the original diagnosis to ensure resolution of tender point.

Anatomy of the Foot & Ankle

The bony anatomy of the ankle includes the distal tibia and fibula held together by the interosseous membrane. The fibrous distal joint is stabilized by the anterior tibiofibular ligament and the posterior tibiofibular ligament (Figures 19 and 20); this is in contrast to the proximal tibiofibular that is a synovial joint. At the distal end of the tibiofibular joint are two bony projections, the medial and lateral malleolus. Between these two structures is the ankle mortise, made up of the curved under surface of the tibia whose articular surface contacts the talus – the tibiotalar joint.

Figure 19

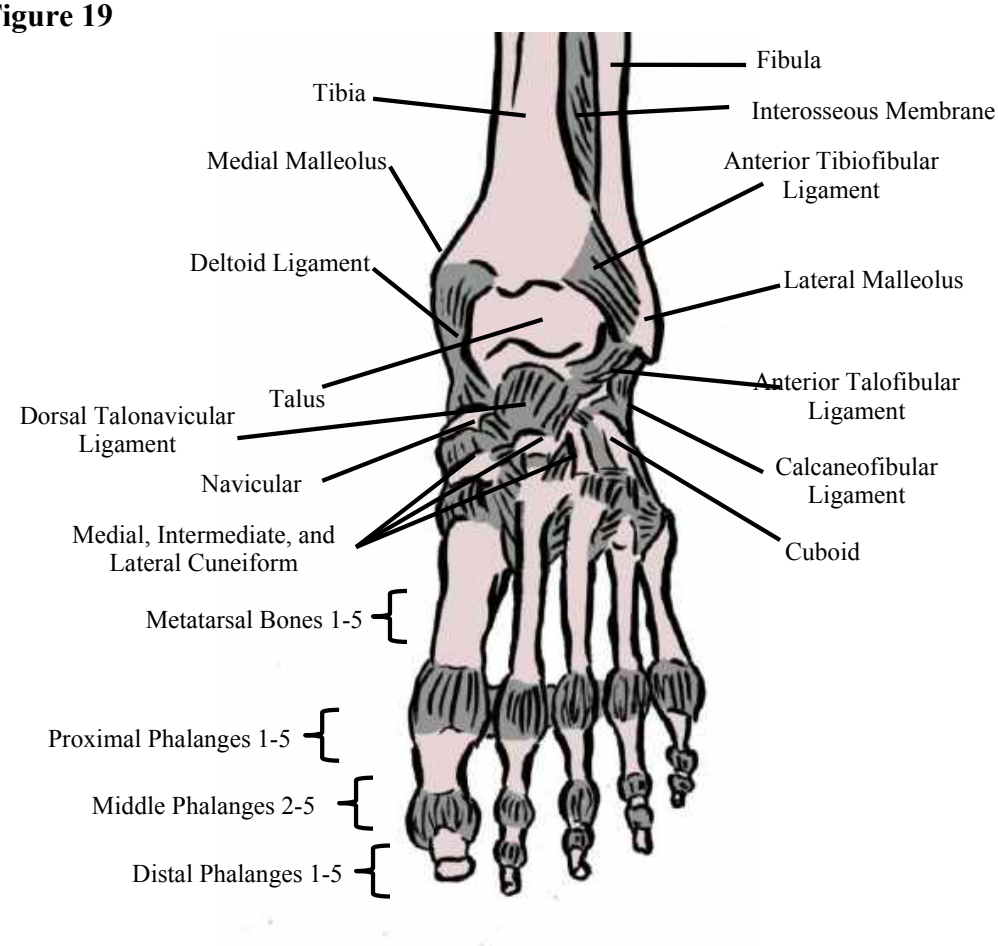


Figure 19: Boney and Ligamentous Anatomy of the Ankle

The talus has a head, neck, and three articular surfaces; the tibiotalar joint moves the joint into dorsi-flexion and plantar flexion, and the subtalar and talocalcaneonavicular joints comprise the two joints of inversion and eversion of the ankle (Figure 21). The tibiotalar joint (Figures 19 and 20) is stabilized to the anterior talofibular ligament (ATFL), posterior talofibular ligament (PTFL), and the deltoid ligament on the medial side. The deltoid ligament starts at the medial malleolus and attaches to the talus, navicular, and below the sinus Tarsi of the

calcaneus. These ligaments, along with the calcaneofibular ligament, stabilize the joint in the anterior posterior direction, as well as with inversion and eversion of the ankle. The joint capsule is loose anteriorly and posteriorly to allow for dorsiflexion and plantar flexion.

Figure 20

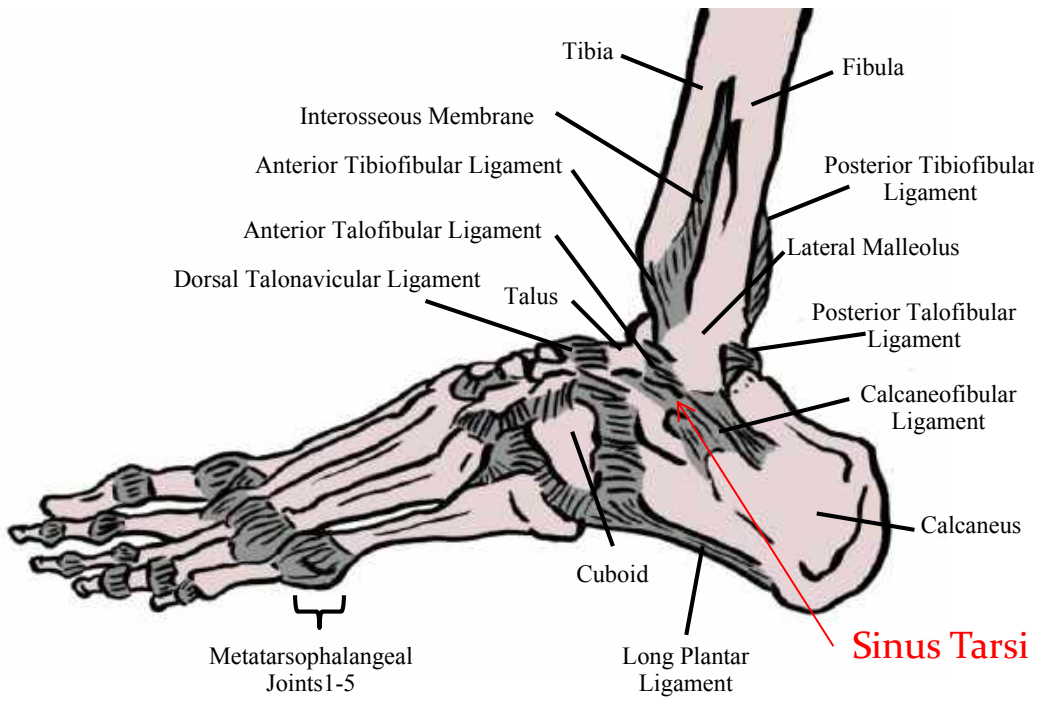
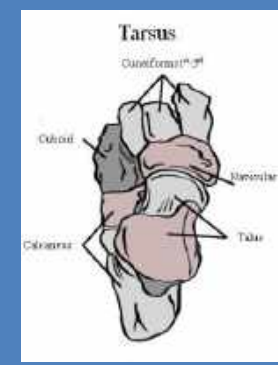


Figure 20: Bone and Ligamentous Ankle and Foot Anatomy, Lateral View

The Tarsus:
Is comprised of the talus, calcaneus, navicular, 1st -3rd cuneiforms, and cuboid.

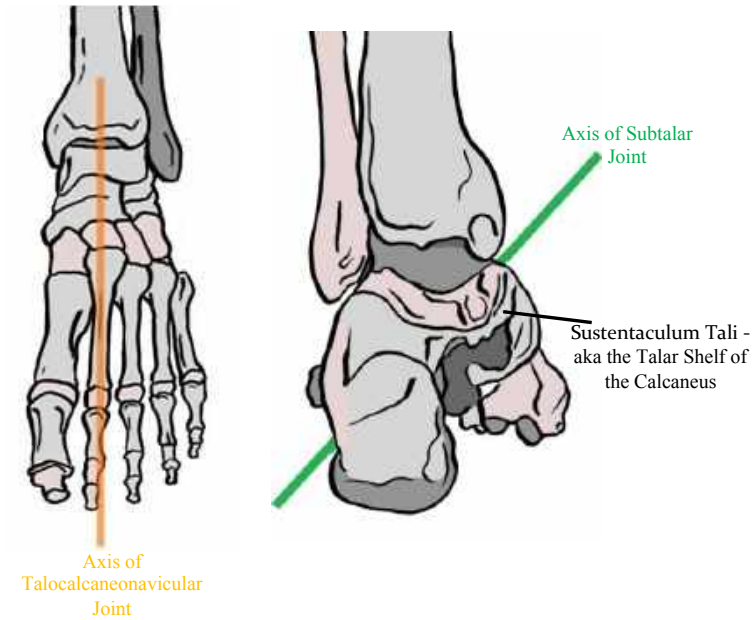


The tarsus is comprised of the talus, calcaneus, navicular, first through third cuneiforms, and the cuboid. The calcaneus (heel) has a posterior portion for Achilles tendon attachment, anterior surface for cuboid articulation, a medial portion called the sustentaculum tali, and an upper portion articulating with the talus on two surfaces; the small portion in front and the larger portion behind form the subtalar joint.

Figure 21: Axes of Inversion and Eversion of the Ankle

Two axes of inversion/eversion of the foot:
1. Subtalar Joint (aka Talocalcaneal Joint)
2. Talocalcaneonavicular Joint

Figure 21

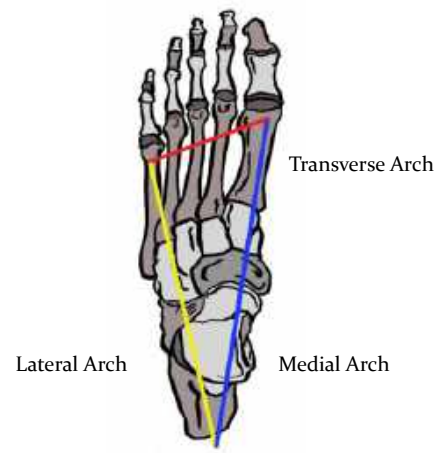


The second joint of eversion/inversion is the talocalcaneonavicular joint. The head of the talus articulates with the anterior calcaneus and proximal navicular and is stabilized by the calcaneonavicular ligament (spring ligament). The motion of inversion/eversion moves about an oblique axis (medial to the long axis of the foot). In addition to the ATFL, PTFL, and deltoid ligaments, the ligaments of stabilization for inversion/eversion are the interosseous-talocalcaneal ligaments that lie between the subtalar and talocalcaneonavicular joints.

The remaining bony anatomy relates to the forefoot. The forefoot includes metatarsals 1-5, with the first one being the biggest, the second the longest, and the fifth unique for its proximal tubercle. The proximal metatarsals form an articulation with the tarsal bones to form the transverse arch of the foot (Figure 22).

Figure 22: Three Arches of the Foot

Figure 22



Distal to the metatarsals are the phalanges. Similar to the hand, the great toe has two phalanges, a proximal and distal, while the remaining digits have three phalanges including the proximal, middle, and distal (Figure 19). The joints involved are the metatarsophalangeal joint and two interphalangeal joints.
The major muscle groups of the foot and ankle include the dorsiflexors, plantar-flexors, invertors, and evertors (Figure 23). The details of muscle function are listed in Table 5.

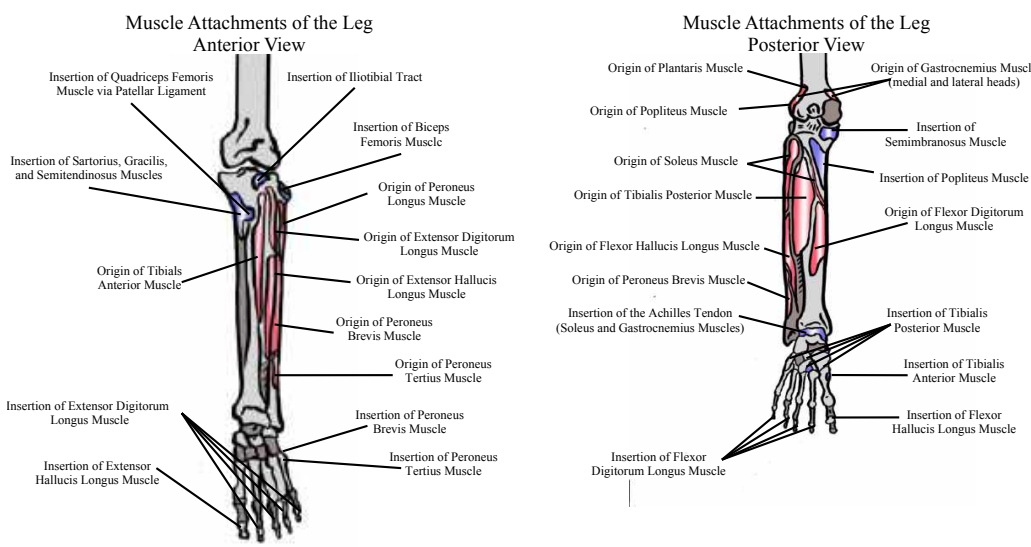
Motions & Muscles of the Foot and Ankle

Table 5: Motions and Muscles of the Foot and Ankle

TABLE 5	
Muscle	Action
Tibialis Anterior	The main dorsiflexor of the foot also inverts the foot. Other minor dorsiflexors include the extensor digitorum longus and the peroneus tertius.
Gastrocnemius and Soleus	Plantar Flexor. The two muscles converge to form the achilles tendon.
Peroneus Longus and Brevis	Plantar Flexor and everts foot
Peroneus Tertius	Everts foot
Extensor Hallucis Longus Extensor Digitorum Longus	Extensor of toes
Flexor Hallucis Longus Flexor Digitorum Longus	Flexor of toes

Figure 23

Figure 23: Origin and Insertion of Leg Musculature



Orthopedic Evaluation of the Foot and Ankle

Evaluation of the foot and ankle should involve a systematic approach with the following sequence of evaluation:

1. Inspection
2. Range of motion
3. Palpation
4. Motor strength testing
5. Neurovascular testing
6. Orthopedic Special Tests
7. Evaluation for Somatic Dysfunction

Inspection

Observe the patient in stance and during gait. Notice any asymmetries related to foot biomechanics, including pes planus, valgus heel, or any acute swelling or ecchymosis post an inversion type of ankle sprain. As with other areas of the lower extremity, observe for an antalgic gait or favoring of one side compared to the other.

Range of Motion

Observe the active and passive ROM of dorsiflexion, plantar flexion, inversion, and eversion. Normal dorsiflexion is approximately 20° and normal plantar flexion is approximately 55°. Inversion and eversion are approximately 10°.

Palpation

Palpate the major bony prominences of the foot and ankle including the medial and lateral malleolus, tubercle of the fifth metatarsal, bony prominence of the navicular bone, sinus Tarsi, the cuneiforms 1 through 3, and the cuboid. In the forefoot, palpate the metatarsals and phalanges. Also palpate the soft tissue structures in the posterior calcaneus and heel at the insertion of the Achilles tendon and plantar fascia. It is important to palpate the lateral ankle ligaments including the ATFL, calcaneofibular ligament, anterior inferior tibiofibular ligament, and posterior inferior tibiofibular ligament. On the medial ankle, the deltoid ligament has broad insertions to the navicular, sinus Tarsi, and calcaneus. Importantly, palpate along the syndesmosis of the tibiofibular joint. Palpate the tendons of the lateral ankle including the peroneal tendons, peroneus longus, and peroneus brevis. Then palpate the medial tendons including tibialis posterior, flexor digitorum longus, and flexor hallucis longus.

Motor strength testing

The main dorsiflexors of the foot include tibialis anterior (deep peroneal nerve, L4 and L5), extensor hallucis longus (deep peroneal nerve L5), and extensor digitorum longus (deep peroneal nerve L5). To test the tibialis anterior, have the patient sit with leg hanging from the table and place the foot in dorsiflexion and

eversion while the operator tries to force the foot into plantar flexion and eversion. To test extensor hallucis longus, place the great toe into dorsiflexion and push down on the great toe, evaluating for weakness. To test the motor strength of the extensor digitorum longus, similarly move the second through fifth toes into dorsiflexion and apply force toward plantar flexion. The major plantar flexors are gastrocnemius, soleus, and plantaris (tibial nerve, S1, S2). Testing the plantar flexor muscles, instruct the patient to bounce up and down on the balls of their feet single weighted, or have them supine on the table with the knee extended and the foot dorsiflexed against the examiner's resistance.

The main evertors are peroneus longus and brevis (superficial peroneal nerve, S1) these muscles are tested with the patient seated and the leg hanging off of the treatment table. The examiner brings the patient's foot into inversion, and the patient everts against resistance. The main evertor of the foot is tibialis posterior, with tibialis anterior providing a minor contribution. To test the strength of tibialis posterior, the patient is seated with the leg hanging off of the treatment table and the foot is brought into eversion; the patient is asked to plantar flex and invert the foot against resistance.

Neurovascular Testing

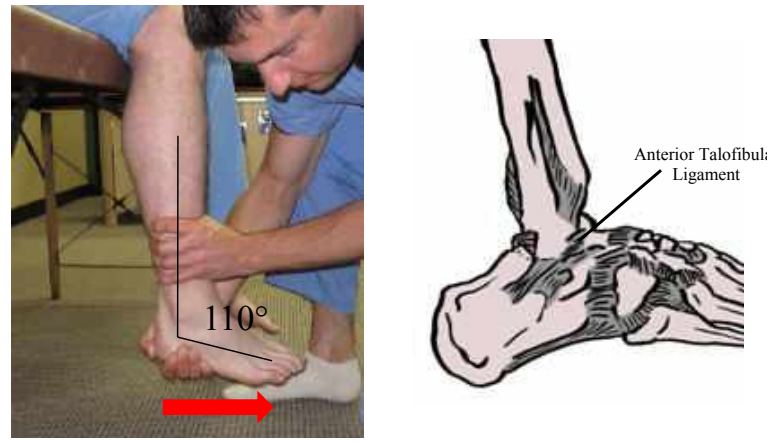
Evaluate using Tinel's test at the tarsal tunnel for nerve entrapment. Palpate arterial pulses of the tibialis posterior on the medial ankle and dorsal pedis on the dorsum of the foot. Evaluate sensation to soft touch and pinprick the following: L4 dermatome on the medial side of the foot to the medial first MTP joint, L5 on the dorsum of the foot, the great toe to the fourth toe, and S1 on the lateral foot and dorsum of the fifth toe.

Orthopedic Special Tests

Anterior drawer test:[19,20] evaluates the integrity of the ATFL (Figure 24). The patient is placed in a seated position with a leg hanging off the table. The foot is placed into approximately 20° of plantar flexion with medial rotation of the foot to take any slack off of the ligament. The operator places one hand at the posterior calcaneus and the other hand bracing the distal shin. The operator then provides an anterior translation of the calcaneus to test the laxity of the ATFL. A positive test occurs when the patient has significant laxity compared to the contralateral side.

Figure 24: The Anterior Drawer Test
- Evaluates the anterior talofibular ligament

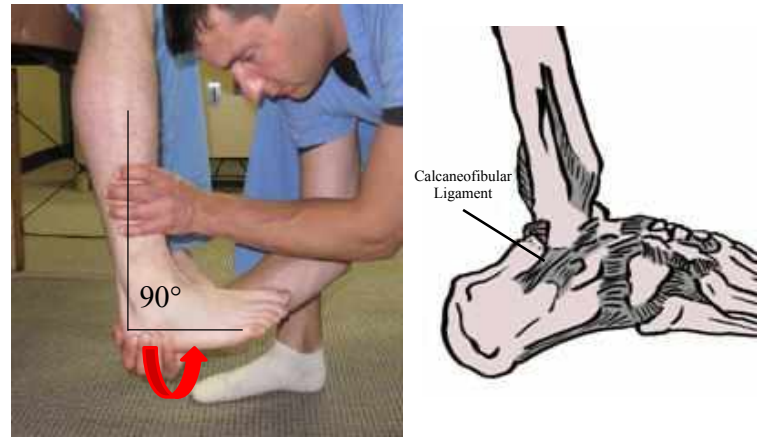
Figure 24



Talar tilt test[21,32] evaluates the integrity of the calcaneofibular ligament. The ankle is kept at 90°, and the operator has a similar handhold as with the anterior drawer test, except in this instance the operator applies an inversion force to the calcaneus to test the calcaneofibular ligament. As with the anterior drawer test, there is excessive laxity and tilt when the test is positive.

Figure 25: The Talar Tilt Test
- Evaluates the anterior calcaneofibular ligament. The arrow indicates inversion.
- Eversion in this same position evaluates the deltoid ligament (i.e. , Reverse Talar Tilt Test)

Figure 25



Reverse talar tilt test evaluates integrity of the deltoid ligament. Perform similarly to the talar tilt test, but the ankle and foot are everted to test for laxity of the deltoid ligament.

Thompson test[22,23] evaluates Achilles tendon rupture (Figure 26). Patient is prone on the treatment table with the knee flexed to 90°. Operator squeezes the mid-calf to note subsequent plantar flexion of the foot. If plantar flexion does not occur, there is suspicion for Achilles tendon rupture.

Figure 26

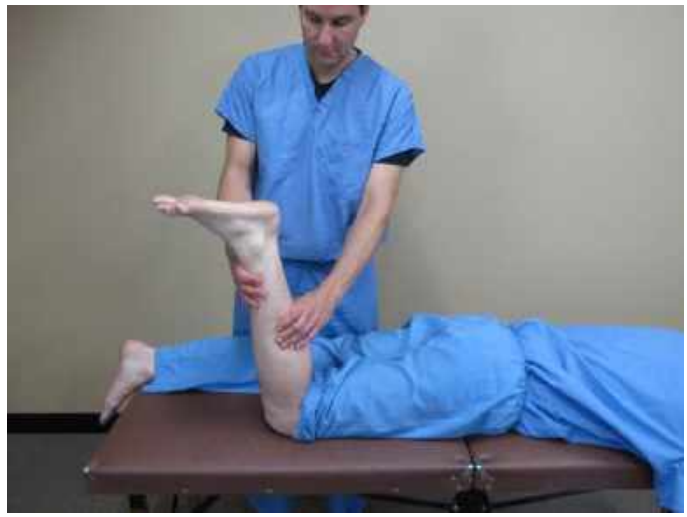


Figure 26: Thompson Test

External rotation test[24,25,31] for syndesmotic injury of the talofibular joint or "high ankle sprain:" ankle in neutral at 90° with a leg hanging off of the table and the operator externally rotates the foot slowly. The test is positive if the patient feels pain at the syndesmosis. This test is also used to perform a stress view on x-ray.

Figure 27

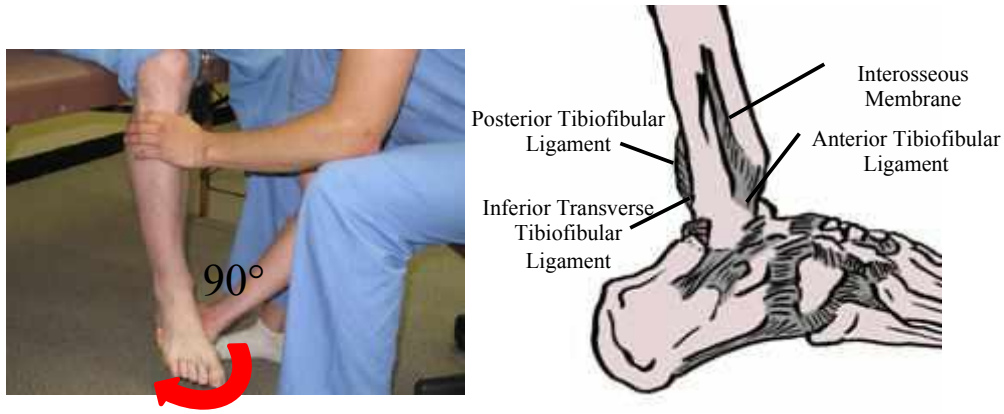


Figure 27: External Rotation Test for syndesmotic injury. The syndesmosis includes the:

- Ant. Tibiofibular Ligament
- Post. Tibiofibular Ligament
- Interosseous Ligament
- Inf. Transverse Tibiofibular Ligament (not seen in diagram)

Peroneal tendon subluxation test: The patient dorsiflexes and plantar flexes; then palpate for subluxation and reproduction of the patient's symptoms.

Table 6: Statistical analysis of Foot and Ankle Orthopedic Tests

Table 6				
Test	Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value
Anterior Drawer	95	84. 2	96. 25	80
Talar Tilt Test	Not reported			
Thompson Test	96	93		
External Rotation Test	Not reported			
Peroneal Tendon Subluxation test	Not reported			

Evaluation of Foot & Ankle Somatic Dysfunction

Musculoskeletal evaluation of the foot and ankle begins with understanding the origin and insertion of the lower extremity musculature. Ankle pathology will often present following trauma. Clinically, we see somatic dysfunction between the tibia and fibula that is either anterior or posterior and between the tibia and femur that is either internally or externally rotated.

Posterior Distal Tibia

In this dysfunction, the tibia is posterior to its typical position related to the talus. To diagnose this dysfunction, the clinician is positioned in front of the patient who is seated at the edge of the table with legs hanging and ankles at 90° of flexion. The physician supports the plantar surface of both feet and places the thumbs over the talus (Figure 28a) as the ankle glides posteriorly. The dysfunctional side will have restricted posterior glide.

The patient then lies in the supine position as the physician uses one hand to grasp the distal tibia and the other hand to cup the calcaneus; then while bracing the calcaneus, the tibia is glided posteriorly. If the tibia is anterior, it will not spring posteriorly. If the tibia is posterior, the calcaneus will not glide posteriorly.

Figure 28

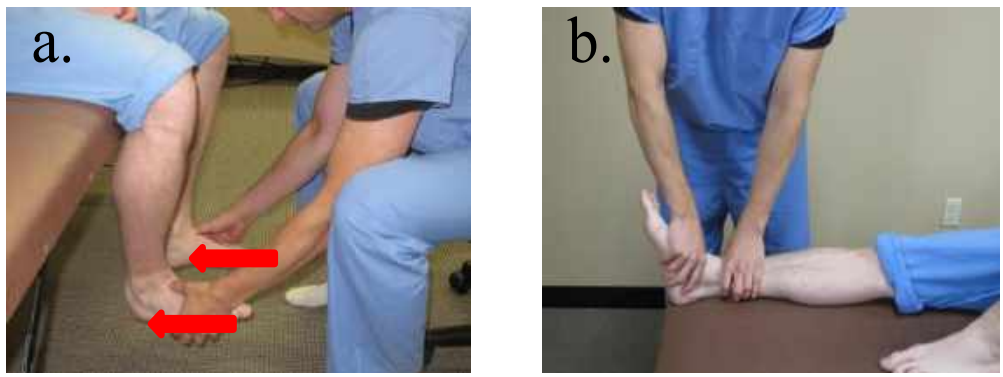
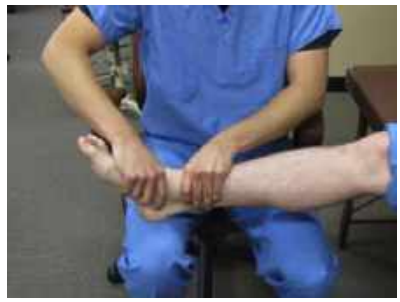


Figure 28: Diagnosis of Posterior Distal Tibia and Anterior Distal Tibia
a. First Step
b. Second Step

Foot/Ankle Treatment Examples of Posterior Distal Tibia:

Posterior Distal Tibia with ME

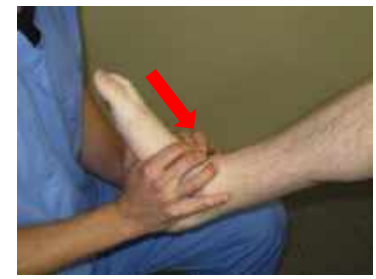
1. The patient remains seated after diagnosis.
2. Establish a fulcrum by holding the tibia with one hand.
3. Create a lever by grasping the foot with the other hand.



4. Reverse the components of the positional diagnosis by dorsiflexing the foot.
5. Once at the closed pack position, slightly release tension to the "feather edge" of the barrier.
6. Create an isometric contraction by having the patient plantar flex his foot as the operator maintains an unyielding counter force for 3-5 seconds. Have the patient relax. Reposition at the new barrier by taking up the slack into further dorsiflexion. Repeat the process of isometric contraction, relaxation, and movement to the new barrier three to five times.
7. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Posterior Distal Tibia with HVLA

1. The patient remains seated after diagnosis.
2. Established a fulcrum by holding the tibia with one hand.
3. Create a lever by grasping the foot with the other hand.
4. Reverse the components of the positional diagnosis by dorsiflexing the foot
5. The clinician "loose packs" the joint by introducing slight internal/external rotation, and flexion/extension of the foot while focusing on the talus.
6. Apply a posterior thrust on the talus.

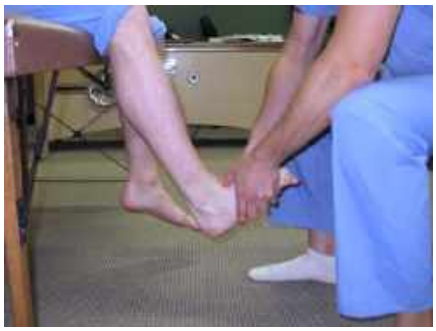


7. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Foot/Ankle Treatment Examples of Posterior Distal Tibia:

Posterior Distal Tibia with Functional

1. The patient remains seated after diagnosis.
2. The clinician inverts the foot to "float" the fibular head



3. The operator then places the fibula in the positional diagnosis by rotating the tibia externally.
4. Effort is made to find the dynamic balance point.
5. An axial compression or distraction is added toward the tibiofibular joint. While maintaining this force, the joint unwinds away from the restrictive barrier, reaches a still point, and then unwinds toward the previous restrictive barrier.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Posterior Distal Tibia with S/CS (aka Extension Ankle[9pp108])

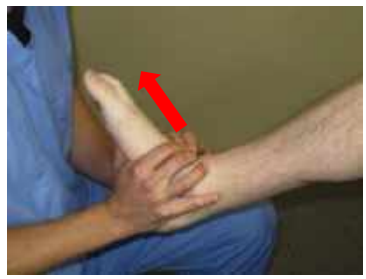
1. Extension ankle tender point on the heads of either the medial or lateral gastrocnemius. Meyers names this the gastrocnemius tenderpoint.[27pp199]
2. The patient is placed in the prone position on the table with the knee flexed to approximately 60° and marked plantar flexion of the ankle as the operator places the dorsum of the foot on his/her thigh.

3. The operator then applies a force on the calcaneus toward the table with one hand while applying a similar downward force on the calf with the other hand, monitoring the tender point until it is relieved by 70%.
4. Maintain this position for 90 seconds.
5. SLOWLY return the patient to the neutral position.
6. Recheck the original diagnosis to ensure resolution of tender point.

Foot/Ankle Treatment Examples of Anterior Distal Tibia:

Anterior Distal Tibia with HVLA

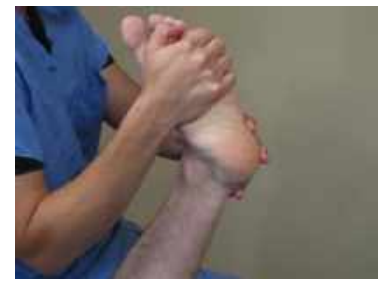
1. The patient remains seated after diagnosis.
2. Establish a fulcrum by holding the tibia with one hand.
3. Create a lever by grasping the foot below the tibia with the other hand.
4. Reverse the components of the positional diagnosis by plantar flexing the foot
5. The clinician "loose packs" the joint by introducing slight internal/external rotation of the ankle and flexion/extension of the extension
6. Apply an anterior thrust on the talus.



7. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Anterior Distal Tibia with S/CS (aka Talus or Astragalus[9pp109])

1. The tender point is on the medial side of the foot located approximately 2 cm anterior to the medial malleolus.
2. Patient is prone on the table with the knee flexed to 90°. The foot is placed into inversion and internal rotation.



3. Fine tune inversion and rotation until the tender point is relieved by 70%.
4. Maintain this position for 90 seconds.
5. SLOWLY return the patient to the neutral position.
6. Recheck the original diagnosis to ensure resolution of tender point.

Lower Extremity Treatment Pearls

In a patient with documented osteoarthritis of the hip who develops a new onset antalgic gait, re-imaging the patient is important to assess for avascular necrosis, stress fracture or other potential surgical indications.

The lumbopelvic region can cause multiple, overlapping pain patterns referred to the lower extremity. In patients with a normal neurological examination, for various soft tissue, tissue/joint dysfunctions it is appropriate to use manual medicine, or injections to act both diagnostically and or therapeutically.

In patients with a seemingly minor ankle sprain, be sure to send the patient for comprehensive rehabilitation that includes activation of the lumbopelvic stabilizing musculature. Many lower extremity injuries can lead to inhibition of these muscles and subsequent compensatory axial spine and peripheral joint pain complaints.

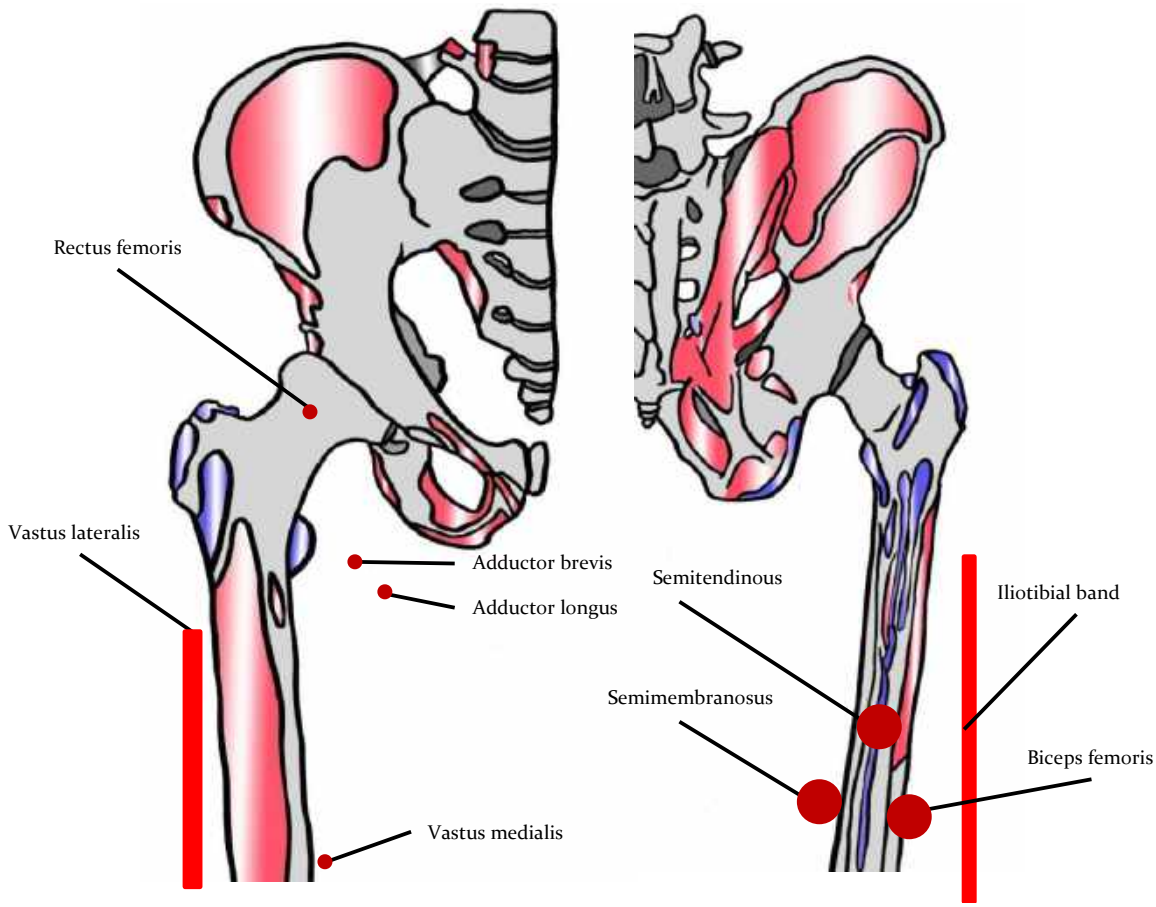
References:

1. Netter, Frank H. *Atlas of Human Anatomy*. Philadelphia, PA: Saunders/Elsevier, 2011.
2. Agur, Anne M. R. , and Ming J. Lee. *Grants Atlas of Anatomy*. Philadelphia: Lippincott Williams, 1999.
3. Harvey, D. "Assessment of the Flexibility of Elite Athletes Using the Modified Thomas Test." *British Journal of Sports Medicine*32, no. 1 (January 1998): 68–70.
4. Maitland, Geoffrey D. *The Peripheral Joints: Examination and Recording Guide*. Virgo Press, 1975.
5. Magee, D J. *Orthopedic Physical Assessment*. Philadelphia: Elsevier, 2011.
6. Bird, P. A., S. P. Oakley, R. Shnier, and B. W. Kirkham. "Prospective evaluation of magnetic resonance imaging and physical examination findings in patients with greater trochanteric pain syndrome." *Arthritis & Rheumatism: Official Journal of the American College of Rheumatology* 44, no. 9 (2001): 2138-2145.
7. Ober, F. R. "The role of the iliotibial band and fascia lata as a factor in the causation of low back disabilities and sciatica." *J. Bone Joint Surg.* 8 (1926): 171.
8. Stiles, EG Lecture notes from Osteopathic Principles and Practice, Kentucky College of Osteopathic Medicine, 1998-1999.
9. Jones, Lawrence H., et al. *Strain-Counterstrain*. Boise, ID: Jones Strain-Counterstrain, 1995.
10. Ward, Robert C. *Foundations for Osteopathic Medicine*. Philadelphia: Lippincott Williams & Wilkins 1998.
11. Kurosaka, M. , M. Yagi, S. Yoshiya, H. Muratsu, and K. Mizuno. "Efficacy of the axially loaded pivot shift test for the diagnosis of a meniscal tear. " *International orthopaedics* 23, no. 5 (1999): 271-274.
12. Ercin, Ersin, Ibrahim Kaya, Ibrahim Sungur, Emrah Demirbas, Ali Akin Ugras, and Ercan Mahmut Cetinus. "History, clinical findings, magnetic resonance imaging, and arthroscopic correlation in meniscal lesions. " *Knee surgery, sports traumatology, arthroscopy* 20, no. 5 (2012): 851-856.
13. Karachalios, Theofilos, Michael Hantes, Aristides H. Zibis, Vasilios Zachos, Apostolos H. Karantanas, and Konstantinos N. Malizos. "Diagnostic accuracy of a new clinical test (the Thessaly test) for early detection of meniscal tears. " *The Journal of Bone & Joint Surgery* 87, no. 5 (2005): 955-962.
14. Harilainen, A. "Evaluation of knee instability in acute ligamentous injuries." In *Annales chirurgiae et gynaecologiae*, vol. 76, no. 5, pp. 269-273. 1987.
15. Torg, Joseph S. , Wayne Conrad, and Vickie Kalen. "Clinical I diagnosis of anterior cruciate ligament instability in the athlete. " *The American journal of sports medicine* 4, no. 2 (1976): 84-93.
16. Rubinstein JR, Richard A., K. Donald Shelbourne, John R. McCarroll, Charles D. VanMeter, and Arthur C. Rettig. "The accuracy of the clinical examination in the setting of posterior cruciate ligament injuries." *The American journal of sports medicine* 22, no. 4 (1994): 550-557.
17. Sallay, Peter I., Jeffery Poggi, Kevin P. Speer, and William E. Garrett. "Acute dislocation of the patella: a correlative pathoanatomic study." *The American journal of sports medicine* 24, no. 1 (1996): 52-60.
18. Karlson, Stig. "Chondromalacia patellae." *Acta Chir Scand* 83 (1939): 347-381.

19. Landeros, Oscar, Harold M. Frost, and Christopher C. Higgins. "Post-traumatic anterior ankle instability." *Clinical Orthopaedics and Related Research* 56 (1968): 169-178.
20. Lindstrand, A. "New aspects in the diagnosis of lateral ankle sprains." *The Orthopedic clinics of North America* 7, no. 1 (1976): 247-249.
21. Rubin, Gustav, and Morris Witten. "The talar-tilt angle and the fibular collateral ligaments." *J Bone Joint Surg Am* 42, no. 2 (1960): 311-326.
22. Thompson, T. Campbell, and John H. Doherty. "Spontaneous rupture of tendon of Achilles: a new clinical diagnostic test." *Journal of Trauma and Acute Care Surgery* 2, no. 2 (1962): 126-129.
23. Maffulli, Nicola. "The clinical diagnosis of subcutaneous tear of the Achilles tendon." *The American journal of sports medicine*26, no. 2 (1998): 266-270.
24. Kleiger, Barnard. "The diagnosis and treatment of traumatic lateral ankle instability." *New York state journal of medicine* 54, no. 18 (1954): 2573.
25. Boytim, Mark J. , David A. Fischer, and Larry Neumann. "Syndesmotic ankle sprains." *The American journal of sports medicine*19, no. 3 (1991): 294-298.
26. Waddell, Gordon. *The Back Pain Revolution*. Edinburg: Churchill Livingstone, 1998.
27. Myers, Harmon L., et al. *Clinical Application of Counterstrain*. Tucson, AZ: Osteopathic Press, a Division of Tucson Osteopathic Medical Foundation, 2012.
28. Shakespeare, D. T., M. Stokes, K. P. Sherman, and A. Young. "Reflex inhibition of the quadriceps after meniscectomy: lack of association with pain." *Clinical physiology* 5, no. 2 (1985): 137-144.
29. Rice, David Andrew, and Peter John McNair. "Quadriceps arthrogenic muscle inhibition: neural mechanisms and treatment perspectives." In *Seminars in arthritis and rheumatism*, vol. 40, no. 3, pp. 250-266. WB Saunders, 2010.
30. Ostrowski, John A. "Accuracy of 3 diagnostic tests for anterior cruciate ligament tears." *Journal of athletic training* 41, no. 1 (2006): 120.
31. Sman, Amy D., Claire E. Hiller, and Kathryn M. Refshauge. "Diagnostic accuracy of clinical tests for diagnosis of ankle syndesmosis injury: a systematic review." *Br J Sports Med* 47, no. 10 (2013): 620-628.
32. Hertel J, Denegar CR, Monroe MM, Stokes WL. "Talocrural and subtalar joint instability after lateral ankle sprain." *Medicine and science in sports and exercise* 31, no. 11 (1999): 1501-1508.

Appendix A: Thigh S/CS Tenderpoints

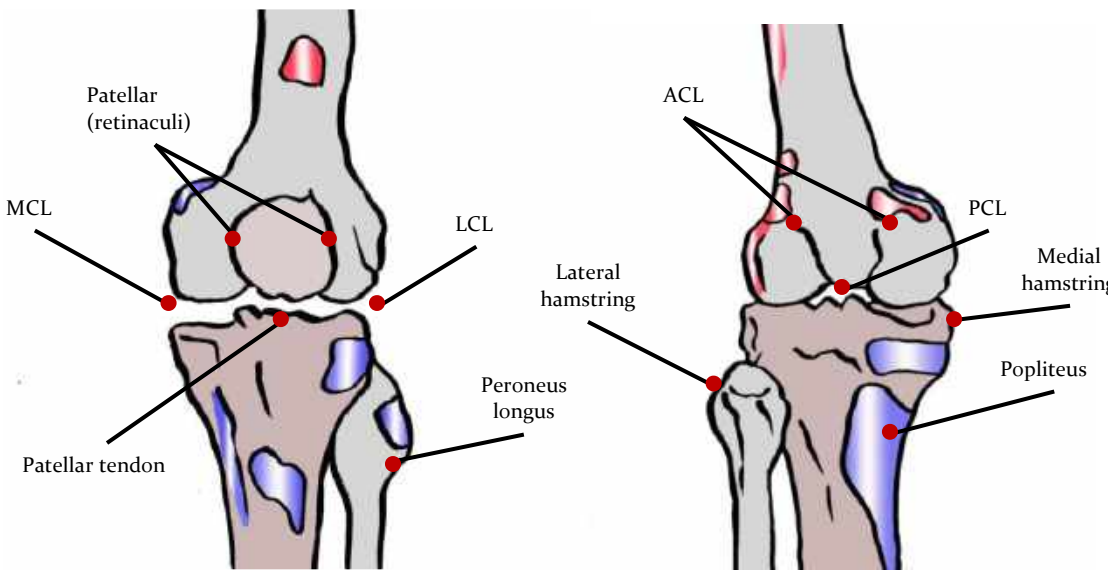
Similar to other tenderpoint (TP) procedures, the thigh has many TPs that are beneficial in the treatment of somatic dysfunction and are specific to knee, hip, and pelvic complaints. Anteriorly, the rectus femoris TP is located at its origin on the AIIS or the tendon overlying the femoral head and neck. The vastus lateralis TP is often confused with the iliotibial (IT) band tenderpoints as their location is similar. Here we point out that the IT band is more posterior, and that numerous tenderpoints can be located along its course. Likewise, the vastus lateralis has innumerable points in the belly of the muscle. The adductor brevis and adductor longus TPs lie in the belly of their accompanying muscles and are both treated with exaggerated adduction. Similarly, the vastus medialis TP lies in the belly of its named muscle.



We point out that the semitendinosus, semimembranosus, and biceps femoris have numerous accompanying TPs indicated by large "red TPs." Clinically, thigh muscles are inhibited following mechanical knee disruption and resultant interarticular effusion[9,28,29] termed arthrogenic muscle inhibition (AMI). Accompanying TPs arise as a result of the effusion and can persist even after the affected knee structures heal and the effusion resolves; at this point, a clinician can "clean-up" the neuro-inhibition caused by AMI that prevents the central nervous system from fully activating these muscles. More physiology is discussed in Chapter 11, the neurophysiology of somatic dysfunction.

Appendix B: Knee S/CS Tenderpoints

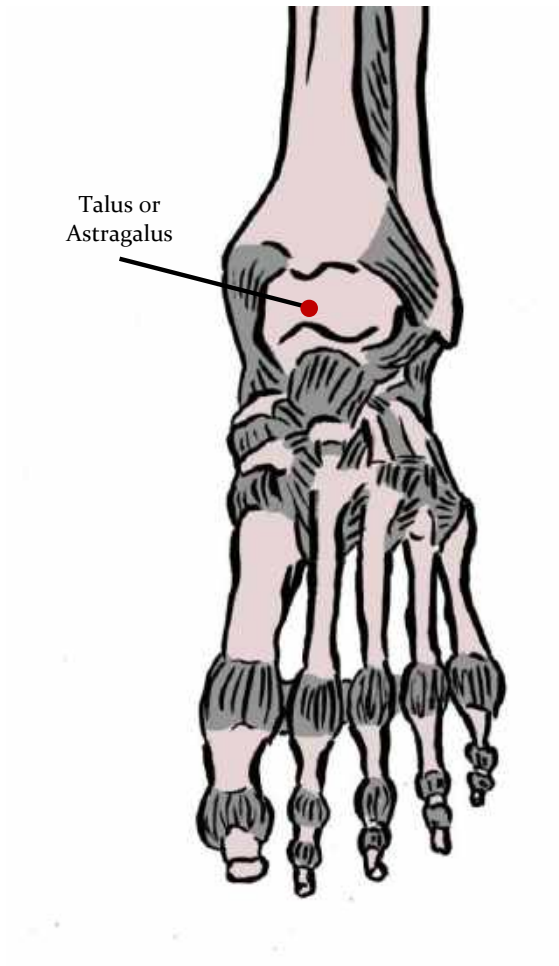
Clinically, authors JB and PJ view the knee in the traditional orthopedic model and test according to the structure and function of the supporting ligaments as detailed in this chapter. While we observe knee TPs in our patient population, we note that tenderness along the medial collateral ligament (MCL), lateral collateral ligament (LCL), anterior cruciate ligament (ACL), and the posterior cruciate ligaments (PCL) usually indicates their disruption, especially when accompanied with joint effusion: the tenderpoints of these ligaments are located along the course of the ligament itself. Likewise, patellar tendon tenderness usually indicates either trauma or misuse of the patellar complex, and we look above and below the joint for abnormal gait, muscle firing patterns, sacroiliac dysfunction, and loss of the foots' arches. Tenderness along the patellar retinacula or abnormal tracking of the patella across the femoral condyles, as in patellofemoral syndrome, usually indicates their disruption. This results in lateral patellar tracking and with chronic irritation, chondromalacia patella. Meyers points out that the MCL and LCL tenderpoints correlate with Jones' medial and lateral meniscus TPs respectively.[27(pp193-194)]

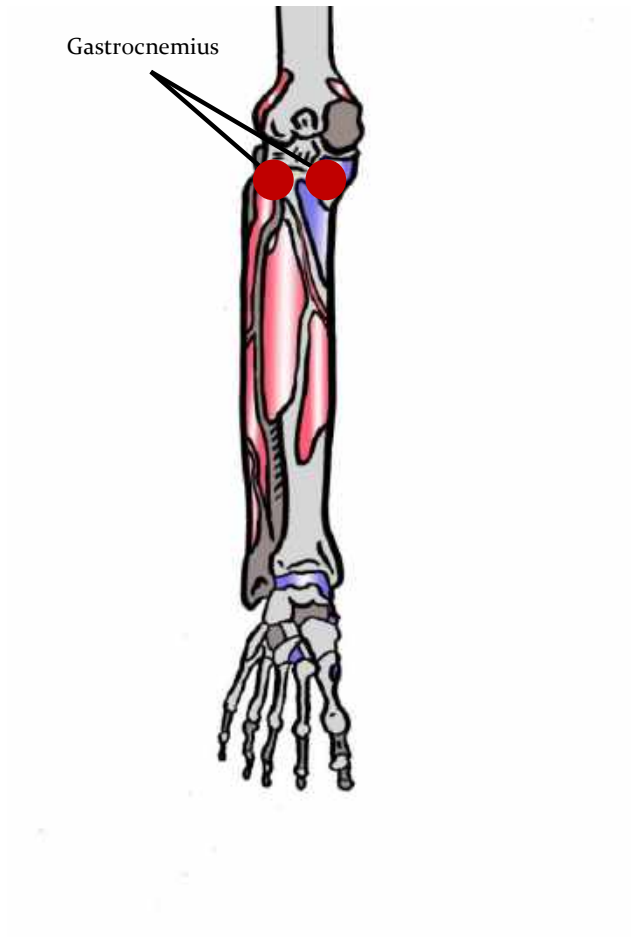


We do frequently observe clinically useful TPs in the peroneus longus, lateral hamstring, and medial hamstring as outlined in treatment examples. However, in our honest clinical assessment, they become more useful after surgery or prolotherapy have restored the ligamentous structure of the knee and residual pain and/or loss of function remain in the joint.

Appendix C: Foot & Ankle S/CS Tenderpoints

We find in this area that restoration of the supporting ligaments and arches usually resolves foot pain, and that many times trigger points and TPs indicate a "back-up" system for failed normal structure. We have included the tenderpoints (see treatment examples above) that we use regularly in clinical practice, but there are many more that Jones and Meyers describe in their texts.





Clinical Vignette:

CC: Knee pain

HxCC: A 16-year-old, white, female presents with bilateral knee pain for 2 years. She was evaluated at an orthopedic clinic for this same injury, underwent normal knee X-rays, and was ultimately diagnosed with patellofemoral syndrome. She was given quadriceps strengthening exercises that have not helped. She is a professional ballerina and notes her pain prevents her from dancing. She denies any injury and hasn't participated in formalized physical therapy or other treatment, besides periodic nonsteroidal anti-inflammatories that minimally alleviate her symptoms.

ROS:
Constitutional: no mental status changes, no travel or sick contacts
Eyes: no change in vision
Endocrine: no fever, night sweats, weight change
Cardiovascular: no chest pain or palpitations
Respiratory: no dyspnea, cough, or sputum
Gastrointestinal: no nausea, vomiting diarrhea; no change in BM, no blood in stool
Genitourinary: no change in urine, no dysuria, no blood in urine
Neurologic: no anesthesia, no paresthesia, no loss of consciousness, no seizures
Musculoskeletal: no weakness
Psychiatric: no homicidal or suicidal ideations, no signs of depression
Allergy: no angioedema, no urticaria, no anaphylaxis, no symptoms of seasonal allergies

PMHx/PSHx:
1. Asthma
2. Dislocation of her right shoulder after hitting the corner of a wall

Allergies:
-none

MEDs:
-none

Immunizations:
-Influenza 2018

Social:
-no smoking; no alcohol; no drugs; work = student

FamHx:
-mother = very flexible

VITALS = 110/80, 110#, 60bpm, 5'11', 98. 6F

GEN = awake, alert, oriented to time, place, situation, self, in no acute distress
HEENT = normal cephalic, a-traumatic, extraocular muscles intact, no otic/nasal discharge
NECK = supple, thyroid w/o masses
HEART = regular rate, no clicks/rubs/murmurs
LUNGS = clear to auscultation, without crackles, wheeze, or rhonchi
GI = soft, non-tender, no rebound tenderness, +bowel sounds x 4 quadrants, no hepato-splenomegaly
CV = no jugular venous distention, no carotid bruits, mucus membrane moist, no edema, 2/4 peripheral pulses, no lymphadenopathy
N/M = cranial nerves 2-12 intact, 5/5 global strength, 2/4 global reflexes, **patient has tremendous flexibility with 9/9 Beighton score (opposition of the thumb to the volar aspect of the thumb ipsilateral and bilaterally (b/l), passive dorsiflexion of fifth metacarpophalangeal >90° b/l, elbow extension 105°, bilateral knee hyperextension of >20°, can forward flex at the waist and place palms of hands on the floor without bending knees.** The Orthopedic knee exam was positive for patellar grind test b/l, but otherwise normal. Noted are bilaterally flattened medial longitudinal arches. Overall good muscle tone, lean habitus.
Manual medicine exam = pertinent exam findings include: negative spinal sweep, SitFBT, SFBT, stork, Trendelenburg, ASIS compression test. Standing exam reveals valgus deformity of bilateral knees.

Assessment/Plan:

1. Hypermobility Spectrum Disorder (formerly named Joint Hypermobility Syndrome)
2. Patellofemoral syndrome (PFS)
3. Fallen medial longitudinal arches bilateral

   →it is likely the patient's hypermobile joints are the underlying cause to her somatic complaints. Her inverted medical arches have likely predisposed her to PFS. Overall, she has the excellent muscle tone necessary to maintain joint stability, and will need to maintain her currently level of fitness lifelong to prevent further joint dislocation and soft tissue rheumatism.

   →refer for orthotics that she will likely need lifelong; this will reverse her valgus knee deformity and prevent further PFS.

Paul Johnson, DO

Review Questions:

1. Which anatomical structure crosses both the SI joint and hip joint?
    a. Gluteus maximus
    b. Iliopsoas
    c. Piriformis
    d. Obturator internus

2. The piriformis muscle arises from the___ and inserts on the ___?
    a. Posterior sacrum, lateral greater trochanter
    b. Posterior sacrum, inner superior portion of greater trochanter
    c. Anterior sacrum, lateral greater trochanter
    d. Anterior sacrum, inner superior portion of the greater trochanter

3. The Thomas test evaluates contracture of which muscle?
    a. Piriformis
    b. Gluteus medius
    c. Gluteus maximus
    d. Iliopsoas

4. The Scour (quadrant) test assesses?
    a. Iliopsoas hypertonicity/contracture
    b. Hip joint internal derangement
    c. Greater trochanteric bursitis
    d. Piriformis syndrome

5. Which physical exam maneuver is most accurate in detecting meniscal tears?
    a. McMurray's
    b. Childress
    c. Thessaly's
    d. Anterior drawer

6. Which physical exam maneuver identifies iliotibial band syndrome?
    a. Thomas test
    b. Thompson's test
    c. Scour (quadrant) test
    d. Ober's test

7. Which physical exam maneuver best identifies an ACL tear?
    a. McMurray's
    b. Valgus stress test
    c. Lachman's
    d. Anterior drawer

8. Which structure is responsible for the "screw home" mechanism of the knee?
    a. Adductor magnus

b. Iliotibial tendon
c. Patellar ligament (tendon)
d. Popliteus

9. Which three structures make of the Pes Anserinus (French for "duck feet")?
    a. ITB tendon, adductor magnus, and patellar ligament
    b. Patellar ligament, sartorius, and semitendinosus
    c. Sartorius, gracilis, and semitendinosus
    d. Gracilis, Sartorius, and semimembranosus

10. From anterior to posterior, in which order do the structures orientate in their insertion on the anterior-lateral knee?
    a. Peroneal nerve, biceps femoris, LCL, Iliotibial band
    b. Iliotibial tendon, LCL, peroneal nerve, semi-tendinosis
    c. LCL, ITB tendon, biceps femoris, peroneal nerve
    d. Iliotibial band, LCL, biceps femoris, peroneal nerve

11. The popliteus arises from the _____?
    a. Adductor tubercle to insert on the posterior medial tibia
    b. Gerdy's tubercle to insert on the posterior medial tibia
    c. Lateral femoral condyle (popliteal notch) superficial to the LCL to insert on the posterior medial tibia
    d. Lateral femoral condyle (popliteal notch) deep to the LCL to insert on the posterior medial tibia

12. The medial patellofemoral ligament and retinaculum attach to _____?
    a. Gerdy's tubercle
    b. Tibial tubercle
    c. Fibular head
    d. Adductor tubercle

13. The posterior drawer tests the integrity of which structure?
    a. ACL
    b. MCL
    c. LCL
    d. PCL

14. The anterior drawer test of the ankle tests the integrity of the _____?
    a. CFL
    b. Spring ligament
    c. Tibialis posterior tendon
    d. Peroneal tendon
    e. ATFL

15. The talar tilt test, tests the integrity of?
    a. ATFL

b. Deltoid ligament
c. Calcaneofibular ligament
d. Bifurcate ligament

16. The external rotation test of the ankle tests?
    a. Syndesmosis and ATFL
    b. ATFL and CFL
    c. Anterior tib-fib ligament and ATFL
    d. Anterior tib-fib ligament, posterior tib-fib ligament and syndesmosis

17. The Thompson test, tests for rupture of the _____?
    a. Achilles tendon
    b. ATFL
    c. Anterior tib-fib ligament
    d. Posterior tib-fib ligament

18. Which soft tissue structures form a sling or tensegrity structure to support the midfoot arch?
    a. Spring ligament and posterior tibialis
    b. Peroneus brevis and posterior tibialis
    c. Peroneus longus and posterior tibialis
    d. Spring ligament and peroneus longus

19. What is the most commonly injured ligament of the ankle?
    a. CFL
    b. Bifurcate ligament
    c. ATFL
    d. Anterior tib-fib ligament

Answers:

1. C
2. D
3. D
4. B
5. C
6. D
7. C
8. D
9. C
10. D
11. D
12. D
13. D
14. E
15. C
16. D
17. A
18. A
19. C

Chapter 9: Sequencing - Identifying Primary Somatic Dysfunction

Principles presented in this chapter:

- Identifying significant primary somatic dysfunction through the use of sequencing
- Tensegrity

Outline:

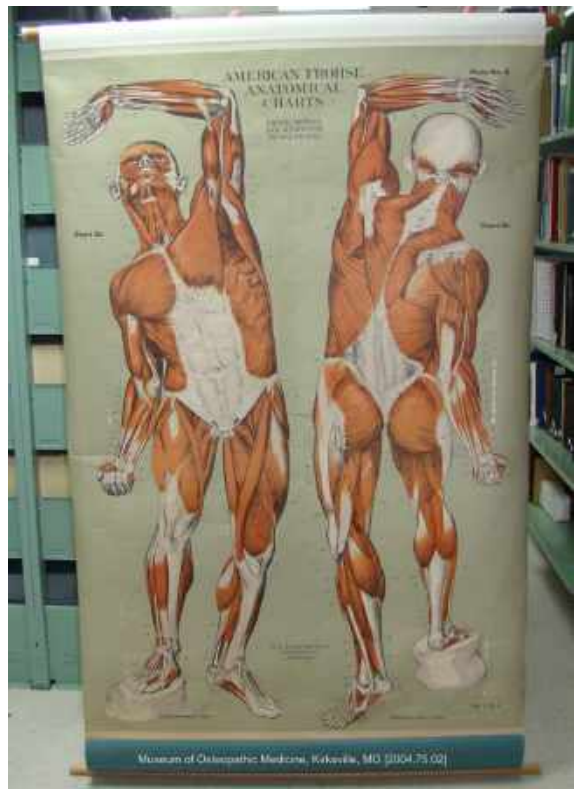
- Tensegrity and the Human Body
- Sequencing Method of Edward Stiles, DO
- Gait Analysis Sequencing
- Static Analysis Sequencing
- Sequencing Pearls
- Clinical vignette - sequencing
- Review Questions

Chapter 9: Sequencing - Identifying Primary Somatic Dysfunction

In this chapter we present sequencing methods for determining the optimal area to begin treatment. We have purposely suspended its discussion until this point in the text, awaiting presentation of the major body regions. As will be seen, sequencing allows a practitioner to put all of the major principles presented in previous chapters to practical application.

Sequencing is the term coined by Edward Stiles, DO, FAAO, to describe the methodology for finding areas of significant primary somatic dysfunction. In the process of determining where to start treatment, practitioners using dissimilar procedures focus on different aspects of somatic dysfunction (i.e., tissue texture changes, range of motion restriction, and areas of tenderness). Clinical findings often do not match the patient's pain complaints, and a practitioner must develop a methodology for examining patients. Stiles is often quoted saying "Don't chase [subjective] pain, pain is the biggest liar." His intent is to emphasize that the pain generator is often different from the location where the patient perceives pain.

Figure 1



From the Museum of Osteopathic Medicine, Kirksville, MO [2004.75.02]

Figure 1: American Frohse Anatomical Chart of the Muscular System, A.J. Nystrom and Co. Publishers; 1922.

Stiles frequently refers to a picture from the Museum of Osteopathic Medicine that displays the anatomical fascial patterns of the body (Figure 1) and remarks how a disruption in the shoulder could have implications in the low back or pelvis via the latissimus dorsi musculature and thoraco-lumbar fascia. The notion of the 'body as a functional unit' becomes clearer when sequencing, not only as a principle of how the body works, but also as a tool of problem solving. Studies relaying the importance of looking for and treating structures outside the patient's subjective painful areas, are found in a physical therapy study which shows that treatment of the thoracic spine improves cervical spine pain.[1] Another publication attests to the need to look above and below a painful joint for areas of pathology.[2]

Stiles noted that his three mentors, Perrin T. Wilson, DO, George Laughlin, DO, and Fred Mitchell, Sr., DO, all sequenced patients in their own way without formal verbalization and had excellent results. Stile's is considered one of the premier educators of manual medicine for two main reasons: first, he teaches principles, and second, he provides the method to apply them. One of his goals is to teach a reproducible method of finding and treating significant primary somatic dysfunction - sequencing. By teaching this method, Stiles gives voice to what experienced practitioners of manual medicine do naturally when they treat patients.

Tensegrity and the Human Body

While at the University of Pikeville, Kentucky College of Osteopathic Medicine (KYCOM), Stiles became interested in the work of Donald Ingber, MD, PhD, a Harvard Medical School Pathologist who applied an engineering concept called tensegrity to living tissues and the musculoskeletal system.[3] Tensegrity, short for tensional-integrity, refers to structural forms based on balanced tension and compression. Buckminster Fuller made the concept of tensegrity famous with his geodesic domes (Figure 2a).

Figure 2

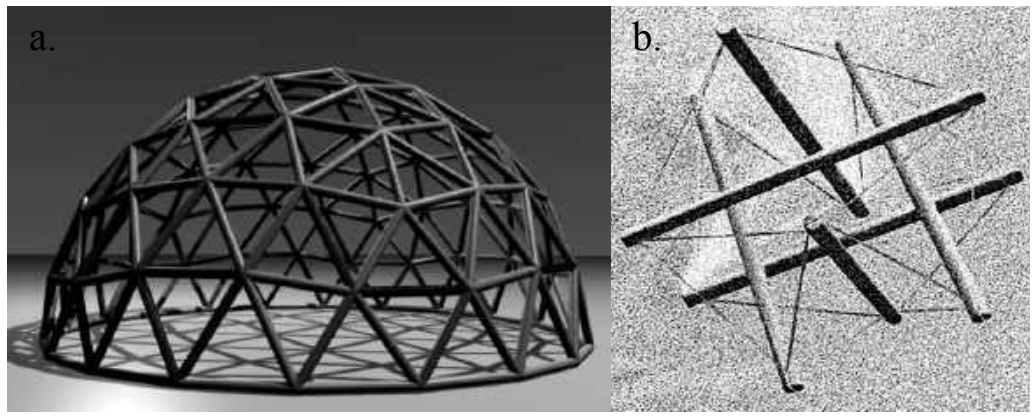


Figure 2: Tensegrity Model.

a. Geodesic dome
b. Tensegrity prism

The basic features of tensegrity include all of the following:[4(pp12-14)]

- A pre-stressed, continuous tension-compression system
- A self-balanced system without a central support column
- A system that is self-correcting

Figure 2b depicts a tensegrity structure in which the rods don't touch and are connected by continuous tension wires such that the structure is self-supporting without internal support. Ingber likens the rods of the structure to bones and the wires to muscle and fascia. He postulates that the 206 bones of our skeleton are held against the force of gravity and stabilized in a vertical form by the pull of muscles, tendons and ligaments.[3] In a tensegrity system, a distortion in one area produces excess load that is distributed equally to all other areas of the system - hence, due to the continuous tension system, it is self-correcting. At some point excessive distortion may be introduced, causing the whole tensegrity system to fail, similar to the "straw that broke the camel's back." It is at this breaking point where Stiles postulates that patients decompensate, perceive severe pain and are self-aware of biomechanical dysfunction.

Stiles further postulates that restoration of the body's tensegrity system is clinically important in normal joint function, interstitial fluid movement, and postural balance. Tendonitis, for example, often results from routine use of an abnormally functioning joint, not its overuse, and is an example of a failed tensegrity system. When clinically analyzing low back pain and its role in disc disease as an etiological factor, the concept of tensegrity forces us to ask the question: is the intervertebral disk a back-up system for a failed tensegrity system, or is the disk a primary spine stabilizer as conventionally thought? Likewise, Michael Kuchera, DO, FAAO, uses the tensegrity model as a method of explaining injury to a foot or ankle that results in the clinical symptoms of back pain or headaches.[5]

Stiles likens the treatment of multiple primary somatic dysfunctions to the layers of an onion: as one layer is removed another is uncovered. As the reader will discover with his sequencing method, since multiple areas will change as the system's resolved dysfunctional load is redistributed and tensegrity is restored, Stiles rescreens after successful treatment of a primary somatic dysfunction.

Ingber shows the importance of tensegrity on a cellular level.[3] He notes that mechanical dysfunction is etiological at a cellular level in a variety of diseases including many cancers. His research has proven that alterations in a cells shape, known as mechanotransduction, can ultimately determine a cell's genetic expression or type.[6] Thus, changes caused by somatic dysfunction have implications not only on the neuro-musculoskeletal system but on the cellular level. Based on the concept of mechanotransduction, as providers of manual medicine we therefore have the potential to impact multiple facets of patient health (Table 1).

Table 1 - Diseases of Mechanotransduction

Cardiology	Angina (vasospasm)	C T
	Atherosclerosis	T M
	Atrial fibrillation	M
	Heart failure	C T M?
	Hypertension	C T M?
	Intimal hyperplasia	C T M?
	Valve disease	T
Dermatology	Scleroderma	T
Gastroenterology	Achalasia	C
	Irritable bowel syndrome	C M?
	Volvulus	C T
Nephrology	Diabetic nephropathy	C T M?
	Glomerulosclerosis	C T M?
Neurology	Cerebral edema	T
	Facial tics	C
	Hydrocephalus	T C?
	Migraine	C M?
	Stroke	C T
	Stuttering	C
Oncology	Cancer	C T M?
	Metastasis	C
Opthalmology	Glaucoma	C T M?
Orthopedics	Ankylosing spondylitis	C T
	Carpal tunnel syndrome	C T
	Chronic back pain	C T
	Dupytren's contracture	C T
	Osteoporosis	T M
	Osteoarthritis	T
	Rheumatoid arthritis	T
Pediatrics	Collagenopathies	T
	Congenital deafness	C T M
	Mucopolysaccharidoses	T
	Musculodystrophies	C T M
	Osteochondroplasias	C T
	Polycystic kidney disease	T M
	Pulmonary hypertension of newborn	C T M?
Pulmonary medicine	ARDS	C T M
	Asthma	C T M?
	Emphysema	T
	Pulmonary fibrosis	T
	Pulmonary hypertension	C T M?
	Ventilator Injury	C M
Reproductive medicine	Pre-eclampsia	C T M?
	Sexual dysfunction (male & female)	C M?
Urology	Urinary frequency/incontinence	C M?

A partial list of diseases that share the feature that their etiology or clinical presentation results from abnormal mechanotransduction. The right column indicates whether the mechanical basis of the disease or condition is likely due to changes in cell mechanics (C), alterations in tissue structure (T), or deregulation of mechanochemical conversion (M); '?' indicates situations where deregulation of mechanochemical conversion is likely but remains to be demonstrated.

The Sequencing Method of Edward Stiles, DO

Stiles' sequencing method utilizes "type 3 spinal motion" using a rigid-straight-thumb, as described in the thoracic spine chapter. The simultaneous introduction of motion to multiple spinal joints in three directions reduces the individual components of flexion/extension, sidebending, and rotation, as Nelson discussed,[8(pp44)] but allows for efficient motion testing over a broad area of the spine. He begins screening a standing or sitting patient at the base of the skull and works inferiorly to the sacrum, repeating this process on the contralateral side.

Figure 3

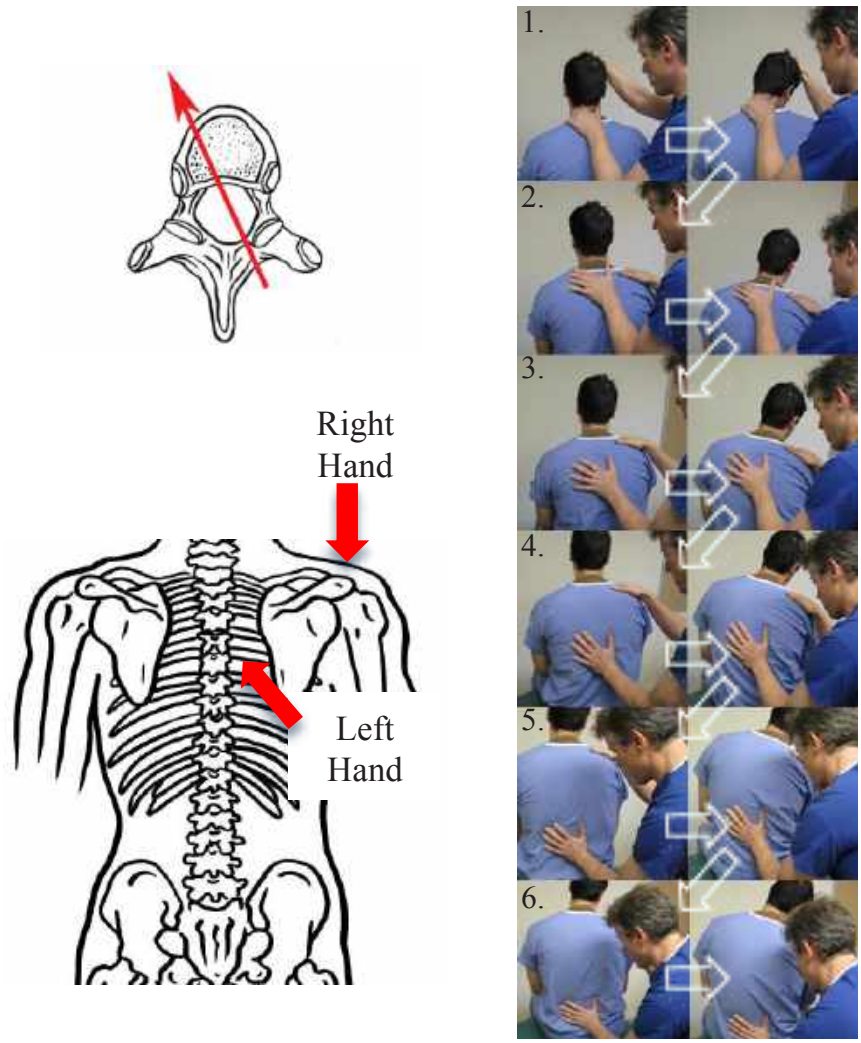


Figure 3: The Spinal Sweep

Spinal segments are examined beginning at the cervical spine and ending in the lumbar spine. In each area, the examiner introduces extension while simultaneously introducing sidebending, and rotation using a diagonal vector (with the thumb). Once completed, this process is repeated on the opposite side.

Notice that the operator's left thumb is testing the right facets in this instance.

Note the whole thumb held in a rigid-straight position is used in the spinal sweep.

We have designated this exam the "spinal sweep" as seen in Figure 3. THIS PROCESS IS MORE FULLY DESCRIBED IN THE THORACIC SPINE CHAPTER; PLEASE REVIEW BEFORE PROCEEDING.

Figure 4: Map of the AGR

Cranial – a tight band is palpated in the nuchal area.

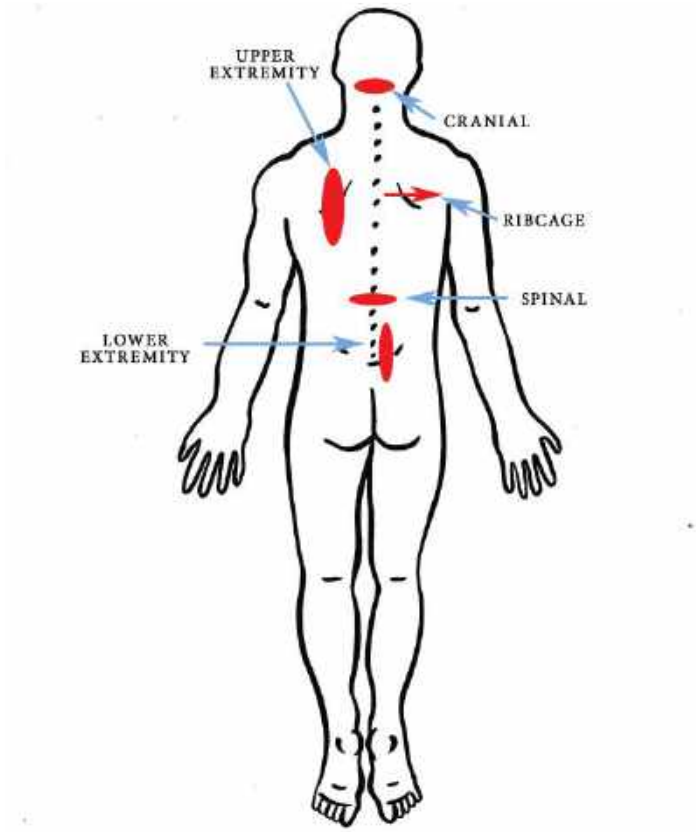
Upper Extremity – a tight band is palpated along the medial border of the scapula.

Ribcage – an increasingly tight band is observed as you palpate medial to lateral along the posterior aspect of the spine.

Spinal – there is symmetrical tightness on the right and left of the spine.

Lower Extremity – there is a unilateral, paravertebral tightness of the lumbar spine, similar to that found in upper extremities.

Figure 4



Gross assessment using the spinal sweep does not diagnose somatic dysfunction, but it allows Stiles to localize regions where significant primary somatic dysfunction is most likely present; he names these regions the area of greatest restriction (AGR). Once the AGR is identified, Stiles determines the cause for the restriction using the diagnostic methods taught in previous chapters. Figure 4 depicts the major regions taught in this book, leaving out the sacrum and pelvis. Note that after completing the initial spinal sweep, Stiles also performs seated (SitFBT) and standing forward bending tests (SFBT). A flow chart summarizing Stiles' method of sequencing is listed in Figure 5 and in steps 1 thru 6 below:

1. Apply the "spinal sweep" to screen for the most restricted area (Figure 4). Test the SitFBT and the SFBT as described in the sacrum and pelvis chapter.
2. Determine if the AGR is in the upper or lower half of the body (i.e., above or below T12).
3. Determine the AGR within the upper or lower half. Diagnose the primary somatic dysfunction within the AGR.
4. Treat the primary somatic dysfunction in the AGR
5. Begin at step 1 and rescreen (i.e., peel away the next layer of the onion). Treat the area with the next greatest restriction.
6. Repeat until the area of greatest restriction resists correction.

Figure 5

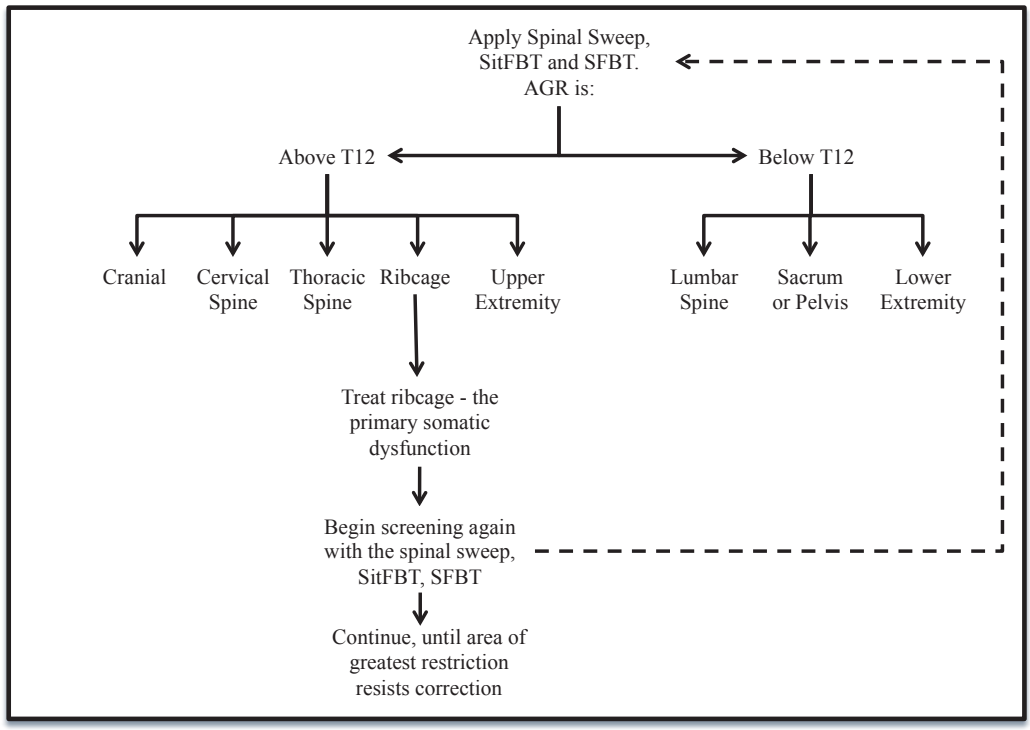


Figure 5: Flow Chart of Stiles' Sequencing Method

In this instance the ribcage is the primary somatic dysfunction. After ribcage treatment, screening is resumed at the beginning.

Upper Half of the Body

Cranial: In the cervical spine, the upper cervical spine will often present with restrictions due to cranial restriction; cranial restrictions could also manifest due to C0-C3 restrictions as the dura attaches to C0, C2 and C3. During the spinal sweep, to test for dural restriction, author JB grasps both mastoid processes with one hand and then adds medial-to-lateral translatory force along the sagittal suture/falx cerebri with the other. If treatment of other areas is yielding poor results, consider cranial somatic dysfunction.

Cervical Spine: Individual areas of TART are first noted with the patient standing or sitting. For better individual testing, place the patient supine and test individual segments with passive ROM testing as in the cervical spine chapter. Upper cervical dysfunction often presents with a horizontal band of contracting tightness over C1-2; in this instance look for atlanto-occipital or atlanto-axial dysfunction. Somatic dysfunction of the lower cervical spine C2-7 is found with segmental testing.

Thoracic Region: If the AGR is believed to be in the thoracic region, there are three treatment considerations: vertebra, ribs or upper extremity. The practitioner must determine which level is most restricted; is it worse palpating laterally or is there a horizontal or vertical band of restriction?

- Thorax: midline restriction of the thorax that decreases with lateral palpation indicates somatic dysfunction of the thoracic spine. To determine which segment is restricted, the practitioner asks the patient to test each set of vertebrae in the AGR, moving through flexion, extension, and neutral.
- Ribcage: a restriction found in the thorax that increases with lateral palpation indicates somatic dysfunction of the ribcage. Note - the AGR found on gross motion testing of the ribs doesn't necessarily correspond to restriction at that rib level. To determine the key rib, the practitioner should place the patient supine and perform individual testing of the ribcage in static and dynamic phases. Ideally, begin by treating out-of-pattern ribs (inhaled right ribs or exhaled left ribs) first. Anecdotally, in patients with back pain, Stiles implicates the ribcage as the AGR ~60% of the time. This makes good sense as the ribcage has connections to the pelvis through the quadratus lumborum, rectus abdominus, diaphragm, and thoraco-lumbar fascia.
- Upper Extremity: a vertical band of restriction along the medial border of the scapula will lead toward the diagnosis of upper extremity somatic dysfunction. Additional information is obtained by using the scapular-clavicle motion test. From this point, a practitioner would need to test the carpal bones, forearm, hand, elbow, acromioclavicular (AC) joint, and sternoclavicular (SC) joint, etc. to pinpoint precise dysfunction.

Lower Half of the Body

Lumbar Region: central and symmetrical para-vertebral muscle tightness in the lumbar region indicates lumbar somatic dysfunction – the detection and diagnosis are identical to the thoracic spine. Check individual segments to diagnose segmental somatic dysfunction.

Lower Extremities: diffuse, unilateral, para-vertebral tightness, like that found in the upper extremities, is a clue for lower extremity somatic dysfunction. This occurs due to the iliopsoas/quadratus lumborum anatomical connection from the lower extremities to the lumbar spine. The key lesion is usually found in the ipsilateral leg or thigh by testing fibular heads, tibial rotation, ankle, and feet as described in the lower extremity chapter.

Pelvis and Sacrum: somatic dysfunction is detected by the stork test, ASIS compression test, the SFBT, and the SitFBT. Many times there will be several simultaneous pelvic dysfunctions. Based off of pelvic axes, treat in the following order:

1. Innominate Shear: since the superior, middle, and inferior transverse axes, and oblique axes as are all disrupted with an innominate shear, begin by treating shears.

2. Pubic Shear: treat pubic shears prior to sacral somatic dysfunction since a pubic shear may cause sacral dysfunctions: Stiles considers the sacrum "the great adaptor." Further, the pubic axis is integral for normal gait.
3. Sacral Dysfunctions: treat prior to innominate rotations since posterior sacral torsions and sacral extensions disrupt normal gait.
4. Innominate Rotations: treat last as these are commonly compensatory to leg length inequalities and sacral somatic dysfunction.

Gait Analysis Sequencing

Often in acute musculoskeletal injuries, the patient's subjective pain is easily identifiable as the source of injury. However, after two to three months an acute, untreated dysfunction becomes chronic, and the body will create neurologically mediated measures (i.e., neuromuscular facilitation and secondary somatic dysfunction) to protect the injured area. These areas often lack movement, and pathological hypo/hyper-mobility will occur around, or in conjunction with, the primary hypo-mobile somatic dysfunction. Gait analysis is especially useful for identifying these chronic primary somatic dysfunctions.

In gait analysis the concept is simple; the patient walks away-from and then toward the practitioner who observes for motion restriction (Figure 6). Areas of restriction impede movement and are visually apparent by their inherent lack of normal motion: diminished arm swing, impaired thoracic rotation, abnormal toe off-heel strike, and abnormal head carry. Initially, the practitioner will watch the patient walk using peripheral vision (i.e., without a central tracking focus); this method allows for easier identification of the immobile-restricted area. The patient repeats the same short walk away-from and toward the practitioner who now observes with central tracking to confirm if indeed this area lacks motion. Final confirmation is made when this area is tested for TART criteria. To perfect this method, knowledge of normal human gait is essential, but is beyond the scope of this text.

Figure 6: Gait Analysis Sequencing

The red circle signifies primary somatic dysfunction in the thoracic area, around which the body compensates.

Figure 6

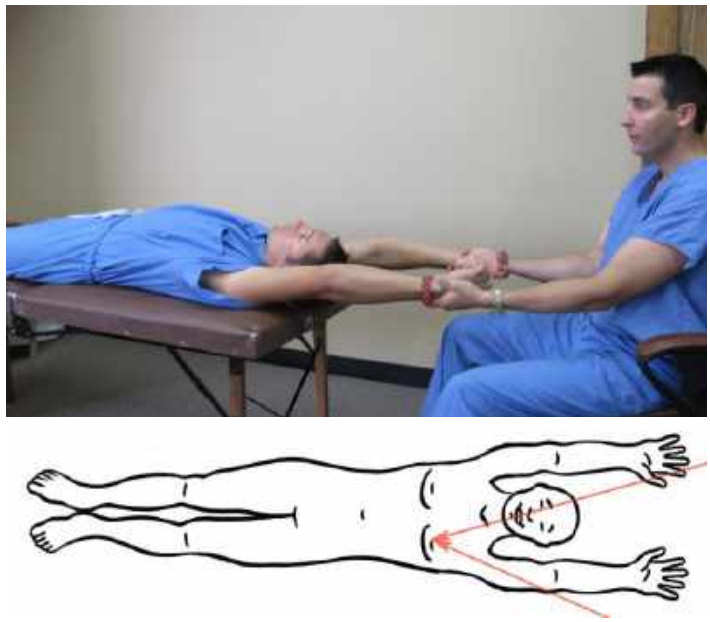
Static Analysis Sequencing

To determine the AGR, static analysis is typically performed on a supine patient. The patient's upper extremities are flexed 180° above their head, and the practitioner applies and holds distraction to them; somatic dysfunction is present if a directional pull of the fascia occurs (Figure 7).

Figure 7: Static Analysis Sequencing

The patient's arms are distracted with symmetrical force and held. An asymmetrical directional pull of the fascia indicates the presence of somatic dysfunction.

Figure 7



Likewise, the operator can apply distraction to the lower extremities to localize the regions of somatic dysfunction. This idea is akin to those aforementioned; identify the area where the body compensates. This method is particularly useful for diagnosing fascial somatic dysfunction.

Sequencing Pearls

The following sequencing pearls are derived from Stiles' teachings and apply to any technique used for sequencing:

- Stiles commented that when the AGR is located and precise diagnosis is treated, the body will "beg to be treated" and respond easily.
- Landmarks will not match the diagnosis when treating out of sequence (i.e., all physical findings of a unilateral shear will not be found). In other words, the practitioner will find odd patterns and uncommon diagnoses.
- If treating out of sequence, treatment will be like "swimming in peanut butter" When you treat in the AGR, ~20% of the body's dysfunctions change. When treating out of sequence, only that specific area changes.

References

1. Cleland, Joshua A., Maj John D. Childs, Meghann McRae, Jessica A. Palmer, and Thomas Stowell. "Immediate effects of thoracic manipulation in patients with neck pain: a randomized clinical trial." *Manual therapy* 10, no. 2 (2005): 127-135.
2. Wainner, Robert S., Julie M. Whitman, Joshua A. Cleland, and Timothy W. Flynn. "Regional interdependence: a musculoskeletal examination model whose time has come." *Journal of Orthopaedic & Sports Physical Therapy* 37, no. 11 (2007): 658-660.
3. Ingber, Donald E. "The Architecture of Life." Sci Am Scientific American 278, no. 1 (1998): 48-57. doi:10.1038/scientificamerican0198-48.
4. Pfluger, C., "The Meaning of Tensegrity Principles for Osteopathic Medicine." Master 's thesis for MS in osteopathy submitted at the Donau University, Krems and the Vienna School of Osteopathy, 2008, published online (www. osteopathicresearch.com/paper_pdf/ Pflueger.pdf), pp. 1-129.
5. Kuchera, M. L., and A. G. Chila. "Postural considerations in osteopathic diagnosis and treatment." *Foundations of Osteopathic Medicine. 3rd ed. Baltimore, MD: Lippincott Williams & Wilkins* (2011): 437-483.
6. Ainsworth, Claire. "Stretching the imagination." *Nature* 456, no. 7223 (2008): 696-699.
7. Ingber, Donald. "Mechanobiology and Diseases of Mechanotransduction." Annals of Medicine 35, no. 8 (2003): 564-77. doi:10.1080/07853890310016333.
8. Giusti, Rebecca, DO. *Glossary of Osteopathic Terminology*. Third ed. Chevy Chase, MD: American Association of Colleges of Osteopathic Medicine, 2017.

Clinical Vignette

Patient Name: CR
June 14, 2020

CC: new patient visit, right upper extremity (RUE) pain

HxCC: Patient is a 35-year-old female who presents as a consult from her primary care physician for RUE pain that began in the second week of August. She notes the pain in her arm waxes and wanes and feels like a tourniquet. The pain ranges from her shoulder to her elbow and is rated a 4-5/10. She denies trauma to this area. Ultrasound to rule-out deep vein thrombosis of her RUE was negative. Tramadol does help. She has no history of connective tissue disease, although her father has sarcoidosis and essentially a flaccid RUE as a neurological complication.

ROS:
Constitutional: no mental status changes, no travel or sick contacts
Eyes: no change in vision
Endocrine: no fever, night sweats, weight change
Cardiovascular: no chest pain or palpitations
Respiratory: no dyspnea, cough, or sputum
Gastrointestinal: no nausea, vomiting diarrhea; no change in BM, no blood in stool
Genitourinary: no change in urine, no dysuria, no blood in urine
Neurologic: no anesthesia, no paresthesia, no loss of consciousness, no seizures
Musculoskeletal: no weakness
Psychiatric: no homicidal or suicidal ideations, no signs of depression
Allergy: no angioedema, no urticaria, no anaphylaxis, no symptoms of seasonal allergies

PMHx/PSHx:
1. Iron deficiency with heavy menses
2. Vaginal birth without problems
3. Sickle cell trait

Allergies:
-no known drug or food allergies

MEDs:
-Tramadol 50mg every 6hrs
-Vitamin D 2000IU daily
-Ferrous Sulfate 325mg, 2 pills daily

Immunizations:
-per her primary care physician

Social:
-no smoking; no alcohol; no drugs; work = airline stewardess

FamHx:
Dad = Sickle cell-trait; sarcoid; unable to move his RUE

VITALS = 5'6", 128#, 100/64, 100 beats per second
GEN = awake, alert, oriented to time, place, situation, self, in no acute distress
HEENT = normal cephalic, a-traumatic, extraocular muscles intact, no otic/nasal discharge
NECK = supple, thyroid w/o masses
HEART = regular rate, no clicks/rubs/murmurs
LUNGS = clear to auscultation, without crackles, wheeze, or ronchi
GI = soft, non-tender, no rebound tenderness, +bowel sounds x 4 quadrants, no hepato-spleenomegaly
CV = no jugular venous distention, no carotid bruits, mucus membrane moist, no edema, 2/4 peripheral pulses, no lymphadenopathy
N/M = CN 2-12 intact, 5/5 global strength **except in her left-forearm extension and left arm abduction, 4/5 in her hip flexion and leg flexion/extension**, 3/4 global reflexes with **4/4 in her L-achilles and L-patellar, cerebellum intact, sensation symmetric, gait normal**.
Biomechanical = abnormal findings include **restricted ribs 1-4 on the right which are inhaled. Gait sequencing analysis reveals no motion in her right shoulder with diminished arm swing phase of gait.** Negative Roos test, negative Spurling compression test, and negative Adson's test.

Assessment/Plan

1. RUE Pain
2. Arthralgia with weakness of her left side
3. Neuralgia
4. Heavy Menses with iron deficiency
   -OMM deferred today due to gross muscle weakness. Note her pain is in her RUE, yet her weakness is in her L-side with left sided hyperreflexia. The differential diagnosis for this includes iron deficiency, hyper/hypothyroid, neuro-sarcoidosis, HIV, Hepatitis B/C, syphilis, bone tumor, cervical stenosis, prior stroke.
   -I will order a right shoulder X-ray, ACE level, complete blood count with differential, comprehensive metabolic panel, rapid plasma reagin, HIV Elisa, Hepatitis B/C panel, ferritin, transferrin, iron, TSH. She also may benefit from imaging of central nervous systems structures such as brain or cervical spine to evaluate for injury, as she is both weak and hyper-reflexive on her L-side.
   -follow up after testing is complete
5. Somatic Dysfunction
   a. Ribs – R-ribs 1-4 inhaled, NO Treatment done.
   b. Thorax – T1 L-rotated, NO Treatment done

Jason Browder, DO

Review Questions

1. While evaluating a patient using Stiles' approach, a taught band is felt across the medial border of the right scapula. This would indicate:
    a. The AGR is in the ribs
    b. The AGR is in the right upper extremity
    c. The AGR is in the cervical spine
    d. The AGR is in the thoracic spine

2. When sequencing for the AGR using Stiles' approach, a practitioner finds that the restriction is worse when she palpates lateral to the thoracic spine at the level of T9 on the right. The key lesion is most likely:
    a. Thoracic spine
    b. Right ribs
    c. Right upper extremity
    d. Right hemi-diaphragm

3. Using the "spinal sweep," a practitioner is using their right hand to introduce sidebending, rotation and extension using the patient's right shoulder. The correct hand placement of a practitioner's left thumb should be:
    a. Overlying the right facets
    b. Overlying the left facets
    c. Overlying the either the left or right facets

4. While sequencing a patient using Stiles' method, the practitioner notes a posterior right sacral base. Treatment of the right sacral extension does not reduce the sacral base. Why?
    a. There is another primary dysfunction
    b. The diagnosis was not correct
    c. The treatment procedure was unsuccessful despite a correct diagnosis
    d. All of the above are possibilities

5. While examining a patient, a practitioner diagnoses the key lesion as a right innominate upshear. The patient complains that her pain is in the lumbar spine, and that there is no way this is possible. What should the practitioner do?
    a. Politely tell the patient that her pain is a result of a primary problem of the pelvis and treat the pelvis.
    b. Politely tell the patient that her pain is a result of a primary problem of the pelvis and treat the pelvis, and then give attention to the lumbar spine to see if this area changes or the patient's perception of pain changes.
    c. Politely tell the patient that her pain is a result of a primary problem of the lumbar spine and focus treatment there.

Answers:

1. B
2. B
3. A
4. D
5. B

Chapter 10: Cranial Manual Medicine

Principles Taught in this Chapter:

- Evidence in Support of Cranial Motion
- Primary Respiratory Mechanism
- Physiologic Cranial Motion
- Diagnosis of Cranial Somatic Dysfunction
- Treatment using Indirect and Direct Cranial Methods

Outline:

- Evidence Supportive of Cranial Motion
- Anatomy of the Head
- Diagnosis of Cranial Somatic Dysfunction
    - Sphenobasilar Synchondrosis (SBS) Somatic Dysfunction
- Treatment of Sphenobasilar Somatic Dysfunction
- Additional Cranial Diagnoses and Treatment
- Treatment of Cranial Somatic Dysfunction other than SBS Dysfunction
- Cranial Treatment Pearls
- Further Considerations in Cranial Manipulation
- Clinical Vignette
- Appendix A: Strain/Counterstrain Tenderpoints for the Head
- Review Questions

Chapter 10: Cranial Manual Medicine

Manual medicine/therapy (MM/T) involving the cranium was first introduced by William Garner Sutherland, DO, (1873-1954, Figure 1) in 1939. In acknowledment of the general subtle nature of cranial manipulation, we first review the supportive concepts and research and then present the anatomy and MM/T procedures. Initially, Sutherland published *The Cranial Bowl*,[1] and then his transcribed lectures were published in *Teaching in the Science of Osteopathy*.[2]

During Sutherland's time, skull sutures were thought to fuse solidly after adolescense. He dedicated 30 years to the development of his concept of cranial anatomy, cranial bone movement, and palpatory diagnosis of head.

Figure 1

Figure 1: Depiction of William Garner Sutherland, DO

Cranial manipulation originated with the work of Sutherland.

Sutherland perceived a rhythmic motion of the head, separate from diaphragmatic and cardiac rhythms, that was perceptibly present through out the body. He named it the primary respiratory mechanism (PRM): "primary" to describe its presence at the cellular or smallest levels, "respiration" to signify its impact on cellular metabolism, and "mechanism" to emphasize its ubiquitousness throughout the body. The PRM is often incorrectly referred to as the cranial rhythmic impulse (CRI), a phrase related to psychiatric patients that was previously coined by James Woods, DO, and Rachel Woods, DO,[3] that has come into popular use.

Sutherland summarized the PRM as five key phenomena.[4]

1. The inherent rhythmic motion of the brain and spinal cord
2. Fluctuating cerebrospinal fluid (CSF)
3. Mobility of the intracranial and spinal membranes (meninges, dura, etc.)
4. Articular mobility of the cranial bones
5. Involuntary mobility of the sacrum between the ilia

In this chapter, we evaluate the Sutherland phenomena and research from the past four decades that support the concept and application of cranial manipulation. We then describe and illustrate the application of cranial manipulation in the diagnosis and treatment/therapy for cranial somatic dysfunction and cranial bone derangement.

Evidence Supportive of Cranial Motion

Cranial manipulation remains a controversial subject in some quarters of the medical-scientific community. Some anatomists, physicians and manual therapists believe that the skull is fused after adolescence and incapable of motion, a notion that seems to preclude further consideration of cranial manipulation.

Do the bones of the cranium have mobility? Several studies have demonstrated objective measurements of cranial bone motion. Some of the studies are cited in bullet format below:

- The maxillary arch was measured to move an average of 1.5mm in a single individual.[5]
- Magnetic resonance tomography and serial roentograms demonstrated 0.388mm changes in intracranial dimension between AP dimensions.[6]
- NASA used ultrasound to measure intracranial distance resulting from variations in arterial pressure.[7] In a later study, tilting a person with their head down 70° from upright-vertical posture changed the internal diameter of the skull 100-200μm.[8]
- MRI images show calvarial area changes over a few minutes.[9]

Does evidence exist to suggest a change in cranial bone position and the clinical efficacy of cranial manipulation? One retrospective study of twelve patients showed differences in cranial landmarks pre- and post-treatment using a fixed positioning device for the radiographs; eleven of the twelve subjects had radiographic change post-treatment at three or more sites.[10] While inconclusive, a systematic review of the clinical application of cranial manipulation showed some positive results.[11] A subsequent response to the Jäkel and von Hauenschild review[11] suggests there is a growing body of evidence supportive of the clinical benefit of cranial manipulation.[12]

What is the driving force(s) of the PRM? To date no one knows definitively, but multiple theories have been proposed. Chaitow[13] summarized potential drivers of the PRM as follows:

1. Intrinsic motility and mobility of the brain and nervous system
2. CSF production through arachnoid granulations
3. Muscles as a motive force
4. Lymphatic contractions
5. Tissue pressure
6. Vasomotion
7. Entrainment
8. Traube-Hering Oscillation

We now take these possible mechanisms in order and evaluate their likelihood.

1. Sutherland's theory of the cranial concept proposes an inherent motion throughout the body as part of the PRM, but to date there is no research to confirm this. It seems unlikely that the brain parenchyma alone has sufficient contractile force to propel cranial bone motion. But if demonstrated, the addition of vascular forces affecting cranial fluid volume in some harmonic fashion could provide a viable explanation for cranial bone motion.[14]

2. CSF production through the arachnoid granulations was proposed by Upledger.[15] Upledger's "pressure-stat" model proposes that CSF production and resorption create a gradient resulting in fluid movement and ultimately a pump. However, too little CSF is produced per minute to account for the necessary gradient required to move the dura mater, much less the whole body.

3. Besides the heart and diaphragm, no muscle is known that causes a rhythmic, whole body contraction. Further, skeletal muscle is not known to contract in a rhythmic method. The PRM is observable in patients with spinal cord injuries and paralysis, making this a less likely phenomenon.

4. Degenhardt and Kuchera[16] proposed that lymphatic contractions potentially explained the PRM. However, the lymphatics do not contract at any of the rates suggested in the PRM literature. The flow of fluid into and through the lymphatics could produce a gradient as palpable as the PRM; however, since lymphatics are found only in the dural membranes of the brain, this phenomenon is not likely to be the cause of the body wide motion palpable in the PRM. In fact, interstitial fluid movement through the lymphatics may be a result of and not the primary mover of the PRM.

5. The tissue pressure model suggested by Norton[17] proposed that activation of cutaneous mechanoreceptors of the patient and practitioner, combined with respiratory and cardiovascular rhythms, create the PRM. The interactions of all

three are thought to harmonize into a frequency, i.e., the PRM. While a promising theory, Norton's apparent attempt to prove this hypothesis with a computer model was unsuccessful.

6. Vasomotion is an oscillation of vascular tone that is generated from within the vascular wall and is not a consequence of heartbeat, respiration, or central nervous input.[18] Vasomotion is a local phenomenon that may occur in any portion of the body. However, vasomotion is thought to be the result of local tissue ischemia rather than a whole body phenomenon.

7. Entrainment, as proposed by McPartland and Mein,[19] holds that all organisms pulsate with myriad electrical and mechanical rhythms. Many of these rhythms emanate from synchronized pulsating cells (i.e., pacemaker cells, cortical neurons). They assert that the PRM is a harmonic frequency that incorporates the rhythms of multiple biological oscillators. It is derived primarily from signals between the sympathetic and parasympathetic nervous systems. So far, though it is often cited as a possible mechanism for the PRM phenomena, there has been no research to substantiate this theory.

8. Another theory has linked the Traube-Hering phenomenon to the PRM.[20] In 1865 using anesthetized animals, Traube first identified cyclical fluctuations of pulse pressure that were independent of heart rate and respiration.[21] In 1869 using similar experiments, Hering independently confirmed this cyclical pressure change.[22] Pulse-pressure is the difference between systolic and diastolic blood pressures. Traube-Hering oscillations, as they are known today, are caused by physiologically normal cyclical changes in pulse-pressure. They have the following characteristics:

Pulse-pressure: the difference between the systolic and diastolic pressure.

PRM has been reported to vary from 5 to 14 cycles per minute as reported in various studies.

- rates of ~ 6 cycles per minutes (CPM)
- independent from respiration or heart rate
- under central nervous system (CNS) control
- occur systemically (as opposed to vasomotion)
- do NOT occur in perfect cycles as seen in sinusoidal waves

We suggest that the above eight theories, though not necessarily exhaustive, do provide evidence in support of Sutherland's five phenomena of the PRM. Hopefully, neuroscience research imagery technology will provide a further and more definitive explication of PRM phenomena.

Nils Lundberg Waves:
A-wave = pathological waves observed with intracranial pressure increases.
B-wave = Mayer oscillations
C-wave = Traube-Hering oscillations

In the 1960s, the physiologists Ingvar and Lundberg identified Traube-Hering oscillations intracranially.[23,24] At that time, Lundberg identified three distinct intracranial oscillations that he named A, B, and C waves. A-waves are indicators of pathology and are seen with intracranial pressures of 50-60 mmHg (normal intracranial pressures in adults are 7-15 mmHg). B-waves are the result of

respiratory variations and occur at a rate of one to two CPM. B-waves have been linked to Mayer oscillations[25] that Osteopathic physicians such as Roland Becker have termed the "slow tide."[26] Lundberg immediately recognized C-waves as Traube-Hering waves that have been consistently recorded at a rate of 4-8 CPM.

In 2001 Nelson et al[20] published a single-blinded study evaluating the Traube-Hering oscillation. The purpose of the study was to determine if the Traube-Hering oscillation is synchronous with the PRM. Results demonstrated that the Traube-Hering oscillation is likely a phenomenon related to the PRM.

Although this study has not been repeated, the results are compelling. The Traube-Hering phenomenon is a body-wide phenomenon under CNS control that occurs at a rate of 6-10 CPM, is independent of heart rate and respiration, and is well studied in multiple disciplines of medicine. Later studies[27] suggest that manual medicine influences the Traube-Hering oscillation, demonstrating that manual medicine influences CNS activity.

Neurophysiology research suggests that CSF is secreted by the choroid plexus and reabsorbed by arachnoid granulations that ultimately allow it to reenter the dural venous sinuses. Recent research suggests that CSF is absorbed not only by arachnoid granulations, but also by lymphatic channels associated with nerve roots exiting the spinal cord and brain.[28,29] Further consideration has been given to CSF absorption through brain capillaries.[30]

Fluctuations in pulse-pressure create ebb and flow, promoting interstitial and CSF movement into arachnoid granulations, lymphatic channels, and possibly capillaries. The Traube-Hering oscillation likewise promotes fluid movement outside of the CNS between cells in soft tissues such as the arms, legs, and the visceral structures. During exercise, it is unlikely that Traube-Hering oscillation plays a significant role in interstitial fluid movement; at rest however, its importance in cellular nutrition and waste appears to be an essential physiological function.

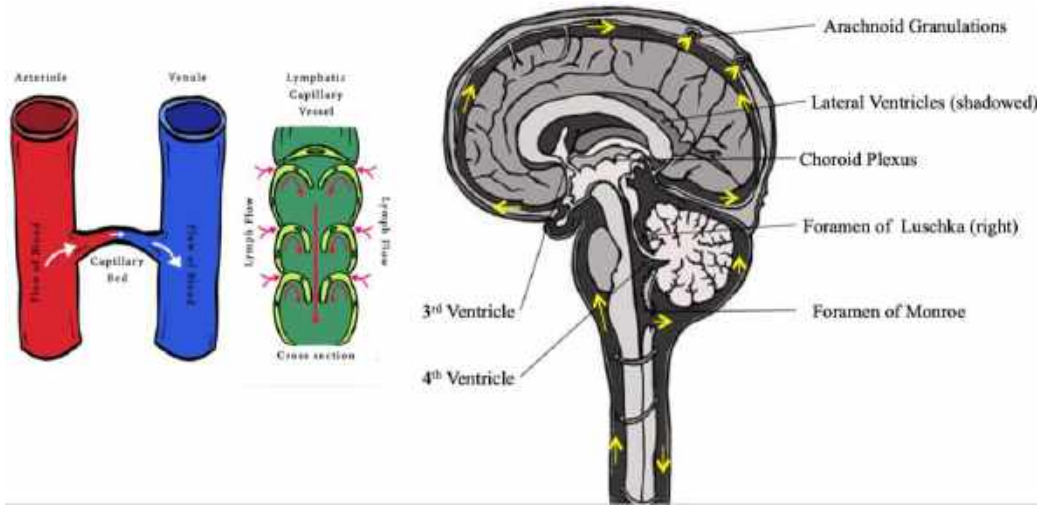
What is the purpose of the Traube-Hering oscillation? The Traube-Hering phenomenon creates a gradient to promote interstitial and CSF fluid movement (Figure 2).

Figure 2: Fluid Movement of Arteries, Veins, Lymphatics, and Cerebrospinal Fluid

Fluid that exits the capillary bed reenters through lymphatic channels.

Yellow arrows indicate CSF flow.

Figure 2



In conclusion to this section, we revisit Sutherland's five phenomena of the PRM in light of the past 40 years of research:

1. The inherent rhythmic motion of the brain and spinal cord:
    i. It is well established that the brain and spinal cord do have intrinsic movement characteristics, but such motion alone is unlikely to be the driving force of the PRM.

2. Fluctuating cerebrospinal fluid:
    i. CSF fluctuation is a well-established phenomenon.

3. Mobility of the intracranial and spinal membranes (meninges, dura, etc):
    i. The mobility of dural membranes is well established in the field.

4. Articular mobility of the cranial bones:
    i. This concept is still controversial, but evidence is mounting that supports the presence of cranial bone motion. Presently, Traube-Hering oscillation appears to be a defensible explanation of the mechanism of CSF movment as well as the contraction and expansion of the cranial structures.

5. Involuntary mobility of the sacrum between the ilia:
    i. Sacral motion is a well-established phenomenon. It is unlikely that the observed flexion and extension of the the SBS are the primary mover causing sacral motion.

ii. The dura mater surrounding the spinal cord may be able to transmit motion causing sacral movement and may also be related to dural tube filling and emptying of CSF.

This brief review of some of the literature related to cranial manipulation theory and clinical application is intended to establish that there is sufficient evidence supporting the continued provision of cranial manipulation in health care. Nevertheless, there is still not enough evidence to provide a systematic review that proves the concept, such as in a Cochrane review.

Anatomy of the Head

In this section, we briefly review the anatomy of the cranium, dural membranes, and central nervous system and their interrelatedness. Anatomy provides a launching-point for the understanding of cranial manipulation and procedures. Figures 3 and 4 depict gross external views of the cranial bones.

Figure 3

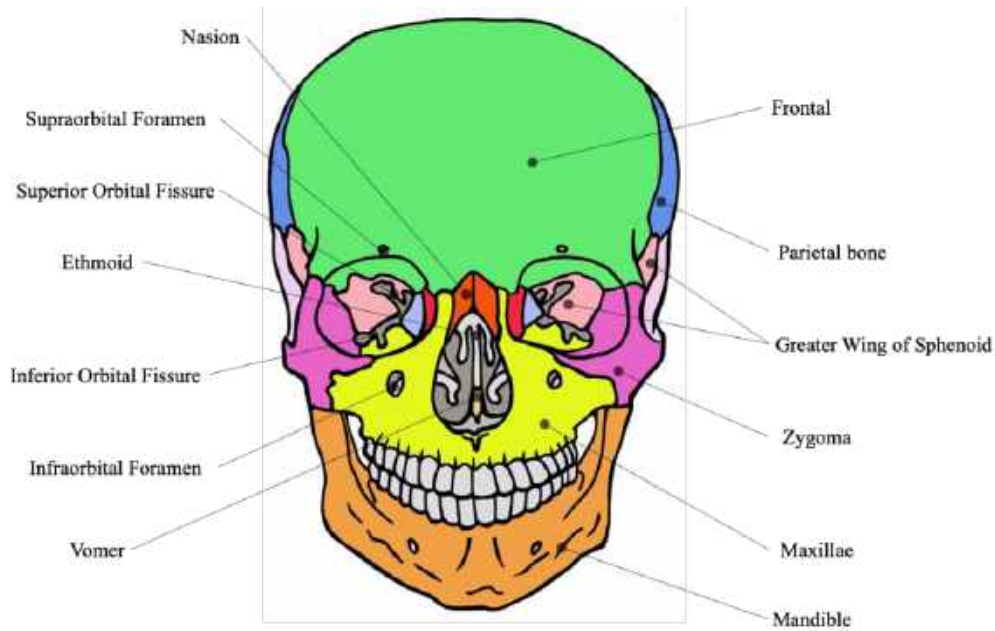
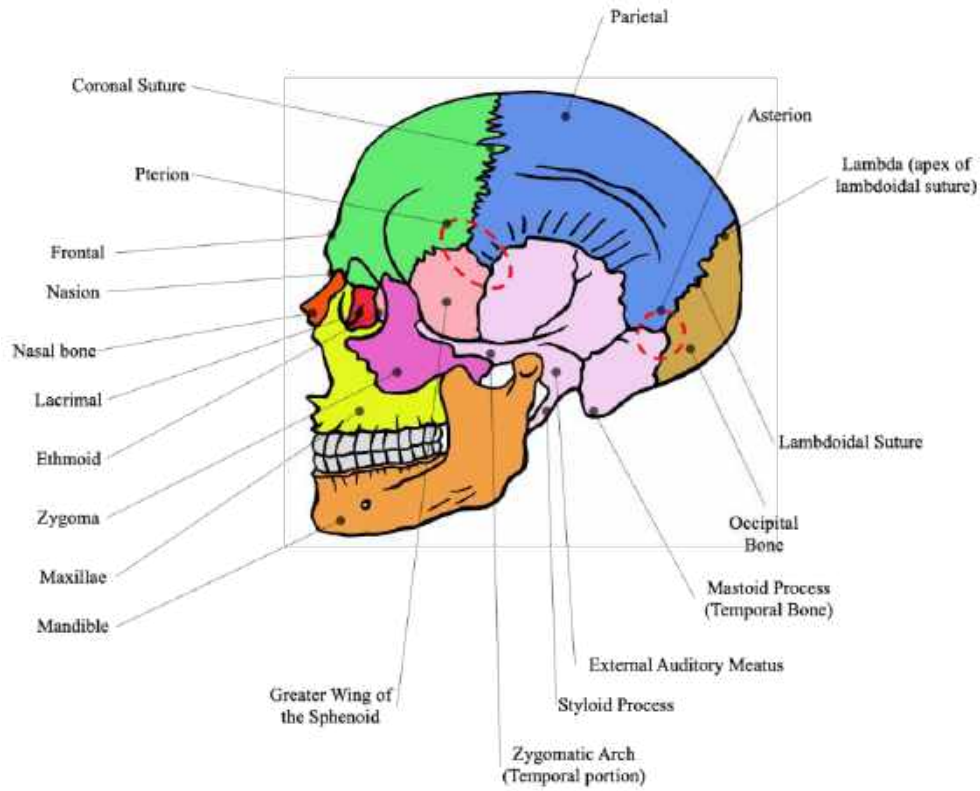


Figure 3: Anatomy of the Skull – Anterior View

Figure 4: Anatomy of the Skull – Lateral View

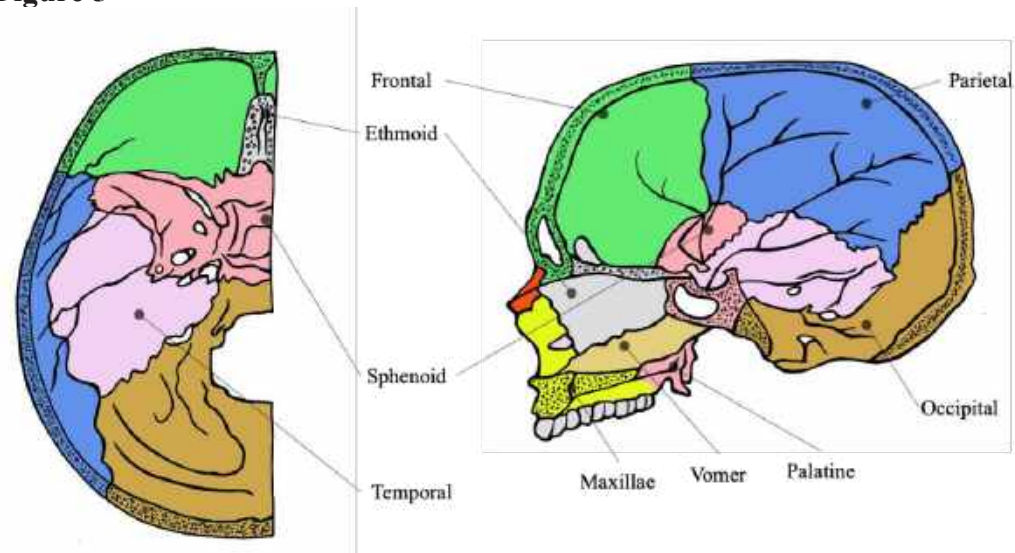
Figure 4



When reviewing the anatomy, note that external palpation allows direct access to all bones except the vomer and the ethmoid (Figure 5).

Figure 5: Anatomy of the Skull – Inner Surface

Figure 5



The sphenoid contacts every cranial bone except the nasal bone; the pituitary gland lies in the sella tursica of the sphenoid. No other structure has as much influence over the

influence over the cranial bones as the sphenoid. Figure 6 shows where the cranial nerves exit the cranial vault through their respective foramina.

Figure 6

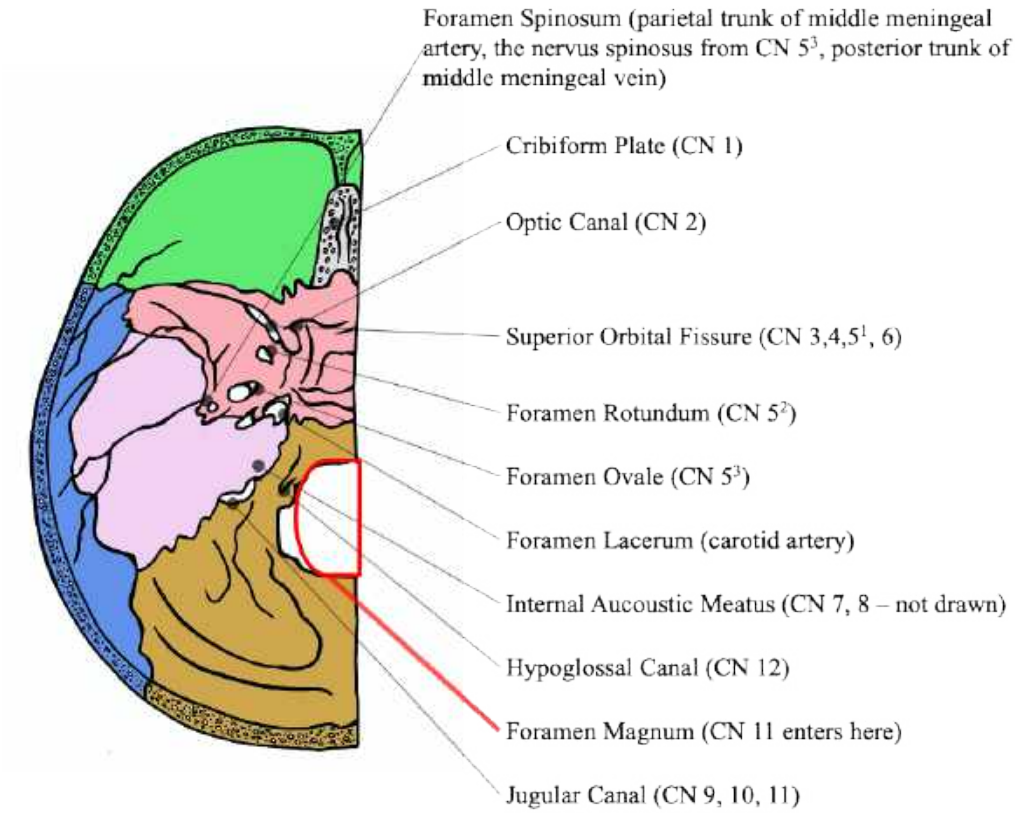
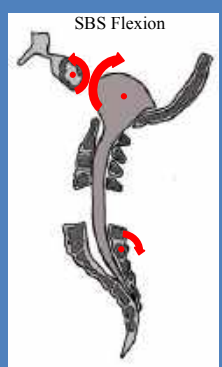


Figure 6: Fossa for Cranial Nerves Exiting the Skull

As described by Sutherland, movement of the cranium has a typical pattern that many have likened to the gear motion seen in clockworks. Figure 7a depicts sphenobasilar synchondrosis (SBS) flexion and the external rotation of related cranial bone motion as the motion of the greater wings of the sphenoid (GWS). Figure 7b shows the opposite motion of SBS extension and the internal rotation of paired bones. In the extension phase of SBS motion, the caudad direction of the occiput allows the sacrum to return to its flexed position through its dural connection.

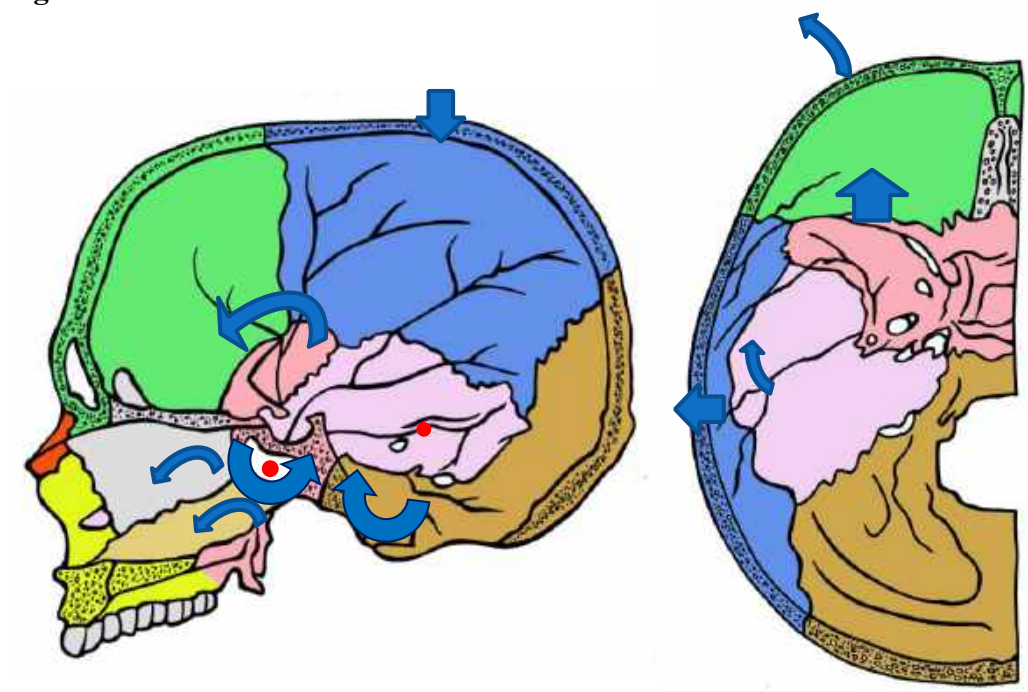
Figure 7a: SBS Flexion

- The net effect is shortening of the skull's vertical height along the sagittal suture and widening of the circumference anteriorly, posteriorly, and laterally.
- The sphenoid drives the palatine and ethmoid bones anteriorly.
- The GWS drives the frontal bone into external rotation.
- The tentorium flattens as the temporal bone externally rotates. The falx-cerebri is driven down and with it the parietal bone.
- The sacrum moves posteriorly into extension around the superior transverse axis (STA)



SBS Elexion = Eat head

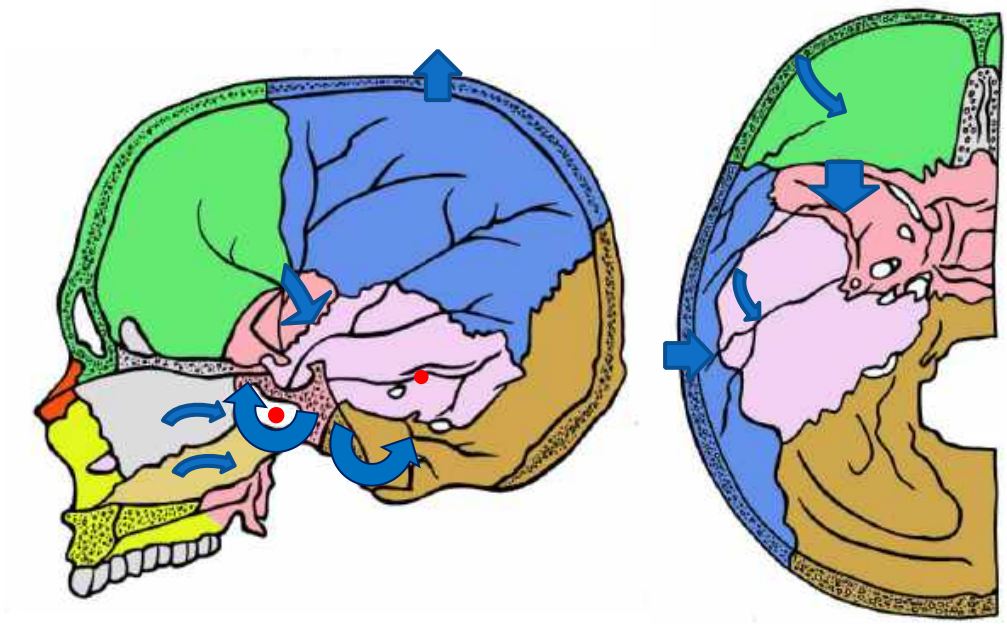
Figure 7a



SBS flexion occurs with cerebral vessel engorgement during the Traube-Hering oscillation; thus, the process is likely hydraulic pressure-driven. A good analogy is the lungs filling with air during inhalation and causing the expansion of the chest cavity. The net effect of SBS flexion is a shortening of the cranium's vertical height along the sagittal suture and widening of the circumference anteriorly, posteriorly, and laterally.

Sutherland's concept of cranial manipulation holds that the rise of the occiput during SBS flexion pulls the sacrum into sacral extension; this occurs through vertical displacement of the dural tube that attaches to the foramen magnum and the body of the second sacral segment. Using the model of the Traube-Hering oscillation, it seems more likely that external rotation of the cranium's paired bones is in response to the increased fluid volume of the cranium. The mechanism further suggests that increased intracranial pressure during cranial flexion displaces CSF into the spinal dural sheath causing its engorgement and ultimately the extension of the sacrum. The displacement of CSF into the dural tube was described in Nelson's research[20] as a capacitor that both receives and propels CSF fluid. It follows that flexion and extension of the sacrum are most likely under the local influences of the Traube-Hering reflex.

Figure 7b



According to the Traube-Hering oscillation model, cranial extension occurs with emptying of the arterial vessels; pressure inside the skull diminishes and the paired bones return to their initial positions. The arachnoid mater of the dura recoils and causes the flow of CSF back into the skull and the sacrum to move to a flexed position. CSF movement into and out of the cranium accurately describes the "tide" that is palpable to practitioners of manual medicine.

Figure 8

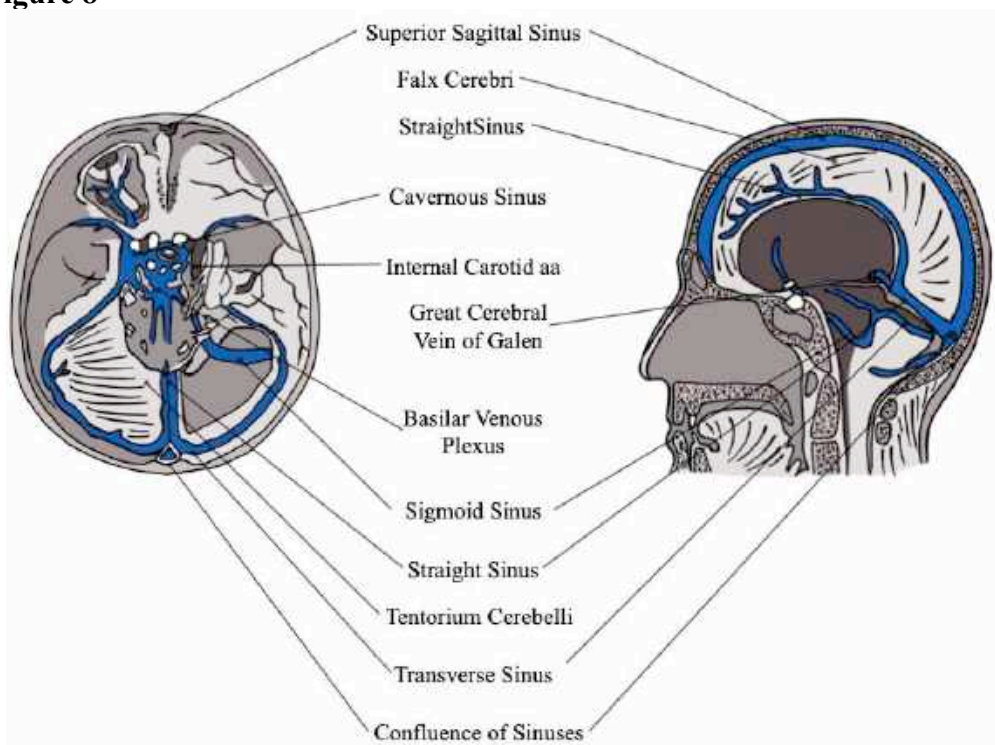


Figure 7b: SBS Extension

- The net effect is lengthening of the skull's vertical height along the sagittal suture and narrowing of the circumference anteriorly, posteriorly, and laterally.
- The sphenoid rotates posteriorly, reversing the motion it imparts to the frontal, ethmoid, and palatine bones.
- The tentorium relaxes as the temporal bone internally rotates. The falx-cerebri relaxes superiorly along with the parietal bone.
- The sacrum moves anteriorly into flexion around the STA.

SBS Extension = long narrow head

Figure 8: Dural Membranes and Dural Veins

Observe the internal relationship of the dural membranes and their formation of the dural veins (Figure 8). A theoretical shifting fulcrum, traditionally named the Sutherland fulcrum, is located along the straight sinus at the junction of the falx cerebri and the tentorium cerebelli.[31]

The Sutherland fulcrum is believed to be a dynamic axis about which the skull moves through its phases of flexion and extension. Sutherland named the internal tension of the falx cerebri, the falx cerebelli, the tentorium cerebelli, and the spinal meninges the reciprocal tension membranes. This term describes the tensegrity phenomenon that allows for flexion-extension movement of the skull around the Sutherland fulcrum and maintenance of its structural shape. The membranes act as tension-wires to maintain internal support during cranial motion.

Observe that the lambdoidal, coronal, and sagittal sutures look like expansion joints on a bridge (Figure 9a). Bridge expansion joints absorb the heat-induced expansion and contraction, absorb vibration, and allow movement due to weather changes, ground settlement, or earthquakes.

Figure 9a

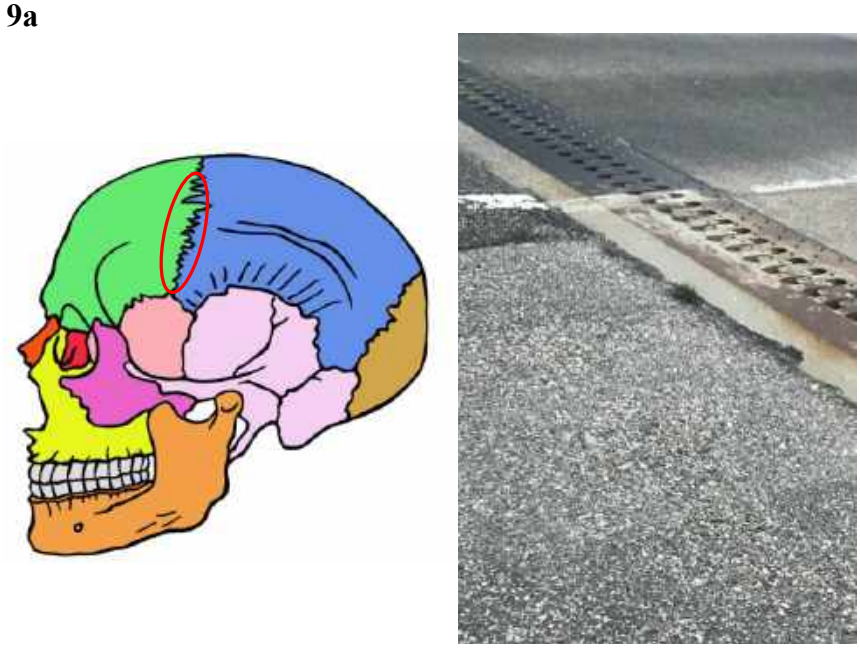


Figure 9a: Comparison of the Expansion Joints of the Head to those of a Bridge

The skull's expansion joints serve a similar purpose, allowing expansion and contraction (i.e., changes in diameter) and providing adaptation due to direct trauma to the frontal bone or increases in intracranial pressure. The zygomatic arch not only provides a channel for the temporalis muscle, but it also acts as a strut to transfer energetic blows directed at the zygoma away from the skulls internal contents.

The sutures themselves are composed of nerves, arteries, veins, and Sharpey's fibers. Sharpey's fibers (Figure 9b) consist of bundles of collagenous fibers that connect periosteum to bone; their elasticity contributes to the cranium's palpated motion.

Figure 9b

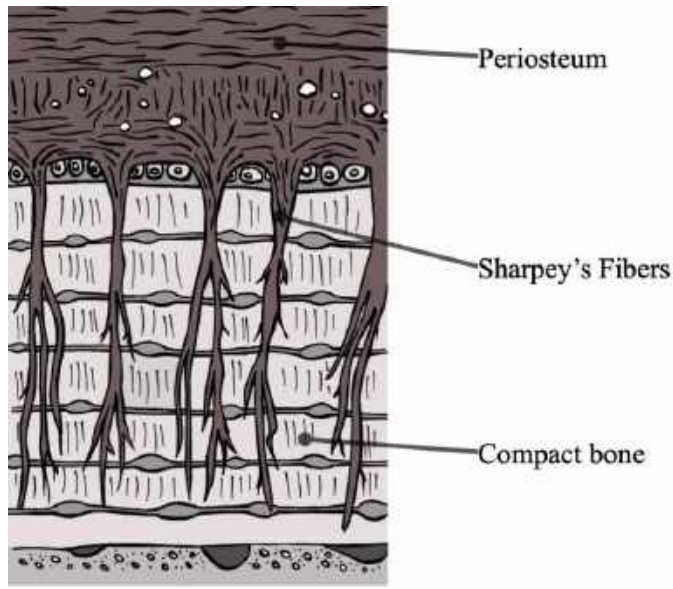


Figure 9b: Sharpey's Fibers Linking Periosteum to Bone

In the living calvarium the boney plasticity and cartilaginous attachments between sutures allow for cranial bone motion

Living bone exhibits plasticity that allows for distortion. If this were not possible, changes due to development and growth, adaption from trauma, and barometric pressure changes would not be possible.

Diagnosis of Cranial Somatic Dysfunction

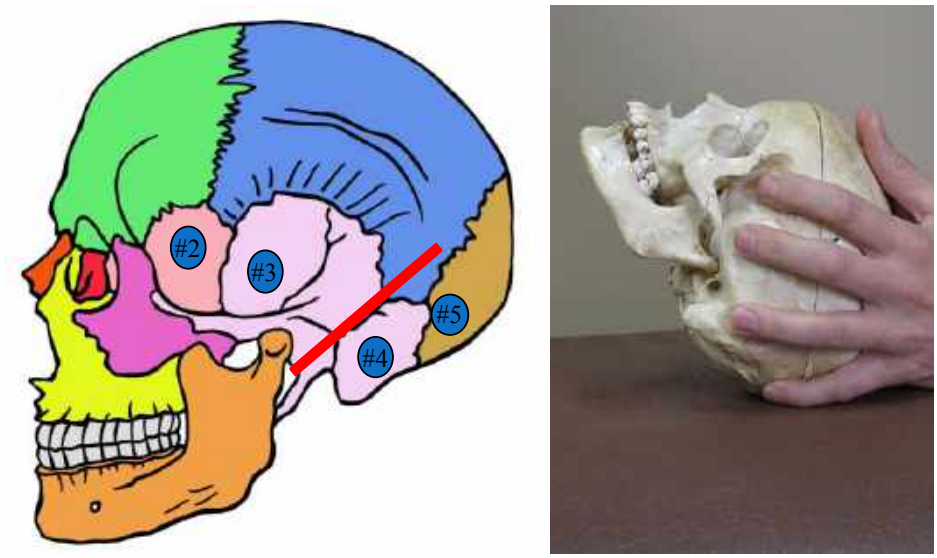
This section describes somatic dysfunction of the head according to Sutherland's formulations and the current teachings of major cranial manipulation training programs. There are three basic handholds that are used in general screening that focuses primarily on the diagnosis of the SBS: the vault hold, the occipital-mastoid hold, and the sphenobasilar hold. In living tissue, the SBS is composed of cartilaginous material and cancellous bone that is compliant and allows movement. The SBS is considered of utmost importance in cranial manipulation due to the sphenoid's link with the skull and the occiput's link to the remainder of the body.

The vault hold (Figure 10a) is performed with bilateral placement of digits #2-5 over the patient's GWS (#2), the anterior (#3) and posterior (#4) temporal bone, and the occiput (#5); the operator's thumbs form an axis above the skull by touching thumb tips together. Normal motion occurs during cranial flexion when digits #2 and #3 move anterior and laterally, the mastoid process moves medially, and the occiput inferior-medially. Recognize that the temporal bone demonstrates paradoxical motion since the anterior portion (#3) and mastoid process (#4) move oppositely around the axis in red; this motion led to its nickname "the wobbling wheel."

Figure 10a: The Vault Hold

Digits 2-5 are placed on the corresponding positions with the thumb tips (i.e., digit #1) touching and forming a central axis above the parietal bone

Figure 10a



Any lack of symmetry between the right and left hands or between fingers usually indicates somatic dysfunction. Specifically, a restriction between digits #2 and #3 on the left side would indicate temporal-sphenoid dysfunction at that suture.

The occipital-mastoid hold (Figure 10b) detects the most common somatic dysfunction of the skull, occipital mastoid suture restriction. Thumb tips are placed bilaterally on the mastoid processes with fingers cradling the occiput and pointing anteriorly.

Figure 10b: The Occipital-Mastoid Hold

Figure 10b

During flexion, the mastoid processes move medially and the occipital base moves anterior and superiorly; the motion is opposite during extension. Restriction is noted when the movement of one side compared to the other is asymmetrical, or when an attempt to glide the joint anterior or posterior is restricted.

In the assessment of the SBS, the vault hold is generally the first procedure taught for this purpose, but the sphenobasilar hold (Figure 10c) is also utilized by many practitioners for SBS assessment.

Figure 10c

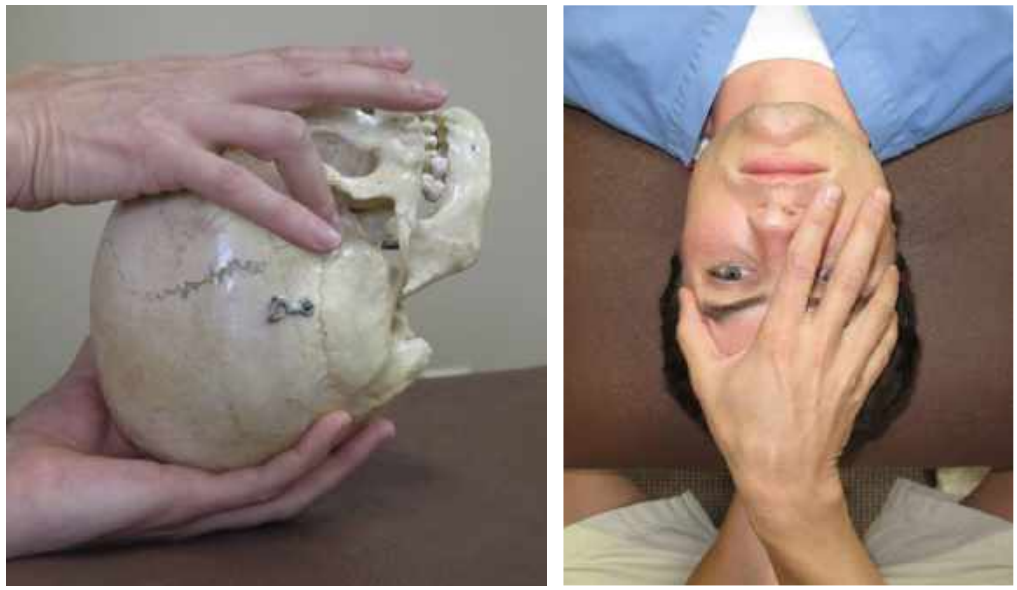


Figure 10c: The Sphenobasilar Hold

Digits #2-5 of one hand are placed on the patient's occiput while the thumb and third or fourth fingers of the opposite hand (depending on operator hand size) are placed on the GWS. This handhold allows for the detection of SBS dysfunction as described further.

Sphenobasilar Synchondrosis Somatic Dysfunction

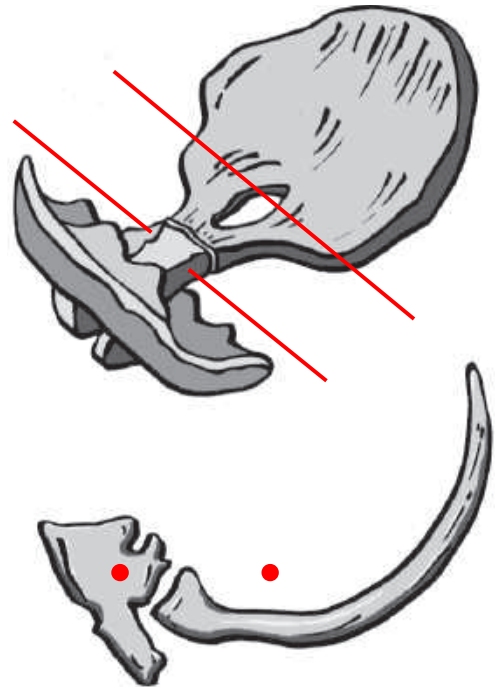
Considered to be the most important articulation of the cranial bones, the SBS often presents the most notable cranial somatic dysfunction. The naming of SBS somatic dysfunction is always referenced from the position of the sphenoid in relation to the basiocciput. Figure 11a depicts the transverse axes of the sphenoid and the occiput.

The naming of SBS dysfunction is based on the position of the sphenoid.

Figure 11a: Transverse Axes Associated with SBS Flexion and Extension

Transverse Axes are in red. There is a sphenoid transverse axis through the body of the sphenoid and a transverse axis of the occiput in the space above the occiput.

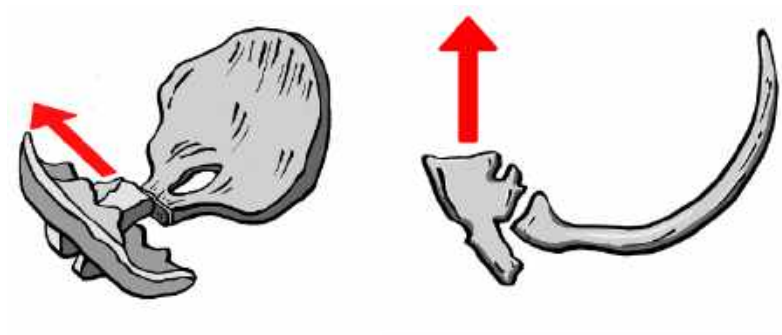
Figure 11a



Strains between the SBS may be vertical or lateral; they are named for the position of the sphenoid in relation to the basiocciput. Figure 11b depicts two of the four possible shears.

Figure 11b: Right Lateral Strain (left) and Superior Vertical Strain (right)

Figure 11b



Often confused, SBS-torsions and side-bending rotations are different entities (Figure 11c). SBS-torsions occur purely around a pathologic A-P-axis where the rotation of the sphenoid and occiput are opposite. Naming is based on the side with the more superior GWS. Side-bending rotations primarily involve side-bending with a rotational component of the sphenoid and occiput in the same direction; they are named according to the side of greater convexity. Another way to remember this terminology is that the greatest angle between the sphenoid and the occiput provides the named side.

Figure 11c

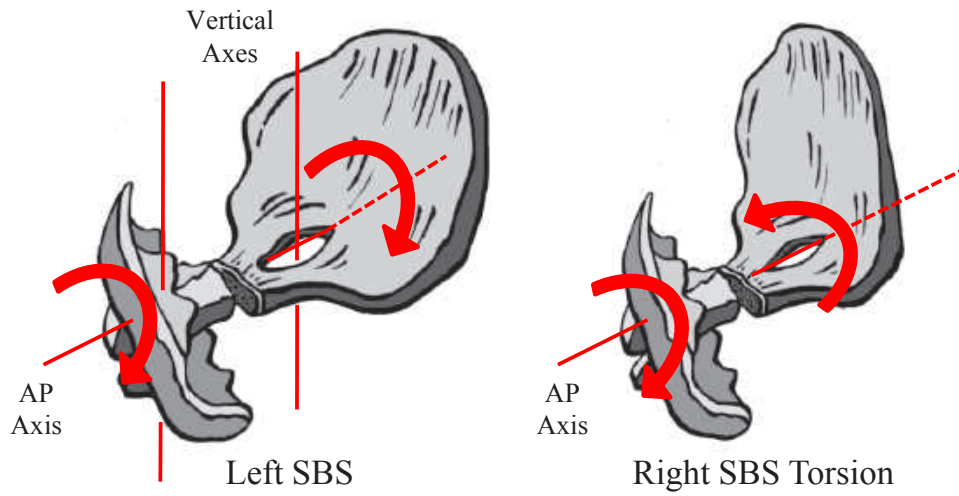


Figure 11c: Left Side-Bending Rotation and Right SBS torsion

SBS torsion is named for the side of superior GWS. SBS side-bending rotation is named for the side of convexity or the greatest angle between the occiput and sphenoid.

Somatic dysfunction of the sphenoid and occiput around their respective transverse axes is described as either the positional diagnoses of "SBS flexion" or "SBS extension," referring to the dominant SBS tendency for a position of flexion or extension. Probably the most debilitating dysfunction of the head is SBS compression; clinically this is most often due to a forceful, anterior-posterior blow to the head. With SBS compression, little or no motion is evident in the PRM, and often the head feels motionless and solid like a bowling ball.

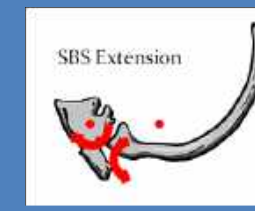


Figure 11d

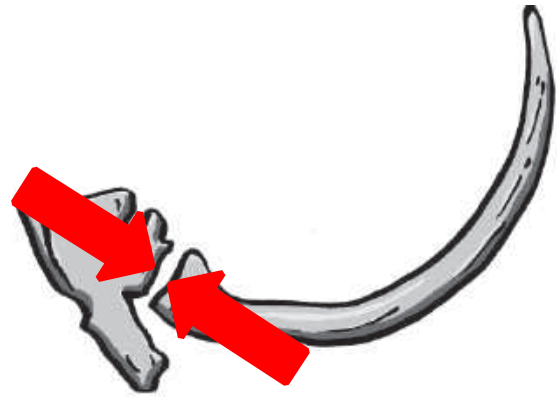


Figure 11d: SBS Compression

In addition to SBS dysfunction, Edward Stiles, DO, teaches that any suture could exhibit somatic dysfunction. If somatic dysfunction is considered to be due to patient history or by restriction found by the three basic cranial holds, he encourages several other screenings - correct cranial diagnosis can be elusive. One method of diagnosis is to exaggerate the somatic dysfunction to the indirect treatment position – this magnifies the positional diagnosis allowing for easier evaluation. For example, a right-SBS-torsion is more easily identified if its rotational components are exaggerated; this also provides the position of treatment using indirect techniques.

Treatment of Sphenobasilar Somatic Dysfunction

Most cranial manipulation training programs primarily emphasize indirect techniques such as exaggeration and hold until a "release" is perceived. For beginning students and when treating infants, this is the prudent approach. That said, we have personally witnessed Philip Greenman, DO,[32] perform direct impulse treatment of the cranium. Likewise, in our presence, Larry Jones, DO, has demonstrated the use of strain-counterstrain (S/CS) to treat sphenoid somatic dysfunction. In this section, we demonstrate direct and indirect cranial techniques with the recommendation to receive training and practice prior to the utilization of direct techniques. It is also possible to treat SBS and cranial suture dysfunction with other indirect techniques such as myofascial release.

Indirect Treatment of SBS Dysfunction

Using the diagnosis of *right-SBS-torsion* on a supine patient, the operator grasps the GWS bilaterally with their second OR third digit and thumb; the other hand cups the occiput. The sphenoid is further rotated along an imaginary AP-axis to a point of relaxation; the occiput is rotated left along this same axis. To review:

1. Obtain the positional diagnosis *right-SBS-torsion*.
2. Place the patient in the position *right-SBS-torsion*.
3. Very gently exaggerate the sphenoid to a position of three-dimensional balance or patient comfort; then, exaggerate the occiput rotation to three-dimensional balance.
4. Hold, add compression or distraction and allow inherent motion to provide treatment

Direct Treatment of the SBS Dysfunction

Direct treatment of a *right-SBS-torsion* requires a good amount of practitioner confidence.

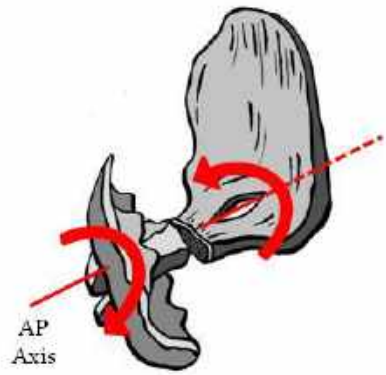
1. Obtain the positional diagnosis *right-SBS-torsion*.
2. Hold the occiput still, creating a fulcrum.
3. Reverse the components of sphenoid with right-rotation around the imaginary AP-axis until tension is felt. Keep in mind that in a *right-SBS-rotation*, the sphenoid is actually rotated left with the right GWS more superior.
4. Create a force by using a right-rotational strain in the sphenoid or an impulse toward the right in the sphenoid.

Treatment Examples: Indirect Procedures

Right-SBS-torsion using Indirect Cranial

1. Grasp the base of the sphenoid with the palms and fingers of your left hand.
2. Grasp the GWS with the second or third digit of your right hand and the thumb of your left hand.

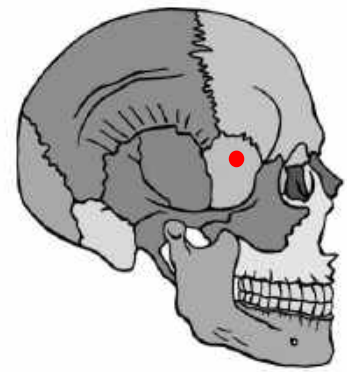
3. Place the patient in the position *right-SBS-torsion*.
4. Exaggerate the rotation of the sphenoid to a position of three-dimensional balance or patient comfort; then exaggerate the occiput rotation right to its three-dimensional balance.



5. Hold, add compression or distraction into the SBS, and allow treatment.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Right-SBS-torsion using S/CS

1. Patient is supine with their head rotated left. The operator is sitting at the head of the patient. Identify the tender point over the GWS.



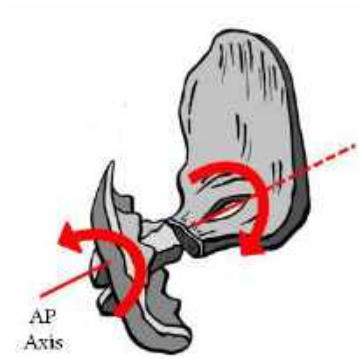
2. Place the index finger of the left hand on the GWS on the **opposite** side of the tender point.
3. The index finger of the right hand lightly monitors the tender point and the other fingers stabilize the head.
4. Apply moderate pressure to the left GWS in a direction toward the tender point. The monitoring finger needs to be especially light or it will block the movement of the sphenoid.
5. Once the tender point is reduced to less than a 2 or 3 on a 10-point pain scale, hold for 90 seconds.
6. Recheck the original diagnosis to ensure resolution of somatic dysfunction.

Treatment Examples: Direct Procedures

Right-SBS-torsion using Direct Cranial

1. Grasp the base of the occiput with the palms and fingers of your left hand creating a fulcrum.
2. Grasp the GWS with the second or third digit of your right hand and the thumb of your left hand.

3. Reverse the components of the sphenoid with right rotation (as depicted below) around an imaginary AP-axis until tension is felt.
4. Create a rotational force by using right-rotational strain in the sphenoid. Hold until the sphenoid rotates further right.

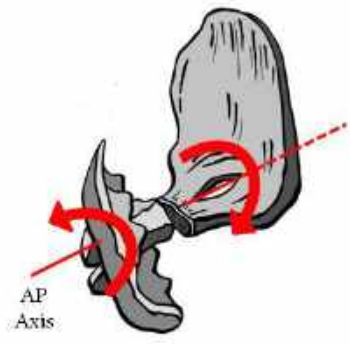


5. Recheck the original diagnosis to ensure the resolution of somatic dysfunction.

Right-SBS-torsion using HVLA

1. Grasp the base of the occiput with the palms and fingers of your left hand creating a fulcrum.
2. Grasp the GWS with the second or third digit of your right hand and the thumb of your left hand.

3. Reverse the components of the sphenoid with right rotation around an imaginary AP-axis until tension is felt (as depicted below).
4. Create a right-rotational impulse in the sphenoid through the barrier.



5. Recheck the original diagnosis to ensure the resolution of somatic dysfunction.

Additional Cranial Diagnoses and Treatment

Assessing cranial somatic dysfunction other than SBS is simple. These dysfunctions are detected by compressing individual sutures between bones and noting a difference from one side to the other, such as a hard-end feel or physical limitation in bone movement. Figure 12a depicts dysfunction of the midline facial bones.

Figure 12a

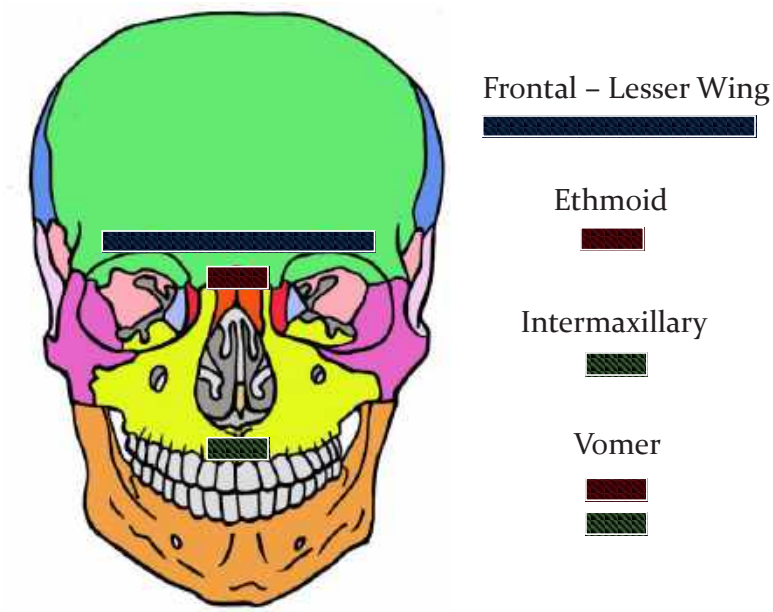


Figure 12a: Skull Articulations

Anterior to posterior compression of the intermaxillary and ethmoid sutures simultaneously tests the vomer's compliance.

The ethmoid is assessed indirectly through the junction of the nasal bones and the frontal bone through anterior-to-posterior compression. Restriction of the ethmoid is almost always in conjunction with the sphenoid or the frontal bone; while differentiation may be difficult, unsuccessful treatment of an ethmoid-frontal restriction should lead a sensible provider toward consideration of ethmoid-sphenoid somatic dysfunction.

Since one cannot directly palpate the vomer, its motion is assessed by applying compression into the skull at the intermaxillary and ethmoid simultaneously. A hard-end feel at both positions indicates vomer somatic dysfunction. Figure 12b depicts the vomer and the palatine bones; note the palatine bone in the orbit is considered by some to function by transmitting a "pumping" action to the orbit. The palatine bones are assessed with a gloved hand in the patient's mouth while simultaneously palpating the GWS and noting a motion restriction between it and the lateral aspect of the palatine bone. Restriction could occur between the two palatine bones or also between a palatine and a maxillary bone. Great care must be exercised in palpation and treatment of the palatine bones, as the pyramidal process that articulates with the sphenoid is easily fractured.

Frontal-lesser wing restrictions are often accompanied by patient history of a "tight-band" across the forehead. Further confirmation is obtained by simultaneous restriction of the GWS in anterior or posterior motion that appears to lock across the frontal bone, just above the orbits.

The zygoma and the palatine bones are often referred to as trauma "speed reducers" and "shock absorbers" since they absorb the greater motion potential of the vault bones (i.e., sphenoid, occiput, temporal, parietal, and frontal). Their restriction can cause confusing and unusual facial pain syndromes.

Speed Reducers = zygoma and palatine bones (note there are two of each).

Figure 12b

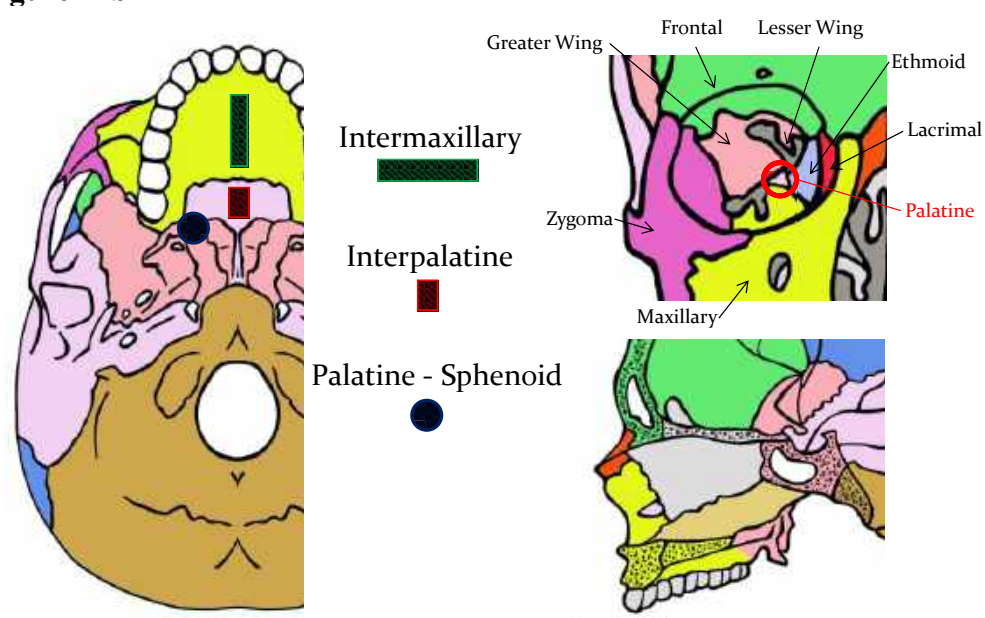


Figure 12b: Skull Articulations

Bones of the orbit and views of the midline bones of the skull.

Note that the palatine has to be assessed through the mouth; be sure to use a gloved hand. Note the palatine bone in the orbit.

Figure 12c demonstrates other potential somatic dysfunctions of the facial bones. Note that palatine dysfunction can sometimes be assessed externally, but must be differentiated from tooth contact pathology and dental abscesses.

Figure 12c

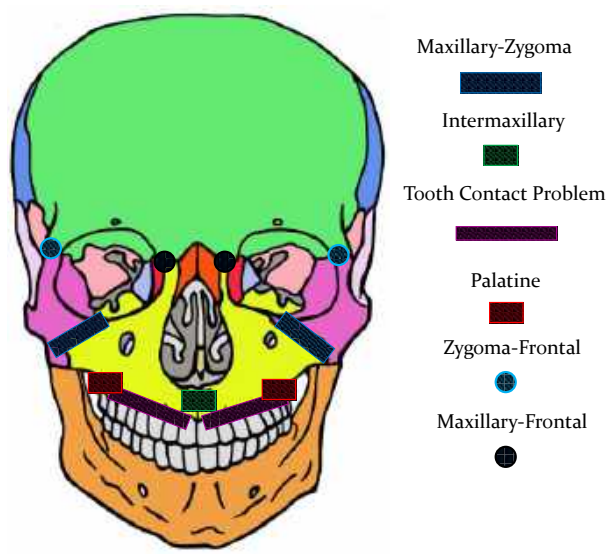
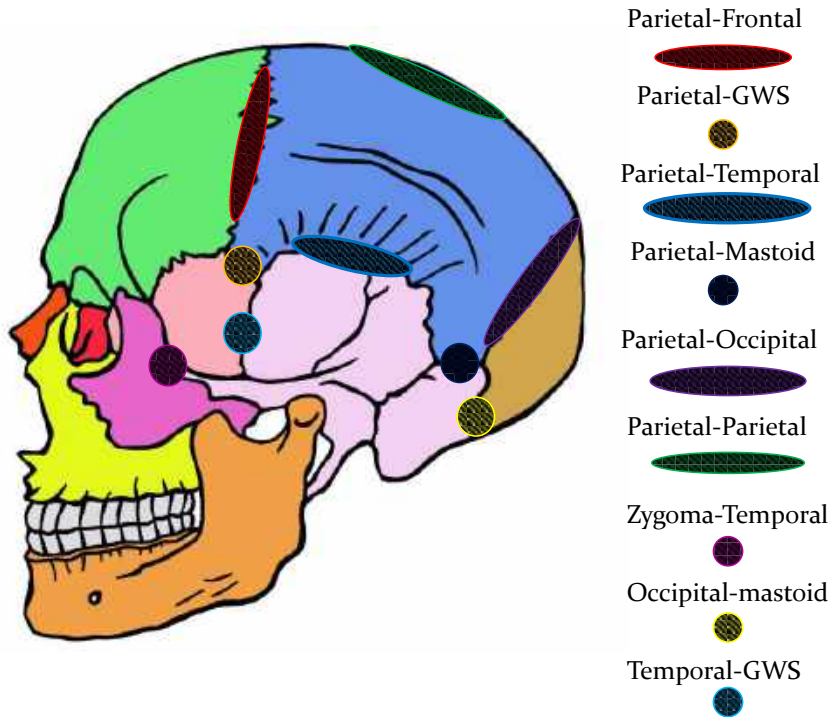


Figure 12c: Somatic Dysfunction of the Facial Bones

Figure 12d: Somatic Dysfunction of the Cranial Bones

The dysfunctions in the areas depicted 12c and 12d are diagnosed by simply palpating over the sutures bilaterally and noting asymmetry between the left and right sides.

Figure 12d



Treatment of Cranial Somatic Dysfunction other than SBS Dysfunction

The treatment of cranial dysfunction between two sutures can be performed early-on in a practitioner's medical career. Restoration of motion to this area can have a great effect on head and facial pain.

Indirect Treatment of non-SBS Dysfunction

To treat the diagnosis of *left temporal-parietal restriction* on a supine patient, the operator places a thumb on either side of the left temporal-parietal suture. This dysfunction is noted by limitation in the vault hold or with direct compression between the sutures; a positional diagnosis is not identified when upon palpitation, only a restriction in motion between the two bones is noted. The more restricted component is moved to a position of ease (POE), assessing anterior versus posterior movement and internal versus external rotation. The other bone is then positioned to ease and then held, or activated by compression or distraction. To review:

1. Obtain the restriction diagnosis *left temporal-parietal restriction*.
2. If the temporal bone is more restricted find its POE.
3. Place the parietal bone in its POE.
4. Hold, add compression, or distraction and initiate treatment.

Direct Treatment of the SBS Dysfunction

Using the diagnosis of *left temporal-parietal restriction* on a supine patient, the operator places a thumb on either side of the left temporal-parietal suture. After assessing anterior versus posterior movement and internal versus external rotation, the less restricted component is moved to a position of strain and held as a fulcrum. The other bone is then put into an equal position of strain. This position is further activated by direct strain to the suture or an impulse. To review:

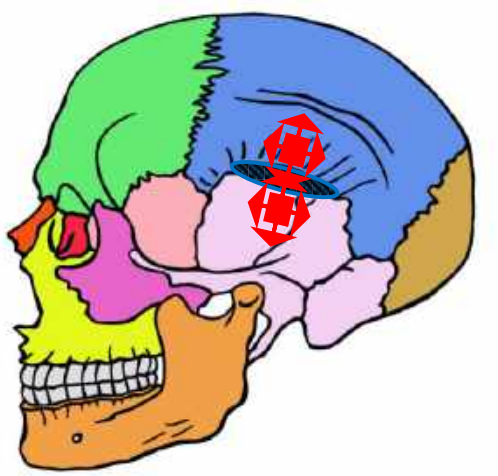
1. Obtain the restriction diagnosis *left temporal-parietal restriction*.
2. If the temporal bone is less restricted, find its position of strain and hold firm as the fulcrum.
3. Place the parietal bone in its position of strain (note: either bone may be used as the fulcrum).
4. Create a force by using strain into the temporal-parietal junction or by adding an impulse to the temporal bone.

See the example provided for direct treatment techniques that allow the practitioner to quickly treat the patient.

Treatment Examples: Indirect Procedure

Left temporal-parietal restriction Functional Procedure

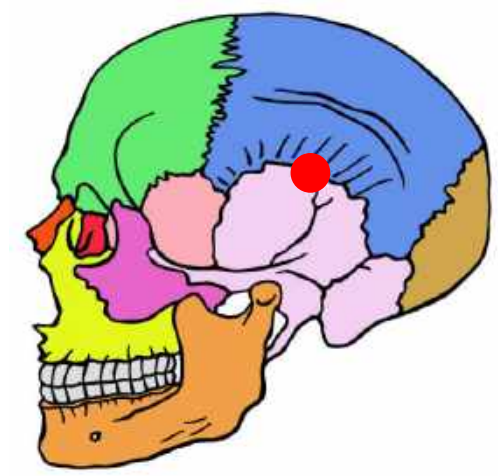
1. The patient is supine on the table.
2. The operator places a thumb on either side of the left temporal-parietal suture.



3. The more restricted component is moved to a position of ease after assessing anterior versus posterior movement, and internal versus external rotation.
4. The other bone is then positioned to its dynamic balance point.
5. Activate treatment by adding compression or distraction into the suture.
6. The area is rechecked for improvement in motion.

Left temporal-parietal restriction S/CS (aka squamosal tenderpoint)

1. The patient is supine on the table.
2. The operator places their left index finger on the tenderpoint located on the left temporal-parietal suture to monitor.

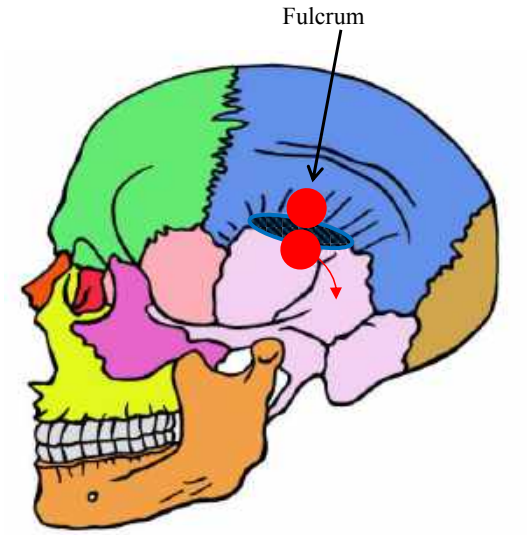


3. The index finger of the right hand applies moderate pressure to the right temporal-parietal in the direction of the tender point. The monitoring finger needs to be especially light or it will block the movement.
4. Once the tender point is reduced to less than a 2 or 3 on 10-point pain scale, hold for 90 seconds.
5. Recheck the original diagnosis to ensure resolution of somatic dysfunction.
6. The area is rechecked for improvement in tenderness.

Treatment Examples: Direct Procedure

Left temporal-parietal restriction Direct Strain Procedure

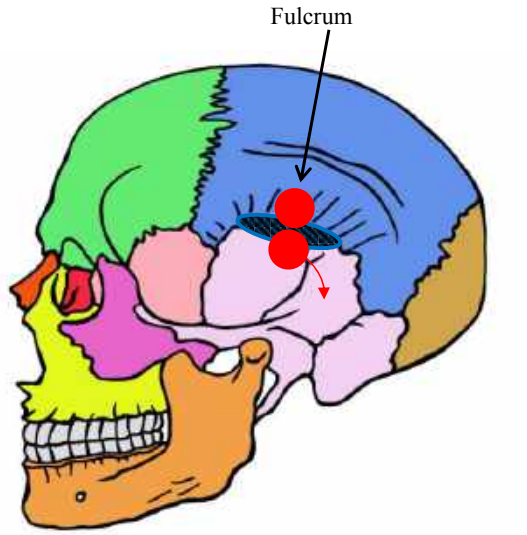
1. The patient is supine on the table.
2. The operator places a thumb on either side of the left temporal-parietal suture.



3. The less restricted (in this instance the parietal bone) component is moved to a position of strain after assessing anterior versus posterior movement and internal versus external rotation and held as a fulcrum.
4. The temporal bone is then put into an equal position of strain.
5. This position is further activated by direct strain separating the suture. In this instance strain is put against the rotational barrier preventing the temporal bone from rotating anteriorly.

Left temporal-parietal restriction HVLA

1. The patient is supine on the table.
2. The operator places a thumb on either side of the left temporal-parietal suture.



3. The less restricted component (in this instance the parietal bone) is moved to a position of strain after assessing anterior versus posterior movement, internal versus external rotation and held as a fulcrum.
4. The temporal bone is then put into an equal position of strain and held.
5. This junction between the bones is tested for the direction of the barrier.
6. An impulse against the restrictive barrier is made. A posterior rotational impulse is used in this instance due to the restriction of posterior rotation.

Cranial Treatment Pearls

With regard to the head, clinical practice has provided treatment "pearls" to aid in determining key areas of somatic dysfunction. These methods are listed below:

- An initial cranial screening utilizing the vault hold, the occipital-mastoid hold, and the sphenobasilar hold provide the practitioner the opportunity to compare and confirm findings and determine the optimal hold for treatment.
- If multiple dysfunctions are noted, treat SBS dysfunctions first that restore the normal mechanics of cranial flexion and extension.
- Once the SBS motion is restored, if other somatic dysfunction is detected, go to the area of greatest restriction and treat it next.
- With practice, it should only take 10-15 seconds to assess all the sutures of the skull. Refer to figures 12a-12d for suture junctions.
- In SBS compression, the skull feels like a bowling ball. Use direct treatment by distracting the GWS while holding the occiput as a stable base (fulcrum).
- Breathing may be introduced into treatments to cause internal and external movement of fascia. Ultimately, this will allow internal fascial movement and remove a restrictive barrier.

Further Considerations in Cranial Manipulation

The models presented in this chapter represent two approaches to manual medicine of the head: bone and dura. In understanding the cranial bones and their interrelated articulation, our goal is to provide a launching point to begin manual medicine of the head. In palpating the PRM through the vault hold, the occipital-mastoid hold, and the sphenobasilar hold, the practitioner learns to palpate the dural membranes. With experience and practice, his skill can be extrapolated to the remainder of the body; the elimination of somatic dysfunction allows movement of the PRM into areas that were previously devoid of normal motion and vitality.

Other cranial techniques using CSF and fluid models are often used in treatment. These procedures are described in *Cranial Manipulation Theory and Practice* by Leon Chaitow,[13] a highly recommended comprehensive text. Although this text is not a procedure book, a good understanding of two procedures is critical for beginners of manual medicine since they serve as the starting point for understanding the CSF fluid model in treatment of cranial manual medicine. The compression of the fourth ventricle (CV4) and the V-spread procedure use CSF fluid to eliminate somatic dysfunction as described below.

The CV4 (Figure 13) is a cranial manipulation procedure in which the lateral angles of the occiput are gently approximated medially. This procedure encourages the cranial bone motion to be held in sustained extension at the SBS, and stimulates the PRM and the person's inherent healing capacity. Bio-mechanically, it is thought that this treatment creates a direct hydraulic force into and narrowing the fourth ventricle (Figure 2) thereby prompting enhanced CSF fluctuant flow throughout the brain and central nervous system (CNS). This in turn stimulates all of the CNS areas contacted by the CSF to perform more efficiently, thereby promoting health.

Figure 13: CV4 Procedure

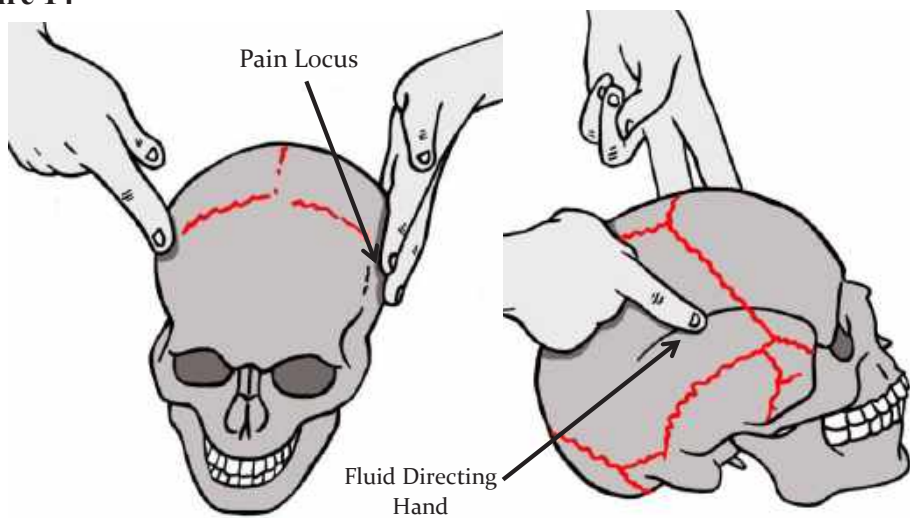
Pressure is medially applied to the lateral angles of the occiput and held causing sustained cranial extension.

Figure 13

The V-spread procedure (Figure 14) propels CSF in the form of a fluid wave across the diameter of the skull to facilitate sutural decompression. Two fingers are spread in the shape of a "V" on either side of a restricted suture; a soft direct push with 2 to 4 fingers or a very light tapping force is directed toward the restricted suture on the opposite side of the skull. Using CSF as a transmitted force mirrors an abdominal fluid wave used in the examination of ascites. To determine the force vector, it is necessary to place a hand opposite the side of restriction and to initially propagate a CSF wave from the site of restriction. The receiving hand palpates a locus generated from the restriction that is then used to propagate a CSF wave toward the sutural restriction.

Figure 14

Figure 14: V-spread Technique



References

1. Sutherland WG. *The cranial bowl*. Mankato, MN; Free Press Company, 1939.
2. Sutherland WG. In Wales AE (Ed.) *Teachings in the science of osteopathy.* Cambridge, MA, Rudra Press, 1990.
3. Woods JM, Woods RH. A physical finding related to psychiatric disorders. *J Amer Osteopath Assoc* 1961; 60:988-993.
4. King HH. Osteopathy in the Cranial Field. In Chila, AG (Ed.) *Foundations of Osteopathic Medicine. 3/e.* Philadelphia, Lippincott, Williams & Wilkins, 2011:728-48.
5. Baker E. Alteration in width of maxillary arch and its relation to sutural movement of cranial bones. *Journal of the American Osteopathic Association* 1970; 70:559-564.
6. Moskalenko Y, et al. Periodic mobility of cranial bones in humans. *Human Physiology* 1999;25: 51-58.
7. Ueno T, et al. Ultrasonic Measurement of Intracranial Pressure Waveforms. Acta Neurochir Suppl. 1998; 71:66-9
8. Uneo T, et al. Cranial Diameter Pulsations Measured by Non-Invasive Ultrasound Decrease with Tilt. Aviation, Space, and Environmental Medicine Vol. 74, no. 8. Aug 2003.
9. Crow WT, King HH, Patterson RM, Giuliano V. Assessment of calvarial structure motion by MRI. *Osteopath Med Prim Care.* 2009;3:8.
10. Oleski SL, Smith GH, Crow WT. Radiographic evidence of cranial bone mobility. *J Craniomandib Pract* 2002;20(1):34-43.
11. Jäkel A, von Hauenschild P. Therapeutic effects of cranial osteopathic manipulative medicine; a systematic review. *J Amer Osteopath Assoc* 2011;111(12):685-93.
12. King HH. Cranial osteopathic manipulation's growing evidence base [Editorial]. *J Amer Osteopth Assoc* 2012;112(1);9.
13. Chaitow L. *Cranial manipulation theory and practice, 2e*. Edinburgh, Elsevier Churchill Livinston, 2005.
14. Lederman A. *Harmonic technique.* Edinburgh, Churchill Livingston, 2000.
15. Upledger JE, Vredevoogd JD. *Craniosacral therapy.* Chicago, Eastland Press, 1983.
16. Degenhardt B, Kuchera M. Update on osteopathic medical concepts and the lymphatic system. *J Amer Osteopath Assoc*. 96(2):97-100.
17. Norton, JM. A tissue pressure model for palpatory perception of the cranial rhythmic impulse. *J Amer Osteopath Assoc.* 1991;91(10):975.
18. Nilsson H, Alkjaer C. Vasomotion: Mechanisms and Physiological Importance. *Molecular Interventions*. 2003;3(2): 27-41.
19. McPartland JM, Mein ES. Entrainment and the cranial rhythmic impulse. *Altern Ther Health Med.* 1997;3(1):40-5.
20. Nelson K, Sergueff N, Lipinski CL, et al. Cranial rhythmic impulse related to Traube-Hering-Mayer oscillation: comparing laser-Doppler flowmetry and palpation. *J Amer Osteopath* Assoc 2001;101(3);163-173.
21. Traube L. Uber periodische Tatigkeitsanderungen des Vasomotorischen und Hemmungs-Nervenzentrums. *Cbl Med Wiss* 1865;56:881-885.
22. Hering E. Uber Athembewegungen des Gefasssystems. *Sitzungb d k Akad d W math naturw* 1869;60:829-856.

23. Ingvar DH, Lundberg N. Paroxysmal Symptoms in Intracranial Hypertension, Studied with Ventricular Fluid Pressure Recording and Electroencephalography. *Brain* 1961;84:446-459.
24. Smith M. Monitoring intracranial pressure in traumatic brain injury. *Anesth Analg.* 2008; 106:240-24
25. Mayer S. Uber spontane Blutdruckschwankungen. *Sitzungb d k Akad d W math naturw* 1876;67:281-305.
26. Becker RE. *Life in motion: the osteopathic vision of Rollin E. Becker.* Brooks RE (Ed.). Portland, OR, Rudra Press, 1997.
27. Sergueef N, Nelson, KE, Glonek T. Changes in the Traube-Herring-Mayer wave following cranial manipulation. *Amer Acad Osteop J* 2001:11:17.
28. Zakharov A, Papaiconomou C, Djenic J, et al. "Lymphatic cerebrospinal fluid absorption pathways in neonatal sheep revealed by subarachnoid injection of Microfil." *Neuropathol. Appl. Neurobiol.* 2003;29(6):563–73.
29. Johnston M. "The importance of lymphatics in cerebrospinal fluid transport.". *Lymphat. Res. Biol.* 2003;1 (1):41-4.
30. Greitz D, Hannerz J. A proposed model of cerebrospinal fluid circulation: observations with radionuclide cisternography. *Am J Neuroradio.* 199617(3):431-8.
31. Educational Council on Osteopathic Principles. *Glossary of osteopathic terminology.* In Chila AG (Ed*.) Foundations of osteopathic medicine*, *3e.* Philadelphia, Lippincott Williams & Wilkins, 2011: 1087-1110.
32. DeStefano LA. *Greenman's principles of manual medicine, 4e.* Philadelphia, Lippincott Williams & Wilkins, 2011:175.

Clinical Vignette – Cranial Chapter

Patient Name: CS
June 14, 2020

CC: new patient visit, medical problems

HxCC: Patient is a 52-year-old, white, male who presents as a new patient. He presents with headaches and migraines which began two months ago following dental work that required him to leave his mouth open for two hours on two separate occasions; this caused dramatic pain and headache. He denies any fillings done during his recent dental appointments. His pain is 7/10, begins at the base of his skull and radiates to his face on the right. He notes a normal MRI of the brain and cervical spine last month as ordered by neurology.

ROS:
Constitutional: no mental status changes, no travel or sick contacts
Eyes: no change in vision
Endocrine: no fever, night sweats, weight change
Cardiovascular: no chest pain or palpitations
Respiratory: no dyspnea, cough, or sputum
Gastrointestinal: no nausea, vomiting, or diarrhea; no change in bowel habit, no blood in stool
Genitourinary: no change in urine, no dysuria, no blood in urine
Neurologic: no anesthesia, no paresthesias, no loss of consciousness, no seizures
Musculoskeletal: no weakness
Psychiatric: no homicidal or suicidal ideations, no signs of depression
Allergy: no angioedema, no urticaria, no anaphylaxis, no symptoms of seasonal allergies

PMHx/PSHx:
-Colonic Polyps with colonoscopies at age 40, 45, and 50yrs

Allergies:
-Penicillin

MEDs:
-none

Immunizations:
-Tetanus 2018
-influenza fall 2013

Social:
-no smoking; no alcohol; no drugs; work = engineer

FamHx:
-Dad = colon cancer diagnosed at age 60

VITALS = 5'11", 218#, 110/74, 107 beats per minute
GEN = awake, alert, oriented to time, place, situation, self, in no acute distress
HEENT = normal cephalic, a-traumatic, extraocular muscles intact, no otic/nasal discharge
NECK = supple, thyroid w/o masses
HEART = regular rate, no clicks/rubs/murmurs
LUNGS = clear to auscultation, without crackles, wheeze, or ronchi
GI = soft, non-tender, no rebound tenderness, +bowel sounds x 4 quadrants, no hepato-spleenomegaly
CV = no jugular venous distention, no carotid bruits, mucus membrane moist, no edema, 2/4 peripheral pulses, no lymphadenopathy
N/M = cranial nerves 2-12 intact, 5/5 global strength, 2/4 global reflexes, cerebellum intact, sensation symmetric, gait normal
Biomechanical exam = abnormal findings include **tender to palpation (TTP) on cervical spine right C2 posteriorly, right C1 laterally, and on right lateral pterygoid muscle. He has a right occipital mastoid restriction. His bite is limited to 1 cm when opening.**

Assessment/Plan

1. Headache
2. Temporal Mandibular Joint (TMJ) Disorder following dental procedure
3. Somatic Dysfunction of head and neck

-the patient exhibits diminished jaw opening and characteristic findings of somatic dysfunction associated with TMJ-disorder. Manual medicine will be directed to these areas. Note: the patient's bite increased from 1 to 2 cm prior to leaving clinic today and his pain is much reduced.
-patient counseled of a normal treatment reaction. A treatment reaction may include muscle soreness, pain, and mild edema that may last two to three days. If patient experiences symptoms that outlie a normal treatment reaction, he should contact a physician or go to the emergency room.
-follow up in one week

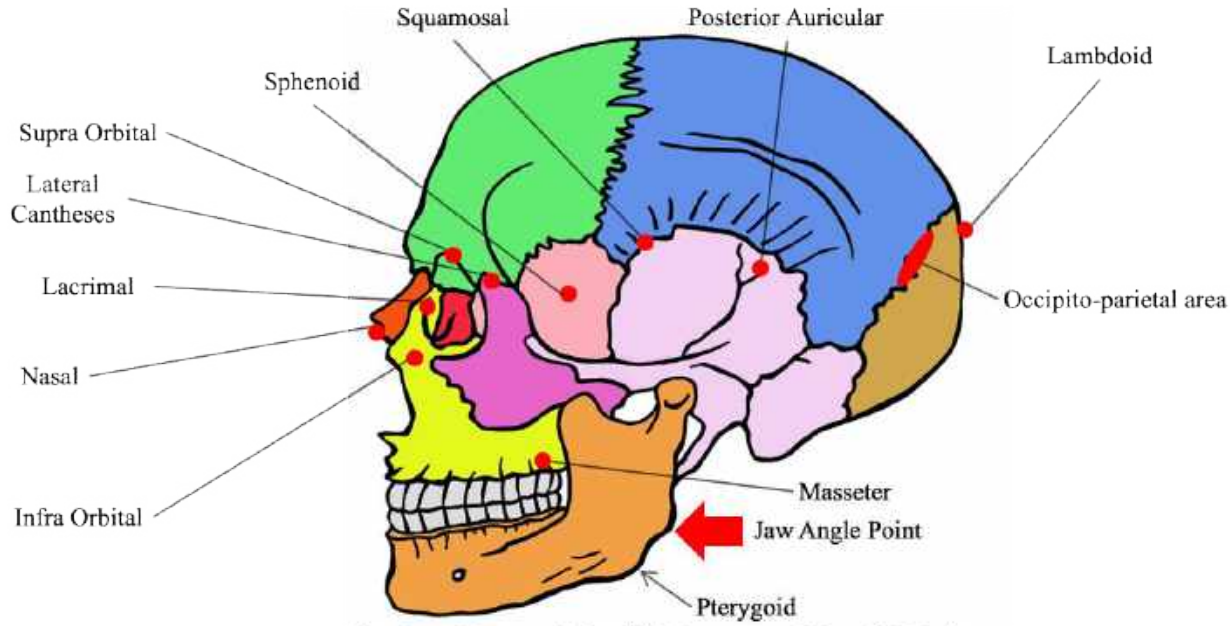
Treatments included:

- C2 TTP right-posterior treated with strain-counterstrain (S-CS)
- C1 TTP right-anterior treated with S-CS
- Right-occipital mastoid restriction treated with indirect cranial
- Right lateral pterygoid TTP treated with S-CS

Hollis King, DO, FAAO

Appendix A: S/CS Tenderpoints for the Head

Jones' treatment of the skull is very similar to the techniques taught by Edward Stiles, DO, FAAO, and regularly used by authors JB and PJ. Jones named the points according to anatomical landmarks he palpated. In most of his treatments, Jones locates a tenderpoint and uses a strain force in the opposing bone to reduce its tenderness; our direct strain procedure mirrors Jones' as previously described.



*from Jones, Lawrence H., Randall S. Kusunose, and Edward K. Goering. Strain-counterstrain. Boise, ID: Jones Strain-Counterstrain,1995.

Of clinical importance, temporomandibular joint (TMJ) disorder is effectively treated with S/CS. The pterygoid tenderpoint is located on the medial side of the ascending ramus and is treated by opening the patient's jaw and deviating it toward the tenderness. Based on this treatment position, Jones is describing treatment of the lateral pterygoid muscle. The jaw angle point is located approximately 2 cm superior to the pterygoid tenderpoint, and is treated by closing the jaw and deviating it away from the tenderness; hence, this is likely the medial pterygoid muscle. The masseter tenderpoint is treated by compression of the closed mouth with slight ipsilateral deviation of the jaw. Lastly, the omohyoid tenderpoint is reviewed in the upper extremities chapter, but the reader is reminded of its importance in TMJ treatment.

Review Questions

1. The primary respiratory mechanism is?
    a. diaphragmatic breathing motion combined with accessory muscles of respiration
    b. equivalent to cranial rhythmic impulse
    c. has correlation with cardiac rate and varies with blood pressure
    d. ubiquitous phenomenon throughout body involving cellular metabolism

2. The primary respiratory mechanism is described as having 5 key phenomena. Which of the choices listed below remains controversial?
    a. The inherent rhythmic motion of the brain and spinal cord
    b. Fluctuating cerebrospinal fluid
    c. Mobility of the intracranial and spinal membranes (meninges, dura, etc)
    d. Articular mobility of the cranial bones
    e. Involuntary mobility of the sacrum between the ilia

3. Cranial bone motion has been demonstarted by which of the following imaging procedures?
    a. MRI, Caliper devices, Ultrasound
    b. MRI, Caliper devices, Serial roentograms
    c. MRI, Ultrasound, Serial roentograms
    d. Caliper devices, Ultrasound, Serial roentograms

4. The evidence base for cranial bone motion …
    a. has been proven
    b. has been disproven
    c. is supportive
    d. is irrelevant

5. Theories of motive force for cranial bone motion include which of the following?
    a. Diaphragmatic respiration, muscular contractins, lymphatic contractions, Traube-Herring oscillations
    b. CSF production, diaphragmatic respiration, lymphatic contractions, muscular contractions
    c. CSF production, diaphragmatic respiration, muscular contractions, Traube-Herring oscillations
    d. CSF producation, diaphragmatic respirations, muscular contractions, lymphatic contractions,

6. Anterior to the external cranial point known as the “Inion,’” on the internal surface of the occipital bone is the _____.
    a. inferior occipital sinus
    b. inferior sagital sinus
    c. sigmoid sinus
    d. confluence of sinuses

7. The anatomy around which cranial strain patterns are orientated is known as?
    a. confluence of sinuses
    b. sphenobasilar synchondrosis
    c. the Sutherland flucrum
    d. temporal mandibular joint
    e. external auditory meatus

8. "Speed reducers" include the _____.
    a. zygoma and palatine bones
    b. sphenoid and occiput bones
    c. zygoma and occiput bones
    d. palatine and sphenoid bones
    e. frontal and sphenoid bones

9. Indirect cranial treatment procedures involve….
    a. pressure to move the bones in the desired direction
    b. applying pressure in some other part of the head than the restriction
    c. forces applied to the roof of the mouth
    d. positioning the bone in the direction of ease

10. Unique OMT procedures frequently employed and introduced in the concept of the cranial bone motion are?
    a. indirect technique and muscle energy
    b. direct technique and CV4
    c. CV4 and V-spread
    d. V-spread and counterstrain
    e. indirect and counterstrain

Answers:

1. D
2. D
3. C
4. C
5. C
6. D
7. B
8. A
9. D
10. C

Chapter 11: Neurophysiology of Somatic Dysfunction

Principles Presented in this Chapter:

- Physiological explanation for somatic dysfunction
- Etiology of viscerosomatic reflexes

Outline:

- The “Osteopathic Lesion” aka Somatic Dysfunction
- The Peripheral Nervous System
    - The A-Afferent System
    - The B-Afferent System
    - Activation of the B-Afferent System
    - Peripheral Sensitization
- Rationale to Explain Criteria of Somatic Dysfunction
- The Korr Model
- Korr Model Refuted
- Central Sensitization
- Viscerosomatic Reflexes
- References
- Clinical Vignette
- Review Questions

Chapter 11: Neurophysiology of Somatic Dysfunction

Osteopathic concepts include "the body is a functional unit," and "structure and function are reciprocally interdependent." The body accomplishes this through a complex equilibrium and tends to be self-regulating and self-healing in the face of disease processes. When the system begins to fail, the complex equilibrium, a series of monitoring feedback pathways, begins to fail. Ultimately, this can lead to an inability to restore the homeostatic mechanism. As Osteopaths or practitioners of manual medicine, our goal is to restore the homeostatic mechanism and thereby optimize the healing potential of the patient. In order to influence the reciprocal relationship of structure and function, we start from where we locate the dysfunction - in the periphery (peripheral nervous system). Practitioners of different treatment techniques emphasize different aspects of somatic dysfunction (i.e., tissue texture changes, increased hypersensitivity, asymmetric range of motion, and anatomic asymmetry (TART) in their approach to treating patients with manual medicine/therapy (MM/T).

Tenets of Osteopathic Medicine:[1]

1. The body is a unit; the person is a unit of body, mind and spirit.
2. The body is capable of self-regulation, self-healing, and health maintenance.
3. Structure and function are reciprocally interrelated.
4. Rational treatment is based upon an understanding of the basic principles of body unity, self-regulation, and the interrelationship of structure and function.

TART Criteria

Tissue texture changes
Asymmetric range of motion
Restricted range of motion
Tenderness

One of this chapter's goals is to present somatic dysfunction as it begins at the peripheral levels of the nervous system and describe its pathological effect on the central nervous system.[11] Furthermore, we will examine how somatic dysfunction changes physiology and the neuro-endocrine-immune system and its implications in psychology, including the development of chronic pain.

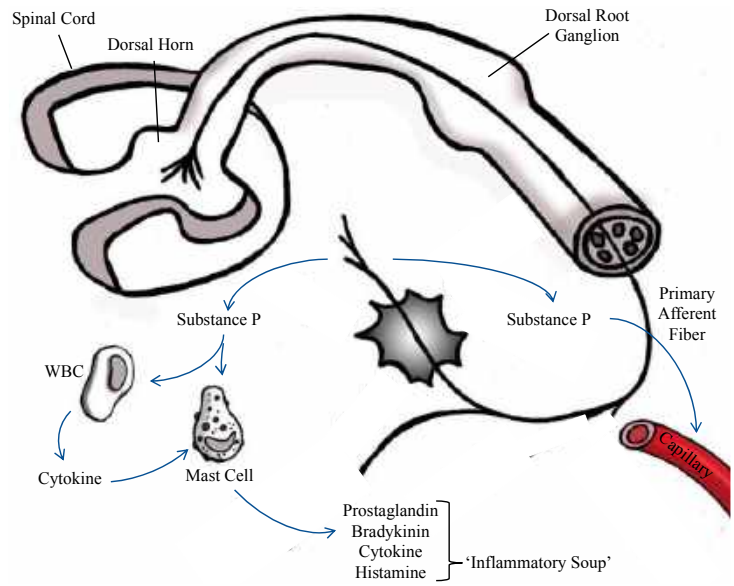
The "Osteopathic Lesion"

Denslow[2] demonstrated how somatic dysfunction has an underlying "sterile inflammation" (Figure 1). In the study, Denslow palpated subjects for somatic dysfunction through TART criteria and biopsied the tissue where somatic dysfunction was detected: tissues with somatic dysfunction were found to have edema and lymphocytic infiltration. Thus, the starting point for physiological principles implicated in the production of pain and somatic dysfunction begins with a palpatory evaluation in the periphery and requires an understanding of both peripheral and central mechanisms of the nervous system.

Figure 1:[4 (pp236),10]
Tissue Injury

TART changes are a manifestation of dysfunction in the nervous system at the level of the peripheral nervous system. Figure 1 depicts tissue injury with release of substance P from primary afferent fibers. This in turn leads to an inflammatory cascade that leads to the release of prostaglandin, bradykinin, cytokines, and histamine to create an "inflammatory soup." Clinically, this is apparent through TART changes in the tissue.

Figure 1



Peripheral Nervous System:

A-afferent system
- A-alpha fibers
- A-beta fibers

B-afferent system
- A-delta
- C-fibers

The Peripheral Nervous System

The peripheral nervous system (PNS) has a direct influence on the central nervous system (CNS). The PNS consists of the A-afferent system and the B-afferent system. Generally, the fibers of the PNS are separate, except in chronic nociception that is discussed later. There are four groups of fibers, two in each group: A-alpha and A-beta in the A-afferent system or large caliber system and A-delta and C-fibers in the B-afferent system referred to as the "small caliber fiber system."

Table 1: Comparison of the Fibers of the Peripheral Nervous System

Table 1

Peripheral Nervous System

A-afferent system	B-afferent system
Large myelinated fibers (encapsulated)	Small caliber unmyelinated ("naked" nerve endings)
"Low threshold"	"High threshold" of activation
Myotactic reflex to ventral horn and dorsal column	Nociception
proprioception/discriminant touch	Necessary for establishing somatic dysfunction, spinal facilitation and its sequele leading from sterile inflammation response
(eg pacinian corpuscle, crude touch)	

A-Afferent System

The A-afferent system is a group of large, myelinated nerves with encapsulated nerve endings. It is a low threshold system, requiring low

activation energy to become operative. The function of the A-afferent fiber system is largely its involvement in proprioception, discriminate touch, and myotactic reflexes to the dorsal column (i.e., pacinian corpuscles).

A-delta and C-fibers are often referred to as the primary afferent nociceptors (PANS)

Nociception: sensation of pain

Facilitated state (aka spinal facilitation):[3(pp67)] the maintenance of a pool of neurons in a state of partial or sub-threshold excitation; in this state, less afferent stimulation is required to trigger the discharge of impulses.

B-Afferent System

The B-afferent system, in contrast to the A-afferent system, consists of small caliber, unmyelinated nerves with naked nerve endings that require a "high threshold" of activation energy. Since they are involved in the experience of pain, collectively A-delta and C-fibers are referred to as the primary afferent nociceptors (PANS). Under appropriate conditions, "high energy" stimulation results in tissue injury and ultimately nociception. As we will see, the B-afferent system is important in the maintenance of somatic dysfunction because of its ability, or tendency, to become sensitized to a lower threshold of activation known as spinal facilitation.

Small caliber fibers are almost ubiquitous: they are present wherever connective tissue is located in the body (Table 2). Note that small caliber fibers are not present on the articular surfaces of the joints, hyaline cartilage, or in the central nervous system.

Table 2

Distribution of the Small Caliber System

Connective tissue	Nerve sheath (epineurium)
Fascia	Joints (synovial sheath)
Dermis	Annulus of vertebral disk
Nerves into muscle (>50% of axons to mm. are small caliber fibers)	Meninges
Arteries, arterioles	Viscera
Fibrocartliage	Ligaments

Note: small caliber fibers not in CNS tissue, hyaline or articular cartilage

Table 2: Distribution of the Small Caliber System

Another aspect of the small caliber system, beyond its involvement in nociception, is its role in tissue homeostasis and the restoration/healing process of injured tissue.

Activation of the B-Afferent System

During tissue injury (heat, cold, or mechanical distortion as in Figure 1), small caliber fibers are activated. These fibers contain receptors sensitive to the compounds generated by membrane breakdown (i.e., the arachadonic cascade that includes the following breakdown products: prostaglandins, phospholipids, leukotrienes, serotonin, histamine, hydrogen ions, potassium, and substance P).[4(pp236)] Many of these tissue injury products are vasodilators that lead to fluid extravasation, mechanoreceptor activation, and ultimately activation of the primary afferent system. As seen in Denslow's study,

chemotactic agents attract white blood cells that produce pro-inflammatory cytokines (IL1, IL6, and TNF), further activating small caliber fibers. Lastly, the B-afferent neuron itself releases neuropeptides, substance P (of which 90% is secreted in the periphery), somatostatin, calcitonin, and C related peptide leading to an increased inflammatory response; Frank Willard, PhD, describes the concoction of these breakdown products as an "inflammatory soup."[10,12]

After an acute injury, such as a torn ligament, these inflammatory products promote tissue repair and physiological splinting through edema. However, prolonged irritation of small caliber fibers through this otherwise normal neuro-secretory function can have a damaging effect. The prolonged tissue chemistry changes that occur with inflammation may result in chronic spinal facilitation.

Peripheral Sensitization

Sensitization:[3(pp62)] an increase in central nervous system response to repeated sensory stimulation that generally follows habituation.

An experiment performed by Schmidt[5] demonstrated how the small caliber fiber system is initiated by somatic dysfunction and can lead to "sensitization" of the peripheral nervous system. In this experiment, recording electrodes were attached to a cat's dorsal and ventral spinal roots to establish how the peripheral nervous system responds to an induced somatic dysfunction. Prior to the injection of the inflammatory compound, the cat's knee was put through passive range of motion; during this motion dorsal fibers were not active (i.e., small caliber fibers were inactive on EMG). After the injection of an inflammatory compound causing a chemically-induced somatic dysfunction, the small caliber, nociceptive, primary afferents became active at the dorsal root ganglion as measured on EMG. This is known as peripheral sensitization.

Rationale to Explain Criteria of Somatic Dysfunction

Thus far, we have shown how tissue injury leads to inflammation, and how fluid extravasation into the extracellular space leads to tissue edema and thus tissue texture change, meeting the TART criteria (asymmetry, restricted range of motion, tenderness, and temperature change) seen in somatic dysfunction. We then explained how activation of the small caliber fiber system accounts for peripheral sensitization. Next, we explain the basic research that provides a scientific basis for chronic somatic dysfunction.

The Korr Model

Until this century, the prevailing theory explaining chronic somatic dysfunction was the gamma-loop theory proposed by Irvin Korr, PhD.[4(pp127-131)] Korr proposed that altered muscle spindle activity, measurable by EMG, explained the physical examination findings in patients with somatic dysfunction. This theory is based on the belief that the large A-afferent system maintains somatic dysfunction. The gamma-loop proposed that injury to the tissue drives a signal through the small B-afferent sensory fibers to the spinal cord. To protect the injured muscle, this in turn causes the large A-alpha efferent motor neuron to contract the injured muscle as well the muscles in the area. In other words, the nociceptive fibers initiate somatic dysfunction, but it is maintained by the large caliber, myelinated fibers that create a reflex loop from the injured tissue to the spinal cord and back; hence the term "gamma-loop" (Figure 2). Ultimately, this model demonstrates that a somatic structure (the portion of the nervous system under voluntary control) produces changes in other somatic structures through the nervous system, a phenomenon termed somatosomatic reflex.

Somatosomatic reflex:[3(pp49)] localized somatic stimuli producing patterns of reflex response in segmentally related somatic structures.

Figure 2

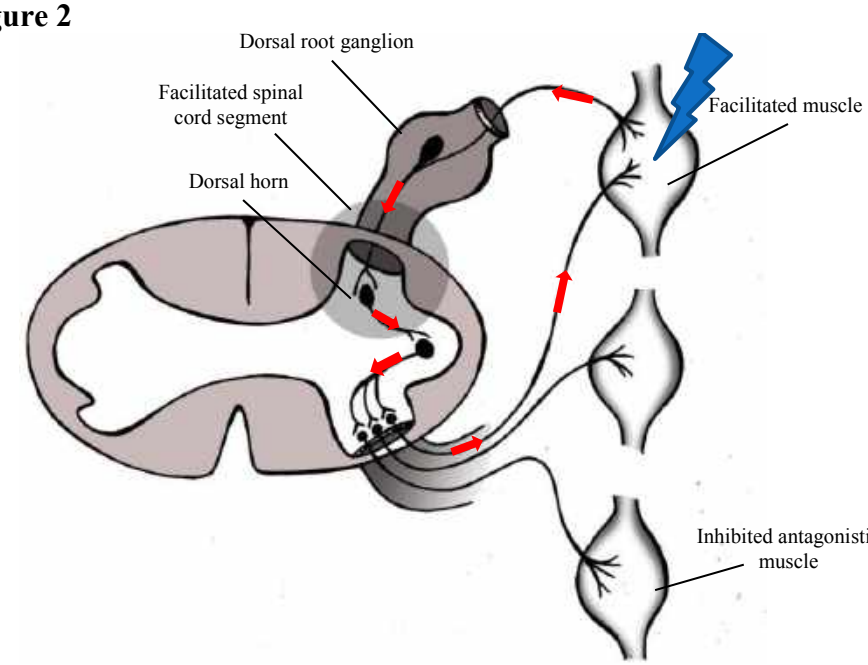


Figure 2: The "Facilitated State" of a Spinal Cord Segment due to an Injured Muscle

Note, that the injured muscle (by electrical current) sends signals to the spinal cord through small B-afferent fibers. The efferent response produces muscle hypertonicity and antagonist muscle inhibition in the surrounding muscles. Acutely, a somatosomatic response is demonstrated in a withdrawal response to a shock. Chronic somatosomatic reflexes are seen in somatic dysfunction after an acute injury leads to dorsal horn change causing "facilitation."

Korr errantly theorized chronic somatic dysfunction was initiated by the small B-afferent fibers, but was maintained by the large A-afferent fibers in a "gamma loop" illustrated by the red arrows.

Based off this model, Korr believed that somatic dysfunction became chronic when maintained by this loop. An additional observation was that a much lower energy stimulus than that of the original injury was needed to maintain the somatic dysfunction. This was termed a "facilitated state" of the CNS, or facilitation of the corresponding cord segment.

Korr Model Refuted

Korr's model was refuted by the work of Anderson[6,7] et al, who challenged the gamma-loop theory. Presumably, if Korr's model was correct, cutting the primary afferent fibers at the dorsal root would break the gamma-loop, and abolish the facilitated segment. In their experiment, Anderson et al, chemically induced somatic dysfunction in a rat's leg and measured the maximal weight that the rat could maintain with a contracted, injured limb. 48 hours after chemical injury to the rat's leg, Anderson performed rhizotomy proximal to the dorsal root, to determine if the facilitated segment persisted. The chemically injured rats, despite interruption of the afferent signal, supported 80% of their weight. Thus, it was observed that after an injury became chronic in nature, the A-alpha afferent system no longer maintained somatic dysfunction. So the question became where and what maintained the facilitated segment?

In an attempt to answer this, researchers transected the cord above the level of the facilitated segment, and no change in the somatic dysfunction was observed. When the ventral/efferent cord was destroyed at the injury level, the somatic dysfunction resolved.

A later experiment by Anderson et al, demonstrated the role of the small caliber fiber system in establishing somatic dysfunction. Dog pups were exposed to capsaicin, a toxic compound that destroys small caliber fibers. After exposure, the dog pups were unable to develop somatic dysfunction when exposed to tissue threatening stimuli.

Therefore, two conclusions came from the work of Anderson et al:

1. Small caliber, nociceptive fibers are necessary to set up spinal facilitation.
2. Once the spinal facilitation becomes chronic, afferent input no longer maintains somatic dysfunction - it is maintained in the spinal cord itself.

Central Sensitization (of the spinal cord)

Thus far, we see how facilitation is maintained at the spinal cord level of the involved segment of somatic dysfunction. So, what CNS change survived rhizotomy after chronic injury?

Excessive activation of the PANS through chronic injury leads to a loss of inhibitory regulation. When the PANS is active at the spinal cord level, a series of reactions including calcium absorption by voltage gated N-Methyl-D-aspartic acid (NMDA) receptors, activation of the phosphorylation

cascade, and altered gene expression lead to increased sensitization of the cell and activation of inhibitory neurons. Activation of the voltage gated channels is important because they can trigger gene expression in the dorsal horn, an event that is termed "activity dependent plasticity." In other words, the neuron is capable of more than just the mechanical function of depolarizing and then re-polarizing: it can make changes in gene expression that sensitize the cell.

During the excessive excitation of the spinal cord by the PANS and the accompanying calcium influx, inhibitory neurons (e.g., gamma-aminobutyric acid (GABA)) become necrotic due to excessive calcium and the ensuing free radical production. So, the loss of inhibitory function and the propagation of facilitation at the cord level results in central sensitization of the dorsal horn.

From a clinical perspective, the dorsal horn is the site of action for medications such as pregabalin and gabapentin in the treatment of chronic pain syndromes such as fibromyalgia.

What Happens When Central Sensitization Is Conducted Upwards?

Once central sensitization occurs, most of the nociceptive information travels to the brainstem, thalamus and cortex via the anterolateral system (ALS) - Figure 3.

Figure 3

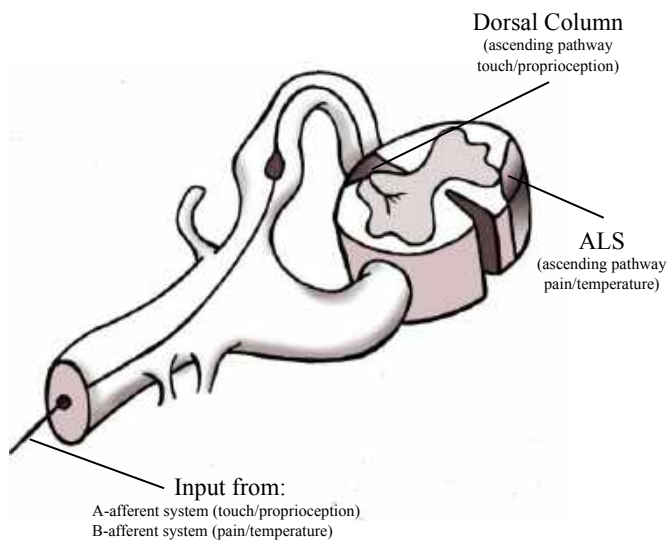


Figure 3: The Relay System of the A-afferent System (via dorsal column) and the B-afferent System (via anterolateral system).

The arousal system in the brainstem is activated through various communication relays. We are especially concerned with this area because of its implication in homeostasis. Within the brainstem, a cluster of cells called the locus coeruleus (blue spot) secretes norepinephrine in response to visual, auditory, somatic and visceral stimuli. These cells of the arousal system have projections into the cortex and hypothalamus. Recall that the hypothalamus has a direct connection to the autonomic nervous system via the brainstem and affects the endocrine system via the anterior pituitary gland. Thus, when the arousal system is activated, numerous physiological effects can be seen: increased cardiac output, respiratory rate, blood pressure, cortisol secretion and decreased gastrointestinal motility to name a few. In the short term, these responses are a part of a protective response or general adaptive response. But in the long term, this response is associated with the loss of feedback pathways and an increased susceptibility to disease (Figure 4).

Figure 4

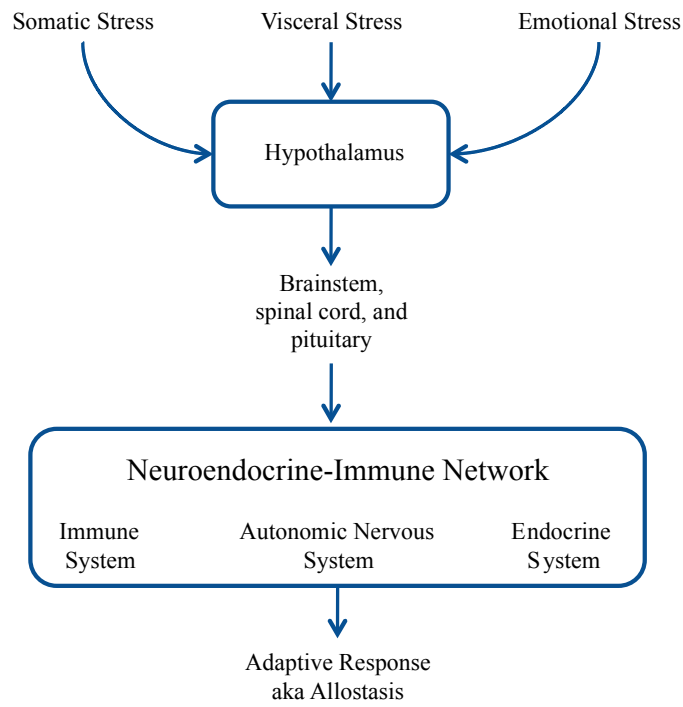


Figure 4: Adaptive Response to Stress through the Neuroendocrine Immune System – i.e., allostasis if the allostatic load is chronic.

This compensatory response, mediated largely by norepinephrine and cortisol, has been termed "allostatic load." Allostasis is the term that describes the neuroendocrine results of chronic allostatic load; it has been linked to various chronic diseases, especially cardiovascular disease that results from increased cortisol or epinephrine.[4(pp257)] Homeostasis, in contrast, includes the brief adaptive increase in cortisol secretion that occurs when undergoing a stressful event such as an examination. With the previous example in mind, consider the consequences of the increased

Homeostasis = acute adaptation to stress

Allostasis = chronic adaptation to stress

glucocorticoids that occur during a three-year residency: cardiovascular disease, weight gain, and hypertension.

As Osteopathic physicians, we are equipped with the skills to address the various aspects, visceral, emotional, and somatic, that enable the patient to optimize their healing potential. This forms the basis of the "holistic" aspect of osteopathic medicine that reinforces Stiles' model where practitioners of manual medicine treat the host component including body, mind, and spirit.

HOST + DISEASE = ILLNESS

Viscerosomatic Reflexes

We want to emphasize the importance of using these concepts clinically while recognizing the practical aspects of Osteopathic medical education. Indeed, in terms of providing a scientific understanding of the interaction of the nervous system with the viscera and soma, this is a unique contribution of osteopathy to the practice of MM/T. In addition, regarding board examinations, Savarese estimates that approximately 20% of the COMLEX questions will come from the topic of autonomic innervation.[8] We estimate that approximately 50% of all osteopathic manual medicine questions will stem from the same topic.

P-ANS does not involve skeletal muscle.

The autonomic nervous system (ANS) depicted in Figure 5 is classically divided into two classifications: parasympathetic autonomic nervous system (P-ANS) and sympathetic autonomic nervous system (S-ANS). Sympathetic responses are mediated by catecholamine release that results in papillary mydriasis, increased chronotropic and inotropic activity, smooth muscle relaxation of the lungs, glucose production by the liver, and increased blood flow to the skeletal muscle. This process is named the "fight-or-flight" response.

The P-ANS is often termed the "rest-and-digest" counterpart to the S-ANS. P-ANS innervation is almost completely visceral and does not involve skeletal muscle. Its response includes ocular miosis, salivary glandular secretion, decreased chronotropic and inotropic heart activity, bronchiolar constriction, and gastric motility and digestion. Together, these two systems must remain in balance to provide homeostasis, but may shift in favor of one or the other to support certain demands or functions of the body.

Viscerosomatic reflexes are the result of either acute or chronic irritation of visceral structures. These spinal cord reflexes are initiated by visceral organ pathology that results in somatic dysfunction of the musculoskeletal system via shared spinal cord levels. Contrast this to the somatosomatic reflexes where only myofascial elements are affected.

A classic example of viscerosomatic reflexes involves left arm and chest pain associated with myocardial infarction; upon injury, cardiogenic injury is relayed by the S-ANS to the dorsal horn. Cardiogenic nociceptive signals enter the spinal cord at levels C6-T7 but can extend as low as T9.[4(pp146)] The nociceptive signal is then sent to the brain through the ALS where pain is perceived. The efferent signal sent from the brain to the ventral cord and back through efferent routes produces the following potential effects:

1. Somatic output produces muscle spasms in, for example, the upper extremities, intercostals muscles, and paraspinal musculature involving T1-5. This is the somatic portion of the so-called viscerosomatic reflex.
2. S-ANS output from T1-5 to the heart results in the secretion of norepinephrine from the post-ganglionic neurons and results in chronotropic and inotropic increases. This is the viscero-sympathetic reflex.

Conversely, somatovisceral reflexes are autonomically mediated by somatic dysfunction that results in the pathology of one or more visceral structures via shared spinal cord levels. They may be either acute or chronic in nature. An example of a somatovisceral reflex is local brain edema causing vagal stimulation and resultant bradycardia.

We urge Osteopathic medical students to know and read the viscerosomatic reflexes for the COMLEX examination. Clinically they are relevant when the same dysfunctions reoccur despite adequate treatment, or in the classic symptoms of heart attack, gallbladder disease, kidney stones, or vagal nerve stimulation.

Figure 5[4(pp136-159), 9 (pp72-73)]

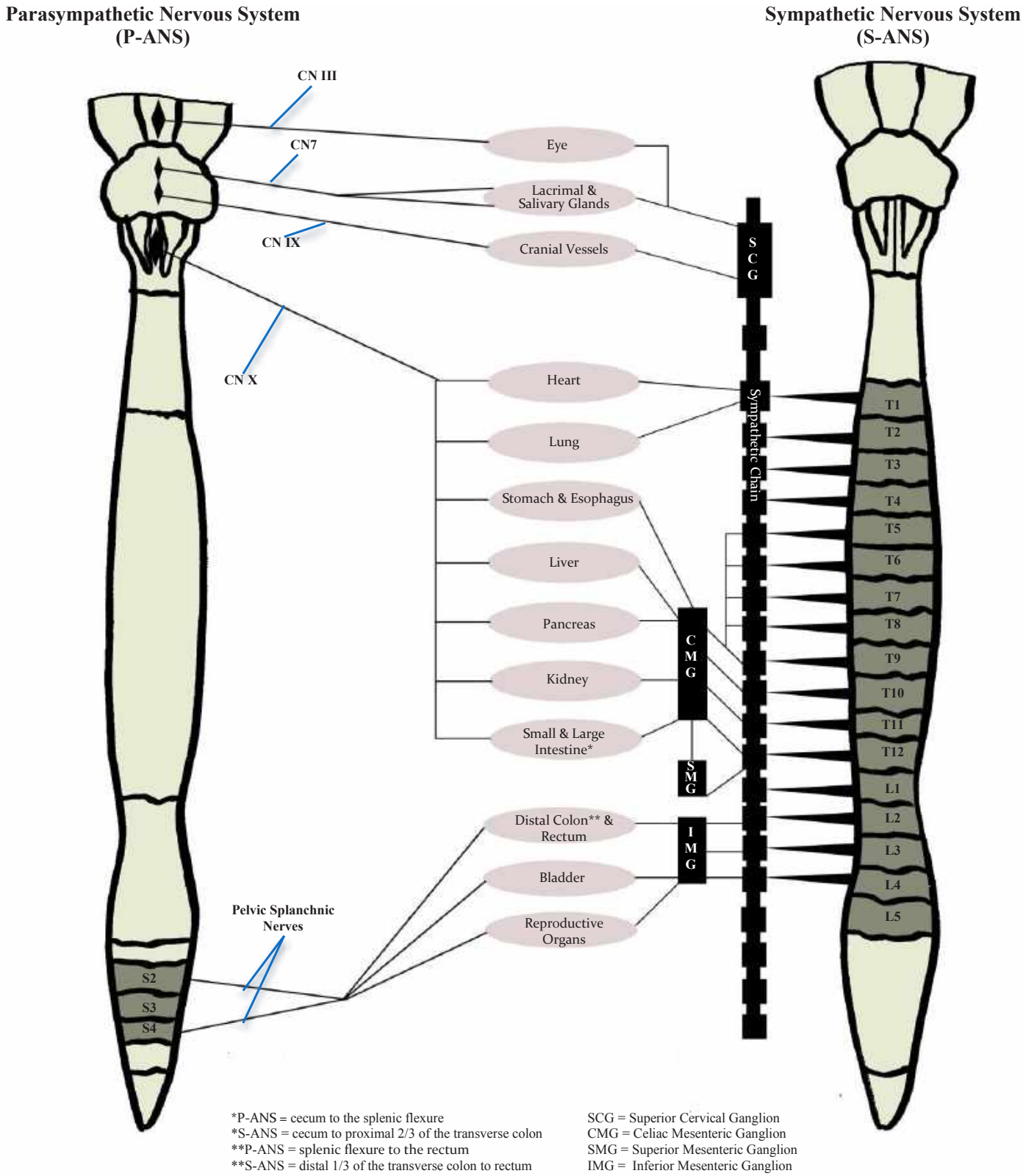


TABLE 3: Visceral organs and their parasympathetic innervation[4(pp136-159)]

Parasympathetic	Organ Affected	Response
CN3 - Ciliary Ganglion	pupil (iris) lens (ciliary muscle)	constriction – miosis bulging of lens for close vision
CN7 - Pterygopalatine/Sub-mandibular Ganglion	lacrimal gland, sublingual gland, submaxillary gland	↑secretory activity
CN9 - Otic Ganglion	parotid gland/mucous membranes mouth	↑saliva secretion
CN10 - Vagal	heart esophagus/stomach trachea, bronchi, lungs liver gallbladder/pancreas adrenal glands kidney ovaries/testes small intestine/large intestine from the cecum to the splenic flexure	↓rate, ↓contractility ↓motility constricts bronchioles bile expulsion contracts lumen, relaxes sphincters, increases motility
Pelvic Splanchnic Nerves - S2, S3, S4	large intestine from the splenic flexure to the rectum bladder penis/clitoris, uterus/prostate prostate	contracts lumen, relaxes sphincters, increases motility detrusor contraction (urination) erection

*note there is no PNS innervation to skeletal muscle

TABLE 4: Visceral organs and their sympathetic innervation[4(pp136-159)]

Sympathetic	Organ Affected	Response
T1-4 Superior Cervical Ganglion	pupil (iris) lens (Ciliary muscle) lacrimal gland, sublingual gland, submaxillary gland, parotid gland, mucous membranes mouth	dilation – mydriasis relaxation for far vision ↓tears/saliva; causes vasoconstriction of blood vessels supplying glands
T1-5	heart	↑rate, ↑contractility
T2-8	esophagus	
T2-7	lungs	dilates bronchioles
T5-7	arms	↑ blood flow
T5-9 via CG	stomach	
T6-9 via CG	liver, gallbladder, spleen	glycogenolysis, gluconeogenesis
T5-11 via CG and SMG	pancreas	↑glucagon, ↓insulin
T9-11 via SMG	small intestine (proximal duodenum from celiac ganglion, distal from SMG), large intestine from the cecum to proximal 2/3 of the transverse colon	relaxes lumen, contracts sphincters, decreases motility
T12-L2 via IMG	Distal 1/3 of the transverse colon to the rectum	relaxes lumen, contracts sphincters, decreases motility
T10	adrenal medulla	catecholamine release
T9-10	testes and ovaries	
T10-12	legs	↑ blood flow
T10-L1	cervix and uterus	
T10-L1	kidneys ureters bladder	vasoconstriction constriction urethral sphincter
T11-L2	penis	ejaculation
L1-2	prostate	

*CG = celiac ganglion, IFM = inferior mesenteric ganglion, SMG = superior mesenteric ganglion

References

1. “Tenets of Osteopathic Medicine.” American Osteopathic Association, n.d. https://osteopathic.org/about/leadership/aoa-governance-documents/tenets-of-osteopathic-medicine/.
2. Denslow JS. Pathophysiologic evidence for the osteopathic lesion: the known, unknown, and controversial. Journal of the American Osteopathic Association. 1975; 74:415-421
3. Giusti, Rebecca, DO. *Glossary of Osteopathic Terminology*. Third ed. Chevy Chase, MD: American Association of Colleges of Osteopathic Medicine, 2017.
4. Chila, Anthony G. *Foundations of Osteopathic Medicine*. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2011.
5. Schmidt, RF, Kniffki, KD, Schomburg, ED. Der Einfluss kleinkalibriger Muskeleafferenzen auf den Muskeltonus. In Bauer, HJ, WP Koella, A. Struppier: Therapie der Spastik. Verlag fur angewandte Wissenschaften, Munchen, 1981.
6. Anderson, M.F., Mokier, D.J., and Winterson, B.J. Inhibition of chronic hindlimb flexion in rat: evidence for mediation by 5-hydroxytryptamine. *Brain Research*, 1991; 541:216–224.
7. Anderson MF, Winterson BJ. Properties of peripherally induced persistent hindlimb flexion in rats: involvement of N-methyl-D-aspartate receptors and capsaicin sensitive afferents. Brain research. 1995; 678:140-150.
8. Robert G. Savarese, D.O., OMT Review, Second Edition, 1999, by Robert G. Savarese.
9. *Atlas of Anatomy Image Collection: General Anatomy and Musculoskeletal System*. Stuttgart: Thieme, 2007.
10. “Neural-Immune Interaction.” Neural-Immune Interaction - an overview | ScienceDirect Topics, n.d. https://www.sciencedirect.com/topics/neuroscience/neural-immune-interaction.
11. Nociception and Somatic Dysfunction lecture notes from Frank Willard, PhD at Kentucky College of Osteopathic Medicine 2000.

Clinical Vignette

Patient Name: AD
June 14, 2020

CC: new patient visit, gastroesophageal reflux disease (GERD)

HxCC: Patient is a 46-year-old, white, male presents as a new patient; he notes a cough for 1 month. He was recently seen at a local hospital for chest pain, had a normal electrocardiogram, serial cardiac enzymes, and myocardial perfusion stress testing. He was given an albuterol metered dose inhaler for the diagnosis of acute bronchitis with a wheeze, yet, failed to improve in his cough. He notes mild, persistent "heartburn." He has no history of seasonal allergic rhinitis nor asthma. He denies fevers, chills, ns, weight loss, history of cancer, or travel.

The patient also complains of mild thoracic pain that began around the same time. He has had similar thoracic pain in the past, when he was a competitive body builder, approximately 15-20 years ago. AD denies recent trauma to his back. He has had a chiropractor "put a rib back" in this area in the past that helped.

ROS:
Constitutional: no mental status changes, no travel or sick contacts
Eyes: no change in vision
Endocrine: no fever, night sweats, weight change
Cardiovascular: no palpitations
Respiratory: no shortness of breath nor sputum
Gastrointestinal: no vomiting, diarrhea; no change in bowel habit nor blood in stool
Genitourinary: no change in urine, no dysuria, no blood in urine
Neurologic: no anesthesia/paresthesia, no loss of consciousness nor seizures
Musculoskeletal: no weakness
Psychiatric: no homicidal or suicidal ideations, no signs of depression
Allergy: no angioedema, urticaria, anaphylaxis, no symptoms of seasonal allergies

PMHx/PSHx:
-Nasal bleeds at age 7-8 status post cautery surgery

Allergies:
-no known drug or food allergies

MEDs:
-Albuterol metered dose inhaler 2 puffs as needed every 4 hours

Immunizations:
-Tetanus booster in 2011

Social:
-no smoking; no alcohol; no drugs; work = engineer

FamHx:
Mom = none
Dad = DM2

VITALS = 6'2", 243#, 128/88, 66 beats per minute
GEN = awake, alert, oriented to time, place, situation, self, in no acute distress
HEENT = normal cephalic, a-traumatic, extraocular muscles intact, no otic/nasal discharge
NECK = supple, thyroid w/o masses
HEART = regular rate, no clicks/rubs/murmurs
LUNGS = clear to auscultation, without crackles, wheeze, or rhonchi
GI = soft, non-tender, no rebound tenderness, +bowel sounds x 4 quadrants, no hepato-splenomegaly
CV = no jugular venous distention, no carotid bruit, mucus membrane moist, no edema, 2/4 peripheral pulses, no lymphadenopathy
N/M = cranial nerves 2-12 intact, 5/5 global strength, 2/4 global reflexes, cerebellum intact, sensation symmetric, gait normal
Biomechanical = positive findings include **T5-8 NR$_R$S$_L$ rubbery in palpation**

A/P

1. Cough
   -I suspect his cough is due to chronic, mild GERD. Will stop his albuterol and have him begin a trial of omeprazole 40mg orally per day for 30 days – this will allow me to determine if his cough is due to GERD and it should resolve in 2-4 weeks.

2. Thoracic Pain
   -I suspect he is having a viscerosomatic reflex from his GERD, given no mechanism of injury (MOI) nor concomitant GERD symptoms. Typically, a group dysfunction in this area without MOI is caused by viscerosomatic reflex. I have considered OMM to this area to decrease sympathetic tone, but I realize I am treating a symptom and not the underlying problem.
   -education material on GERD with a GERD-friendly diet
   -omeprazole as above; if he fails to improve will check stool H. pylori antigen, serum lipase, and consider other etiology for his pain. Consider gastroenterology referral for evaluation of esophagogastroduodenoscopy (EGD) need.
   -follow up in 4 weeks.

   Paul Johnson, DO

Review Questions

1. Which of the following are true regarding stimulation of the vagal nerve?
    a. It will cause constriction of the pupil
    b. It will cause bronchodilation
    c. It will cause ejaculation
    d. It will cause an increase in heart rate

2. Which of the following are true regarding stimulation of the sympathetic nervous system at T6?
    a. Tachycardia
    b. Bronchodilation
    c. Increased release of stomach acid
    d. Gallbladder relaxation

3. A man presents to the ER with complaints of a racing heart. An EKG confirms supraventricular tachycardia. In which area would you expect to see somatic dysfunction causing a viscerosomatic reflex?
    a. OA
    b. C2-5
    c. T6-T8
    d. T9-11
    e. T12-L2

4. Treatment of which segments will have the least effect on pulmonary function:
    a. OA
    b. AA
    c. T1
    d. T2
    e. T3

5. Treatment of the sacrum could affect which of the following?
    a. Heart
    b. Lungs
    c. Small Intestine
    d. Descending colon

6. Treatment of the OA area could affect the following structures:
    a. Pupils
    b. Parotid gland
    c. Bladder
    d. Pancreas

7. A 38yr old female presents to your office with right-lower-quadrant abdominal pain; she has a positive McBurney's point. Which of the following areas may demonstrate a viscerosomatic reflex?
    a. T1-4
    b. T6-T9
    c. T9-T12
    d. T12-L2
    e. Sacrum

8. Sympathetic innervation to the gallbladder is through which of the following?
    a. Ciliary ganglia
    b. Celiac ganglia
    c. Superior mesenteric ganglia
    d. Inferior mesenteric ganglia

9. Parasympathetic stimulation affects which of the following?
    a. Mydriasis
    b. Salivation
    c. Bronchodilation
    d. Inotropic and chronotropic changes
    e. Colon relaxation

10. _____ influence(s) parasympathetic supply to the testicles.
    a. CN-X
    b. T9-12
    c. T12-L2
    d. S2-4

11. A chronically facilitated segment is maintained by:
    a. Afferent input by A-afferents
    b. Afferent input by B-afferents
    c. Gamma loop feedback
    d. Dorsal horn of the spinal cord

12. Nociception is transmitted to the brain through which tract?
    a. Anterolateral system of the spinal cord
    b. Dorsal horn of the spinal cord
    c. Posterior columns of the spinal cord
    d. Sympathetic chain ganglion

13. The B-afferent system innervates which of the following?
    a. Articular cartilage
    b. Central nervous system
    c. Connective tissue
    d. Hyaline cartilage

14. The B-afferent system includes fibers that have the following characteristics:
    a. Large caliber and myelinated
    b. Small caliber and myelinated
    c. Large caliber and unmyelinated
    d. Small caliber and unmyelinated

15. A 34-year-old male presents to your clinic with complaints of epigastric pain associated with nausea. He is treated with omeprazole 40mg by mouth daily and his symptoms improve. On initial presentation, a viscerosomatic reflex would be expected in which area?
    a. Right lower lumbar, L3-5
    b. Right upper thoracic, T1-3
    c. Left middle thoracic, T5-6
    d. Left lower thoracic, T10-12
    e. Sacrum

Answers:

1. A
2. D
3. A
4. C
5. D
6. D
7. C
8. B
9. B
10. A
11. D
12. A
13. C
14. D
15. C

Chapter 12: Evidence-Based Osteopathic Manipulative Treatment

Principles Taught in this Chapter:

- Evidence in support of Osteopathic Manipulative Treatment
- Analysis of Evidence Manual Medicine and Osteopathic Manipulative Treatment
- Evidenced Based Medicine Terminology

Outline:

- Early Osteopathic Research
- Musculoskeletal Disorders
- Women's Health
- Immune System Functions
- Systemic Disorders and Physiologic Functions
    - Gastrointestinal System
    - Cardiac Function
    - Respiratory Function
    - Neurological Functions
- Evidence Based Medicine Terminology
    - Types of Clinical Trials
    - Phases of Clinical Trials
    - P-Value
- Review of Clinical Trials
    - Treatment Trials
        - Example Study #1
        - Example Study #2

Chapter 12: Evidence-Based Osteopathic Manipulative Treatment

In Chapter 10 we reviewed the research supportive of the concepts of Osteopathic Cranial Manipulative Medicine (OCMM). In this chapter we review the research showing the benefit of Osteopathic Manipulative Treatment (OMT) in chronic low back pain and in musculoskeletal conditions and systemic disorders. Then we review the basic principles of Evidence Based Medicine and their application generally in the field of manual medicine and particularly in OMT.

Early Osteopathic Research

Andrew Taylor Still envisioned osteopathy as a system of healthcare for the whole person and not just as a set of techniques focused mainly on the correction of musculoskeletal disorders. Early osteopathic texts were orientated towards all the disease entities typically encountered in healthcare at that time.[1,2] Osteopathic treatment procedures were described for such disorders as hepatitis, colic, and bradycardia.[2] The McConnell text and most other publications followed in this genre, basing much of the treatment on spinal segmental lesions now called somatic dysfunction. Probably the first discussion of spinal segmental malalignment as a cause of systemic disorders was in 1892,[3] and it was expanded upon in 1899.[4]

The osteopathic medical profession has played a major role in the preservation and development of the concept of viscerosomatic/ somatovisceral interactions in health and disease. As research on this phenomenon continues in the 21st Century, the concept of a neurologically based mechanism of action presents a compelling argument for why manual medicine and manual therapy are likely to become more important in healthcare. Little has been written about where A.T. Still derived the ideas he described above, but the idea of viscerosomatic interactions has been around for millennia, first appearing in the Sanskrit writings of Sushruta around 600 BCE in Sushruta Samhita. In this ancient text Sushruta describes "Hritshoola" that literally means "heart pain" or cardiac ischemia perceived as somatic pain.[5]

As discussed in the Foreword, if the neuromusculoskeletal system is optimally aligned, then the physiology coordinated by the autonomic nervous system will work normally. This perspective underlies two of the main tenets of osteopathy:

1. The body is self-regulatory and self-healing.

2. There is a reciprocal relationship between the structure and function of the body.

Among the more influential early osteopathic researchers was Louisa Burns, DO, who published several books based on her work at the A. T. Still Research Institute. She was one of the first to empirically demonstrate in an animal model the mechanism of viscerosomatic and somatovisceral interactions. Dr. Burns surgically prepared dogs and stimulated their stomachs. She observed contraction of the spinal muscles near the sixth to the ninth thoracic vertebrae; stimulation of the tissues near the fifth to the eighth thoracic vertebrae was followed by muscular and secretory activity in the stomach, and stimulation near the eighth to the twelfth thoracic vertebrae was followed by activity of the intestines.[6]

Along with Burns' groundbreaking work on the autonomic nervous system and its interaction with visceral functions, other significant osteopathic research projects involving large scale patient populations were conducted and have been largely overlooked by the mainstream of medical and scientific research. An observational study published in 1920 showed that during the 1917-18 Spanish flu epidemic 110,120 patients receiving OMT had a mortality rate of only 0.25% while the mortality rate due to influenza in patients receiving traditional medical care was ultra-conservatively estimated to be 5% to 6%. Among patients with pneumonia treated medicinally, mortality was estimated at 33% and even as high as between 68% and 78% in some large centers. The death rate due to pneumonia among 6258 patients cared for by osteopathic physicians was 10%.[7]

As suggested below, OMT has much to offer in the care of women's health. An early example was a compilation of data by S. V. Robuck, DO, at the A.T. Still Research Institute. Jones[8] reported that 13,816 pregnant women received prenatal OMT and when delivered by osteopathic physicians had a mortality rate of 2.2 per thousand living births. This compared with a rate of 6.8 deaths per thousand mothers in the group that did not receive OMT, as reported in the government bulletins of the time.

There are other examples of high-quality osteopathic research done in the 20th Century, but the above citations illustrate that the osteopathic professionals of the time were laying down a foundation of research. Given space constraints, the following sections are intended to highlight the research progress in a number of areas and suggest directions for future research.

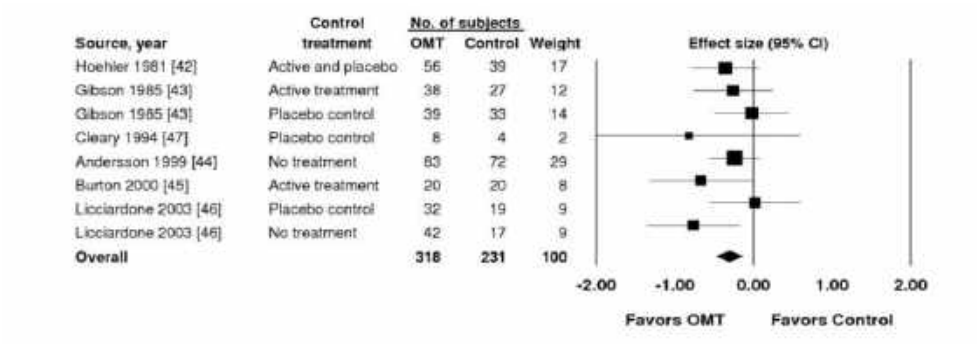
Musculoskeletal Disorders

Since the days of the "lightning bonesetters," (a phrase A.T. Still once used to advertise his services), the treatment of bodily pain and restricted

movement has been the predominate reason to seek osteopathic care.[9,10] A Cochrane Review level study was published by Licciardone et al. in 2005 that presents "proof" of the benefit of OMT in the care of chronic low back pain.[11] This systematic review and meta-analysis was based on randomized clinical trials on treatment of the low back by Hoehler et al.,[12] Gibson et al.,[13] Cleary and Fox,[14] Andersson et al.,[15] Burton et al.,[16] and Licciardone et al.[17] Figure 1 presents the meta-analysis that shows overall benefit for OMT (P = .001).

Figure 1: Effect size for low back pain.

Figure 1



Effect size for low back pain. CI denotes confidence interval; OMT, osteopathic manipulative treatment. Overall effect size, -0.30; 95% CI, -0.47 – -0.13; P = .001.

Virtually all educational texts on OMT, including this one, illustrate OMT procedures for every part of the neuromusculoskeletal system. The list of procedures in Osteopathic literature, including this text, covers the whole body and discusses many of the extant OMT procedures. Indeed, the manual medicine known as OMT is the most comprehensive of any of the professions that use their hands in healthcare.

Randomized controlled clinical trials have been conducted that support the benefit of OMT in: neck pain (McReynolds and Sheridan[18] and Schwerla et al.[19]) shoulder pain (Knebl et al.[20]) carpal tunnel syndrome (Sucher[21]) and ankle sprains (Eisenhart et al.[22]). But above all, OMT in the treatment of low back pain has been the most thoroughly studied.

One of the largest low back pain clinical trials was the UK Beam trial that had an N = 1334. It compared exercise classes and spinal manipulation (by osteopaths, chiropractors and physical therapists) and a combination of both. The outcome was that relative to standard care, manipulation showed moderate benefit at 3 months and a small benefit after 12 months, thus the benefit of spinal manipulation was supported by the UK BEAM Trial Team,[23] and Vogel et al.[24]

Another large randomized controlled trial by Licciardone et al.[25] compared OMT to sham OMT and to both real and sham ultrasound in a 2 x 2 factorial design. Six treatment sessions were provided over 8 weeks and reviewed at 12 weeks for outcome measures. Patients receiving OMT were more likely to achieve moderate improvements in low back pain ($P = .002$) than patients receiving sham OMT. These results demonstrate that OMT met the Cochrane Review Group criteria for relieving chronic low back pain, yielding a "medium" effect size.

Based in large part on the Licciardone et al.[11] meta-analysis and to a degree on the UK BEAM trial,[23] practice guidelines for treatment of chronic low back pain were published in 2010 by both the American Osteopathic Association Clinical Guideline Subcommittee on Low Back Pain[26] and the US Agency for Healthcare Research and Quality.[27] The implication of these guidelines is that OMT should be included in the treatment plan for patients with chronic low back pain. It is now appropriate to say that there is "proof" that OMT is beneficial in the treatment of chronic low back pain. In fact, the NIH Center for Complementary and Integrative Health no longer accepts research proposals for clinical trials on the effectiveness of OMT as it is considered a proven modality.

Since this text is an introduction to OMT for learners who have not been to Osteopathic medical school or have not utilized OMT in their medical practice, the emphasis is on learning the basics of manual palpatory skill and application to improve the alignment of the musculoskeletal system. But it also may be useful to know that broader benefits of OMT have been reported in the literature.

Women's Health

As previously noted, early osteopathic research included a large-scale observational study on the relationship between OMT and obstetrical outcomes. This is an area where the application of OMT offers much promise for continued research, as other early osteopathic research supports the potential for benefit in the obstetrical field. Whiting[28] reported that prenatal OMT reduced the time of labor in both prima-gravida and multiparous women; also, Hart[29] reported shorter labor times and a reduced need for the use of forceps in deliveries if the woman received prenatal OMT.

Recent obstetrical research has studied prenatal OMT. King et al.[30] reported, in a case control study, that women who received prenatal OMT had significantly fewer instances of preterm delivery and meconium-stained amniotic fluid. Guthrie and Martin[31] reported significant reduction in lumbar area pain during labor with OMT. Licciardone et al.[32] found that women

receiving prenatal OMT during the third trimester did not deteriorate in the functions measured by the Roland-Morris Disability Questionnaire; whereas, women receiving either sub-therapeutic ultrasound or standard care only significantly worsened related to the OMT group.

The PROMOTE study[33] was a randomized controlled trial with 400 women treated during the third trimester with OMT, placebo ultrasound, or standard care only. The results showed significant treatment effects with reduced functional deterioration and pain for both the OMT and placebo ultrasound groups compared to the standard care only group ($P < .001$).

Another promising area of research on women's health is in the treatment of primary dysmenorrhea. In a small clinical trial, Boesler et al.[34] demonstrated support for the hypotheses that OMT reduced low back pain and menstrual cramping. In a pragmatic, treat what you find, randomized controlled OMT trial, Schwerla et al.[35] found that women receiving OMT had significant reduction in pain and improved quality of life as compared to the control group. OMT has also been shown effective in reducing urinary tract symptoms[36] and pelvic pain in women.[37]

Immune System Functions

Some of the OMT procedures described in the Rib Cage Chapter were used by osteopathic physicians during the 1917-18 influenza epidemic, as described above. The Spanish Flu, as it was called, resulted in approximately 50 million deaths (recent estimates are as high as 100 million deaths) around the world, with about 675,000 Americans among that number.[38]

In the early 1930s, Castlio and Ferris-Swift[39,40] reported immune system enhancement utilizing a procedure called the "splenic pump." By using this procedure, they demonstrated an 80% increase in leucocytes, a 95% increase in opsonization, an 80% increase in IgM and agglutination, and a 90% increase in complement activity. These earlier studies provided a very promising line of research demonstrating the affects of the thoracic lymphatic pump on humans and the abdominal lymphatic pump on animals.

Measel[41] applied thoracic lymphatic pump treatment twice a day for 7 days following pneumococcal immunization and found that 14 days after immunization, antigen specific Ab titers were significantly increased when compared to the no treatment subjects. Jackson et al.[42] administered thoracic lymphatic pump treatment 3 times a week for 2 weeks after Hepatitis B vaccination and found significant increases in Hepatitis B titers by week 13 after immunization. Other human studies show thoracic lymphatic pump enhancement of both basophilia[43] and flu vaccinations.[44]

Systemic Disorders and Physiologic Functions

The following studies are very briefly described and cited here to give the reader some appreciation for the potential of the application of OMT.

Gastrointestinal System

Postoperative Ileus is the uncomplicated ileus that occurs after surgery and generally resolves spontaneously in 3 to 4 days. There are three studies showing the impact of OMT on postoperative ileus. Hospital stays were shortened compared to control groups for patients with postoperative ileus.[45-47] Hospital stays were also shortened in a small group of patients hospitalized with pancreatitis.[48]

Cardiac Function

Heart rate variability was improved by OMT.[49,50] In a hospital based random controlled trial, compared to a control group that did not receive OMT, O-Yurvati et al.[51] reported significantly improved hemodynamic function after coronary artery bypass graft surgery if OMT was applied post surgery.

Respiratory Function

A large clinical trial was done on the treatment of pneumonia in the elderly called the Multicenter Osteopathic Pneumonia Study in the Elderly (MOPSE).[52] The MOPSE study was based on two preliminary studies[53,54] that found a significant reduction in the prescription of oral antibiotics in hospitalized elders with pneumonia, as well as a significantly shorter hospital stay in the OMT group. While the MOPSE study did not demonstrate a dramatic decrease in the length of stay for those in the OMT protocol, there was a decrease in mortality.

Asthma treatment in the pediatric population was shown to be more effective, with significantly improved peak expiratory flow after OMT.[55,56] Patients hospitalized for gall bladder surgery who received thoracic lymphatic pump treatment returned to preoperative forced vital capacity (FVC) and forced expiratory volume in one second (FEV1) level faster than control group.[57]

Neurologic Functions

Several studies have shown the benefit of OMT in improving balance and equilibrium in healthy elders[58] and in patients diagnosed with dizziness.[59]

When utilizing cranial manipulation, Cutler et al. (2005) reported reductions in sleep latency and in sympathetic muscle activity. There is much interest in applying OMT, especially cranial manipulation, in cases of post-concussion syndrome and traumatic brain injury. Besides the two studies cited above suggestive of the possible application in nervous system disorders, there is research showing the benefit of OMT in head pain and migraine cases,[60-62] and in the treatment of Parkinson's disease.[63]

Evidence Based Medicine Terminology

Evidence based medicine (EBM) is a term used to describe statistical analysis of the scientific method as it pertains to the medical field and its use in clinical practice. The Centre for Evidence Based Medicine defines it as "the conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients."[64] EBM has been made popular in recent times by Dr. Archie Cochrane through his book *Effectiveness and Efficiency: Random Reflections on Health Services*. Cochrane's epidemological work led to the formation of the Cochrane Collaboration that provides systematic reviews of healthcare interventions. Thus, the Cochrane Collaboration provides meta-analysis of multiple similar studies to establish a comprehensive assessment on topics such as the efficacy of manual medicine for back pain. The following are some examples of EBM statistical analysis terms:

Sensitivity is the proportion of actual positives that are correctly identified; mathematically it is expressed in Figure 2. Despite misconceptions, sensitivity is NOT true positives. Specificity is the proportion of negatives that are correctly identified as expressed in Figure 3; it is NOT just true negatives.

Figure 2

$$\text{Sensitivity} = \frac{\text{Number of True Positives}}{\text{Number of True Positives} + \text{Number of False Negatives}}$$

Figure 2: Sensitivity[66]

Figure 3

$$\text{Specificity} = \frac{\text{Number of True Negatives}}{\text{Number of True Negatives} + \text{Number of False Positives}}$$

Figure 3: Specificity[66]

Too often in clinical conversation, the terms sensitivity and specificity are used interchangeably and thus incorrectly. Mathematically they are not the same, and they must be understood correctly to make good clinical decisions.

Gold Standard Test: a stand-alone test that makes a diagnosis

Pretest Probability: the probability of the sought disorder before a diagnostic test result is known.

Sensitivity and specificity are clinically useful when comparing one test against a "gold standard test." A gold standard test is a stand-alone test that makes a diagnosis. Ideally, a gold standard test is performed on every patient when confirming a diagnosis, but often that is not cost effective, convenient, or safe. For example, to determine the sensitivity and specificity of a D-dimer in the detection of a deep venous thrombosis (DVT), it must be compared to venography, the gold standard.

Prior to continuing, the concept of pretest probability requires discussion. Pretest probability uses the patient's chief compliant, history of chief complaint, past medical history, medications, social history, and family history to determine the probability of a disorder before a diagnostic test result is known. For example, performing a Lachman test to detect an injury of the anterior cruciate ligament (ACL) in a patient with the absence of knee pain or injury makes little sense because the patient has a very low pretest probability of ACL injury. Conversely, a negative or positive Lachman test would have high value in a patient with knee effusion following an injury incurred during a football game.

The concepts of sensitivity, specificity, and pretest probability are best illustrated in the following clinical scenario:

JD is a 29-year-old male that presents to the emergency room with complaints of right leg pain. He has just arrived in Portland, OR after a 6-hour flight from Washington, D.C. He has no history of trauma to his knee, no knee pain, is a nonsmoker, and has no risk factors for clotting disorders. He has no edema and no palpable phlebitis. The physician recognizes that the risk of an ACL injury is extremely low due to the absence of knee pain and injury mechanism; he foregoes a Lachman test. He however recognizes JD has risk of right leg venous thrombosis (DVT) due to his plane ride.

While JD's risk of DVT technically falls in the category of low pretest probability (his only risk factor is the plane flight), the ER physician realizes that a missed diagnosis of a DVT could result in a life-threatening pulmonary embolism. Detection of an ACL injury should follow more potentially catastrophic causes of leg pain. The physician appropriately decides to order a Nyco-Card D-dimer as a screening test for the detection of a DVT.

The following worked example (Figure 4) compares the sensitivity and specificity of D-dimer detection using the Nyco-Card semi-quantifiable method[69] when compared to the gold standard venography test.

Figure 4

		Patients with DVT Detected by Venography: Positive Venography	Patients with DVT Detected by Venography: Negative Venography	
Nyco Card D-Dimer	Positive Nyco Card	TP =98	FP = 69	PPV = TP / TP + FP = 98 /(98 + 69) = 98 / 167 ≡ 59%
	Negative Nyco Card	FN = 2	TN = 31	NPV = TN / TN + FN = 31/(31 + 2) = 31/33 ≡ 94%
		Sensitivity = TP / TP + FN = 98 / (98+ 2) = 98/100 ≡ 98%	Specificity = TN / FP + TN = 31 / (31 + 69) = 31/ 100≡ 31%	

Figure 4: DVT detection of Nyco Card compared to the gold standard of venography[65]

FP = false positive
FN = false negative
TP = true positive
TN = true negative
PPV = positive predictive value
NPV = negative predictive value

Overall, the of Nyco Card D-dimer is 98% sensitive and 31% specific. This means that the test will detect 98% of DVT (venography detects 100%) when the test is positive. While the test does an excellent job of detecting DVT, it also produces a high proportion of false positive results (i.e., positive Nyco Card D-dimer with negative venography). Comparing the true positive results to the false positive yields a mathematical concept termed positive predictive value (PPV) seen in Figure 5.

Figure 5

$$PPV = \frac{\text{Number of True Positives}}{\text{Number of True Positives + Number of False Positives}}$$

Figure 5: Positive Predictive Value (PPV)[66]

Revisiting our previous clinical scenario, a positive D-dimer does not necessarily indicate that JD has a DVT, because the Nyco Card test has a high proportion of false positives, as indicated by a PPV of 59%. Because JD has a low pretest probability, a positive D-dimer should prompt a confirmatory test such as venography or ultrasound directed at detection of DVT prior to treatment with an anticoagulant.

Suppose JD has a negative D-dimer; how would this change the clinical scenario? The specificity of the Nyco Card D-dimer is 31%, however in the setting of a low pre-test probability, this negative test result has great value (Figure 6).

Figure 6

Figure 6: Negative Predictive Value (NPV)[66]

$$\text{NPV} = \frac{\text{Number of True Negatives}}{\text{Number of True Negatives} + \text{Number of False Negatives}}$$

The negative predictive value (NPV) compares the proportion of patients with negative test results who are correctly diagnosed. Using JD's scenario, a negative Nyco Card D-dimer would likely indicate the absence of DVT since the NPV is 94% and he has low risk for DVT.

Using a similar situation, consider JD's brother, TL, who was on the same trip:

TL is a 45year old male that presents to the emergency room with complaints of right leg pain. He has just arrived to Portland, OR after a 6 hour flight from Washington, D.C. He has a history of trauma to his leg one week ago and a large contusion overlying his calf area. He is in considerable pain and obvious pitting edema is present from his thigh to his ankle. He has a history of small cell carcinoma and is a twenty-pack/year smoker. The physician palpates phlebitis in his thigh and calf.

The ER physician recognizes that TL has a high pretest probability of DVT and orders a D-dimer as a screening test for the detection of a DVT. The result is negative.

Posttest Probability: the probability assigned after the relevant evidence is taken into account.

This is the dilemma of clinical medicine; a negative screening test with a high suspicion for DVT. Before proceeding, consider the concept of posttest probability. Posttest probability is the probability allocated after the relevant evidence is taken into account. In this situation, the posttest probability is the chance that a DVT is present despite a negative D-dimer.

With a negative D-dimer, the posttest probability of a DVT in JD, a low risk patient, is 0-3% (note: calculation not provided for low risk, post-test probability). In a high-risk patient, such as TL, the posttest probability is up to 20%), despite a negative D-dimer (note: calculation not provided for high-risk, post-test probability). In JD's clinical scenario, a negative D-dimer should effectively rule-out DVT and no further work-up is warranted due to a low post-test probability. A negative D-dimer in TLs situation leaves a 1 in 5 chance of a potentially life-threatening DVT, thus, a negative

test does not offer clinical value. A physician who understands that TL has a high pretest probability of DVT will likely forgo a D-dimer test and order a more specific (and usually more expensive) screening test such as DVT-ultrasound.

Confused??? The flow chart in Figure 7 gives a conceptual framework for using tests in the determination of a diagnosis.

Figure 7

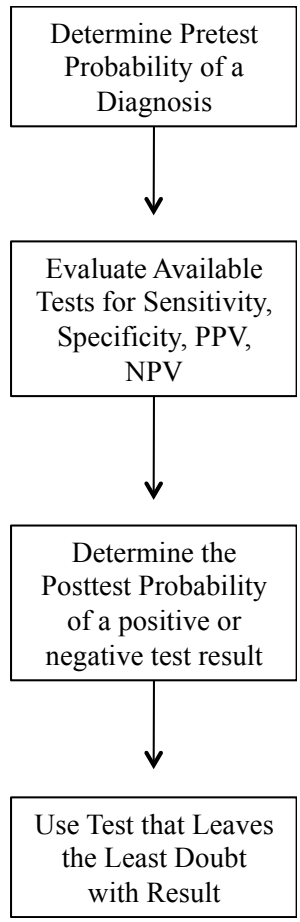


Figure 7: Methodology for Test use in Clinical Practice

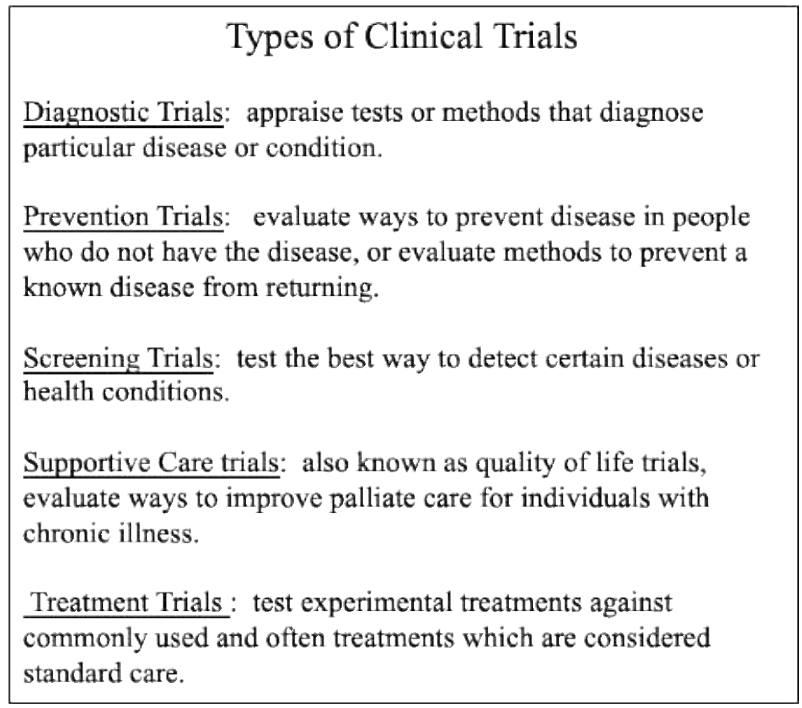
Experienced practitioners realize that physical exam tests, lab tests, etc. mean little outside of a clinical setting. In other words, tests should confirm or deny the pathology that a physician suspects based on the patient's history.

Types of Clinical Trials

To further narrow the scope of this chapter, the following review discusses the types of medical trials. Five types of trials[70] are used for medical research as listed in Figure 8.

Figure 8: Types of Clinical Trials

Figure 8



Two trial types are of particular importance to this text - diagnostic and treatment. We have already discussed many of the important points of diagnostic trials: sensitivity, specificity, PPV, NPV, and pre- and posttest probability. When we consider the classical orthopedic methods of examining the knee and shoulder, diagnostic trials play a very important role in manual medicine. They also play an important role in demonstrating a correlation of inter-practitioner palpatory diagnosis, or diagnostic reproduction amongst practitioners. The information previously provided will aid in understanding the upper and lower extremity chapters of this text.

Treatment trials evaluate the efficacy of one treatment compared to another. In the context of the text, manual medicine treatment outcomes are compared to traditional methods of care, such as physical therapy, education, medication, surgery, etc.

An important concept when evaluating any trial is prospective analysis. Prospective analysis is the evaluation of the trial's hypothesis or endpoint. Retrospective analysis evaluates other aspects of the trial that were noted after the trial began. Traditionally, retrospective data holds less value than prospective. For instance, the Heart Outcomes Prevention Evaluation (HOPE) trial compared the efficacy of two ACE inhibitors with an endpoint of cardiovascular outcomes among high-risk patients with known cardiovascular disease. In the HOPE trial, Ramipril was retrospectively noted to prevent patients' progression to type 2 diabetes. In the author's (JB) opinion, this retrospective data does not alter practice in the prevention of

diabetes, because it was determined retrospectively. Had the study been originally designed to evaluate Ramipril's effect on the prevention of type 2 diabetes, we may have considered the data with more fervor.

Phases of Clinical Trials

In humans, clinical trials are conducted in phases. Under normal circumstances, the phases progress sequentially from I to II to III. This stepwise approach using ever increasing patient numbers allows for several advantages: if the treatment or test is harmful then fewer patients are injured. Researchers are also able to use retrospective data to help design the next phase (Figure 9).

Figure 9

Figure 9: Phases of Clinical Trials

Phases of Clinical Trials
Phase I Trials - 20-80 patients
Phase II Trials - 100-300 patients
Phase III Trials - 1,000-3,000 patients
Phase IV Trials - post marketing studies evaluate risks, benefits, and optimal use.

After a treatment or test has been researched in Phases I-III, it may undergo post-market analysis in phase IV trials. These trials evaluate risk, benefits and optimal use of the test or treatment. Examples of phase IV trials that resulted in a FDA recall include cerivastatin (Baycol) and rofecoxib (Vioxx).

P-Values

A p-value is a probability ranging from zero to one;[67] it answers the question put out by the null hypothesis. Using the previous example of D-dimer compared to venography in the detection of DVT, the null hypothesis could be represented by the following formula:[68]

$$\mu_1 - \mu_2 = 0, \text{ or}$$

$$\mu_1 = \mu_2$$

Where μ_1 is the ability of venography to detect DVT and μ_2 is the detection of DVT by D-dimer. In this instance, the assumption of the null hypothesis is that no difference exists between the two groups. Obviously, μ_1 will be larger than μ_2 and the null hypothesis will ultimately be refuted, but studies are commonly designed to refute the null hypothesis.

The p-value, in this instance, is the probability that D-Dimer is as sensitive as venography in the detection of DVT. The p-value is thought to be the hypothetical reproduction of the study; it is NOT, however, the probability that the null hypothesis is true.

Commonly, the accepted limit of a p-value is less than 0.05. Using the same DVT example, a p-value of <0.05, that refutes the null hypothesis, indicates that if 20 similar studies were done under the same circumstances, 19 of them would show a statistical difference between D-dimer and venography in the detection of DVT, and that one study would show no difference.

There is no hard-fast rule over the set limit of a p-value; some studies set the value at a $p<0.1$. If this were true in the same example, then if 10 similar studies were done under the same circumstances, 9 of them would show a difference between D-dimer and venography in the detection of DVT, and one study would show no difference. For most clinicians, the limit of 10% reproducibility is too high, and thus the common threshold of acceptance is a p-value of <0.05, or 5%.

Prior to a clinical trial, if the limit is set on a p-value to <0.05, and the p-value is determined to be .35 at the trial's conclusion, then the null hypothesis is said to be accepted. Using the same example, we would say that there is no statistical difference between DVT detection using venography or D-dimer,[69] and this is called the alternative hypothesis.

Listed below are six common misconceptions of p-values; these are FALSE beliefs listed to prevent error in understanding:[71]

- The p-value is NOT the probability that the null hypothesis is true.
- The p-value is NOT the probability that a finding is a chance.
- The p-value is NOT the probability of falsely rejecting the null hypothesis.
- The p-value is NOT the probability that a replicating experiment would NOT yield the same conclusion.
- 1 − p-value is NOT the probability of the alternative hypothesis being true.

- The clinical significance of the test is NOT determined by the p-value. The statistical significance level of a test is a value that should be decided upon by the agent interpreting the data before the data are viewed. It is compared against the p-value or any other statistic calculated after the test has been performed. The p-value does not indicate the size or importance of the observed effect.

Review of Clinical Trials

Treatment Trials

The following text is a review of two treatment trials. These reviews were performed by the author JB and are given as examples for beginning students to model their own literature reviews. As in the DVT Diagnostic trial previously discussed in this chapter, the main points are highlighted for easy review and later access.

Example Study #1

"Effects of Clopidogrel in Addition to ASA in Patients with Acute Coronary Syndromes Without ST-Segment Elevation," N Engl J Med. 2001 Aug 21; 345(7):494-501.

Study Objective: to determine if ASA combined with clopidogrel provides benefits of decreased mortality and morbidity for pts with NSTEMI versus treatment of ASA alone.

Study Design: randomized, double blind, placebo-controlled, prospective, 12,562 patients, begun December 1998-September 2000 at 482 centers in 28 countries; Canadian Cardiovascular Collaboration Project Office, McMaster University, Hamilton, Ontario headed project.

Patient Inclusion: patients hospitalized within 24hrs of symptoms onset of NSTEMI that did not have an ST-elevation MI.

Patient Exclusion: patients with potentially high bleeding risk, contraindications to antithrombotic or antiplatelet therapy, patients taking oral anticoagulants, patients who had undergone coronary revascularization in last 3months, patients who had received IIb/IIIa receptor inhibitors in the last 3days.

Group Randomizations: Two groups randomly selected and followed for 3-12 months (mean 9 months):

- 6303 pts: ASA 75 to 325mg po qday + placebo
- 6259pts: ASA 75 to 325mg po qday + Clopidogrel 300mg po loading dose followed by 75mg po qday

Study Checkpoints/Internal Review: at discharge, 1 month, 3 months, 6 months, 9 months, 12 months.

Primary End Points: death from cardiovascular cause, nonfatal myocardial infarction, ischemic chest pain, Troponin/CK-MB/other at least 2x upper limit of normal or 3x upper limit of normal after reperfusion, EKG Changes, CVA, new neurological change lasting >24hrs, intracranial hemorrhage, ischemia seen on CT or MRI.

Secondary End Points: severe refractory ischemia, heart failure, need for revascularization.

Safety End Points: life-threatening bleed, major bleed (requiring two or more units transfused), minor bleed (no transfusion).

Studies' Conclusion: "Clopidogrel significantly reduces the risk of the composite outcome of death from cardiovascular causes, nonfatal MI, or CVA as well as a range of related ischemic events. The use of the drug, in addition to ASA is associated with an increased risk of bleeding."

Author's (JB) Review of Article:
Q: Were all patients who entered the trial accounted for at its conclusion, and were they analyzed in the groups where they were randomized?
A: YES, the 6 lost in clopidogrel group and 7 lost in placebo group were lost in initial follow-up (99.9% of patients accounted for).

TABLE 1. BASE-LINE DEMOGRAPHIC CHARACTERISTICS, MEDICAL HISTORY, ELECTROCARDIOGRAPHIC CHANGES, AND DRUG THERAPY.*

CHARACTERISTIC	CLOPIDOGREL GROUP (N=6259)	PLACEBO GROUP (N=6303)
Age — yr	64.2±11.3	64.2±11.3
Female sex — no. (%)	2420 (38.7)	2416 (38.3)
Time from onset of pain to randomization — hr	14.2±7.2	14.1±7.1
Heart rate — beats/min	73.2±14.8	73.0±14.6
Systolic blood pressure — mm Hg	134.4±22.5	134.1±22.0
Diagnosis at study entry — no. (%)		
Unstable angina	4690 (74.9)	4724 (74.9)
Suspected myocardial infarction	1569 (25.1)	1579 (25.1)
Associated myocardial infarction — no. (%)†	1624 (25.9)	1659 (26.3)
Medical history — no. (%)		
Myocardial infarction	2029 (32.4)	2015 (32.0)
CABG or PTCA	1107 (17.7)	1139 (18.1)
Stroke	274 (4.4)	232 (3.7)
Heart failure	462 (7.4)	492 (7.8)
Hypertension	3750 (59.9)	3642 (57.8)
Diabetes	1405 (22.4)	1435 (22.8)
Current or former smoker	3790 (60.6)	3841 (60.9)
Electrocardiographic abnormality — no. (%)‡		
Any	5863 (93.7)	5921 (93.9)
ST segment		
Depression ≥1 mm	2642 (42.2)	2646 (42.0)
Elevation ≤1 mm	203 (3.2)	199 (3.2)
Transient elevation >2 mm	38 (0.6)	37 (0.6)
T-wave inversion		
Major (≥2 mm)	1589 (25.4)	1635 (25.9)
Other (<2 mm)	721 (11.5)	713 (11.3)
Other	670 (10.7)	690 (10.9)
Medications at time of randomization — no. (%)		
Aspirin	4168 (66.6)	4134 (65.6)
Heparin or LMW heparin	4522 (72.3)	4605 (73.1)
ACE inhibitor	2347 (37.5)	2309 (36.6)
Beta-blocker	3678 (58.8)	3690 (58.5)
Calcium-channel blocker	1784 (28.5)	1771 (28.1)
Lipid-lowering agent	1599 (25.6)	1586 (25.2)
Intravenous nitrate	2836 (45.3)	2906 (46.1)

*Plus–minus values are means ±SD. CABG denotes coronary-artery bypass grafting, PTCA percutaneous transluminal coronary angioplasty, LMW low molecular weight, and ACE angiotensin-converting enzyme.

†An associated myocardial infarction was defined as a myocardial infarction associated with the episode of pain that occurred before randomization.

‡Data on the particular type of abnormality were missing for one patient in the placebo group.

Table 1: Base-Line Demographic Characteristics, Medical History, Electrocardiographic Changes, and Drug Therapy.

Q: Were patients and clinicians kept "blind" to what treatment was being received.

A: YES, study was double blinded, placebo controlled.

Q: Aside from the experimental treatment, were the groups treated equally?
A: YES, no therapeutic option was withheld from either group for the sake of the study.

Absolute Risk Reduction: Control Event Rate – Experimental Event Rate

Absolute Risk Reduction (ARR)
(Control Event Rate – Experimental Event Rate) =
11.4% - 9.3% = 2.1%

Number Needed to Treat: 1/Absolute Risk Reduction

Number Needed to Treat (NNT)
1 / ARR = 1 / 2.1% = 47.6 patients for 1 year.
Typically, in an intervention study the treatment is worth employing if the NNT is ≤ 15-20.

Table 2: Main Study Outcomes

TABLE 2. INCIDENCE OF THE MAIN STUDY OUTCOMES.*

OUTCOME	CLOPIDOGREL GROUP (N=6259)	PLACEBO GROUP (N=6303)	RELATIVE RISK (95% CI)	P VALUE
	no. (%)			
First primary outcome: nonfatal myocardial infarction, stroke, or death from cardiovascular causes	582 (9.3)	719 (11.4)	0.80 (0.72–0.90)	<0.001
Second primary outcome: first primary outcome or refractory ischemia	1035 (16.5)	1187 (18.8)	0.86 (0.79–0.94)	<0.001
Death from cardiovascular causes	318 (5.1)	345 (5.5)	0.93 (0.79–1.08)	
Myocardial infarction†	324 (5.2)	419 (6.7)	0.77 (0.67–0.89)	
Q-wave	116 (1.9)	193 (3.1)	0.60 (0.48–0.76)	
Non–Q-wave	216 (3.5)	242 (3.8)	0.89 (0.74–1.07)	
Stroke	75 (1.2)	87 (1.4)	0.86 (0.63–1.18)	
Refractory ischemia‡	544 (8.7)	587 (9.3)	0.93 (0.82–1.04)	
During initial hospitalization	85 (1.4)	126 (2.0)	0.68 (0.52–0.90)	
After discharge	459 (7.6)	461 (7.6)	0.99 (0.87–1.13)	
Death from noncardiovascular causes	41 (0.7)	45 (0.7)	0.91 (0.60–1.39)	

*The number of patients who died from cardiovascular causes or had a nonfatal myocardial infarction was 539 (8.6 percent) in the clopidogrel group and 660 (10.5 percent) in the placebo group (P<0.001; relative risk, 0.81; 95 percent confidence interval, 0.72 to 0.91). The corresponding numbers at 30 days were 241 (3.9 percent) and 305 (4.8 percent) (relative risk, 0.79; 95 percent confidence interval, 0.67 to 0.94; P=0.007). CI denotes confidence interval.

†Some patients had both a Q-wave and a non–Q-wave myocardial infarction.

‡Only the first ischemic event was counted for each patient.

Number Needed to Harm: 1/(test group harmed – control harmed)

Number Needed to Harm (NNH)
1 / difference of two groups = 1/(8.5-5) = 1/3.5% = 28.6
Thus, one in 28.6 patients treated will have a hematologic event at 1 year.

Table 3: Bleeding Complications

TABLE 3. BLEEDING COMPLICATIONS.*

VARIABLE	CLOPIDOGREL GROUP (N=6259)	PLACEBO GROUP (N=6303)	RELATIVE RISK (95% CI)	P VALUE
	no. (%)			
Major bleeding	231 (3.7)	169 (2.7)	1.38 (1.13–1.67)	0.001
Necessitating transfusion of ≥2 units of blood	177 (2.8)	137 (2.2)	1.30 (1.04–1.62)	0.02
Life-threatening	135 (2.2)	112 (1.8)	1.21 (0.95–1.56)	0.13
Fatal	11 (0.2)	15 (0.2)		
Causing 5 g/dl drop in hemoglobin level	58 (0.9)	57 (0.9)		
Requiring surgical intervention	45 (0.7)	43 (0.7)		
Causing hemorrhagic stroke	7 (0.1)	5 (0.1)		
Requiring inotropic agents	34 (0.5)	34 (0.5)		
Necessitating transfusion of ≥4 units of blood	74 (1.2)	60 (1.0)		
Non–life-threatening	96 (1.5)	57 (0.9)	1.70 (1.22–2.35)	0.002
Site of major bleeding				
Gastrointestinal	83 (1.3)	47 (0.7)		
Retroperitoneal	8 (0.1)	5 (0.1)		
Urinary (hematuria)	4 (0.1)	5 (0.1)		
Arterial puncture site	36 (0.6)	22 (0.3)		
Surgical site	56 (0.9)	53 (0.8)		
Minor bleeding	322 (5.1)	153 (2.4)	2.12 (1.75–2.56)	<0.001
Total with bleeding complications	533 (8.5)	317 (5.0)	1.69 (1.48–1.94)	<0.001

*The number of patients with bleeding that met the criteria for major bleeding established by the Thrombolysis in Myocardial Infarction trial[11] was 68 in the clopidogrel group and 73 in the placebo group (relative risk, 0.94; 95 percent confidence interval, 0.68 to 1.30; P=0.70). The number with bleeding that met the criteria for life-threatening or severe bleeding established by the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries trial[12] was 78 in the clopidogrel group and 70 in the placebo group (relative risk, 1.12; 95 percent confidence interval, 0.81 to 1.55; P=0.48). Some patients had more than one bleeding episode. CI denotes confidence interval.

Author's Conclusion (JB): This is well designed clinical trial. Clopidogrel given to patients undergoing a NSTEMI show a reduction in mortality based on the study's designated primary end points for 1 in 48 patients who receive treatment. The primary outcome may be statistically significant based on the p-value (we could reproduce this trial over 1000 times and only get a different result once); however clinically, the risks of serious life-threatening bleed outweigh the benefits.

Example Study #2

"A Comparison of Osteopathic Spinal Manipulation with Standard Care for Patients with Low Back Pain," N Engl J Med. 1999;341:1426-31

Study Objective: to determine whether osteopathic care, including manipulative therapy, would benefit patients with low back pain (LBP) of 3 week to 6 months duration when compared with standard allopathic care.

Study Design: randomized, prospective, un-blinded study, from August 1992-December 1994 at 2 HMO offices in Chicago, IL.
Patient Inclusion: LBP lasting 3 weeks to 6 months

Patient Exclusion: Nerve root compression, systemic inflammatory disorder, scoliosis, serious medical condition such as cancer, myocardial infarction, diabetic neuropathy, neurovascular disease, alcohol or drug abuse, known psychiatric or psychological illness, no identifiable "somatic dysfunction" (TART), pregnant, currently undergoing litigation, receiving worker's comp, undergone manipulation in past 3 weeks.

Group Randomizations: 178 selected patients of 1193 identified; ages 20-59yrs. Computer Randomized patients into two groups:

- 87 standard care, control group
  - standard allopathic treatment
  - analgesics (ASA, acetaminophen, codeine, oxycodone)
  - anti-inflammatory (naproxen, ibuprofen, piroxicam)
  - muscle relaxant (cyclobenzaprine)
  - Physical Therapy
  - Ultrasonography
  - diathermy (high £ current to generate heat)
  - hot/cold packs
  - corset
  - TENS

- 93 OMT intervention by three physicians from CCOM
  - "Osteopathic manipulation was applied to areas that the osteopathic physician determined to be related in some way to the patient's back; treatment was individualized."
  - Modalities of treatment included: standard allopathic treatment and high velocity-low amplitude (HVLA), muscle energy (ME), Articular, strain-counterstrain (S/CS), myofascial release (MFR).

Study Checkpoints/Internal Review: Questionnaires performed at initial visit, at all treatments, and 12 weeks.

- Visual Analogue Pain Scale
- Roland-Morris Questionnaire
- Oswestry Questionnaire
- Range of motion (ROM) testing with goniometry

Primary End Points: 12 weeks of treatment, LBP resolved prior to 12 weeks, discontinued participation. The final evaluation at the primary end point was performed by an evaluator who was blinded to the treatment assignments and not involved with either group.

Secondary End Points: none

Safety End Points: none

Visual Analogue Pain Scale (scored 0-100):
$\Delta = 26.3 \pm 24.1$ standard
$\Delta = 32 \pm 23$ OMT
$p = 0.19$

Figure 10

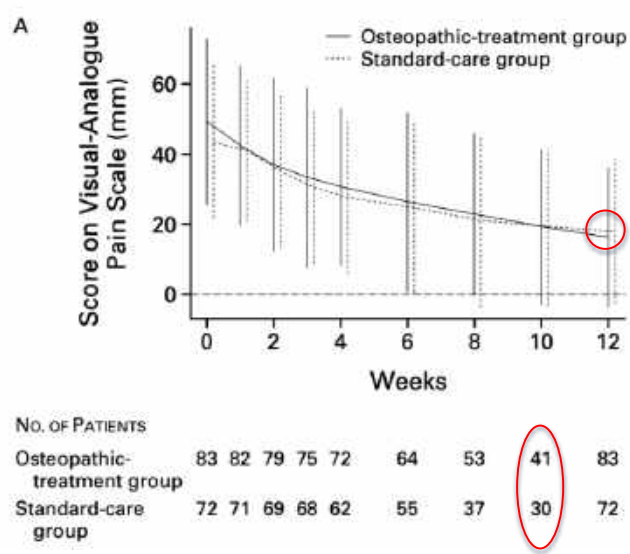
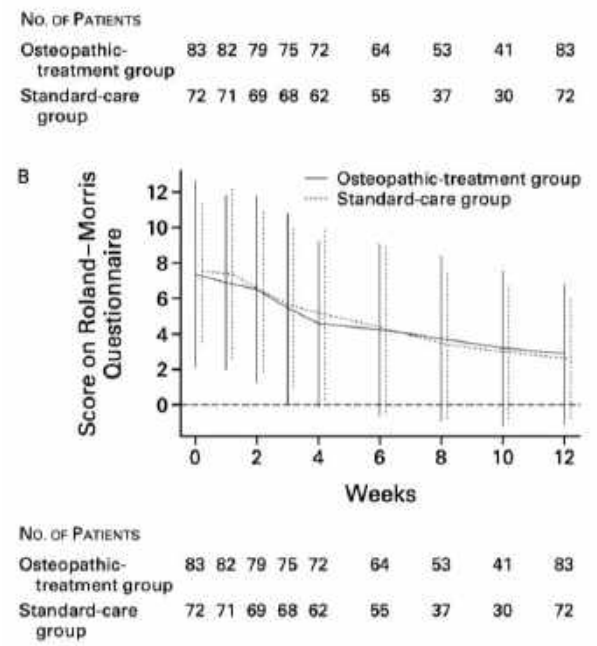


Figure 10: Visual Analogue Pain Scale.

Roland-Morris Questionnaire
$\Delta = 5$ standard
$\Delta = 5$ OMT
$p = 0.16$

Figure 11: Roland-Morris Questionnaire

Figure 11

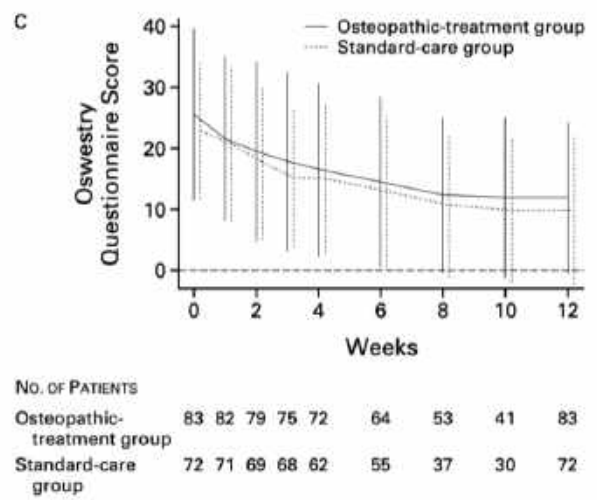


From New England Journal of Medicine, Gunnar B.J. Andersson, Tracy Lucente, Andrew M. Davis, et al, "A Comparison of Osteopathic Spinal Manipulation with Standard Care for Patients with Low Back Pain," volume 341, pp 1430. Copyright © 1999 Massachusetts Medical Society. Reprinted with permission from the Massachusetts Medical Society.

Oswestry Questionnaire
$\Delta = 12.9 \pm 13.4$ standard
$\Delta = 13.6 \pm 13.4$ OMT
$p = 0.97$

Figure 12: Oswestry Questionnaire Score

Figure 12



From New England Journal of Medicine, Gunnar B.J. Andersson, Tracy Lucente, Andrew M. Davis, et al, "A Comparison of Osteopathic Spinal Manipulation with Standard Care for Patients with Low Back Pain," volume 341, pp 1430. Copyright © 1999 Massachusetts Medical Society. Reprinted with permission from the Massachusetts Medical Society.

Goniometry:

Straight leg raise

Supine: $\Delta = 1.3 \pm 9.1$ standard
$\Delta = 2.8 \pm 9.7$ OMT
$p=0.4$

Sitting: $\Delta = 5.2 \pm 10.4$ standard
$\Delta = 6.6 \pm 12.7$ OMT
$p=0.94$

Flexion
$\Delta = 4.2 \pm 21.3$ standard
$\Delta = 1.9 \pm 22$ OMT ($p=0.64$)

Extension
$\Delta = 1.7 \pm 11.1$ standard
$\Delta = 0.8 \pm 11.9$ OMT ($p=0.65$)

Study's Conclusion: "We found no difference in clinical outcome between standard care and osteopathic care among patients with LBP of at least three weeks in duration."

Author's (JB) Evaluation of Study:

Q: Was the assignment of patients to treatments randomized? And, was the randomization list concealed?
A: Yes, by computer - list was concealed.

Q: Were all patients who entered the trial accounted for at its conclusion? And, were they analyzed in the groups where they were randomized?
A: Yes, excluded all pts from initial and final analysis if dropped out (9 in OMT, 12 in Standard).

Q: Were patients and clinicians kept "blind" to what treatment was received?
A: This was not a blinded study.

Q: Aside from the experimental treatment, were the groups treated equally?
A: Yes, no therapeutic option was withheld from either group for the sake of the study aside from OMT.

Q: Were the groups similar at the start of the trial?
A: Randomization was excellent, except for age (shown to be not significant with $p<0.091$). I believe 10yrs of age difference is important.

Q: Are the results of this therapeutic trial important?
A: Since there was no statistical significance between these groups, we cannot calculate Absolute Risk Reduction or Number Needed to Treat.

Author's (JB) Conclusion: according to this study, Osteopathic modalities provided little additional patient benefit when compared to allopathic treatments using the Visual Analogue Pain Scale, the Roland-Morris Questionnaire, the Oswestry Questionnaire, nor ROM testing with goniometry in LBP patients with duration of 3week-6months.

This study would have been improved with an increased number of patients, although by comparison, similar studies have an "N" of only 20 to 40 patients. One factor to consider in designing a future study of LBP is that 90% of LPB will improve spontaneously after three months, regardless of treatment modality.[72] Thus, a study evaluating LBP of greater than three months duration (termed "chronic LBP"), may result in a different outcome. Another important factor that could be studied in the future would be the cost and time spent in each treatment group. As practitioners of manual medicine, we understand that every patient is unique and entitled to freedom of treatment choice, but the absence of designated treatment modalities (i.e., ME, HVLA, S/CS, etc.) or of a standard treatment protocol lowers the credibility of this study. These issues continue to be debated: is it better to design a "protocol defined" study in which every subject/patient receives the same intervention or a "pragmatic study" where the intervention is to treat the unique malalignment(s) found in each individual? To date, more pragmatic studies are being accepted in peer-reviewed journals.

One final point to convey to readers is the difficulty in blinding studies when they involve a procedure. At the very best, we can only hope to blind the patient through sham-treatment and then use the results of the sham as a control treatment group. The alternative is the straightforward method of either inclusion or exclusion of manual medicine in conjunction with standard care. In contrast, blinding the physician and the patient is simple if the treatment involves a medication, as in the clopidogrel study previously reviewed.

Of interest, one bright spot did appear in the retrospective analysis of this study – a decreased use of physical therapy, NSAIDS, and narcotics. Since this was not a primary or secondary endpoint of the study, its value is somewhat diminished. When study results are in conflict with professional care, the question may arise "Do these results indicate that we should not use manual medicine in patients with LBP?" The answer in short is NO, as author HK has described above.

References:

1. Still AT. *Osteopathy: research and practice*. Originally published by the Author 1910, citation from edition published Seattle: Eastland Press, 1992 Introduction.
2. McConnell CP (Ed.). *Clinical osteopathy*. Chicago: The A.T. Still Research Institute, 1917.
3. Still AT. *Philosophy and mechanical principles*. Kirksville, MO: AT Still, 1892, pp 45-50.
4. Still AT. *Philosophy of osteopathy.* Kirksville, MO: AT Still, 1899, pp 213-220.
5. Dwivedi G, Dwivedi S. Sushruta — the Clinician — Teacher par Excellence. *Indian J Chest Dis Allied Sci.* 2007;49:243-244.
6. Burns L. Viscero-somatic and somato-visceral spinal reflexes. *J Am Osteopath Assoc.* 1907;7:51-60.
7. Smith RK. One hundred thousand cases of influenza with a death rate of one-fortieth of that officially reported under conventional medical treatment [reprint of *J Amer Osteopath Assoc*. 1920;19:172-175. *J Am Osteopath Assoc*. 2000;100: 320-323. Available at: http://www.jaoa.org/cgi/reprint/100/5/320. Accessed March 3, 2009.
8. Jones M. Osteopathy and obstetrical mortality and stillbirth and infant mortality: symposium on osteopathy in obstetrics chaired by S.V. Robuck, DO. *J Am Osteopath Assoc*. 1933;33: 350-353.
9. Trowbridge C. *Andrew Taylor Still 1828-1917*. Kirksville, MO: Thomas Jefferson University Press. 1991.
10. O'Brien J. *Bonesetters: a history of British osteopathy*. Turnbridge Wells, UK: Anshan Publishers, 2012.
11. Licciardone JC, Brimhall AK, King LN. Osteopathic manipulative treatment for low back pain: a systematic review and meta-analysis of randomized controlled trials. *BMC Musculoskelet Di*. 2005;6:43.
12. Hoehler FK, Tobis JS, Buerger AA. Spinal manipulation for low back pain. *J Am Med Assoc*. 1981;245:1835-1838.
13. Gibson T, Greahmer, Harkness J et al. Controlled comparison of short-wave diathermy treatment with osteopathic treatment in non-specific low back pain. *Lancet*. 1985; I: 1258-1261.
14. Cleary C, Fox JP Menopausal symptoms; an osteopathic investigation. *Complement Ther Med.* 1994;2**:**181-186
15. Andersson GBJ, Lucente T, Davis AM, et al. A comparison of osteopathic spinal manipulation with standard care for patients with low back pain. *N Engl J Med*. 1999;341:1426-1431.
16. Burton AK, Tillotson KM, Cleary J. Single-blind randomized controlled trial of chemonucleolysis and manipulation in the treatment of symptomatic lumbar disc herniation. *Eur Spine J.* 2000;9:202-207.

17. Licciardone JC, Stoll ST, Fulda KG, et al. Osteopathic manipulative treatment for chronic low back pain: a randomized controlled trial. *Spine.* 2003;28:1355-1362.
18. McReynolds TM, Sheridan BJ. Intramuscular Ketorolac versus osteopathic manipulative treatment in the management of acute neck pain in the emergency department: a randomized clinical trial. *J Am Osteopath Assoc.* 2005;105:57-68.
19. Schwerla F, Bischoff A, Nürnberger A, Genter P, Guillaume JP, Resch KL. Osteopathic treatment of patients with chronic non-specific neck pain: a randomized controlled trial of efficacy. *Forsch Komplementmed.* 2008;15:138-145.
20. Knebl JN, Shores JH, Gamber RG, et al. Improving functional ability in the elderly via Spencer technique, an osteopathic manipulative treatment: a randomized controlled trial. *J Am Osteopath Assoc.* 2002;102:387-396.
21. Sucher BM. Palpatory diagnosis and manipulative management of carpal tunnel syndrome. *J Am Osteopath Assoc.* 1994;94:647-663.
22. Eisenhart AW, Gaeta TJ, Yens DP. Osteopathic manipulative treatment in the emergency department for patients with acute ankle injuries. *J Am Osteopath Assoc*. 2003;103:417-421.
23. UK BEAM Trial Team. United Kingdom back pain exercise and manipulation (UK BEAM) randomized trial: effectiveness of physical treatments for back pain in primary care. *Brit Med J.* 2004: doi:10.1136/bmj.38282.660225.AE.
24. Vogel S, Dear J, Evans D. The UK BEAM trial – a review and discussion. *Intern J Osteopath Med.* 2005;8:62-68.
25. Licciardone JC, Minotti DE, Gatchel RJ, et al. Osteopathic manual treatment and ultrasound therapy for chronic low back pain; a randomized controlled trial. *Ann Fam Med*. 2013;11:122-129.
26. American Osteopathic Association Clinical Guideline Subcommittee on Low Back Pain. *J Am Osteopath Assoc.* 2010;110: 563-576. http://jaoa.org/issue.aspx#issueid=932115
27. National Guideline Clearing House. Agency for Healthcare Research and Quality. Guideline for evidence-informed primary care management of low back pain. *U.S Department of Health & Human Services* 2010. http://www.guideline.gov/content.aspx?id=37954
28. Whiting LM. Can the length of labor be shortened by osteopathic treatment? *J Am Osteopath Assoc.* 1911;11:917-921.
29. Hart LM. Obstetrical practice. *J Am Osteopath Assoc*. 1918;18:609-614.
30. King HH, Tettambel MA, Lockwood MD, Johnson KH, Arsenault DA, Quist R. Osteopathic manipulative treatment in prenatal care: A retrospective case control design study. *J Am Osteopath Assoc* 2003;103(12):577-82.
31. Guthrie RA, Martin RH. Effect of pressure applied to upper thoracic (placebo) versus lumbar areas (osteopathic manipulative treatment) for inhibition of lumbar myalgia during labor. *J Am Osteopath Assoc.* 1982;82:247-251

32. Licciardone JC, Buchanan S, Hensel KL, et al. Osteopathic manipulative treatment of back pain and related symptoms during pregnancy: a randomized controlled trial. *Am J Obstet Gynecol.* 2010;202:43.e1-8.
33. Hensel KL, Buchanan S, Brown SK, Rodriguez M, Cruser dA. Pregnancy research on osteopathic manipulation optimizing treatment effects: the PROMOTE study. *Am J Obstet Gynecol.* 2015;212:108.e1-9.
34. Boesler D, Warner M, Alpers A, Finnerty EP, Kilmore MA. Efficacy of high-velocity low-amplitude manipulative technique in subjects with low-back pain during menstrual cramping. *J Am Osteopath Assoc.* 1993; 93:203-4.
35. Schwerla F, Wirthwein P, Rütz M, Resch KL. Osteopathic treatment in patients with primary dysmenorrhea: a randomized controlled trial. *Int J Osteopath Med.* 2014;17:222-231.
36. Franke H, Hoesele K. Osteopathic manipulative treatment (OMT) for lower urinary tract symptoms (LUTS) in women. *J Bodyw MovTher.* 2013;17(1):11-18.
37. de Almeida BS, Sabatino JH, Giraldo PC. Effects of high-velocity, low amplitude spinal manipulation on strength and the basal tonus of female pelvic floor muscles. *J Manipulative Physiol Ther.* 2010;33(2):109-116.
38. Billings M. The influenza pandemic of 1918. https://virus.stanford.edu/uda/ accessed March 28, 2015.
39. Castlio Y, Ferris-Swift L. Effects of splenic stimulation on normal individuals on the actual and differential blood cell count and the opsonic index. *Coll J (Kansas City).* 1932;16:111-120.
40. Castlio Y, Ferris-Swift L. Effects of direct splenic stimulation on the cells and antibody content of the blood stream in acute diseases. *Coll J (Kansas City).* 1934;18:196-211.
41. Measel JW Jr. Introduction: thoughts on osteopathic practice and infectious disease. *Osteopath Ann.* 1982;10:92-94.
42. Jackson KM, Steele TF, Dugan EP, Kukulka G, Blue W, Roberts. Effect of lymphatic and splenic pump techniques on the antibody response to hepatitis B vaccine: a pilot study. *J Am Osteopath Assoc.* 1998;98(3): 155-160.
43. Mesina J, Hampton d, Evans J, et al. Transient basophilia following the application of lymphatic pump techniques; a pilot study. *J Am Osteopath Assoc.* 1998;98:92-94.
44. Breithaupt T, Harris K, Ellis J, et al. Thoracic lymphatic pumping and the efficacy of influenza vaccination in health young and elderly populations. *J Am Osteopath Assoc.* 2001;101:21-25.
45. Herrmann E. Postoperative ileus. *The DO.* 1965:163-4.
46. Crow WT, Gorodinsky L. Does osteopathic manipulative treatment (OMT) improves outcomes in patients who develop postoperative ileus: a retrospective review. *Intern J Osteopath Med.* 2009;12:32-37.

47. Baltazar GA, Betler MP, Akella K, et al. Effect of osteopathic Manipulative treatment on incidence of postoperative ileus and hospital stay in general surgery. *J Am Osteopath Assoc*. 2013;113:204-209.
48. Radjieskie JM, Lumley MA, Cantieri MS. Effect of osteopathic manipulative treatment on length of stay for pancreatitis: a randomized pilot study. *J Am Osteopath Assoc.* 1998;98:264-272.
49. Giles PD, Hensel KL, Pacchia CF, Smith ML. Suboccipital decompression enhances heart rate variability indices of cardiac control in healthy subjects. J Altern Complement Med. 2013;19(2):92-6.
50. Henley CE, Ivins d, Mills M, et al. Osteopathic manipulative treatment and its relationship to autonomic nervous system activity as demonstrated by heart rate variability: a repeated measures study. *Osteopath Med Prim Care*. 2008:2.7.
51. O-Yurvati AH, Carnes MS, Clearfield MB, et al. Hemodynamic effects of osteopathic manipulative treatment immediately after coronary artery bypass graft surgery. J Am Osteopath Assoc. 2005;105:475-481.
52. Noll RN, Degenhardt BF, Morley TF, et al. Efficacy of osteopathic manipulation as an adjunctive treatment for hospitalized patients with pneumonia; a randomized controlled trial. *Osteopath Med Prim Care.* 2010;4.2.
53. Noll RN, Shores JH, Gamber RG, et al. Benefits of osteopathic manipulative treatment for hospitalized elderly patients with pneumonia. *J Am Osteopath Assoc.* 2000; 100:776-782.
54. Noll RN, Shores JH, Bryman PN, et al. Adjunctive osteopathic manipulative treatment in the elderly hospitalized with pneumonia: a pilot study. *J Am Osteopath Assoc.* 1999;99:143-144.
55. Guiney PA, Chou R, Vianna A, et al. Effects of osteopathic manipulative treatment on pediatric patients with asthma: a randomized controlled trial. *J Am Osteopath Assoc.* 2005;105:7-12.
56. Bockenhauer SE, Julliard KN, Lo KS, et al. Quantifiable effects of osteopathic manipulative techniques on patients with chronic asthma. *J Am Osteopath Assoc.* 2002;102: 371-375.
57. Sleszynski SL, Kelso AF. Comparison of thoracic manipulation with incentive spirometry in preventing postoperative atelectasis. *J Am Osteopath Assoc.* 1993;93:834-836.
58. Lopez D, King HH, Knebl JA, et al. Effect of comprehensive osteopathic manipulation treatment on balance in elderly patients: a pilot study. *J Am Osteopath Assoc*. 2011;111(6):382-388.
59. Fraix M, Gordon A, Graham J, et al. Use of the SMART Balance Master to quantify the effects of osteopathic manipulative treatment in patients with dizziness. *J Am Osteopath Assoc*. 2013;113:394-403.

60. Anderson RE, Seniscal C. A comparison of selected osteopathic treatment and relaxation for tension-type headaches. *Headache.* 2006;46:1273-1280.
61. Schbert EC, Crow W T. Impact of osteopathic manipulative treatment on cost of care for patients with migraine headache: a retrospective review of patient records. *J Am Osteopath Assoc.* 2009;109(8):403-407.
62. Arnadottir TS, Sigurdardottir AK. Is craniosacral therapy effective for migraine? Tested with HIT-6 Questionnaire. *Complement Ther Clin Pract.* 2013;19(1):11-4.
63. Wells MR, Giantinoto S, D'Agate d, et al. Standard osteopathic manipulative treatment acutely improves gait performance in patients with Parkinson's disease. *J Am Osteopath Assoc.* 1999;99:92-98.
64. Sackett DL, Rosenberg WM, Gray JA, Haynes RB, Richardson WS. Evidence based medicine: what it is and what it isn't. *Brit Med J.* 1996;312(7032):71-72.
65. Black Well Publishing Ltd. *Brit J Haematol.* 2004;124:15-25.
66. http://clinicaltrials.gov/ct2/info/understand. Accessed August 10, 2008.
67. Sackett, D. L, W. M C Rosenberg, J A M. Gray, R B. Haynes, and W S. Richardson. "Evidence Based Medicine: What It Is and What It Isn't." *Bmj*312, no. 7023 (1996): 71–72. https://doi.org/10.1136/bmj.312.7023.71.
68. Null Hypothesis (1 of 4). Accessed September 7, 2019. http://davidmlane.com/hyperstat/A29337.html.
69. Keeling, David M., Ian J. Mackie, Alan Moody, Henry G. Watson, and. "The Diagnosis of Deep Vein Thrombosis in Symptomatic Outpatients and the Potential for Clinical Assessment and D-Dimer Assays to Reduce the Need for Diagnostic Imaging." *British Journal of Haematology*124, no. 1 (2004): 15–25. https://doi.org/10.1046/j.1365-2141.2003.04723.x.
70. "Learn About Clinical Studies." ClinicalTrials.gov. Accessed September 7, 2019. https://clinicaltrials.gov/ct2/about-studies/learn.
71. "P Values." StatsDirect. Accessed September 7, 2019. https://www.statsdirect.com/help/basics/p_values.htm.
72. Wheeler, Stephanie G. UpToDate. Accessed September 7, 2019. https://www.uptodate.com/contents/evaluation-of-low-back-pain-in-adults.

Glossary of Terms

Absolute Risk Reduction (ARR): control event rate – experimental event rate = ARR

Active Range of Motion Testing: patient initiated movement.

Allen's Test: test for thoracic outlet syndrome. The patient is seated with the shoulder abducted and externally rotated at 90° and the elbow flexed to 90°. The patient rotates their head toward the unaffected side and the clinician palpates to determine if the radial pulse is present, absent or diminished.

Allostasis: chronic adaptation to stress.

Allostatic Load: refers to the physiological consequences of chronic or prolonged allostasis.

Analysis, Prospective: the evaluation of the trial's hypothesis or endpoint.

Analysis, Retrospective: evaluates other aspects of the trial, which were noted after the trial began.

Anterior Apprehension Test: used to evaluate for anterior glenohumeral instability or dislocation. The patient is supine with shoulder abducted and externally rotated to 90°. The shoulder is externally rotated further; if the patient feels like "shoulder is going to come out" the test is positive. This test is done in conjunction with the relocation test – see below.

ASIS Compression Test: a test used to evaluate the horizontal pelvic axes for motion restriction.

Axis, Pelvic: as defined by Fred Mitchell, Sr. DO, FAAO:

Left Oblique Axis (LOA) = axis about which L/L, R/L torsions occur.

Right Oblique Axis (ROA) = axis about which R/R and L/R torsions occur.

Superior Transverse Axis (STA) = sacral axis at the level of S2; axis responsible for craniosacral motion.

Middle Transverse Axis (MTA) = sacral axis at the level of S2; axis about which sacral flexion and extension occur.

Inferior Transverse Axis (ITA) = sacral axis at the level of S3; axis about which innominate rotation occurs.

Barrier: a limit to range of motion:

Anatomical: limit of normal passive ROM; represents bone contacting bone.

Elastic: found during passive ROM, located between anatomical and physiological barriers; represents barrier created by joint capsule and supporting ligaments.

Physiological: limit of normal active ROM; represents end of muscle fiber stretching and fascia.

Pathologic: also known as somatic dysfunction and indicates hypo/hyper-mobility.

Belly Press: a test used to evaluate subscapularis muscle strength. The patient holds the palm of their hand on their belly and presses against the abdomen while the examiner maintains maximal resistance.

Childress Test: assesses meniscal pathology. During this test the patient is asked to perform a "duck walk." A test is positive when the patient reproduces their typical symptoms along the medial or lateral joint line.

Common Compensatory Fascial Pattern: the alternating fascial motion predilection at transitional regions of the body described by Dr. Zink.[1]

Compression of the Fourth Ventricle (CV4): a cranial manipulation procedure in which the lateral angles of the occiput are gently approximated medially. This procedure encourages the cranial bone motion to be held in sustained extension at the SBS, and stimulates the PRM and the person's inherent healing capacity.

Counternutation: describes posterior movement or position of the sacral base that occurs with sacral flexion.

Coupled Movements (aka Coupling): describe the rotation or sidebending of a vertebral body about or along one axis that is consistently associated with its rotation or sidebending about another.

Contralateral Coupling: in spinal mechanics, rotation that is opposite sidebending.

Ipsilateral Coupling: in spinal mechanics, rotation that is to the same side as sidebending.

Closed Pack Position: a maximal tautness of the articular capsule and the ligaments that describes the ideal location for direct HVLA.

Cranial Rhythmic Impulse (CRI): the primary respiratory mechanism (PRM) when appreciated in the head. This phrase relates to psychiatric patients that was coined by James Woods, DO, and Rachel Woods, DO, that has come into popular use.

Crank Test: evaluates for shoulder labral pathology. During this test, the shoulder is abducted to ~160° in the scapular plane. While externally rotating the shoulder, an axial compression from the elbow toward the glenohumeral joint is applied. If the patient feels a clunk or pain deep inside the shoulder, it is a positive test.

Cross-arm Adduction Test (aka Apley-Scarf Test): assesses for pathology at the AC joint. The hand of the affected side is placed on the opposite shoulder and the clinician raises the elbow to 90°, loading the AC joint. A test is positive if pain occurs at the AC joint.

Diagnosis of Somatic Dysfunction:

Positional Diagnosis: named for the location of somatic dysfunction in three-dimensional space.

Restriction Diagnosis: named for the limitation in motion of somatic dysfunction and contains the term "restriction" or "restricted."

Direct Treatment: a treatment which begins at the restriction barrier.

Drawer Test:

Anterior: evaluates integrity of the anterior cruciate ligament (ACL). The patient is supine on the table with the knee flexed at 90° and their foot on the table. The examiner stabilizes the leg by partially sitting on the dorsum of the foot and grasps the patient's lower leg at the tibial plateau. The examiner then provides an anterior translation to the tibia to test for laxity or increased movement. ACL injury is suspected if increased motion is noted when compared with the opposite side.

Posterior: evaluates the posterior cruciate ligament (PCL) and is performed from the same position as the anterior test but with the translation of the tibia on the femur in a posterior direction. The test is considered positive when there is laxity compared to the contralateral side.

Drop Arm Test: effectively tests for supraspinatus tendon rupture. The shoulder is passively abducted to 170° and patient lowers the arm back down to side. Inability to lower the arm can indicate a significant rotator cuff tear.

Dynamic Balance Point (DBP): with reference to an indirect treatment, the point where the somatic dysfunction is balanced in three-dimensional space.

Dynamic Milking Maneuver: evaluates ulnar collateral ligament instability/pain. The shoulder is positioned at 90° of abduction and at varying degrees of elbow flexion (usually from 30 to 60°). The examiner holds the thumb of the affected extremity with one hand and stabilizes the elbow on the lateral side with the other hand; they then externally rotate the elbow while applying a valgus load to the elbow. The test is positive if the patient has a reproduction of pain symptoms.

Ely's Test: assesses for rectus femoris spasticity. The patient is placed prone on the table and the heel is passively flexed toward the buttock. The test is considered positive if the heel cannot touch the buttocks.

Extension: posterior movement of a structure in relation to another.

Sacral Extension: describes posterior movement or position of the sacral base in relation to the innominates during sacroiliac somatic dysfunction.

Evidence Based Medicine (EBM): a term used to describe statistical analysis of the scientific method as it pertains to the medical field, and its use in clinical practice.

External Rotation Test: used for syndesmotic injury of the talofibular joint or "high ankle sprain." The ankle is neutral at 90° with a leg hanging off of the table as the operator externally rotates the foot slowly. Test is positive if the patient feels pain at the syndesmosis.

Homeostasis: acute adaptation to stress.

Finkelstein's Test: evaluates for DeQuervain's tenosynovitis. The patient makes a fist with the fingers wrapped around the thumb. The clinician braces the forearm with one hand and grasps the patient's fist with the other hand, moving the wrist into a position of ulnar deviation to reproduce the radial sided symptoms.

Flexion: anterior movement of a structure in relation to another

Sacral Flexion: describes anterior movement or position of the sacral base in relation to the innominates during sacroiliac somatic dysfunction.

Fourth Ventricle Compression (CV4): a cranial manual medicine technique in which the lateral angles of the occiput are approximated medially. This technique places the cranium in sustained extension, locking the PRM of the head.

Gamma-Loop Theory: proposed by Irvin Korr, PhD. Korr proposed that altered muscle spindle activity, measurable by EMG, explained the physical examination findings in patients with somatic dysfunction.

Hawkin's – Kennedy Test: a test for shoulder impingement syndrome. The shoulder is positioned at 90° of forward flexion and then internally rotated. Test is positive if pain symptoms reproduce.

High Velocity, Low Amplitude (HVLA): classically taught as a direct treatment technique in which a fulcrum, lever, and impulse are used by a practitioner to move through a barrier of somatic dysfunction.

Indirect Treatment: a treatment that begins away from the restriction barrier in a position of ease, or by exaggerating the positional diagnosis.

Jobe's Relocation Test: for evaluation of shoulder stability. The clinician repeats the anterior apprehension test with the exception of placing an anterior-to-posterior stress over the humeral head, stabilizing the joint. The test is positive if the operator can externally rotate the arm further before the patient feels apprehension.

Key Lesion: see somatic dysfunction

Lachman's Test: evaluates for anterior cruciate ligament (ACL) injury; the patient is supine with the knee flexed to approximately 20-30°. The operator braces the thigh with one hand, and the other hand is placed on the posterior proximal tibia. The operator then applies anterior translation of the tibia on the femur to determine if there is a firm endpoint to the ACL. If laxity or absence of firm endpoint is noted (compared with the normal side), then ACL injury is suspected.

Leg Length Discrepancies:

Anatomical: leg length discrepancy caused by unequal length of the femur, tibia, or foot.

Functional: leg length discrepancy that is caused by somatic dysfunction and resolves with OMT.

Pathological: same as anatomical.

McMurray's test: (active axial compression, circumduction maneuver) evaluates meniscal pathology. Performed with the patient in a supine position and the hip flexed to 60° and the knee flexed to 90°. The examiner palpates the medial and lateral joint line with one hand and applies axial compression with the other hand. From the sole of the patient's foot, the examiner then applies the axial load with a circumduction maneuver to load each knee compartment. A palpable clunk is a positive test, and frequently patients experience pain with this maneuver.

Movement: contralateral and ipsilateral – see coupled movements.

Muscle Energy (ME): classically taught as a direct treatment technique in which a patient makes a muscle effort that is resisted by a practitioner and results in an isometric contraction made at the barrier of somatic dysfunction.

Myotomal Pain: pain generated from acute muscle injuries that is later manifested by trigger and tender points.

Negative Predictive Value (NPV): the proportion of patients with negative test results who are correctly diagnosed.

Neer Test: for shoulder impingement syndrome. This test is performed by forced, passive flexion of the humerus while stabilizing the shoulder girdle. If symptoms are reproduced, the test is positive.

Nociception: the stimulation of peripheral pain carrying nerve fibers (C and A-delta) and the transmission of impulses along peripheral nerves of the central nervous system, where the stimulus is perceived as pain.

Null Hypothesis: in evidence based medicine, an assumption made that no difference exists between the two groups studied.

Number Needed to Treat: 1/absolute risk reduction.

Number Needed to Harm: 1/test group harmed – control harmed.

Nutation: describes anterior movement or position of the sacral base that occurs with sacral extension.

Ober's Test: evaluates contracture or tightness of the iliotibial band. Patient is side lying on the treatment table with the top leg bent. This leg is abducted by the physician and then released. If there is ratcheting, slow return, or the leg does not drop back to the neutral position, this indicates a contracture of the iliotibial band.

Obrien's Test (aka Active Compression Test): a two step test used to evaluate labral pathology. To perform, the shoulder is flexed to 90°, adducted approximately 20°, and internally rotated (thumbs down). The patient resists the clinician's downward force. This is then repeated in the externally rotated position with the thumbs up. If the patient feels pain "deep inside" the shoulder on the first part of the test and the pain is relieved on the second part, then there is suspicion for labral pathology.

Open Pack Position: with respect to HVLA, any position that is not the closed pack position.

Osteopathic Manipulative Medicine (OMM): the use of Osteopathic philosophy, diagnosis, and OMT in the management of patient care.

Osteopathic Manipulative Treatment (OMT): the treatment of a somatic dysfunction with manual medicine.

Osteopathic Principles and Practice (OPP): the delivery of health care using the four tenants of Osteopathy:

1. The body is a functional unit, and the person represents a dynamic combination of body, mind, and spirit.
2. The body is capable of self-regulation, self-healing, and health maintenance.
3. Structure and function are reciprocally interrelated.
4. Rational treatment is based on the first three principles.

P-value: a probability that ranges from zero to one; it answers the question of the null hypothesis.

Passive Range of Motion Testing: examiner-initiated movement without effort from the patient.

Patellar Apprehension Test: evaluates for patellar subluxation/dislocation. The test is performed with the patient supine, the knee in full extension. The examiner grasps the patella and translates it laterally to medially. Should the patient feel reproduced symptoms, the test is positive.

Patellar Grind Test: evaluates patellofemoral syndrome (PFS) pathology caused by lateral traction of the patella due to quadriceps muscle imbalance or medial retinaculum laxity. The patient is positioned supine with the knee extended. The examiner grasps the patella and pushes it posteriorly; the patient is directed to contract their quadriceps muscle. The patient may experience discomfort, as the pain is usually severe in the PFS.

Peroneal Tendon Subluxation Test: evaluates peroneal tendon. The patient dorsiflexes and plantar flexes; then palpate for subluxation and reproduction of the patient's symptoms.

Phalen's Test: assesses for median nerve entrapment. The patient is seated with the dorsal aspect of both hands in contact as both wrists are flexed. Patient is held in this position for one minute and the test is positive if the patient has paresthesias in the median distribution of the affected hand.

Positive Predictive Value (PPV): the proportion of patients with positive test results who are correctly diagnosed.

Posterior Apprehension Test: evaluates for posterior shoulder instability. To perform this test, the patient is supine and the shoulder is adducted approximately 20°. The clinician provides an axial compression from the elbow towards the ground. If the patient has the sense of the shoulder "coming out," it is a positive test.

Posttest Probability: is the probability determined after the relevant evidence is taken into account.

Pretest Probability: the probability of the sought disorder being present before a diagnostic test result is known.

Primary Respiratory Mechanism (PRM): coined by William Garner Sutherland, DO. "Primary" describing its presence at the cellular or the smallest levels, "respiration" signifying cellular metabolism, and "mechanism" to emphasize the ubiquitousness of the phenomenon in the body. The PRM is classically summarized in five key elements:

- Inherent motility of the brain and spinal cord
- Fluctuating cerebrospinal fluid
- Motility of the intracranial and spinal membranes (meninges, dura, etc.)
- Mobility of the bones of the skull
- Mobility of the sacral motion between the ilia that is interdependent with that of the sphenobasilar synchondrosis

Principles of Spinal Motion: the first two principles were originally published in *Principles of Osteopathic Technic* by Harrison H. Fryette, DO, in 1954. The third principle was described originally by Nelson.[2(pp44)] Note, these principles are under scientific scrutiny and may not apply clinically.

I. When in neutral, sidebending produces rotation opposite that of the sidebending. The direction of rotation is toward a convexity.
II. When in flexion or extension, sidebending and rotation occur in the same direction. The direction of rotation is toward a concavity.
III. Introducing motion in one plane limits its mobility in the other two planes.

Prolotherapy: short for "proliferative-therapy," involves injecting irritant solutions, such as dextrose, in the area of ligaments, tendons, and joints to strengthen weakened connective tissue and promote healing.

Prospective Analysis: the evaluation of the trial's hypothesis or endpoint.

Radial Collateral Ligament (aka Lateral Collateral Ligament) Stability - Varus Stress Test: performed at 20° of elbow flexion – the operator then applies varus force to assess laxity of lateral collateral ligament.

Radial Head Subluxation - Lateral Pivot Shift Test: evaluates for posterolateral elbow instability. The patient is supine with the forearm supinated while the clinician holds the elbow in full extension, maintaining a valgus load on the elbow. From the extended position (elbow), the clinician introduces an axial load to the elbow while supinating and flexing the elbow to approximately 40°. During this maneuver, which is optimally performed under anesthesia, there is a palpable

subluxation, then reduction of the radial head. Without anesthesia, the patient feels "apprehension" when positive.

Radicular Pain: pain emanating from spinal nerve roots.

Range of Motion (ROM):

Active ROM: patient-initiated movement.

Passive ROM: examiner-initiated movement (without patient effort).

Reciprocal Inhibition, Law of: a basic concept in which the dysfunctional hypertonic muscle is induced to relax after contraction of its paired (antagonist) muscle.

Reciprocal Tension Membranes: the internal tension (i.e., tensegrity) between the falx cerebri, falx cerebelli, the tentorium cerebelli, and spinal meninges that allows for a movement of the skull with maintenance of its integrity.

Relocation Test: used to assess for anterior shoulder instability. The clinician repeats the anterior apprehension test, and now places an anterior-to- posterior stress over the humeral head, which effectively stabilizes the joint. The test is positive if the operator can externally rotate the arm further before the patient feels apprehension.

Retrospective Analysis: evaluates other aspects of a trial that were noted after the trial began.

Reverse Lachman's Test: evaluates the posterior cruciate ligament (PCL). The patient is supine with the knee flexed to approximately 20-30°. The operator braces the thigh with one hand, and the other hand is placed on the posterior proximal tibia. The tibia is translated posteriorly to test for laxity or firm endpoint of motion.

Reverse Talar Tilt Test: evaluates integrity of the deltoid ligament. Performed similarly to the talar tilt test, but the ankle and foot are everted to test for laxity of the deltoid ligament.

Roos Test: attempts to reproduce these symptoms - both shoulders are abducted and externally rotated to 90° and the elbows flexed to 90°. The patient is asked to repeatedly clench the fist for 3 minutes. If the patient cannot maintain the position for the duration of the test or develops paresthesias, then the test is positive for thoracic outlet syndrome.

Scapular-Clavicle Motion Test: test used to determine somatic dysfunction of the scapula-thoracic joint. Performed by placing the thenar eminence of both hands across the spine of the scapula. Digits two through four are placed comfortably across the medial to lateral clavicle; a spring is added posterior-medially using the thenar eminence and the fingers, almost as if unscrewing a lid from a large jar. If somatic dysfunction is present, one side will have restricted ROM compared with the other. Next, observe position of the scapular spines - the abnormal side is compared with respect to position (inferior or superior) to the normal side.

Sclerotomal Pain: pain arising from skeletal, arthrodial, and ligamentous structures.

Scour Maneuver (aka Quadrant Test): evaluates femoral acetabular impingement or labral pathology. The patient lays supine on the table, the hip and knee flexed to 90° and adducted. A compressive force is applied along the length of the femur toward the

acetabulum, and the examiner moves the femur through a circular arc of motion. Reproduction of pain or "catching" indicates anterior hip subluxation, or labral pathology.

Sensitivity: the proportion of actual positives that are correctly identified.

Sensitization: an increase in central nervous system response to repeated sensory stimulation that generally follows habituation.

Sequencing: the term used to describe methods for finding areas of primary somatic dysfunction coined by Edward Stiles, DO, FAAO.

Shoulder Impingement Syndrome: refers to irritation of the supraspinatus tendon as it passes under the acromion and attaches to the humerus.

Sitting Forward Bending Test (SitFBT): used to test for sacral somatic dysfunction. Performed with the patient sitting, feet resting on a level surface; physician behind the patient with thumbs palpating both PSIS. As the patient bends forward, the physician maintains contact with the PSIS. With normal motion, the PSIS should move symmetrically cephlad. When somatic dysfunction is present, the pathological PSIS will move more cephlad than the normal PSIS.

Somatic Dysfunction: "impaired or altered function of related components of the body framework system: skeletal, arthroidal, and myofascial structures, and their related vascular, lymphatic and neuronal elements."[2(pp63)]

Primary Somatic Dysfunction: ideal place to begin manual medicine treatment. Somatic dysfunction which maintains the secondary patterns of somatic dysfunction. Also known as the "key" somatic dysfunction.

Secondary Somatic Dysfunction: somatic dysfunction that develops as a result of another pathological process or primary somatic dysfunction.

Somatic Pain: non-nerve root associated pain arising from the skin, musculoskeletal system and connective tissue.

Somatovisceral Reflex: an autonomically mediated spinal cord reflex caused by somatic dysfunction that results in pathology of one or more visceral structures via shared spinal cord levels.

Somatosomatic Reflex: localized somatic stimuli producing patterns of reflex response in segmentally related somatic structures.

Specificity: the proportion of negatives that are correctly identified.

Speed's Test: evaluates for pathology of the long head of the bicep as it passes through the bicipital groove. The elbow is maintained in extension and supination while the patient actively flexes the arm against resistance. Pain reproduced at the biceps groove is a positive test.

Sphenobasilar Synchondrosis (SBS): Approximation of the basilar portion of the sphenoid and the basilar portion of the occiput.

Spinal Facilitation: the maintenance of a pool of neurons in a state of partial or sub-threshold excitation; in this state, less afferent stimulation is required to trigger the discharge of impulses.

Spurling's Test: attempts to reproduce symptoms of nerve root compression in the cervical spine. The test is performed with the patient seated with his head extended and side-bent toward the same side, while the clinician applies a compressive/downward force through the head effectively further narrowing the

intervertebral foramen. The test is positive if pain or paresthesia are reproduced down the arm, neck, head, or shoulder.

Standing Forward Bending Test (SFBT): used to test for innominate somatic dysfunction. Perform with patient standing with feet about 15cm apart and the physician behind the patient with thumbs palpating both PSIS. As the patient bends forward, the physician maintains contact with the PSIS. With normal motion, the PSIS should move symmetrically cephlad. When somatic dysfunction is present, the pathological PSIS will move more cephlad than the normal PSIS.

Sternoclavicular (SC) Shrug Test: evaluates SC motion. While standing in front of the patient, the operator places the finger pads of their index fingers on the superior-medial edge of both clavicles, observing the position of the clavicle for symmetry. The patient then shrugs his shoulder by contracting the levator scapula. A normal test is evident by symmetrical inferior movement during the shoulder shrug. The SC joint is diagnosed as "*inferior*" when it is initially inferior (compared to the opposing clavicle), and if during the shoulder shrug it does not glide further inferiorly. The SC joint is diagnosed as "*superior*" when it is initially superior (compared to the opposing clavicle), and if during the shoulder shrug it does not glide inferiorly.

Still-Point: a temporary interruption of the primary respiratory mechanism that may occur during treatment with manual medicine.

Stork Test (aka Gillet Test): the stork test is performed with patient standing and feet about 15-30 centimeters apart. The physician stands behind the patient with thumbs palpating both PSIS inferiorly. The patient flexes the knee of one leg toward the chest while the physician maintains contact with the PSIS. The test is then repeated with the opposite leg. With normal motion, the PSIS should move symmetrically caudad. When somatic dysfunction is present, the PSIS will fail to move caudad and may in fact move cephlad, indicating dysfunction on that side.

Sutherland Fulcrum: a theoretical, shifting fulcrum located along the straight sinus at the junction of the falx cerebri and the tentorium cerebelli.

Talar Tilt Test: evaluates the integrity of the calcaneofibular ligament. The ankle is kept at 90°, and the operator has a similar handhold as with the anterior drawer test, except in this instance the operator applies an inversion force to the calcaneus to test the calcaneofibular ligament. If there is excessive laxity and tilt, the test is positive.

TART: pneumonic that describes somatic dysfunction. T = tissue texture abnormality, A= asymmetry, R = restriction of motion, T = tenderness to palpation.

Tender point (TP): an area of tenderness less than 1cm located in ligaments, tendons, and muscle ends. They are edematous, non-radiating, and tender to palpation, yet non-tender when not palpated. Their presence results in restricted ROM and muscle weakness. TP are considered somatic dysfunction.

Tensegrity: short for tensional integrity, refers to structural integrity based on balanced tension and compression.

Thessaly's Test: evaluates meniscal pathology. The operator attempts to reproduce joint pain by grinding the meniscus. The patient stands on the affected knee with approximately 20° of joint flexion and introduces internal and external rotation of

the femur on the tibia three times. The test is positive if the patient can reproduce the symptoms on the medial or lateral side of the loaded extremity.

Third Digit Resisted Extension Test: used to detect lateral epicondylosis. The elbow is extended and the clinician resists as the patient extends the middle finger. The test is positive if the patient experiences reproduction of lateral elbow pain with resisted 3rd digit extension.

Thomas Test: assesses for restriction or hypertonicity of the iliopsoas muscle. Patient is placed in the supine position on the treatment table with their legs off the table and the crease of the posterior knee at the edge of the table. Patient sits up, grabs the knee of one lower extremity and rolls back to the supine position. The other lower extremity remains on the table. If there is a contracture, or the posterior leg does not lie evenly with the table, this is evidence of hypertonicity of the iliopsoas muscle. The test is performed again for the contralateral side.

Thompson Test: evaluates for Achilles tendon rupture. Patient is prone on the treatment table with the knee flexed to 90°. Operator squeezes the mid-calf to note subsequent plantar flexion of the foot. If plantar flexion does not occur, there is suspicion for Achilles tendon rupture.

Thoracic Outlet Syndrome (TOS): clinical symptoms produced by compression of the components of the brachial plexus, the subclavian artery, or the subclavian vein.

Tinel's Test: evaluates for nerve entrapment at multiple locations, including, but not limited to the carpal tunnel, cubital tunnel, radial tunnel. Perform by percussing over the nerve to elicit radicular symptoms in the nerve's distribution.

Treatment: in this textbook refers to manual medicine.

Direct: treatment begins at the pathological barrier.

Indirect: treatment begins away from the pathological barrier, usually at the position of ease (POE).

Treatment Reaction: usually occurring 24 to 72 hours after a musculoskeletal treatment, a painful reaction manifest as tolerable local or diffuse muscle ache, or pain similar to beginning a new work-out program.

Trendelenburg Test: assesses weakness of the gluteus medius on the weight-bearing side. Patient is in the standing position with the examiner observing from behind to assess for a shift of the pelvis when the patient stands single weighted on one leg. Ideally, the pelvis should remain level. The patient will have a positive test when the hip of the non-weightbearing leg drops down.

Trials, Clinical: five basic types:

Diagnostic Trials: appraise tests or methods that diagnose particular disease or condition.

Prevention Trials: evaluate ways to prevent disease in people who do not have the disease or evaluate methods to prevent a known disease from returning.

Screening Trials: test the best way to detect certain diseases or health conditions.

Supportive Care Trials: also known as quality of life trials, evaluate ways to improve palliate care for individuals with chronic illness.

Treatment Trials: test experimental treatments against commonly used or "standard of care" treatments.

Trigger Points: are described as either active or latent, depending on their chronicity.

Active Trigger Points: characterized by focal, discrete, hyperirritable spots located

in a taught band of skeletal muscle that produce referred pain and tenderness.[3] Classically, they cause pain with and without palpation and have a "local twitch response."

Latent Trigger Points: similar to active trigger points, but do not cause tenderness without palpation. They do restrict ROM and cause muscle weakness and many regard them to be the same as tender points.

Ulnar Collateral Ligament Valgus Stress Test: performed to detect laxity. The clinician places one hand on the lateral elbow and the other hand on the distal forearm and applies a valgus load at 0 and 30° of flexion. The test is performed on the contra-lateral side for comparison. The test is positive when the joint opens up compared to the opposite side, or the patient experiences reproduction of painful symptoms.

Ulnocarpal Stress Test (TFCC Grind Test): assesses for TFCC injury. The clinician braces the distal radial-ulnar joint with one hand and grasps patient's hand with the other. The patient's wrist is placed in maximal ulnar deviation while adding a rotational axial load. If painful clicking or if typical pain symptoms are present, TFCC injury or disruption is suspected.

V-spread Procedure: this treatment propels CSF in the form of a fluid wave across the diameter of the skull to accomplish sutural gapping. Two fingers are spread in the shape of a "V" on either side of a restricted suture; a tapping force is directed toward restricted suture on the opposite side of the skull.

Viscerosomatic Reflex: an autonomically mediated spinal cord reflex caused by visceral organ pathology that results in somatic dysfunction of the musculoskeletal system via shared spinal cord levels.

Watson's Test: evaluates for scapho-lunate subluxation secondary to ligament injury. The patient is seated with the elbow flexed at 90° while the clinician palpates the scaphoid tubercle on the volar side with his thumb, holding the wrist in ulnar deviation. The patient is then instructed to move the wrist into radial deviation as the clinician discerns a palpable subluxation and reduction of the scaphoid (the scaphoid will move dorsally) - this indicates scapholunate dissociation. If the patient only experiences pain with this maneuver, then there is suspicion for scapholunate enthesopathy.

Yergason's test: evaluates for pathology of the long head of the bicep as it passes through the bicipital groove. This test is performed with the elbow flexed to 90° while the forearm is pronated. The clinician palpates the bicipital groove with one hand and provides resistance to active supination at the wrist. The test is positive if pain is perceived in the bicipital groove.

Zink, Lines of: see Common Compensatory Fascial Pattern.

1 Foundations in Osteopathic Medicine,2nd edition, 2003, glossary. Ward, Robert, ed. Lippincott Williams & Wilkins Company, Philadelphia, PA.

2 Giusti, Rebecca, DO. *Glossary of Osteopathic Terminology*. Third ed. Chevy Chase, MD: American Association of Colleges of Osteopathic Medicine, 2017.

3 Janet Travell, MD. Myofascial pain and dysfunction: the trigger point manual, 2nd edition. Baltimore Williams and Wilkins, 1999.

Index

N

O

P

R

S

T

U

V

W

X

Z