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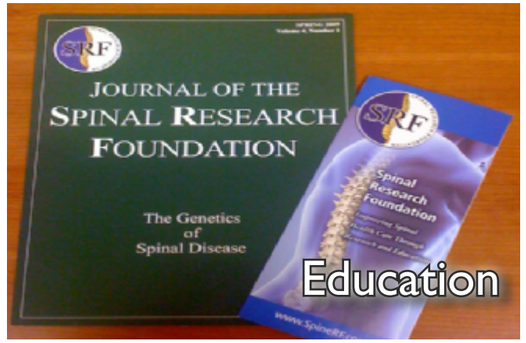

JOURNAL OF THE SPINAL RESEARCH FOUNDATION
SPRING 2011
VOLUME 6 NUMBER 1

Volume 6, Number 1

JOURNAL OF THE SPINAL RESEARCH FOUNDATION

Spine Support:
Muscles, Tendons, and Ligaments

2011

THE JOURNAL OF THE SPINAL RESEARCH FOUNDATION
A multidisciplinary journal for patients and spine specialists

"Improving the health of my patients is my lifelong commitment. My goal is always to get my patients healthy and back on their feet. I connect with my patients on a personal level—when they hurt, I hurt. I will continue to work tirelessly to improve their lives and provide them exceptional spine care."

SPINAL CHAMPION

Rick C. Sasso, M.D.

Indiana Spine Group

The Spinal Research Foundation recognizes our outstanding clinicians and researchers in the field of spine research and profiles them as Spinal Champions. These dedicated spine care professionals embrace excellence in both research and education, contributing significantly to improvements in the diagnosis and treatment of spinal disorders. We recognize Rick C. Sasso, M.D., of Indiana Spine Group in Indianapolis, IN as a Spinal Champion.

THE JOURNAL OF THE SPINAL RESEARCH FOUNDATION
Volume 6, Number 1

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From the Editor

Brian R. Subach, M.D., F.A.C.S.

This edition of the *Journal of The Spinal Research Foundation* is truly exciting. We have contributions from numerous different experts in the field of spinal health care, focusing on the supporting structures of the spine. When one thinks of the spine, the focus is typically on the bony skeleton. Our research has identified that the supporting structures of the spine, specifically muscles, ligaments, tendons and discs are key culprits in many of the pain issues affecting our patients.

In our Spine Tale, we tell the story of a patient who has dealt with chronic pain, misdiagnoses, and being told simply that nothing could be done for her. Once she found the spinal health care providers who could help, it was if a light bulb was turned on for her. Now, more than a year has passed since her treatment, and she is back to a normal life without pain. Look for the Spine Tale to read the details of her story.

It is my pleasure to introduce Rick C. Sasso, M.D., who is our Spinal Champion for this issue. Dr. Sasso is a Board Certified orthopaedic surgeon and the president of the Indiana Spine Group. He is internationally renowned as both a surgeon and educator in the field of spinal implants and minimally invasive spinal surgery. He received his undergraduate medical degree from the Indiana University School of Medicine, followed by an orthopaedic surgery residency at the University of Texas Medical Center. He, then, completed spinal surgery fellowships at Northwestern University and The Baylor College of Medicine. Aside from being an outstanding spinal surgeon and educator, he is also a true patient advocate. He was chosen as the Spinal Champion for this issue primarily for his efforts in helping his patients return to normal lives.

It is my pleasure to draw attention to the Spinal Research Foundation's annual "We've Got Your Back" race/walk events supporting research and celebrating the accomplishments of those who have overcome back or neck pain. The Washington, DC Metropolitan Area is the site of this year's first race/walk on Saturday, May 14, 2011. It will be held on the grounds of The Virginia Spine Institute who for the fourth consecutive year is hosting the event in Reston, Virginia. Other "We've Got Your Back" race/walk sites include Philadelphia, Pennsylvania; San Francisco, California; and Springfield, Massachusetts, with more being planned. These annual events allows patients and their families to join their spinal health care providers in raising funds that directly contribute to life changing research targeting those afflicted by spinal disorders, as well as educational programs for patients and providers alike. Few realize that 90% of the population suffers from severe neck or back pain at some point during their lifetime, and that 8% of these people develop chronic pain. In the United States, thirty-five million people are directly affected by disabling spinal pain. To learn more, find SRF on the web at www.spineRF.org and join us in raising funds for research in spinal disorders and innovative therapies for conditions that could affect you or someone you love.

From the President

Thomas C. Schuler, M.D., F.A.C.S.

The Fallacy of Evidence-Based Medicine

The current trend to embrace evidence-based medicine is creating a crisis for access to many medical therapies in America. Academic medicine has coined the term evidence-based medicine to describe a scientific process to prove which treatment best solves a specific medical condition. On the surface, this is a very sound and appropriate goal. Unfortunately, political agendas which are driving this initiative are creating disasters for many patients who will be denied proper care in the future.

Comparative effectiveness is the Obama Care equivalent of evidence-based medicine. The basis of evidence-based medicine, as well as comparative effectiveness, is to scientifically prove what treatment is the best. A significant problem with this concept is that one cannot generalize treatments for all patients. Another important problem is that many of the "experts" who "interpret" research are discrediting quality studies because of their personal bias. In discounting many outcome studies that have already confirmed the effectiveness and success of a given therapy, patients are subsequently denied access to life changing treatment. There are many reasons that a reviewer would choose to discredit a procedure. In my opinion, the two most common consist of an individual bias against a procedure since in that reviewer's perspective, he or she was unable to obtain the desired result when such technologies were employed by their own hands. This would not be uncommon in cases where the individual performing the review was more gifted as a writer than as a surgeon. "Those who can, do, and those who can't, write." This well known quote among physicians emphasizes a common reality. The other reason for a reviewer's bias can be linked to the funding source for their studies. Many of the "experts" of evidence-based medicine, including some new academic positions entitled "professor of evidence-based medicine" receive the funding for their research from parties who have a vested interest to reduce health care expenditures. There is no easier way to reduce health care expenditures than to declare an individual treatment unsuccessful, thereby justifying an insurance company or government's position statement that they will not fund that specific technology. The more treatments a professor of evidence-based medicine discredits, the more funding he or she receives.

The basis of the scientific method for proving or disproving a theory is to test a hypothesis with a control and a variable. In the laboratory, one can do this simply with test tubes and petri dishes. However, in dealing with humans, especially in the operating room, this creates significant ethical dilemmas. The best evidence-based medicine studies are those that are randomized and prospective. In simple terms, this means that the individual receiving a treatment does not know which treatment they will receive until they are randomly given one treatment or another. Furthermore, to meet the highest level of validation, according to evidence-based medicine, not only must the studies be randomized, but they must all be done prospectively. Specifically meaning all of the questions being asked must be asked prior to the start of an experiment.

If medical research is done in a prospective randomized fashion, it is considered to be of the best quality. This is logical assuming the studies are well constructed. The problem is that many questions and answers arise as studies progress and as data is analyzed at completion of the study. Writing research papers from information found out at the end of the study or reanalyzing the data with new questions is considered retrospective and not valued as much as the prospective data. This is where the fallacy of evidence-based medicine starts to show. Furthermore, many studies, which are not randomized but are excellent collections of data obtained prospectively and/or retrospectively from a variety of treatments, are

extremely valuable. However, the political powers of comparative effectiveness and the academic powers of evidence-based medicine, choose to disregard, discredit, or minimize many of these valuable and essential outcome studies since they do not support their desired agenda.

At a national spine meeting one of the newly appointed professors of evidence-based medicine proposed that the best surgical research would include sham operations where a surgeon opened a patient, but performed no surgery. Not only is this impractical, but it is completely unethical. Furthermore, without performing the complete surgical dissection involved in a given surgery, a sham incision would not produce comparable surgical morbidity. The fact that an "expert" in evidence-based medicine dreams of this possibility frightens me and is a sign that patients across America should be concerned about their future access to time-proven treatments and surgeries.

To elicit my point more clearly, a retrospective study of NFL football players who had undergone discectomies for lumbar disc herniations during the past thirty years was compiled. Sixty-six NFL players were identified as having disc herniations and underwent discectomy surgery to treat their symptoms. Eighty percent of these professional athletes were able to return to the same or better level of performance after surgery. The purpose of the article was to show that lumbar discectomy surgery is a viable and safe option in these elite athletes. This wonderful and important thirty year study was criticized by the academic reviewer since it was not prospective and it was not randomized. The reviewer went on further to state that the study needed to be done in a prospective randomized fashion before any conclusions could be drawn as to the safety of NFL players undergoing discectomy surgery and their ability to return to play. I have been the spinal surgeon to the Washington Redskins for eighteen years. In my experience with these professionals, I have never encountered one player who was ever willing to enter into a randomized research protocol. All of these athletes want to identify the safest and best way for them to return to their careers as rapidly as possible. For an individual reviewer to propose that this study should be performed in a prospective randomized fashion shows a complete disconnect from reality. Unfortunately, this disconnect exists across many levels of academic medicine and many levels of the political arena.

The Spinal Research Foundation continues to develop the best outcome measures to prove the safety and efficacy of non-operative and operative treatments of spinal disorders. We feel that quality research is of value, but all quality research does not require human experimentation and/or sham procedures. Rather, quality questions and detailed analysis of prospective and retrospective treatments is what will continue to provide us with the best answers for all patients. We will continue to empower patients across America to understand what works and what does not work. We will not lose sight of the fact that individuals are being treated, and that many large studies speak to generalizations but not to the specifics that affect an individual patient's condition. They do not frequently address the sociologic and physiologic details that the physician and his or her patient must consider to determine what will work most effectively for that individual's specific problem. **It is the physician's knowledge, experience, and understanding of scientific data, combined with a specific patient's individual and social needs, which allows the two of them to develop the optimal treatment.** "Evidence-based experts" and political appointees are not the answer to the best treatment options for an individual or for all Americans.

Ask the Expert

Richard A. Banton, P.T., D.P.T., A.T.C.
Virginia Therapy and Fitness Center

Can targeted muscle strengthening improve low back pain?

Yes, but some types of strengthening are more effective than others. The musculature that has been found to be most effective in improving, as well as preventing, low back pain are the deep stabilizing muscles of the trunk, specifically, the transverses abdominus, multifidus, and pelvic floor muscles. The activation of these muscles is specific and may require seeking the assistance of a health care professional to ensure proper form. Once proper activation and control of this musculature is achieved, strengthening the superficial musculature will also be important. This musculature includes the external and internal obliques, gluteus maximus, gluteus minimus, and back extensors. Strengthening during or following an episode of low back pain can worsen symptoms, so caution must be taken. Consulting a health care professional before beginning a strengthening program is advised.

How does a low back muscle strain occur and how is it treated?

When force exceeds a muscle's threshold for strength production, injury can occur. Regarding the lumbar spine musculature, lifting from a position of flexion and rotation is the most common mechanism of injury, often creating forces greater than eight times a person's body weight. The pathogenesis of overloaded muscles can lead to involuntary shortening, loss of oxygen supply, loss of nutrient supply, and trigger point formations. When muscle is strained from severe trauma, overuse, or mechanical overload, there is a disruption of the muscle's basic elements myosin and actin. In a normal muscle, these two proteins slide on each other as the muscle contracts. When injured, these proteins remain stuck on one another and often lead to trigger points or shortened muscle fibers. Treatment for muscle strain begins with control of swelling, pain, and edema. The first three days of treatment following injury should consist of ice, compression, and avoidance of activities that may create more injuries. Day three up until six weeks should consist of gentle range of motion, soft tissue mobilization via dry needling or cross friction massage, and strengthening within the patient's tolerance.

How long do muscle strains usually take to heal?

Grade I muscle strains mean that only 25% of the muscle fibers were injured. These types of strains present with pain when stretched, but no strength loss during resisted testing. The strains heal between 3 days and 2 weeks. Grade II tears infer that 50% of the muscle fibers are involved and normally take 4 to 6 weeks to fully heal. Grade II tears are diagnosed by pain upon stretch and pain and weakness against resistance. Grade III muscle tears usually require surgical intervention and may require from 12 weeks to one year of rehabilitation to fully heal. These tears are diagnosed when 75% to 100% of the muscle fibers are injured, there is a complete loss of strength when manually tested, and the response to stretch or contraction is painless since the muscle's nerve supply was also disrupted during the injury.

What are myofascial trigger points and how are they treated?

Myofascial trigger points are defined as taut, hyperirritable bands within a skeletal muscle that are painful with palpation. Myofascial trigger points often refer pain to other regions surrounding the tissue. They also may twitch or involuntarily contract when palpated. Research has identified an increased metabolic demand within tissues containing trigger points. In my opinion the most effective method of treating myofascial trigger points is through dry needling. Dry needling releases shortened muscles, produces local inflammation necessary for healing, and removes chemical elements responsible for pain production.

Richard A. Banton, P.T., D.P.T., A.T.C

Richard Banton has served as co-clinic director for Virginia Therapy and Fitness Center since its inception in 2004. He has been practicing physical therapy since 1998, working with a variety of orthopedic, neurologic, and pediatric conditions. His extensive experience includes the treatment of athletes from the high school to collegiate and professional levels; including Olympic athletes, Washington Redskins football players, and other athletes from NASCAR and the LPGA.

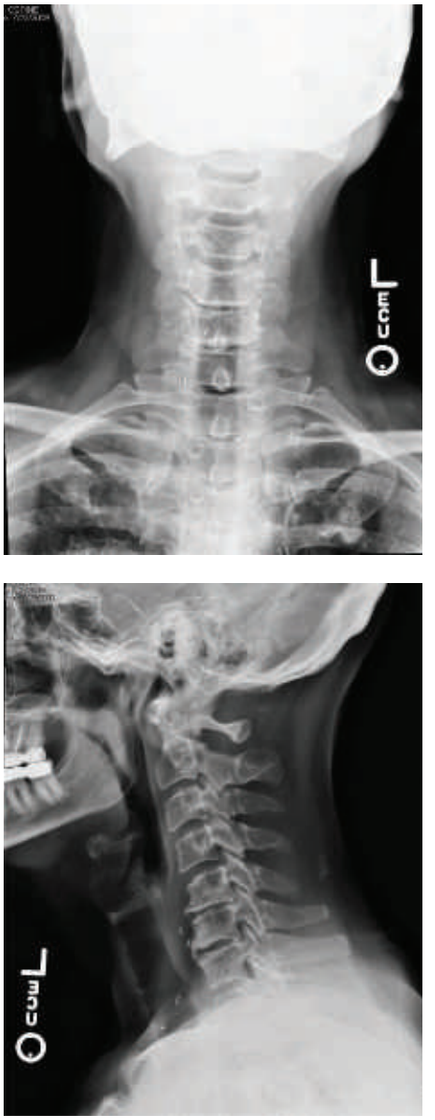
Spine Tale

Brian R. Subach, M.D., F.A.C.S.

It is a pleasure to present the story of Nancy Klepper. Nancy is the focus of our Spine Tale for this issue of the *Journal of The Spinal Research Foundation*. She is a sixty-three year old woman who was initially referred to my office for complaints related to her neck. Nancy has had significant neck problems, off and on, for the past fifteen years, stemming back to a car accident she was in as a teenager. She had pain, tingling and numbness in both arms, as well as a burning feeling in her neck and shoulders, and difficulty with her balance. The pain in her neck caused Nancy to have disabling headaches.

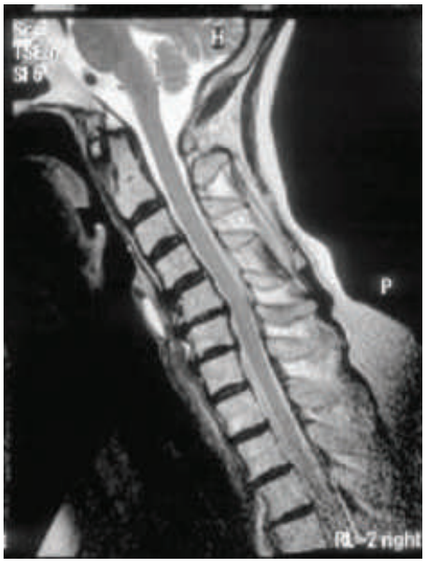
The pain was so severe that it bothered her almost every day. She found some relief by lying on her side, but essentially was unable to sleep at night due to the pain. Sitting at the computer and even sitting while watching TV were intolerable.

Nancy does have a family history of spinal disease, and essentially presented with severe degenerative changes in her cervical spine. She had failed the typical conservative management strategies, including physical therapy, anti-inflammatory agents, massage, pain medications, and muscle relaxants. Her neck x-rays showed reasonable posture, however, she clearly had severe degenerative changes at the C4/5, C5/6 and C6/7 disc spaces. Her range of motion x-rays demonstrated that arthritis had essentially stiffened her spine and the grinding arthritis pain was forming bone spurs, compressing both her spinal cord and the exiting nerve roots. Nancy's MRI scan showed, similarly, that disc herniations were compressing her spinal cord.



Anterior-posterior and lateral pre-surgery x-rays showing severe arthritis.

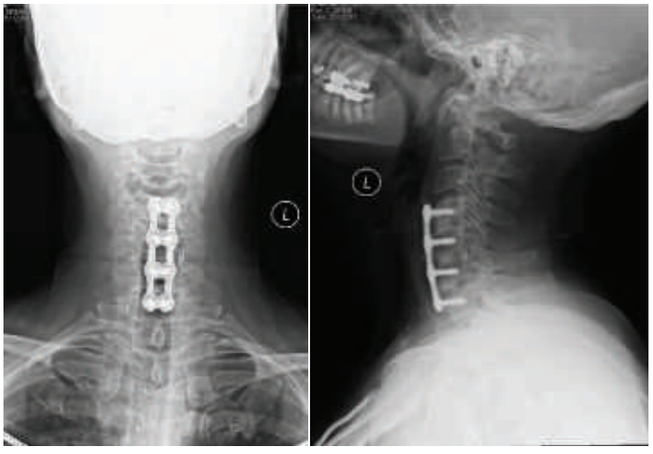
She had previously undergone a cervical fusion using donor bone and plate fixation, which failed to incorporate properly at the C4/5 and C6/7 levels. Upon review of her studies, we recommended a revision surgery, which essentially consisted of removing the plate, using an osteotome to cut through the areas in which scar tissue had intermingled with the bony fusion, and performing the fusion a second time at C4/5 and C6/7 to accomplish a solid incorporation. This would be the best way to restore her posture,



Sagittal pre-surgery MRI showing advanced arthritis and stenosis.

alleviate her neck pain, and stabilize her spine in a permanent fashion. To perform the revision procedure, we utilized rh-BMP-2 (recombinant human bone morphogenetic protein) bone graft, which is essentially a human bone-forming protein produced by transfecting human chromosomes into Chinese hamster ovary (CHO) cells. The cells mass produce this protein, which is absolutely identical to native human protein, and there is no risk of disease transmission. The protein was placed along with bone grafts and plate fixation to help promote healing. At this point in time, Nancy has not yet completely healed but is clearly on the road to recovery.

Nancy Klepper is the Spine Tale for this edition of the *Journal of The Spinal Research Foundation* since she has overcome adversity and suffered from years of disabling neck pain before finally finding a solution to her problems. The solution entailed a cervical fusion utilizing the state of the art rh-BMP-2 protein along with a very technically advanced plating system to stabilize her spine. We applaud Nancy's efforts in dealing with spinal disease and wish her the best as her recovery progresses.



Anterior-posterior and lateral post-surgery x-rays showing the fixation plate and the correction of the cervical curvature.

Nancy has recovered a good range of motion in her neck.

Issue Overview

Marcus M. Martin, Ph.D. and Anne G. Copay, Ph.D.

The current issue of the *Journal of The Spinal Research Foundation* covers the pathology of the soft tissues supporting the spine. These conditions are often treatable with the intervention of well-trained physical therapists. Physical therapists may be able to identify soft tissue dysfunctions, which act as pain triggers or physiological generators of spinal instability. They are trained to treat many of these conditions non-invasively, so that patients may delay the need for more aggressive treatment. They treat patients prior to spine surgery and assist with their rehabilitation after surgical interventions.

In this issue, we get valuable input from exceptional physical therapists covering cervical, thoracic, and lumbar soft tissue dysfunction. Larry Grine, Richard Banton, and Michael McMurray provide us with an outline of some common conditions that affect the tissues in specific regions of the spine. Erin Friend presents an article providing insight into proper workstation ergonomics to promote spine health. This issue also includes articles from three pain management physicians: Michael Cicchetti, Thomas Nguyen, and Neil Chatterjee. Their topics are the novel mechanisms of treating pain by botox injection into soft tissue, alternative methods of pain therapy through the use of acupuncture, and the use of EMG in the diagnosis and treatment of spine diseases. We have also included an article by one of the top researchers in the field of myofascial trigger points, Lynn Gerber, who, along with her research partners at The National Institutes of Health, is dedicated to unlocking the mechanism of myofascial trigger point formation.

Most spine disease incorporates or is initiated by some form of soft tissue dysfunction. This may include the muscles, ligaments, tendons, or the intervertebral disc. Maintaining optimal back muscle, tendon, and ligament health are key ingredients to maintaining a healthy spine. Proper care for back muscles includes muscle strengthening to avoid deconditioning, which is a precursor to many spine conditions. We can consider the spine as a multi-jointed center pole holding up a circus tent. In this analogy, the tent material represents the muscles and ligaments, and the ropes that anchor the post and the tent to the ground represent the tendons. A tear in the tarp, or a weakened or overstretched rope contribute to instability of the tent. Interventions to restore proper function of the supporting structures of the spine can often alleviate aberrant mechanical stress applied to the spine, add to the longevity of its discs, and improve the overall stability of the spine.

This issue provides some background anatomy of the structural support provided by the soft tissue surrounding the spine. It also gives us important information related to common pathologies in these regions and treatments used to address these conditions.

Marcus M. Martin, Ph.D.

Dr. Martin's research interests include virology, immunology and neuroimmunology. He is engaged in collaborative research through SRF, with the Medical University of South Carolina Children's Hospital, geared toward the development of neuroprotective and neuroregenerative compounds for the treatment of nerve pathology.

Anne G. Copay, Ph.D.

Dr. Copay studies the outcomes of surgical and non-surgical spine treatments. She published several articles on the outcomes of spine fusion. She has ongoing research projects concerning the effectiveness of new spine technologies and the long-term outcomes of surgical treatments.

Soft Tissue Structures Supporting the Cervical Spine

E. Larry Grine, P.T., M.S.P.T., A.T.C., C.S.C.S.

The human neck is considered the most mobile region of the spinal column as it is in nearly continuous motion throughout the entire day and even while we sleep. It is remarkable that through an area allowing such exceptional movement pass some of the most delicate and vital structures to sustain human life, such as the spinal cord, carotid and vertebral arteries, jugular veins, esophagus, and trachea. In order for movement to occur, without injury, precise reflexive motor control of the head and neck is provided by intricate and sophisticated neuromuscular machinery. This machinery consists of bones, joints, and the soft tissue structures supporting the cervical spine.[1] The purpose of this article is to review the soft tissue structures that support the cervical spine and provide a brief discussion on the relation of cervical soft tissue structures to common symptomatic conditions of the neck region. The cervical spine has the potential to lead to numerous symptom presentations including pain and numbness in the face, neck, shoulder, upper back, and upper extremity, headache, vertigo, incoordination, and muscle spasticity.[2] In many states, direct access legislation allows patients to arrange an appointment directly with a physical therapist without a prescription. Physical therapists are able to comprehensively examine each patient for biomechanical dysfunction in the spine and extremities and are able to differentially diagnose conditions that are not appropriate for physical therapy. A comprehensive examination of the cervical spine includes a proper history, assessment of active joint movements, neurological testing, special tests, and a hands-on (manual) biomechanical joint assessment of the cervical spine from the base of the skull down to the thoracic spine. Despite having direct access, it is imperative for all physical therapists to utilize a team approach and communicate effectively with every patient's physicians.

Cervical Spine

The cervical spine requires optimal function of all of its supporting structures to allow for proper balance between mobility and stability for each articulating segment.[3] Adequate segmental stability in the neck

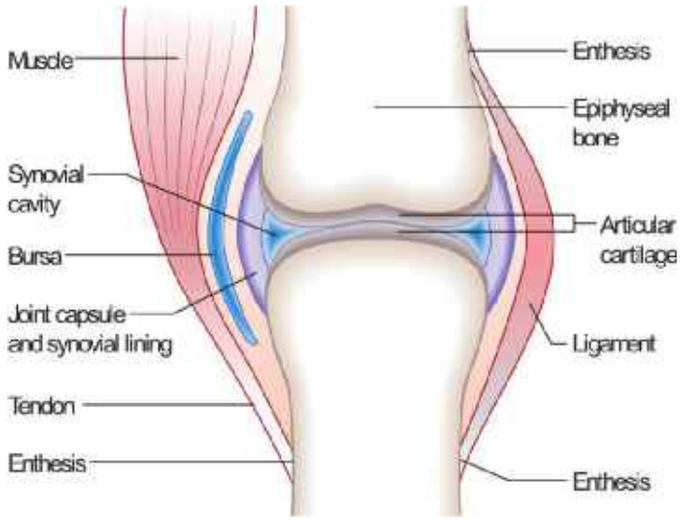


Figure 1. Components of a joint. *Image Courtesy of Wikimedia Commons.*

is necessary for normal and pain-free joint motion. According to Panjabi, stability is achieved by the passive, active, and control systems of the body through joint approximation (bringing the joint surfaces closer together).[3] The passive system describes ligaments and fascia, the active system describes the muscular system, and the control system describes the interplay and interaction of these systems. Stability is achieved through a combination of *joint form closure and joint force closure.* The amount of approximation required is variable and is essentially dependent on an individual's joint bone structure and supporting muscular control. The term "form closure" is used to describe how the joint's structure, orientation, and shape contribute to stability and potential mobility.[3] All joints have a variable amount of form closure which will dictate how much additional force, generated by muscles supporting the joint, is needed to allow stabilization when loads across the joint increase.[4] The force needed to provide additional stabilizing forces is called force closure. A delicate balance needs to exist between joint stability and mobility to allow the body to adapt to imposed demands of different environmental situations. The skilled manual physical therapist is able to properly evaluate and determine when muscles are too active (spasm), not active enough (weakness), and when

additional supportive structures and joints are not moving adequately.

Every anatomical tissue of the human body has an exhaustive potential, a breaking point, whereby it can no longer function normally without being damaged. If the force delivered to a specific area of the body exceeds the force specifications of that tissue it will surpass the exhaustive potential and become damaged and injured.[5] In addition, anatomical tissue must be subjected to nondestructive forces to maintain an optimal state of health. When tissues are subject to excessive loads or extremely minimal loads, tissue breakdown may occur. This may arise either through injury (destructive force) or as a result of catabolic effects of inactivity (weakness). The boundaries of the optimal loading zone can be altered by several factors including: age, adaptive changes of the soft tissue structures, and injury.[6] These factors have the ability to lower the threshold for destructive loading and reduce the body's ability to absorb or attenuate forces, making the body more susceptible to an initial or subsequent injury.

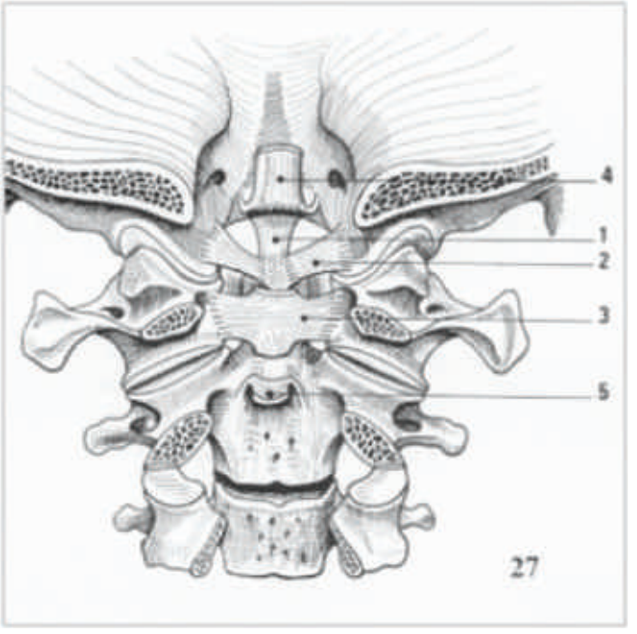


Figure 2a. Dissection of several posterior craniovertebral ligaments. *Image was published in* Physiology of Joints*, Vol. 3, Kapanji, p. 189, Churchill Livingstone 1974.*

Specialized Connective Tissue

In this article, specialized connective tissue refers to bone, articular cartilage, and non-contractile connective tissues such as the intervertebral discs, supporting ligaments, fascia, and joint capsules. Collectively the connective tissues described form a synergistic relationship with each other to promote movement either by providing support or guiding motion.[2] The primary function of the specialized connective tissues, especially the bones and intervertebral discs, is to reduce and distribute the forces of gravity and movement.[3] The bones of the upper quadrant take many shapes and forms. The various articulations within the upper and lower cervical spines provide a structure that allows for considerable mobility. The cervical spine also meets the demands of stability by counteracting the weight of the head and offering a rigid lever system for muscle attachments.

Although certain anatomical features of the articulations of the cervical spine have similar counterparts to the thoracic and lumbar spines, there are several aspects that are unique to the cervical region. The com-

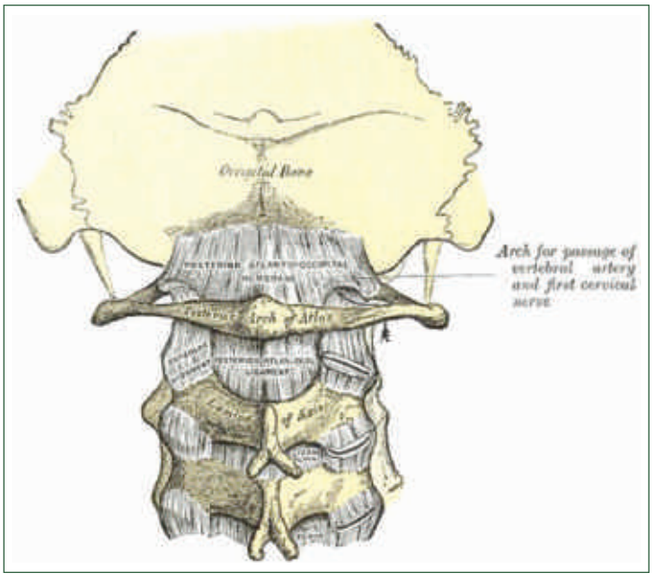


Figure 2b. Posterior view of craniovertebral joints and ligaments. *Image courtesy of Bartleby.com from Henry Gray's* Anatomy of the Human Body.

pressive forces and weight-bearing requirements of the cervical spine are absorbed from the weight of the head and actions of muscles crossing the cervical spine.

The cranio-vertebral joints (upper cervical region), the cervical facet joints, and intervertebral discs demonstrate distinct anatomical features that contribute to the unique biomechanics of the cervical spine. The upper cervical region of the neck articulates with the skull and has proximity to several vital structures including the lower portions of the brain, vertebral arteries, and neurovascular bundles. The sensory feedback from this region to the brain dictates the interpretation of our head position in space, prompting continuous modifications to our visual and balance systems. The upper cervical region provides joint motion that accounts for nearly 50% of all flexion/extension and more than 50% of left/right rotation of the entire cervical spine range of motion.[4] The arrangement of the bony congruency provides only minimal stability. Motion and stability in this region is controlled by the ligamentous and muscular network, making it particularly vulnerable to instability resulting from diseases that affect connective tissue, such as rheumatoid arthritis.[1] The weight-bearing through the middle and lower cervical spine is distributed through three primary loading areas consisting of the intervertebral disc anteriorly and the two facet joints posteriorly.

An ***intervertebral disc*** has a disc-like form. The disc itself is composed of a gelatinous core, the nucleus pulposus, surrounded by a fibrous ring, the annulus fibrosis. Water makes up roughly 90% of the gelatinous core. The discs account for about a fourth of the total length of the spinal column. In relation to its surface area, the cervical disc height is relatively high, which in part allows for the large amount of motion present in the cervical spine. The cervical discs are wedge-shaped, having a greater anterior height than posterior height. This difference in height between the front and back of the cervical disc promotes the normal cervical lordotic posture.[1]

The movements of the spinal column occur in segments. These segments consist of vertebrae, discs, ligaments, and vertebral facet joints. A single segment is itself capable of only very restricted movement, but

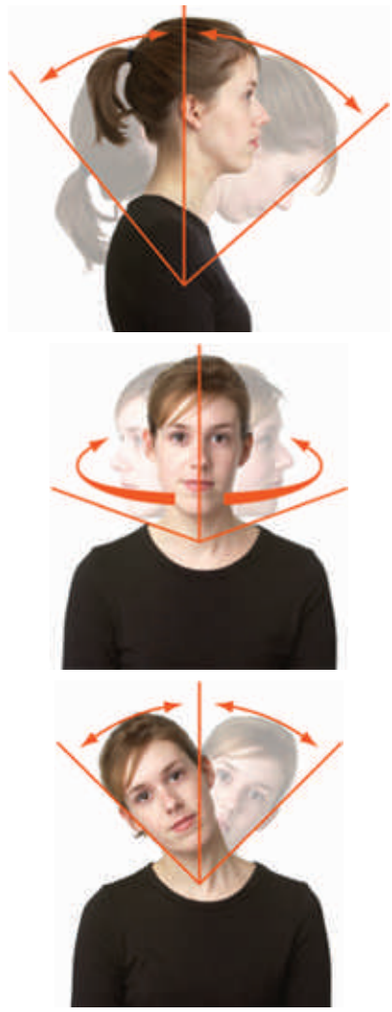


Figure 3. Movements of the cervical spine. *Image Courtesy of Medtronic.*

all the segments together allow for the great flexibility of the spinal column as a whole. The intervertebral discs accommodate each movement as well as possible. The give and take between nucleus pulposus and annulus fibrosis allow for the movements to be smooth. The nucleus pulposus acts as an incompressible pillow of water. The core of a healthy disc is centered when at rest and moves outward during movement. The annulus fibrosis guides the gelatinous core during such movement. Vertical pressure such as exerted by gravity is absorbed by the discs. The discs are, in fact, excellent shock absorbers, capable of snapping back into place and adjusting to various kinds of movement.

E. Grine/Journal of the Spinal Research Foundation 6 (2011) 8–16

The ***joint capsule*** of the cervical ***facet joints*** is vital to the function of the cervical spine. It seals the joint space, provides passive stability by limiting movement, provides active stability via its proprioceptive nerve endings, and may form articular surfaces for the joint. It is a dense fibrous connective tissue that is attached to the bones via specialized attachment zones and forms a sleeve around the joint. It varies in thickness according to the stresses to which it is subject, it is locally thickened to form capsular ligaments, and may also incorporate tendons. The capsule is often injured, leading to laxity, constriction, and/or adhesion to surrounding structures. The facet joints are most commonly affected by osteoarthritis. Cervical facet joint pain can be felt over the affected joint but can also be referred to the shoulder girdle, shoulder blade or arm. It tends to be worse with extension of the spine (bending backwards). Cervical facet joint pain is also present in rheumatic diseases, including rheumatoid arthritis and osteoarthritis, crystal deposition disorders, bony spur formation, and ankylosing spondylitis.[2]

Articular cartilage of the cervical spine facet joints is loaded by compression forces of gravity and muscular contraction.[6] With compression, fluid is expelled from the articular cartilage. With decompression, fluid seeps back into the cartilage. This cyclical activity of compression and decompression maintains the health of the articular cartilage. Excessive compression that cannot be unloaded diminishes the capacity for joint fluid and associated nutrients to seep back into the articular cartilage, thereby accelerating the degenerative process. The degenerative process of articular cartilage places an increased stress on the subchondral bone.[6] The subchondral bone will then begin to take an increased compressive load. Cervical spondylosis (arthritis of the neck), bony sclerosis, facet joint arthritis, and osteophytes (bone spurs) are the eventual result of this process (Figure 4).[6]

Fascia is a specialized non-contractile tissue that encases muscles and organizes muscles into different layers as separated fascial planes. The fascia in the cervical spine is an organized system of layers and forms complex synergistic relationships

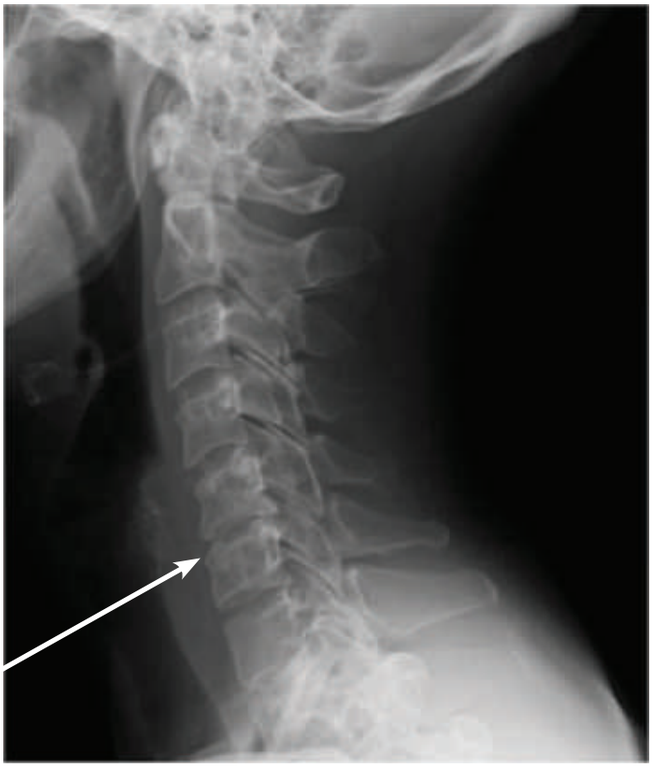


Figure 4. X-Ray of the neck with progressive degeneration of spine (spondylosis) at C5-6 level.

with muscles and joints of the neck. Healthy fascia networks are key in directing and distributing the forces associated with muscle contraction through weight-bearing tissues such as articular cartilage of joints, vertebral bodies, and intervertebral discs. As in other areas of the musculoskeletal system, the fascial network is intimately related to the muscles of the cervical spine.

The sternocleidomastoid muscle is related to the investing fascia of the neck. It is usually one of the soft tissue structures most damaged during an acceleration injury to the neck, particularly when the impact is from behind.[5] Muscle pain and trigger points related to the sternocleidomastoid muscle have the potential to induce ringing in the ears and cause postural and spatial disturbances such as dizziness and vertigo.[6]

Non-contractile supporting tissues (consisting of ligaments, fascia, and the intervertebral discs)

provide stability of the joints passively by restricting motion. *Viscoelasticity* is a property which describes that, within a specific range of stress (stretch), a tissue has elasticity allowing it to return to its original shape and making it resistant to deformation. If stress to a tissue exceeds its viscoelastic capacity, it will no longer return to its original shape and will remain permanently elongated or deformed. When ligaments of the neck are stressed to such a degree that they are permanently deformed, the static and dynamic stability between adjacent cervical vertebrae is lost. The loss of stability allows for excessive translational movements between spinal segments.[4] This can occur due to injury to the spine (such as whiplash from a car accident), from repetitive movements affecting specific segments, or from dysfunctional postures. Excessive translational movements (*segmental hypermobility*) between adjacent spinal segments can cause local joint and nerve irritation allowing for an inflammatory reaction. Lack of joint stability can also cause accelerated degeneration at that spinal segment. Segmental hypermobility may progressively worsen and progress to *segmental instability*. If the segmental movement of adjacent spinal segments is not adequately controlled by the surrounding ligaments, then the spinal segment will rely significantly more on the local muscular support to gain stability in that hypermobile segment. If a spinal hypermobility has progressed to a spinal instability, then the passive restraints of that segment have been lost and the neuromuscular system is not able to maintain the integrity of the involved segment, often causing significant episodic or chronic pain. The best treatment of spinal instability may be spine fusion surgery.

Neuromuscular System

The neuromuscular system refers to the supporting muscles of the cervical spine. Muscles direct forces through the specialized connective tissues and fascia by way of their attachments to various bony levers of the skull, cervical spine, and upper extremity. The actions of the muscles related to the cervical spine

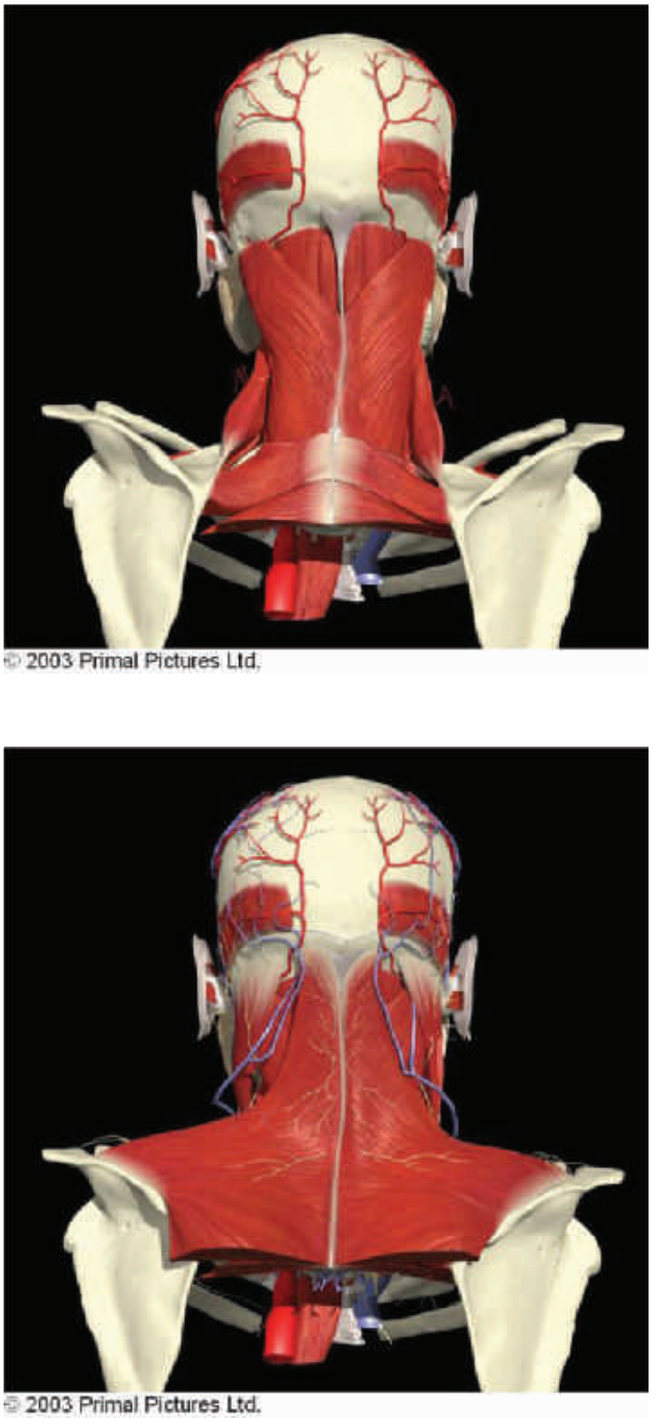


Figure 5. Synergistic relationship between the Trapezius muscle and the Longus Capitis and Longus Colli muscles. *Permission Pending.*

ultimately depend on the afferent information supplied from the joint and muscle receptors associated with the head. An especially sophisticated network of neuromuscular reflexes is integrated with the cervical spine because of the significant number of reflex connections with the auditory, visual, and vestibular (dizziness & balance) systems.

Muscle activity helps to counterbalance the forces of gravity and movement and, often times, works in complex synergies with other muscles surrounding the neck region. This allows for movement patterns that minimize abnormal stresses to the specialized connective tissues. Precise control and discrete functions of the neuromuscular system allow shock absorption to be distributed to muscles. When the central nervous system perceives painful stimuli, there is an increased control of movement patterns, which restricts available motion and redirects forces away from injured tissues. Therefore, the neuromuscular system plays an important role in redirecting potentially destructive forces away from an injured area.

Because of the elaborate sensory system of the head, the neck is active in nearly every activity we encounter on a daily basis. A disruption in neck function can have a significant impact on normal activities of daily living or on occupational demands. Complaints of neck pain can be influenced by numerous factors that can be unique to each patient. Some common influencing factors are psychological, emotional, social, and cultural in nature. Afferent influences refer to influences that are sensed by the body through specialized receptors found in the body's connective tissues and then are reported to the nerve center of our brain for interpretation. Based on our brain's interpretation of these stimuli, an effective response is initiated through our efferent nerves. The efferent nerves connect to specific muscles to initiate a response to the stimuli. The muscles of our bodies are constantly receiving stimulation based on our responses to our immediate surroundings and situations. Many of the muscles of the cervical spine form synergistic relationships with one or more muscles to provide a balanced collective response when initiating movement.

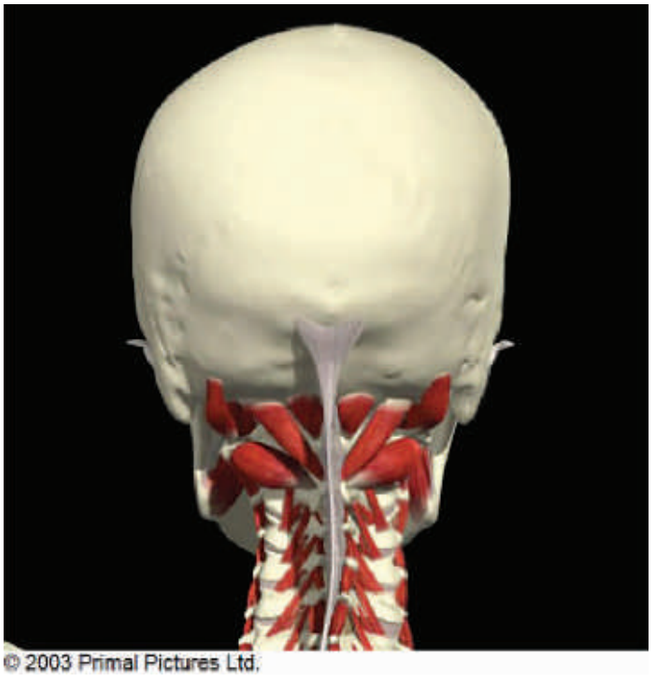


Figure 6. Sub-occipital musculature.

Synergistic Relationships of the Cervical Spine

Cervical Synergy #1: The trapezius muscle (Figure 5) exists in a synergistic relationship with the two anteriorly placed muscles, the longus capitis and longus colli. The longus capitus and longus colli muscles counterbalance the trapezius muscles from the front and the back of the neck. With neck pain and injury, the neural mechanisms of the longus capitis and longus colli "turn off", causing the trapezius to promote an imbalance. This imbalance leads to common patient complaints of neck pain at the base of the neck, above the shoulder blade, and headaches. Travell and Simons consider the trapezius muscle to be the cervical spine muscle most often inflicted with trigger points. Trigger points often are a source of irritation leading to temporal headaches.[7]

Cervical Synergy #2: The upper trapezius also has a synergistic relationship with the levator scapulae. Both muscles have the ability to work together as synergists and elevate the shoulder girdle. However, complete abduction of the shoulder requires

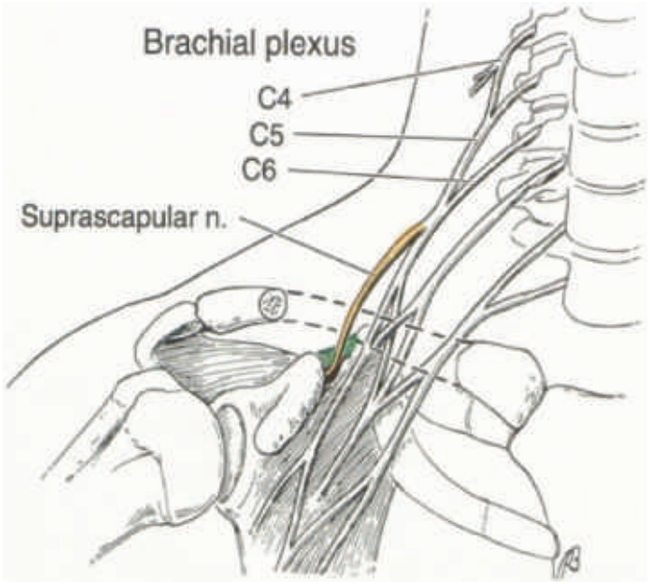


Figure 7. Brachial Plexus of the Cervical Spine in relation to the upper extremity. *Image courtesy of Dr. Jon J.P. Warner, www.bosshin.com.*

a movement of upward rotation of the scapula. For the scapula to rotate upwardly, the upper trapezius is actively shortened and a lengthening contraction occurs with the levator scapulae muscle. Therefore, for the scapula to rotate upwardly, the upper trapezius and levator scapulae must work against one another as antagonists.

Cervical Synergy #3: The cervical spine, scapular retractors, and abdominals also have a synergistic relationship. The cervical spine is subjected to anterior shearing forces resulting from the pull of gravity caused by the normal cervical posture of cervical lordosis. The pull of gravity is accentuated by forward-head posturing. The levator scapulae muscle is oriented to help provide a dynamic restraint to this force. The scapula provides a base of attachment for the levator scapulae. Because the scapula is not fixed like the pelvis, the position of the scapula is maintained in part by muscular control of the rhomboid major, rhomboid minor, and trapezius muscles. These three muscles have the ability to pull back (retract) the scapula. A rounded-shoulder posture commonly is attributed to the lengthening or weakness of the scapular retractors. Attention should also be given to the abdominal wall muscles and the role they play as they work synergistically with the scapula retractors, diaphragm, and pelvic floor muscles to align the abdomen and thorax as well as the relation of the scapula to the thorax. This synergistic relationship between the cervical spine and lower regions of the body links the cervical spine to the thoracic and lower lumbar spine and highlights an important relationship between these areas in order to maintain proper cervical balance.

Cervical Synergy #4: The sub-occipital muscles (Figure 6) are positioned to move the upper cervical joints using complex relationships that are independent of the lower cervical spine. This function allows the lower cervical spine to be positioned and fixated while the upper cervical spine moves into positions that optimize the placement of the various sense organs such as the eyes and ears. For example, when people attempt to track a moving object with their eyes, it is essential that there be a concurrent, coordinated movement of the cervical spine to allow the eyes to follow the object. Also, when people are subjected to a sound, they often reflexively move the position of their head to increase the chances of the sound waves reaching their auditory apparatus.

The nerves and neural tissue of the cervical spine can be affected by changes in the dimensions of the spinal canal and openings where the cervical nerves exit the cervical spine segmentally. The dimensions of these openings can be altered with spinal motion. The peripheral nerves that exit the cervical spine typically innervate the neck, chest, shoulder, upper back, and upper extremity. Neural tissue is very sensitive to inflammation and compression. Nerve irritation can trigger an inflammatory response that causes pain locally and referred to distal points that share the same nerve innervations. Nerve compression and irritation can cause a condition referred to a neural tension. *Neural tension* presents a scenario where a nerve is irritated and thereby causes tightness in muscles that surround the nerve or causes inflammation to the muscles that the nerve connects. Nerve compression can also cause symptoms that refer to distal

Decision Rules for Cervical Treatments

Examination Findings	Proposed Interventions
• Recent onset of symptoms • No radicular or referred symptoms in the upper quarter • Restricted range of motion with side-to-side rotation or discrepancy in lateral flexion range of motion, or both • No signs of nerve root compression or peripheralization of symptoms in the upper quarter with cervical range of motion	Interventions designed to improve range of motion and to decrease pain and disability • Cervical and thoracic spine mobilization/manipulation • Active range-of-motion excercises
• Radicular or referred symptoms in the upper quarter • Peripheralization or centralization of symptoms with range of motion, or both • Signs of nerve root compression present • May have pathoanatomic diagnosis of cervical radiculopathy	Interventions desgined to centralize symptoms and to decrease pain and disability • Mechanical or manual cervical traction • Repeated movements to centralize symptoms
• Lower pain and disability scores • Longer duration of symptoms • No signs of nerve root compression • No peripheralization or centralization during range of motion	Interventions designed to improve endurance, strength, and flexibility • Strengthening and endurance exercises for the muscles of the neck and upper quarter • Aerobic conditioning exercises
• High pain and disability scores • Very recent onset of symptoms • Symptoms precipitated by trauma • Referred or radiating symptoms extending into the upper quarter • Poor tolerance for examination or most interventions	Interventions designed to decrease pain and disability and to permit further examination • Gentle active range of motion within pain tolerance • Range of motion exercises for adjacent regions • Physical modalities as needed • Activity modification to control pain
• Unilateral headache with onset preceded by neck pain • Headache pain triggered by neck movement or position • Headache pain elicited by pressure on posterior neck	Interventions designed to reduce headaches • Cervical spine mobilization/manipulation • Strengthening of neck and upper quarter muscles • Postural education

Reference: Cleland JA, Markowski AM, Childs JD *Current Concepts of Orthopaedic Physical Therapy* 2nd Edition "The Cervical Spine: Physical Therapy Patient Management Utilizing Current Evidence. Orthopaedic Section APTA. 2006 pg. 7

areas away from the cervical spine that remain consistent with common nerve connections. Nerve irritation and compression have the ability to present with similar symptoms but upon careful clinical exam, a clinician can determine differences based on a complete neurological exam. Significant nerve compression will differ from nerve irritation: a compressed nerve will demonstrate changes in the clinical exam with decreased sensation, decreased reflexes, or decreased strength, and reduced nerve conductivity in the involved anatomy.

Manual Physical Therapy

Through a comprehensive biomechanical examination the manual physical therapist can identify muscular imbalances and improper loading patterns of the spine simply by using their hands. The manual

physical therapist, through extensive training, gains the knowledge of how joints should feel when they move properly, how to identify when they are not, and how to manually improve dysfunctional biomechanical imbalances using treatments of soft tissue manipulation, joint mobilizations, and targeted therapeutic exercise. Even though many patients carry the same tagged diagnosis, such as cervical disc degeneration, each patient requires a unique treatment plan based on their individual needs and condition. The fact of the matter is that many people carry the same diagnosis but the reasons for their pain stems from genetic factors of their anatomy, relevant injuries they have incurred over their lifetime, and movement and postural habits that lead to dysfunction that are unique to each person. Based on these factors, every person has the ability to have a unique presentation and respond differently to physical therapy and the manual physical therapist must have the ability to successfully adapt to each and every patient.

Manual physical therapists assess each spinal segment with their hands. They use specific stress tests relying on the biomechanics of each segment to guide their assessment. The cervical segment stress tests determine if the segments are moving too much or too little. If a segment is moving too little, proper treatment would be to mobilize the segment using manual therapy techniques. If the segment has been identified to be moving too much, proper treatment would be to stabilize the segment using therapeutic exercises.

Many conditions arise from cervical dysfunction and injury to the cervical region. Symptom presentation can be broad and diffuse in some scenarios and in others the symptoms can be localized and intense. Several conditions have been discussed throughout this article. Treatment for all cervical conditions begins with an extensive biomechanical evaluation to determine which segments of the cervical spine are involved and whether the cervical segments are either moving too little or too much. A progression for treatment will involve the following steps: (1) Modulation of Pain and Inflammation, (2) Promotion of Active Movement, (3) Enhancement of Neuromuscular Performance, and (4) Patient Education and Biomechanical Counseling. Patients with cervical dysfunction should expect a successful outcome when they subscribe to appropriate guidance from their medical team which consists of their primary care physician, orthopedic spinal surgeon, and their manual physical therapist.

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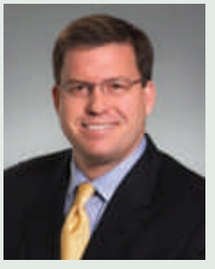

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Larry Grine is an orthopedic manual physical therapist. He has served as co-clinic director for Virginia Therapy and Fitness Center since its inception in 2004. He has been practicing physical therapy since 2000, working with a variety of orthopedic and neurologic conditions. His degrees in physics, athletic training, and physical therapy have allowed him to identify biomechanical dysfunction within his patient presentations and have enabled him to successfully treat high school, collegiate, and professional athletes. He has also assisted treatments for the Washington Redskins and the United States men's World Cup team.

The Soft Tissue Structures of the Lumbar Spine

Michael McMurray, P.T., M.P.T., D.P.T., F.A.A.O.M.P.T.

The soft tissue structures of the lumbar spine include the ligaments, the musculature and the fascia. These soft tissue structures can be grouped into three systems as proposed by Panjabi: the active system, the passive system, and the control system.[1,2] The passive system consists of the ligaments, which offer increased restraint toward the end range of motion. The active system consists of the muscles, which generate force to stabilize the spine. The control system receives information from both systems and makes adjustments through muscle timing and coordination.[2] Each of these structures has different roles, but all play a part in optimal functioning of the lumbar spine. If the integrity of any of these structures is disrupted or injured, then altered functioning, and often symptoms, will arise.

This article will review the anatomy of both the active and passive control systems, as well as further discuss stabilization systems of the lumbar spine, and present the effect of pathology on these systems. The mechanisms by which these systems work together during spinal stabilization will also be discussed. Additionally, treatments that may improve the function of these systems and improve lumbar symptoms will be discussed.

Anatomy

The main supporting **ligaments** of the lumbar spine are the anterior longitudinal ligament (ALL), posterior longitudinal ligament (PLL), sacrotuberous ligament, iliolumbar ligament, and ligamentum flavum.

The **sacrotuberous ligament** is composed of three bands: lateral, medial, and superior. The lateral band connects the ischial tuberosity and the posterior inferior iliac spine and spans the piriformis. The medial band spirals and attaches to the transverse tubercles of S3-5 and lateral margin of the lower sacrum and coccyx. The superior band runs superficial to the interosseous ligament and connects the coccyx with the posterior superior iliac spine. The function of this ligament is to prevent sacral nutation and control posterior rotation of the innominate. This ligament also serves as an attachment for the gluteus maximus muscle (Figure 1).

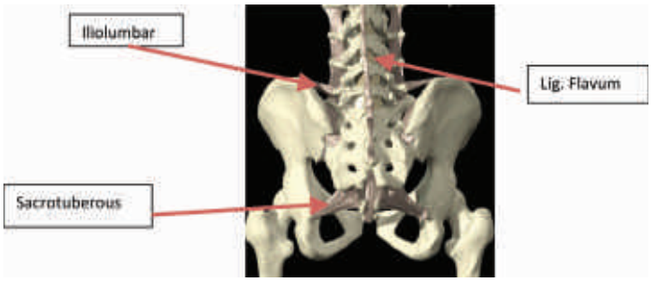

Figure 1. Iliolumbar ligament, sacrotuberous ligament, ligamentum flavum (*Primal Pictures*)

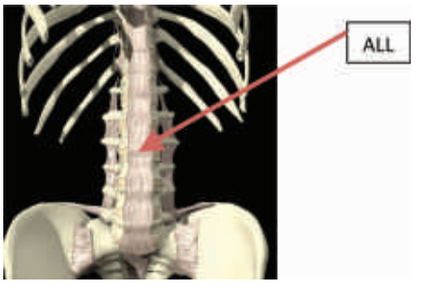

Figure 2. Anterior longitudinal ligament (ALL) (*Primal Pictures*)

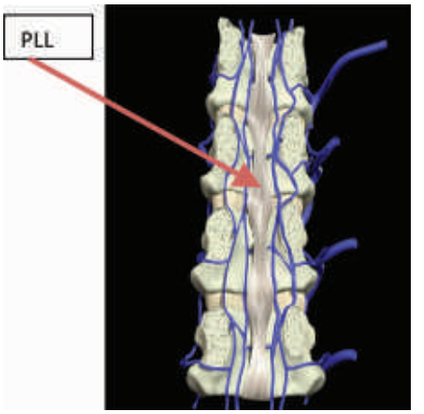

Figure 3. Posterior longitudinal ligament (PLL) (*Primal Pictures*)

The iliolumbar ligament consists of five bands: anterior, posterior, superior, inferior, and vertical. It connects from the tip of the L5 transverse process to the anteriomedial surface of the ilium and the inner lip of the iliac crest. The function of this ligament is to minimize the torque forces at the lumbosacral junction and resist forward sliding of L5 on the sacrum. Unilaterally, it will resist side bending as well as some twisting and forward bending (Figure 1).

The **ligamentum flavum**, otherwise known as the yellow ligament, is a short and thick ligament which connects the laminae of consecutive vertebrae. Its

function is to prevent flexion, as well as pre-stress the disc for functional activities. It is known as the yellow ligament because it is comprised of 80% elastin and 20% collagen which gives it a yellowish hue and makes it more elastic than other ligaments in the body. This is important functionally so that during backward bending the ligament does not buckle and put pressure on the spinal cord (Figure 1).

The **anterior longitudinal ligament** (ALL) lies on the front of the spine and attaches segmentally to the vertebral bodies. The function of this ligament is to prevent extension of the spine (Figure 2).

The **posterior longitudinal ligament** (PLL) is a narrow band that attaches segmentally to the back of the vertebral bodies. It widens over the discs and is narrower over the vertebral bodies. The function of this ligament is to resist flexion (Figure 3).

Musculature

The musculature of the trunk is vitally important to control the lumbar spine during activities. The multifidus, transversus abdominus, external and internal obliques, piriformis and gluteus maximus are important muscles in the stability and function of the lumbar spine.

The **multifidus** in the lumbar spine contains superficial and deep aspects. The deep fibers of the multifidus attach from the posteroinferior aspect of the lamina and articular capsule of the zygapophyseal joint and insert onto the mammillary process two levels below. The superficial fibers insert three levels below; for example, the fibers attaching at L1 insert on to L4 and the medial iliac crest. Multifidus is the largest muscle at the lumbosacral junction. Here it passes distal to the posterior SI ligaments and blends with the sacrotuberous ligament (Figure 4). Functionally, the superficial multifidus is phasic while the deep multifidus is tonic and anticipatory. The multifidus has been found to be a segmental stabilizer of the lumbar spine and plays an important role in recovery from low back pain. Evidence has shown that the multifidus atrophies in patients with chronic low back pain and does not spontaneously recover following low back pain. Patients that do not regain the strength and muscular girth of multifidus have an increased risk for recurrent

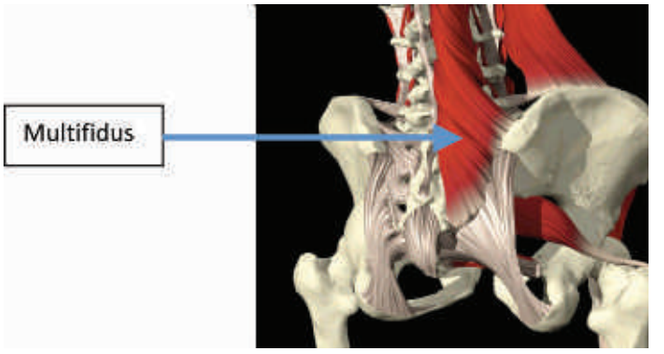

Figure 4. Multifidus (*Primal Pictures*)

low back pain.[6] A targeted exercise program is needed for recovery of this muscle.[6] This type of intervention will be discussed later in this article.

The **transversus abdominus** is the deepest abdominal muscle and has a transverse fiber direction. It arises from the lateral one-third of the inguinal ligament, the anterior two-thirds of the inner iliac crest, the thoracolumbar fascia, and the inner aspect of the lower six costal cartilages, and blends with fibers of the diaphragm. It blends with the internal oblique muscle inferiorly to attach to the pubis crest and superiorly with fibers of the contralateral transversus abdominus

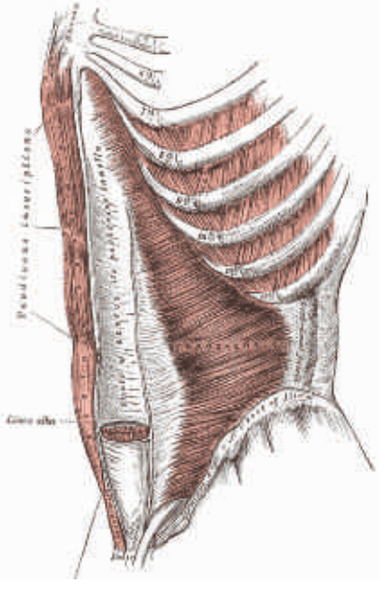

Figure 5. Transversus abdominus (*Henry Gray's* Anatomy of the Human Body, *courtesy of Bartleby.com*)

(Figure 5) and internal oblique (Figure 7). The transversus abdominus has been found to increase intraabdominal pressure as well as play a role in stabilization of the lumbar spine and pelvis due to its connections to the thoracolumbar fascia.

The **external oblique** has two layers, superficial and deep, and runs in an inferomedial direction. The external oblique arises from the external borders of the lower eight ribs and blends with fibers from the serratus anterior and latisimus dorsi muscles. The external oblique is bilaminar. The two layers cross the midline to blend with the fascia of the opposite side. The deep layer is continuous with the contralateral internal oblique, while the superficial layer merges with the superficial layer of the contralateral external oblique (Figure 6).

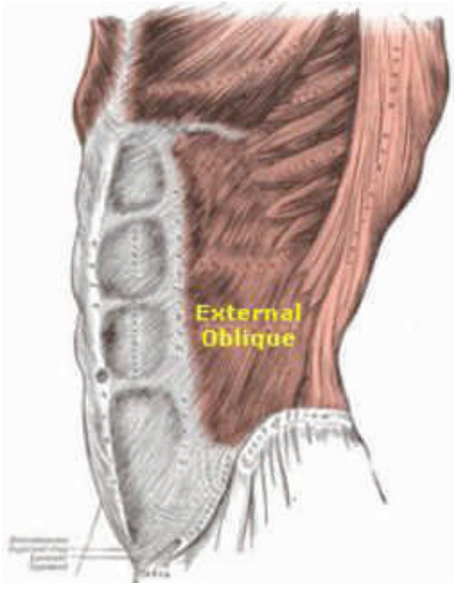

Figure 6. Internal oblique (*Henry Gray's* Anatomy of the Human Body*, courtesy of Bartleby.com*)

The **internal oblique** lies between the external oblique and transversus abdominus. It arises from the lateral two-thirds of the inguinal ligament, anterior two-thirds of the iliac crest and thoracodorsal fascia. It has fibers which attach onto the tips of the tenth through twelfth ribs as well as fibers which blend with the transversus abdominus and the external obliques. This complex attachment system forms a network of abdominal fascia (Figure 7).

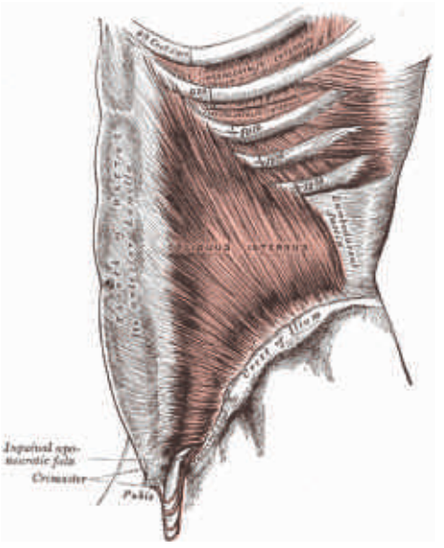

Figure 7. External oblique (*Henry Gray's* Anatomy of the Human Body*, courtesy of Bartleby.com*)

The **piriformis** muscle arises from the sacrum, sacrotuberous ligament, superior margin of the greater sciatic notch, and the medial edge of the sacroiliac joint capsule, and attaches onto the greater trochanter of the femur. The action of the piriformis is to laterally rotate the hip and stabilize the head of the femur in the acetabulum. It has also been found that the piriformis contributes to the stability of the lumbosacral complex by tensioning the sacroiliac joint capsule and pulling the sacrum against the ilium (Figure 8).

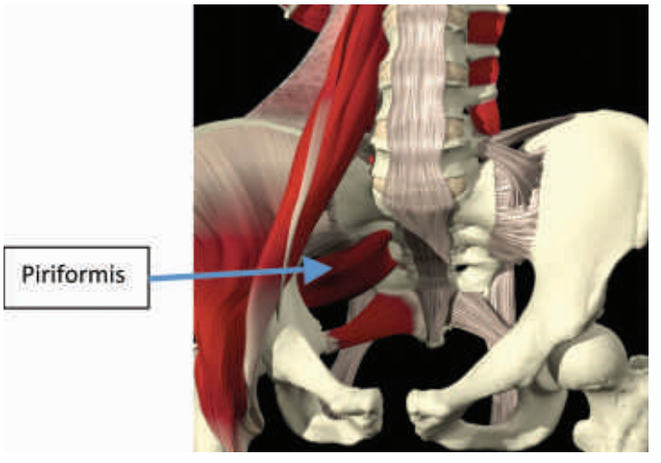

Figure 8. Piriformis (*Primal Pictures*)

The **gluteus maximus** is the largest skeletal muscle in the body. It originates from the posterior surface of the ilium and iliac crest, the thoracolumbar fascia, sacrotuberous ligament, and lateral sacral crest, and attaches to the gluteal tuberosity of the femur and blends with the iliotibial band. Through its attachments, it is coupled to the ipsilateral multifidus and contralateral latissimus dorsi muscles. Gluteus maximus is primarily a hip extensor and has been found to also have a role in lumbar extension. Because of its attachments into the ligaments and fascia of the lumbosacral complex, the gluteus maximus has been found to also have a role in stabilization of the lumbar spine.

Mechanisms of Stability

As previously mentioned, Panjabi theorized that for optimal stability, three systems must function properly: the active, passive and control systems.[2] These systems can be incorporated into another model of stability, force closure and form closure.

Force closure is described as when "extra forces are needed to keep the object in place". These extra forces can be directly applied at the joint in the form of resting tone or co-contraction of muscles that cross the joint. They can also be applied indirectly in the form of resting tone or co-contraction of muscles that do not cross the joint, but increase tension of the surrounding fascia.[1] The active system is incorporated into the force closure model by incorporating the muscles that were previously described.

Form closure is how a joint's structure, orientation and shape offer stability.[1] This is accomplished by a combination of the joint's anatomy and the capsular and ligamentous integrity. The passive system is incorporated in the form closure model and incorporates many of the ligaments described early in this article. According to Lee,[5] the function of the lumbopelvic hip complex is to transfer loads from the lower quarter to upper quarter safely while performing a task.[1]

One of the most important tasks in which all three systems work together is stabilizing the spine. Authors and researchers have found that the spine itself is inherently unstable. So unstable that, without these stabilization systems, the spine would collapse under approximately 9 kg of load.[1,3]

For many years, research has been directed at how the spine achieves stability and how to effectively retrain this stabilization system in individuals with low back pain. Early research revealed that causing stiff joints would prevent the buckling of the spine in static environments. Co-contraction of multiple superficial multi-segmental muscles surrounding the spine was the most effective way to accomplish this. This translated into the rehabilitation arena where patients were instructed in "muscle bracing" techniques to cause a "stiff spine." However, this technique did not carry over into functional dynamic activities. As research continued, it was revealed that the deep muscles of the spine, the transversus abdominus, multifidus and pelvic floor muscles, were more effective and anatomically suited for specific spine stabilization. Activation of these muscles did not cause the reduced joint mobility and increased torque that accompanied superficial muscle "bracing" techniques.[1] Additionally, activation of these deep muscles of the spine for stabilization allowed for functional trunk mobility while maintaining adequate stability. These findings revealed that true spinal stabilization requires not only static resistance to buckling, but also segmental control during motion.[1,3,5]

Current research has determined that the deep muscles of the trunk are vitally important to both static and dynamic control of the lumbosacral complex. Prior to any movement, these deep muscles, the transversus abdominus, pelvic floor muscles, and diaphragm, activate before any other muscle in the body.[4] This occurs regardless of type or direction of the activity being performed. If the activity is continued, these muscles continue to work in a tonic manner throughout the activity. Another deep trunk muscle, the multifidus, also demonstrates activation prior to the onset of the activity, but not with every activity. There seems to be direction specificity to its activation that is not found with the other deep trunk muscles. This was also found with the superficial muscles such as internal oblique, external oblique, erector spinae, and rectus abdominus.[1,3,4]

Generally, it has been revealed that the deep muscles coactivate and work synergistically to prepare

the body for load by increasing the intraabdominal pressure and fine tuning the segmental stiffness of the intervertebral, interpelvic, and hip joints. This occurs prior to activation of the superficial system so that the forces exerted by the superficial muscles cause uniform motion of the spine, evenly distribute forces, and maintain proper axes of rotation for each joint.[1,3,4,5]

In order for an individual to display optimal control and stability in both static and dynamic situations, the superficial muscle system must work in synergy with the deep muscle system. This occurs through multiple linkings of muscles, otherwise known as slings. These slings have been described as mechanisms that serve to transfer loads through the trunk and produce stabilization of the trunk during dynamic activities. This occurs by the superficial muscles working in synergy with each other and with the deep muscle system. Two of the trunk slings are the posterior oblique and anterior oblique. The anterior oblique sling connects the external oblique, anterior abdominal fascia, and contralateral adductor muscles (Figure 9). The posterior oblique sling connects the lattismus dorsi and the gluteus maximus muscles through the thoracolumbar fascia (Figure 10). These muscles connect through the fascia to produce vectors of force, which provide optimal alignment of bones and joints, and assist in the transfer of load when they are properly balanced. The disruption of this balance causes altered vectors and disrupted transference of loads, which commonly will result in dysfunction and pain.[1]

Research to determine the specific mechanism that the spine uses for stability has proven difficult. The current models do not fully explain the spinal stabilization system, but as more research is completed, more understanding of this complex system is achieved.[1,3,4] It has been theorized that there are many components to spinal stabilization, and therefore, many approaches may be used by clinicians to improve spinal stability.[1] The use of multiple strategies may be the most effective treatment course due to the complex nature of this system. For example, use of bracing techniques during predictable, high load situations may be an effective strategy. However, during unpredictable dynamic activities, activation of the deep stabilization system may be more effective.[1]

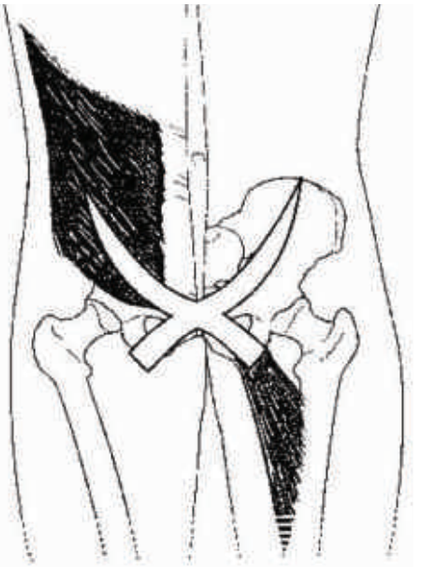

Figure 9. Anterior oblique sling (*Courtesy of Diane Lee's The Pelvic Girdle*)

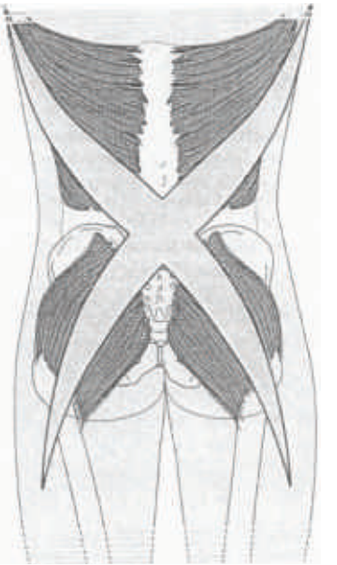

Figure 10. Posterior oblique sling (*Courtesy of Diane Lee's The Pelvic Girdle*)

Injuries

Research has demonstrated that at the first onset of low back pain, the deep layer of supporting musculature stops working effectively. The timing of these muscles becomes altered and instead of being anticipatory muscles, they activate after an activity has

begun, therefore becoming less effective.[3,4,5] This has been found to be true for varying diagnoses. In relation to what was previously discussed, this would be considered a dysfunction in the force closure mechanism. Research has also concluded that dysfunctions of the force closure system can be reversed and can be treated successfully in many patients.[4,5]

Low back pain can also be a result of a dysfunction in the form closure system. Many common diagnoses, such as osteoarthritis, degenerative disc disease, herniated discs and stenosis can affect the form closure system. These are diagnoses in which the integrity of the lumbar spine structures is disrupted, therefore disrupting the ability of the form closure system to work effectively.

Osteoarthritis

Osteoarthritis, or degenerative joint disease, commonly involves synovial joints of the cervical and lumbar spines as well as hip, knee, carpometacarpal, and metatarsophalangeal joints. It is described as a "slow, progressive degeneration of joint structures which can lead to loss of mobility, chronic pain, deformity, and loss of function".[7] Osteoarthritis is not always symptomatic. It is estimated that up to 85% of individuals greater than 60 years old have some degree of osteoarthritis, but only 15%–25% are symptomatic.[7] Osteoarthritis is characterized by the breakdown and thinning of the articular cartilage of the joint. Patients with diagnosed osteoarthritis typically report deep achy pain, stiffness after inactivity, and loss of flexibility.[7] Mild to moderate osteoarthritis of the spine can be effectively managed through a course of physical therapy including education of proper posture to reduce stress through the spine, use of assistive supports if needed, an exercise program including strength, flexibility, and aerobic components, and a home exercise program.

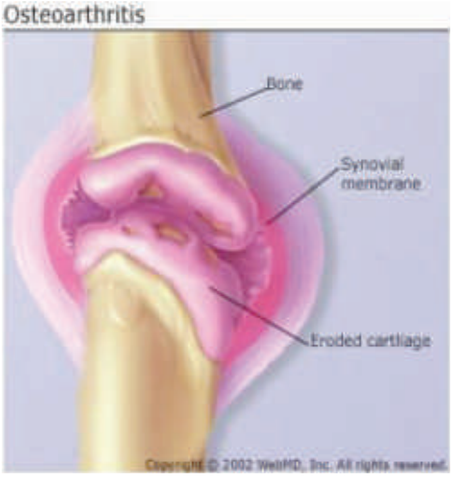

Figure 11. Osteoarthritis of a joint (*Courtesy of WebMD, Inc.*)

Degenerative Disc Disease

The degenerative process described with osteoarthritis can also occur to the intervertebral disc. With degenerative disc disease, the disc undergoes changes in volume, shape, and composition.[7] These changes are not always symptomatic, similar to osteoarthritis, but will cause alterations in mobility and change the mechanical properties of the spine. The disc consists of the nucleus pulposus surrounded by the annulus fibrosus. The nucleus pulposus undergoes the most significant changes. It shows decreased water content as well as fragmentation. In turn, this leads to loss of disc height. As this condition progresses, bulging of the annulus fibrosus, osteophyte formation, and development of associated pathologies, such as degenerative spondylolisthesis and spinal stenosis, may occur.[7] Symptomatically, individuals with degenerative disc disease can have wide ranging complaints depending on the severity of the disease. Dull, achy low back pain, lower extremity pain, weakness, and difficulty walking and standing are all common complaints. Mild to moderate cases of degenerative disc disease can be effectively treated through a course of

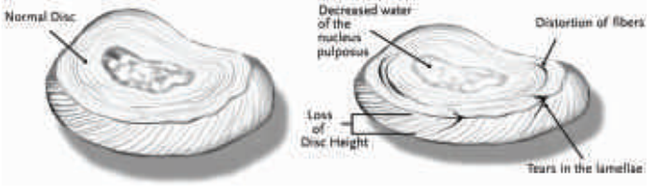

Figure 12. Normal disc (left) and degenerative disc (right) (*Courtesy of Medtronic*)

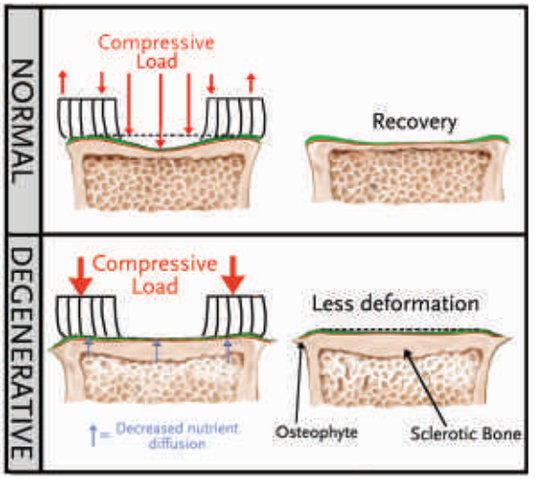

Figure 13. Effect of compressive load on healthy and degenerative discs. (*Courtesy of Medtronic*)

physical therapy including the components as with osteoarthritis.

Assessment and Treatment

Once the symptoms of low back dysfunction begin, either from an insidious onset or due to an injury, many people wonder what the next step is. The majority of people begin by seeing their general practitioner. From there, they are typically referred to a specialist for further examination and diagnostics. This may result in a recommendation of medication and rest and/or possibly a referral to an allied health professional such as a physical therapist or chiropractor. This scenario has worked successfully for countless individuals; however, a new pathway has developed as a result of leg-

Decision Rules for Lumbar Treatments

Key Examination Findings	Treatments
• No symptoms distal to the knee • Recent onset of symptoms • Low levels of fear-avoidance beliefs • Hypomobility of the lumbar spine • Increased hip internal rotation (greater than 35) or discrepancy in hip internal rotation range of motion between the right and left hip	• Manipulation or mobilization techniques targeted to the sacroiliac or lumbar region • Active range of motion exercises
• Frequent prior episodes of low back pain • Increasing frequency of episodes of low back pain • Instability catch or painful arc during lumbar flexion and extension range of motion • Hypermobility of the lumbar spine • Positive prone segmental instability test	• Promoting isolated contraction and co-contraction of the deep stabilizing muscles • Strengthening of large spinal stabilizing muscles
• Symptoms distal to the knee • Signs and symptoms of nerve root compression • Symptoms centralize with lumbar extension • Symptoms peripheralize with lumbar flexion	• Extension exercises • Mobilization to promote extension • Avoidance of flexion activities
• Older age (greater than 65 years) • Symptoms distal to the knee • Signs and symptoms of nerve root compression, neurogenic claudication, or both • Symptoms peripheralize with lumbar extension • Symptoms centralize with lumbar flexion	• Flexion exercises • Mobilization to promote flexion • Deweighted ambulation • Avoidance of extension activities
• Visible frontal plane deviation of the shoulders relative to the pelvis • Asymmetrical side bending active range of motion • Painful and restricted extension active range of motion	• Pelvic translocation exercises • Non-weight-bearing shift correction exercises
• Signs and symptoms of nerve root compression • No movements centralize symptoms	• Mechanical traction

Reference: Fritz JM. *Current Concepts of Orthopaedic Physical Therapy* 2nd Edition "The Lumbar Spine: Physical Therapy Patient Management Utilizing Current Evidence. Orthopaedic Section APTA. 2006 pg. 3.

islative changes. In most states, an individual may see a physical therapist directly without a referral from an MD, otherwise referred to as "direct access". Physical therapists are highly trained in assessment of musculoskeletal dysfunctions as well as screening for more serious pathologies. Physical therapists are extensively trained in anatomy, pathology, and biomechanics. They also have specialized training in assessment and treatment of joint mechanics, the muscular system, and the nervous system, as well as interactions of these systems. Current research has investigated if seeing a physical therapist immediately following an injury can have a positive impact on recovery. Multiple studies have concluded that seeing a physical therapist close to the time of onset of symptoms has given improved prognosis for quicker resolution of symptoms and return to the prior level of function.[3]

During your assessment with a physical therapist, they should examine your active range of motion, joint mobility, flexibility, strength, integrity of your neurological system (reflexes, sensation, etc), and screen for signs and symptoms that may require a referral to a MD or specialist. Special testing may also be incorporated into the exam to confirm or rule out a certain dysfunction, and to test the integrity of the form and force closure systems that were previously discussed. The physical therapist will discuss their findings, the plan for your care, and your individual goals following this thorough examination.

Your physical therapy sessions may include various interventions such as dry needling, joint mobilization or manipulation, stretching, neural glides, and core stabilization, to name a few. These interventions, as well as others that may be used by physical therapists and other allied health professionals, are discussed in the article "Physical Therapy for Soft Tissue Dysfunctions" by Richard Banton in this volume of the *Journal of The Spinal Research Foundation*.

Conclusion

The soft tissue system is vitally important to the optimal functioning of the lumbar spine. The deep and superficial musculature working in concert allow for movement and task accomplishment while maintaining stability of the spine. Without the optimal and synergistic functioning of the musculature and ligamentous systems, symptoms and limited function will result.

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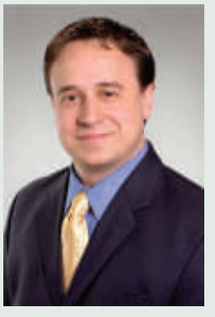

Michael McMurray, P.T., M.P.T., D.P.T., F.A.A.O.M.P.T.

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Physical Therapy for Soft Tissue Dysfunction

Richard Banton, D.P.T., C.M.P.T., A.T.C.

Management of Soft Tissue Dysfunction

Soft tissues of the body include skin, muscles, ligaments, and tendons. Proper physical therapy management and treatment of soft tissue injuries begin with a comprehensive evaluation to determine the origin of dysfunction. A comprehensive evaluation must assess all of the systems that can possibly affect the soft tissues of our body. These systems include the neurological system and the musculoskeletal system. The purpose of this article will be to discuss some of the interventions available to physical therapists treating soft tissue dysfunction.

The Musculoskeletal System

Recent research[1,2] is helping to classify muscles into one of two classification systems: global and local systems. Muscles that belong to the global system appear to be more involved in regional stabilization between the thoracic spine and upper extremities, or the pelvis and lower extremities. Muscles that belong to the local system perform more of an anticipatory role as they are more important in segmental control or intrapelvic stabilization.[2]

Muscles that are considered part of the global system and are responsible for regional movement between the spine, the pelvis, and the extremities are external obliques, latissimus dorsi, serratus anterior, rectus abdominus, the gluteals, hamstrings, adductors, the pectorals, upper trapezius, and levator scapulae.[3]

Muscles that support the spine are considered part of the local system and anticipatory in nature. These are the transverse abdominus, the pelvic floor musculature, and the deep multifidus. Research is suggestive that other muscles are also anticipatory in nature such as psoas, the quadratus lumborum, posterior fibers of internal oblique, the diaphragm, and the lumbar portions of lumbar longissimus and iliocostalis.[2]

Function would be significantly compromised if the human body was not capable of mobility with rigidity. An intricate balance of stability and mobility must exist within the human body for movement to occur. The role of the physical therapist in evaluating the musculoskeletal system is to determine where the breakdowns between stability (form closure) and mobility (force closure) exist.

Treatments for Excessive Force Closure

When assessing the soft tissue structures of the spine, the physical therapist must identify the problems regarding force closure. Force closure refers to the intricate interaction between the local and global systems and their ability to provide stability to joints, but at the same time allow for the body to move freely in space with control and without injury. When there is too much force closure, there will be excessive compression within the system, causing rigidity without mobility. When there is too little force closure within the system, there will be insufficient compression within the system, allowing instability to occur. Kendall et al.[4] simply state that when a muscle is short (through its passive connective tissue elements) or strong (contractile element) and its antagonist (opposing muscle group) is not, it will create "a position of deformity".[11] When a muscle is long (passive elastic element) or weak (contractile element), and its antagonist is not, it will allow for "positions of deformity". The mechanisms creating problems with force closure can be mechanical, chemical, or neurological. A comprehensive physical therapy evaluation is necessary to determine the exact cause of dysfunction so that a specific plan of care can be tailored to the patient's needs. The following section will discuss effective interventions appropriate to utilize when force closure is considered excessive.

Dry Needling

According to the American Academy of Orthopaedic Manual Physical Therapy (AAOMPT), "*Dry needling is a neuro-physiological evidence-based treatment technique that requires effective manual assessment of the neuromuscular system. Physical therapists are well trained to utilize dry needling in conjunction with manual physical therapy interventions. Research supports that dry needling improves pain control, reduces muscle tension, normalizes biochemical and electrical dysfunction of motor endplates, and facilitates an accelerated return to active rehabilitation*".[4]

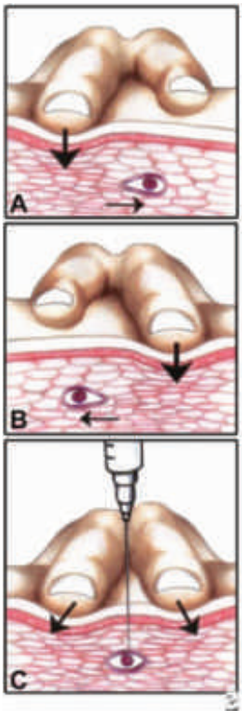

Figure 1. Palpating myofascial trigger points for dry needling. Image reprinted with permission from Medscape.com, 2011. Available at http://emedicine.medscape.com/article/89095-overview.

Dry needling results in positive treatment outcomes for patients when combined with other manual therapy treatments. When combined with manual therapy and exercise, dry needling has been proven to be an effective treatment for low back pain, whiplash, headaches, chronic pelvic pain, complex regional pain syndromes, and fibromyalgia.[5] The effectiveness of a dry needling intervention is highly determined by the skill level of the clinician. Currently, there are only two physical therapy curriculums that offer entry level training in dry needling. These curriculums ultimately enable the clinician to palpate myofascial trigger points and then to use the needle as a palpation tool to appreciate changes in the firmness of those tissues requiring treatment.[4]

The primary goals of dry needling are to desensitize soft tissues, to restore motion and function, and to possibly induce a healing response in the tissue. These goals are achieved by:

1. Releasing shortened muscles
2. Removing the source of irritation by needling paraspinals muscles
3. Promoting healing by triggering local inflammation
4. Decreasing spontaneous electrical activity at trigger points[6]

The mechanical effects of dry needling abnormal muscles are thought to involve disruption of a dysfunctional motor endplate. It is plausible that accurately placing a needle provides a local stretch to the contracted muscle elements.[7] Pistoning the needle up and down is done to elicit a local twitch response within the muscle, which is thought to deplete the muscle cell of its excessive acetylcholine, a neurotransmitter that facilitates muscle contraction and has been found in excess within trigger points.[6]

The exact mechanism of the formation of trigger points or myofascial tightness remains unclear. How dry needling actually eliminates these trigger points also remains unclear. Recent research by Shah et al. (2005)[8] notes that there is an increased concentration of substances (substance P, Bradykinin, interleukin-1, etc.) that intensify the response from nociceptors (pain receptors) located within trigger points and surrounding tissues. Shah also noted an immediate reduction in these pain substances following treatment by dry needling.[6]

Trigger point dry needling has been recognized by prestigious organizations such as the Cochrane Collaboration and is recommended as an option for the treatment of persons with chronic pain. Several clinical outcome studies have demonstrated the effectiveness of trigger point dry needling, however, questions remain regarding the mechanisms of needling procedures.[4]

Active Release Technique (ART)

The goal of ART is to restore optimal texture, motion, and function of the soft tissue and release any entrapped nerves or blood vessels. This is accomplished through the removal of adhesions or fibrosis in the soft tissues via the application of specific protocols.[9] Adhesions can occur as a result of acute injury, repetitive motion, and constant pressure or tension. ART eliminates the pain and dysfunction associated with these adhesions. When adhesions form in soft tissues, they become stiffer, tighter, and shorter.[8] The muscle cells lose the ability to eliminate waste materials and can become painful. The muscular tension created by adhesions may compress joints, causing neuropathy symptoms due to nerve compression. In an ART treatment, the provider uses his or her hands to evaluate the texture, tightness, and mobility of the soft tissue. Using hand pressure, the practitioner works to remove or break up the fibrous adhesions with the stretching motions generally in the direction of venous and lymphatic flow, although the opposite direction may occasionally be used.[8]

In the first three levels of ART treatment, as with other soft-tissue treatment forms, movement of the patient's tissue is done by the practitioner. In level four, however, ART requires the patient to actively move the affected tissue in prescribed ways while the practitioner applies pressure. Involvement of the patient is seen as an advantage of ART, as people who are active participants in their own health care are believed to experience better outcomes.[8] One disadvantage of ART regards the comfort of the patient when ART is being applied. Many of the techniques can be uncomfortable as the practioner attempts to tear adhesions within the muscle. Another question regarding the practicality of ART intervention and its perceived benefit is when it is used in the absence of trauma. Trauma is necessary for adhesions to develop in muscles. If a physical therapist is using ART on atraumatic tissues, minimal benefit should be expected for this intervention.

Strain and Counterstrain

The strain and counterstrain approach is an excellent intervention choice for acute soft tissue dysfunction because it is gentle, atraumatic, and can be used without contraindications. When using strain and counterstrain, the patient's body is moved slowly in non-painful directions until the therapist identifies positions of decreased muscular tension, reported relief, and palpable trigger points.[5] Dramatic changes in pain relief, range of motion, and muscular guarding can be achieved with strain and counterstrain when applied appropriately.

The mechanism of action regarding strain and counterstrain is not clearly understood, but it is thought to involve interaction between the body's natural mechanoreceptors, the spinal cord, and the brain. Somatic dysfunction, i.e. trigger points, is directly related to how the brain perceives information from the body's mechanoreceptors. For example, nociceptors are high threshold pain nerve fibers found in joint capsules, blood vessels, and articular pads. They can be stimulated by chemical changes (as with inflammation), increases in pressure (as with disc herniations), or subluxation of articular joints. When stimulated, nociceptors increase tone in muscles of their corresponding joints via tonic reflexgenic effects. Strain and counterstrain stimulates other mechanoreceptors, muscle spindles, and golgi tendon organs that cause inhibition of tonic muscles via their organic spinal cord and brain reflexes. The relaxation effect on tonic muscles allows the physical therapist to mobilize, stretch, or manipulate the affected joints and normalize the patient's mobility. The following receptors in Table 1 play an important role in somatic dysfunction that is manifested with the tender point of strain and counterstrain.[10]

Table 1. Articular Receptors.

Type	Morphology	Location	Parent Nerve	Function
1	Thinly encapsulated globular corpuscles in 3–6 clusters	Superficial layers of the joint capsule	6–9u small and myelinated	Static and dynamic mechanoreceptors of low threshold and slowly adapting; Proprioceptive
2	Thickly encapsulated conical corpuscles in 2–4 clusters	Deep layers of joint capsule and fat pads	9–12u medium and myelinated	Dynamic mechanoreceptors of low threshold and rapidly adapting; Kinesthetic
3	Thinly encapsulated fusiform corpuscles	Intrinsic and extrinsic joint ligaments	13–17u large and myelinated	Dynamic mechanoreceptors of high threshold and very slowly adapting; Acts as the joint counterpart to the Golgi tendon organ; Inhibits antagonistic muscles to the stretched ligament
4	Simple nerve endings found in plexi and individually.	Fibrous capsule, ligaments, fat pads, blood vessel walls, bone, periosteum	2–5u very small and myelinated and <2u extremely small and unmyelinated	Nociceptors of high threshold and non-adapting; Pain sensors

Joint Mobilizations

Once a muscle has been released, a joint's true mobility can be assessed. The neutral zone of a joint can be determined without influence from excessive force closure (myofascial compression/tension). Similar to strain and counterstrain, joint mobilizations and manipulation have been proven to inhibit muscular tension through reflexes between the spinal cord, muscle spindles, and golgi tendon organs.[11] Joints that prevent mobility through articular restrictions often have varying degrees of limitation depending on the direction of the movement. Therefore, varying grades of joint mobilization are used by physical therapists depending on the acuteness or chronicity of restriction, irritability of the joint, or type of joint dysfunction. The following identifies the varying grades of joint mobilizations and their mechanism of action.[12]

- Grade I—Activates type I mechanoreceptors with a low threshold and responds to very small increments of tension. Activates cutaneous mechanoreceptors and thus decreases pain. Oscillatory motion will selectively activate the dynamic, rapidly adapting receptors, i.e. Meissner's and Pacinian Corpuscles. The former respond to the rate of skin indentation and the latter respond to the acceleration and retraction of that indentation.
- Grade II—By virtue of the large amplitude movement, it will affect type II mechanoreceptors resulting in inhibition of pain and reducing muscle tension or spasm.
- Grade III—Selectively activates more of the muscle and joint mechanoreceptors as it goes into resistance, and less of the cutaneous ones as the slack of the subcutaneous tissues are taken up. Grade III begins to stretch joint capsules which allows for improved range of motion.
- Grade IV—With its more sustained movement at the end of range, Grade IV will activate the static, slow adapting, type I mechanoreceptors whose resting discharge rises in proportion to the degree of change in joint capsule tension.

Figure 2. Joint mobilization (*Courtesy of Neurodynamic Solutions, www.neurodynamicsolutions.com*)

- Grade V—This is the same as joint manipulation. High velocity thrust techniques break joint adhesions and allow for improved joint mobility, decreased pain, and promote normal muscle tone.

Neural Mobilizations

All soft tissues of the human body are connected in some way to the nervous system and the nervous system has complex biomechanics just like the structures it innervates. Nerves can be injured by mechanical, chemical, or physiological consequences of friction, compression, stretching, or disease. Traumas do not have to be severe injuries; they can be a result of repetitive muscle contraction, unphysiological movement, or body postures. There may not be a direct mechanism of injury to a nerve. Often nerve injury results from secondary injury to the nervous system as a result of blood or edema.[13]

Neural mobilization is an intervention that has been around since the beginning of the century. In the late 1880's, surgeons in France and England joined together to develop a tool called a "nerve stretcher." A small incision was made in the patient's gluteal region to expose the sciatic nerve. The "nerve stretcher" was then used to hook the sciatic nerve

and pull it until it was exposed six inches above the skin.[14] Fortunately for patients, the art of mobilizing the nervous system has become more delicate and refined over the years. Neural mobilizations now involve a delicate delivery of technique that involves many factors such as handling and palpation skills, patient communication, knowledge of biomechanics, and reassessment skills.

In regards to the nervous system, movement must be broken down into two types: (a) gross movement, such as how the median nerve glides through the carpal tunnel (b) intraneural movement, refers to movement of neural tissue elements in relation to their connective interface. For example, the brain can move in relation to the surrounding cranial dura mater and the spinal cord can move in relation to the dura mater.[15]

Tension and increased pressure are created as a consequence of elongation and occur in all tissues and fluids enclosed by the epineurium and dura mater.

Physical therapists incorporate these adaptations of nervous system mobility into interventions for treatments in the presence of nerve injury. Intraneural scarring from swelling or mechanical compression from disc injury are only a few examples of pathology that can interfere with normal nervous system mobility. Nerve mobilizations afford physical therapists a way to increase movement of a nerve and at the same time provide enhanced nutrition to the nerve to promote pain relief, improve range of motion, and increase neural input into the tissues that the nerve innervates. Below is an example of a neural mobilization technique to the sciatic nerve.

Neural mobilization techniques can be very powerful and care must be used by the physical therapist to identify worsening of symptoms from their patients. For example, in the presence of a large disc herniation, neural mobilization techniques will likely elicit painful symptoms and result in a poor treatment outcome for the patient. The key in using neural mobilizations is to focus on the word "mobilizations," not "stretch." This way, the physical therapist will focus their treatment on the resistance of the nerve glide and not depend on patient feedback, ensuring that nerve irritation will not result from the intervention.

Figure 3. Neural mobilization (*Courtesy of Neurodynamic Solutions, www.neurodynamicsolutions.com*).

Treatments for Inefficient Force Closure

The key components for promoting force closure are to:

1. "Wake up" and coordinate the deep and superficial systems.
2. Use functional, new strategies for posture and movements based upon the patient's specific needs.[16]

Core training for any individual suffering from or recovering from spine injury must involve activation of the deep musculature system: pelvis floor, transversus abdominus, and multifidus. Research has shown that individuals capable of isolating their deep musculature system have a decreased incidence of recurrence of back pain.[17] Hodge's research determined that the deep musculature system does not contract to stiffen the spine, but instead responds in a coordinated manner to balance the flexion and extension forces acting on the spine, which in turn provides an appropriate strategy for the body to ensure dynamic stability or, as Hodges describes, "mobility without rigidity."[18]

Patients sustaining trauma, surgery, or injury to their spine often lose the ability to efficiently activate their core muscles and thus develop inefficient

strategies to provide proper force closure. Physical therapists possess many tools (taping, electrical stimulation, bracing, diagnostic ultrasound, etc.) and strategies to retrain proper core control and must make this a beginning part of any stability program that their patients enter.

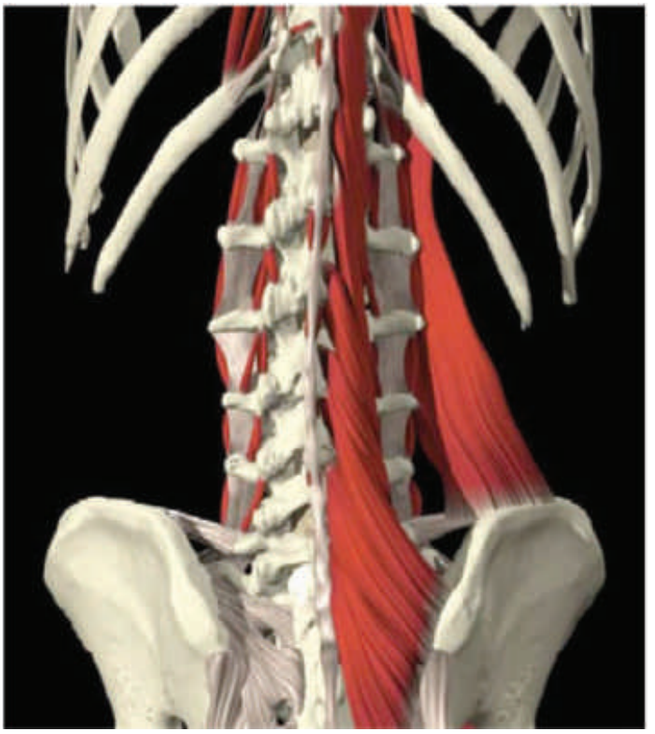

Figure 4. Multifidus (*Primal Pictures*)

Proprioceptive Neuromuscular Facilitation (PNF)

PNF is a philosophy of treatment that incorporates the body's sensory receptors, nerves and muscles, and functional movements into an exercise program tailored to a patient's specific needs. Once the patient identifies how to properly recruit their deep core musculature system, the physical therapist must re-train the patient on how to incorporate this core control into functional movements. Using PNF as an intervention involves the therapist providing manual contact, resistance, or facilitation through a desired functional pattern of movement. The therapist may change their contact, resistance, speed, or verbal cues during the exercise to train the patient's proprioceptive awareness. With repeated practice and training, the patient's brain and nervous system adapts to the movements with improved strength, awareness, and anticipation, thus making them less likely to re-injury themselves as they return to their sport or activities of daily living.[19]

PNF incorporates the patient's visual system, timing, and natural reflexes to stretch, traction, and joint compression to promote stability, increased strength, and facilitate coordination between the trunk and the extremities. The goal of PNF is to promote functional movement through facilitation, inhibition, strengthening, and relaxation of muscle groups, thus creating the ultimate balance of force closure.

PNF Stretching Techniques

Contract-relax is a PNF relaxation technique that uses the body's natural stretch reflex to increase range of motion. Following the contraction of the muscle, the local muscle spindle relays information through the spinal cord to the brain that the muscle is being shortened. Reflectively, the brain relays a message via gamma neurons to the shortened muscle to relax. A skilled physical therapist can feel this relaxation and follow with a gentle stretch to improved flexibility. Likewise, a skilled physical therapist can use the same reflex to facilitate contraction of a muscle. By providing a quick stretch to a muscle the muscle spindle then relays information that it is being stretched too far and, in return, the brain relays a signal for the muscle to contract. By using PNF in this manner, the physical therapist is able to facilitate muscle activation and improve the patient's ability to contract a muscle.

PNF Strengthening Techniques

Along with stretching, PNF strengthens the body through diagonal patterns, often referred to as D1 and D2 patterns. It also applies sensory cues, specifically proprioceptive, cutaneous, visual, and auditory feedback, to improve muscular response. The diagonal movements associated with PNF involve multiple

Figure 5a. The patient and therapist assume the position for the stretch, and then the physical therapist extends the body limb until the muscle is stretched and tension is felt (*Courtesy of The Stretching Institute © 2011, www.thestretchinginstitute.com*).

Figure 5b. The patient then contracts the stretched muscle for 5–6 seconds and the physical therapist inhibits all movement (*Courtesy of The Stretching Institute © 2011, www.thestretchinginstitute.com*).

Figure 5c. The muscle group is relaxed, then immediately and cautiously pushed past its normal range of movement for about 20 to 30 seconds (*Courtesy of The Stretching Institute © 2011, www.thestretchinginstitute.com*).

joints through various planes of motion. These patterns incorporate rotational movements of the extremities, but also require core stability if patients are to successfully complete the motions.

Two pairs of diagonal patterns exist. These patterns can be performed in flexion or extension and are often referred to as D1 flexion, D1 extension, D2 flexion, or D2 extension techniques for the upper or lower extremities. Although patients can perform these patterns with many forms of resistance, the interaction between patient and clinician is critical to early success of PNF strengthening.

This interaction requires manual resistance throughout the range of motion through carefully positioned hand placement and appropriately choreographed resistance. By placing the hands over the agonist (primary mover) muscles, the clinician applies resistance to the appropriate muscle group, while guiding the patient through the proper range of movement.

Using manual resistance, the clinician can make minor adjustments as the patient's coordination improves or fatigue occurs during the rehab session. In general, the amount of resistance applied is the maximum amount that allows for smooth, controlled, pain-free movement throughout the range of motion. In addition to manual resistance strengthening, PNF diagonal patterns enhance proper sequencing of muscular contraction, from distal to proximal. This promotes neuromuscular control and coordination.[13]

Conclusion

The first step for success in rehabilitation starts with accurately assessing the problems in form and force closure, and accurately indentifying the pathology responsible for a patient's dysfunction. The second step is choosing the best intervention to promote wellness. The above techniques are only a few methods used by physical therapists to treat soft tissue dysfunction and were chosen because they are the most commonly used and have been supported by evidence-based research.[20–22] It is important to note that rarely is one technique by itself effective for complete resolution of a patient's pain. The more skilled interventions

a physical therapist has to choose from to treat their patients, the more successful they will be regarding their patient outcomes. Patients suffering from chronic or acute soft tissue dysfunction and in need of physical therapy should seek practitioners that have experience with many of the above mentioned techniques to ensure the best prognosis for a successful non-surgical outcome.

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Medical Acupuncture for Pain Management

Thomas T. Nguyen, M.D., D.A.B.P.M.

Introduction and History

Acupuncture is among the oldest healing arts in the world, as it has been practiced in China and other Asian countries for thousands of years. It is a discipline extracted from a complex heritage of Chinese medicine that includes massage, manipulation, stretching, breathing exercises, and herbal remedies. The earliest source of acupuncture teaching and theory is the *Huang Di Nei Jing* (the Yellow Emperor's Inner Classic) dating as old as the Han dynasty in the 2nd century BC. The Nei Jing theory regarded the human body as a microscopic reflection of the universe and considered the acupuncturist's role that of maintaining the body's harmonious balance. A second text, the *Nan Jing* (the Classic of Difficult Issues) written later, during the Han dynasty in the 1st and 2nd centuries AD, further expanded and advanced the theories of points and channels to address the etiology of illness, diagnosis, and therapeutic needling.

The term "acupuncture" describes a family of procedures involving the stimulation of anatomical points on the body using a variety of techniques. The acupuncture technique that has been most often studied scientifically involves inserting thin, metallic, specialized needles into the skin which are then manipulated manually or electrically. In traditional Chinese medicine (TCM), the body is seen as a delicate balance of two opposing and inseparable forces: Yin and Yang. Yin represents the cold, slow, or passive principle, while Yang represents the hot, excited, or active principle. In TCM, good health is achieved by maintaining the body in a balanced state. Disease, illness, and pain are attributed to an internal imbalance of Yin and Yang. This imbalance leads to blockage in the natural flow of *Qi*, vital energy, along certain pathways referred to as meridians. Acupuncture is used at specific points along the certain meridians where the *Qi* is felt to be blocked.

Figure 1. Representation of Yin and Yang principles. *Image courtesy of www.tcmhealthbc.com.*

Medical acupuncture is acupuncture that has been successfully incorporated into the medical or allied health practices in Western countries. In the United States, acupuncture came to be more accepted and embraced by practitioners after a New York Times reporter, James Reston, described his experience for his post-appendectomy pain control using acupuncture needles.[1] Since that time, guidelines for education, practice, and regulation in acupuncture have been established and implemented within state, national, and international societies.

Acupuncture and Pain

Pain is a feeling triggered in the nervous system. It can be experienced as sharp or dull, intermittent or constant, localized or diffuse. Pain can be a signal from the body to indicate illness or injuries. Although most pain resolves once the underlying insult is addressed, it has the potential to last for a prolonged period of time, from months to even years. Occasionally, pain can persist chronically due to abnormal activity in the pain-sensing regions of the brain from phenomena such as central sensitization and neuroplasticity.

Physical pain is a common occurrence for many Americans. To relieve their pain, people try various treatments and regimens. Many pain sufferers take over-the-counter anti-inflammatory medications and even stronger prescription narcotic analgesics. Other people try non-medicinal approaches to help with their pain such as physical and occupational therapy, cognitive behavioral therapies, and complementary alternative medicine (CAM). Examples of CAM therapies include spinal manipulation, osteopathy, and acupuncture.

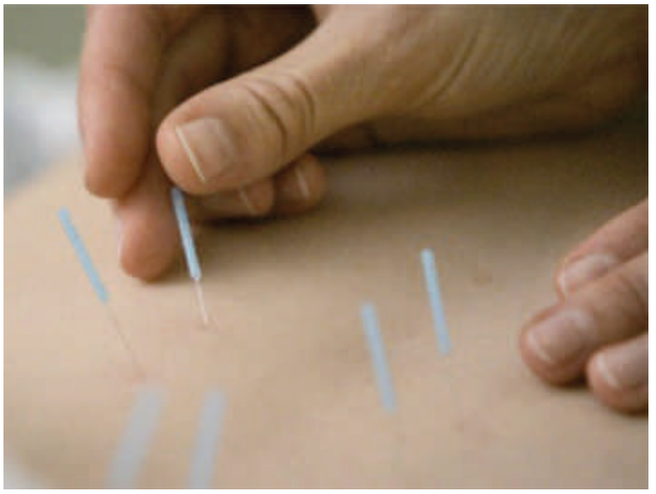

Figure 2. Acupuncture needles. *Image courtesy of www.helpmystyle.ie.*

While acupuncture has been widely studied for various painful conditions such as postoperative pain, dental pain, carpal tunnel syndrome, tennis elbow, headache, sinusitis, osteoarthritis, fibromyalgia, and low back pain, the exact mechanism is unknown. It is believed that the acupuncture can activate the endogenous opioid peptide system to influence the body's regulatory system by changing the processing and perception of painful, noxious stimuli at various levels of the central nervous system. Two models of systems of acupuncture have been hypothesized with one involving an endorphin-dependent system and the other involving a monoamine-dependent system.[2]

Evaluation and Treatment

In an acupuncture evaluation, the initial consultation with the patient is similar to that of any conventional allopathic medical interview and examination. In addition, the practitioner may ask you at length about your health condition, lifestyle, and behavior. For acupuncture, several diagnostic somatotropic systems such as the tongue, radial pulses, and external ear may be used to evaluate the balance of relative strengths or weaknesses within the organs.

Treatment strategies are aimed at activating the appropriate layers of the energy circulation network to address each problem and blockage of *Qi*. For simple strains and sprains, a treatment may consist of a dispersion of needles surrounding the local region or along the appropriate tendinomuscular meridian. Long-standing musculoskeletal pain may need placement of needles around one of the principal meridian circuits, occasionally with additional electrical stimulation, to encourage energy flow through the flow obstruction.

Acupuncture needles are metallic, solid, and hair-thin. Patients experience acupuncture differently. Most patients feel no or minimal pain as the needles are inserted superficially. Needles are inserted to the depth necessary to elicit the patient's sensation of *de qi* or needle grab, a dull ache that radiates from the point. Acupuncture needles remain inserted for 15 to 30 minutes. Patients receiving acupuncture treatment may either feel energized or relaxed after the treatment. Patient visits are usually scheduled once a week for four to six weeks initially. However, the frequency and length of treatments may vary depending on the condition being addressed.

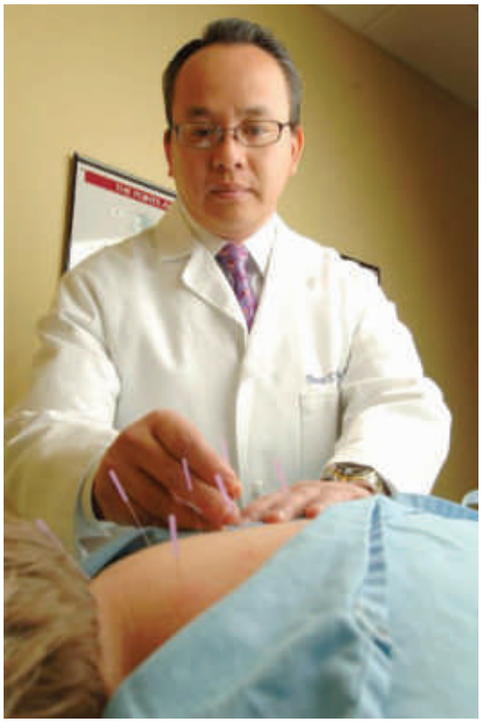

Figure 3. Physician performing acupuncture.

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Conditions for Acupuncture

In the United States, acupuncture has found its greatest acceptance and benefit in the treatment and management of musculoskeletal pain. Acute musculoskeletal and myofascial conditions such as sprains, strains, spasms, and contusions are among the problems most frequently and successfully treated to resolution with acupuncture. In such cases, acupuncture can be legitimately considered as a first line of treatment. Chronic musculoskeletal pain problems are also commonly and appropriately treated with acupuncture, but more as an adjunctive therapy. According to the practice guidelines issued by the American Pain Society and the American College of Physicians in 2007, acupuncture is one of the CAM therapies that practitioners should consider for patients with chronic low back pain that has been refractory to conventional treatment.[3]

Acupuncture can help people with chronic low back pain feel less bothered by their symptoms and function better in their daily activities, according to the largest randomized trial of its kind, published in the May 11, 2009 Archives of Internal Medicine and called the SPINE (Stimulating Points to Investigate Needling Efficacy) trial. "This study of 638 adults who had never had acupuncture suggests that acupuncture is about as effective as other treatments for chronic back pain that have been found helpful," said SPINE trial leader Daniel C. Cherkin, PhD, a senior investigator at Group Health Center for Health Studies in Seattle.[4]

Figure 4. *Image courtesy of the British Medical Acupuncture Society.*

Acupuncture has also been studied for various painful conditions. Some examples of pain conditions treated with acupuncture include, but are not limited to, carpal tunnel syndrome, fibromyalgia, headache/migraine, low back pain, menstrual cramps, neck pain, osteoarthritis/knee pain, postoperative pain, and tennis elbow. Other non-painful conditions treated with acupuncture include depression, sinusitis/allergies, hypertension, and infertility.

Adverse Effects and Risks of Acupuncture

In the hands of a medically trained practitioner, acupuncture is a safe therapy. Relatively few complications from acupuncture have been reported. Serious adverse events related to acupuncture are rare. These risks arise from consequences of penetrating the body with a needle, including syncope, punctured organ, infection, and retained needle. Pneumothorax (air in the chest causing lung collapse) is the most frequently reported complication.

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Workplace Ergonomics

Erin M. Friend, P.T., D.P.T., C.E.A.S.

Introduction

Ergonomics is the science of blending the workstation with the worker in order to increase health and productivity and decrease work-related musculoskeletal disorders.[1,2] This article reviews probable work-related musculoskeletal disorders and basic guidelines to correctly setup a workstation in the office environment.

Records show that ergonomics dates back to the 5th century when Hellenic civilizations used ergonomic principles to design tools and workplaces. Similarly, Hippocrates, the father of medicine, described how surgeons' workplaces should be arranged.[3] Today, as technological advances such as laptops, tablet computers, and cell phones are becoming increasingly popular ways to conduct business on the go and as musculoskeletal disorders are on the rise, ergonomics is in even more demand.

Importance of Implementing Ergonomics for Computer Workers

Poor workstation setup and posture lead to pain, dysfunction, and tissue breakdown. Ultimately, this cascading event increases a worker's number of sick days, decreases productivity, and increases worker compensation costs. Every year, companies are spending an exorbitant amount on worker compensation claims due to worker injuries exceeding the cost of group health care insurance.[2]

The Occupational Health and Safety Administration (OSHA) set minimal standards that all employers should follow to decrease the risk of injury to workers. Even with guidelines in place, the reported injury rates for work-related musculoskeletal disorders in the industrial arena are high. Statistics for musculoskeletal disorders in the office environment are less known.[4] A three-year study completed by Gerr et al. in 2002 reported that 50% of computer users in North America develop some type of musculoskeletal symptoms during their first year on a new job.[4]

Work-Related Musculoskeletal Disorders

Work-related musculoskeletal disorders (WMSDs) is a collective term that describes disorders relating to muscles, tendons, and nerves, typically caused by awkward postures and which are potentially painful during work or rest.[5] Movements that increase a person's chance of developing a musculoskeletal disorder, when constant (2 hours or more) or repetitive in nature, include bending, straightening, gripping, holding, twisting, clenching, and reaching.[2,5] Individuals will not injure themselves just by performing these motions once or twice a day, or if they take a break during the day. Common syndromes related to WMSDs include tendonitis, muscle strain, cervicothoracic dysfunction, thoracic outlet syndrome, low back pain, and carpal tunnel syndrome.[4]

Efforts are being made by OSHA and researchers to fine-tune guidelines and improve standards for workers in all industries, including computer workers. Computer workers increasingly suffer from musculoskeletal disorders related to mental and physical fatigue, poor physical fitness levels, static work and home offices, and longer working hours.[2] The workforce as a whole is also aging as 25.6 million workers are currently over age 55, and it is predicted that in 2014, 33% of the workforce will be over 50 years old.[2]

Poor Posture and its Effects on the Body

Posture is defined as "the relative position of the body at any one period of time."[6] Correct posture or neutral posture is a position in which minimal stress

Figure 1. Examples of good and poor posture. *Figure courtesy of www.trainawaythepain.com.*

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is applied to the body's muscles, tissues, joints, and ligaments, providing the most energy efficient movement.[7]

The central nervous system (CNS) begins to make musculoskeletal adaptations to the postures and movements performed most often. These muscular imbalances cause some muscles to be in a constant state of contraction while other, opposing muscles remain weak and overstretched, which leads to pain.[8,9] To better understand how the CNS controls our movements, do this quick experiment. Cross your arms or clasp your hands together and see which is on top. Now do that a couple times and you will find that it is the same hand or finger on top each time. We are creatures of habit. Now do the same things again, but purposefully make sure the other hand or finger is on top this time. How does it feel? It might feel awkward or maybe even uncomfortable. This is what happens when you try to correct your poor posture to achieve better posture. You simply aren't used to good posture, but you can improve it. In addition to creating new muscle patterns with poor posture, the body's fascial system also adapts.

Fascia is tough connective tissue that supports and lubricates every muscle and organ in the human body. However, if poor posture, trauma, or inflammation is involved, the fascia will bind down and create more fascia in response to stress or demand. When fascia binds down, extra pressure is also being placed on nerves, blood vessels, bones, and organs.[10,11] For instance, a right hand dominant person will have more fascia over the central and right side of the sternum or breastbone. This means the chest area of a right-handed person may be tighter or more contracted, possibly causing rounded shoulders if poor posture is evident. In addition to creating stress on the fascial system, poor posture can also lead to stress on the ligaments and joint capsules. Ligaments and joint capsules are formed by collagen bonds, which begin to breakdown with a 5% elongation and fail at 7% elongation, causing chronic deformation.[8]

The most common examples of poor posture seen in computer workers are a forward head, rounded shoulders, and slouched spine. Therefore, when one is sitting in this awkward posture, it is not uncommon to see tightening of the fascia and muscles of iliopsoas, pectineus, occipitals, pectoralis, trapezius, sternocleidomastoid, levator scapularis, adductors, and piriformis.[8] Eye strain, difficulty breathing, poor circulation, and headaches are also a result of poor posture.

What is a Good Posture for Computer Users?

To decrease a person's chance of creating breakdown in the body, proper posture and desk setup

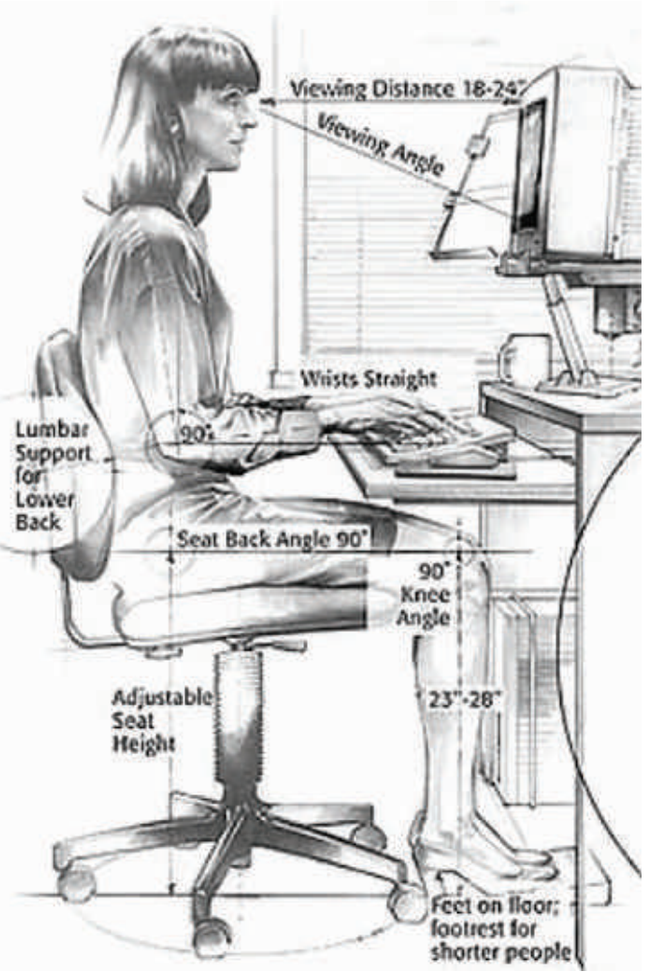

Figure 1. Components of a comfortable workstation. *Figure courtesy of Wikimedia Commons.*

are important. A good workstation is one that has an adjustable chair, mouse/keyboard and computer monitor.

Chair Setup

When sitting, a person's bottom needs to be all the way to the back of the chair with their feet resting firmly on the ground and feel they have equal pressure on the balls of their feet and their ischial tuberosities (sitz bones). A good chair is one in which the seat depth is adjustable to allow 2–3 fingers from the back of the knees to the front of the chair. The lower back and upper back should be well supported by the back of the chair. A lumbar support isn't always adjustable, but if it is, the support needs to be placed in the small of the back. If using the armrests of the chair, a person needs to able to adjust the height, width, and pivot for proper arm placement when typing.

Keyboard and Mouse Setup

Arms need to be relaxed and resting by the person's side with elbows between 90–110 degrees. This is also where the keyboard and mouse need to be placed. Wrists and hands need to be in a neutral or slightly flexed position as if you were playing the piano. Typically, the legs of the keyboard do not need to be up as they cause extension of the wrist. Gel pads in front of the keyboard and mouse also decrease pressure on the carpal tunnel and bring hands into a more neutral position. If you move your arms in and out from this position, it is where 90% of your daily work or accessories' (coffee, phone) need to be placed. When you reach out beyond an arm's distance, posture must change to reach the object.

Computer Monitors

The computer monitor should be placed at an arm's distance away, typically 18–20″ for standard monitors. For larger monitors, place it another couple of inches away. The correct height of the monitor should be where your eyes are at the center to top third of the screen with your head looking forward. A simple monitor riser or books can be added if your screen is not adjustable. For those who use bifocals or progressive lenses, the monitor should be set to where you are looking through the bifocal/progressive part of the lenses.

Additional Setup

When inputting data, documents need to be placed on a person's dominant eye side and monitor level. To determine your dominant eye, make a circle with your fingers and place an object in the center with both eyes open. Now, close one eye, open, then close the other eye. Whichever eye was open when the object remained in the center is your dominant eye.

Frequent phone use also leads to dysfunction. For long phone conversations, utilize a speaker phone or purchase a headset to decrease neck pain. Keep cell phone or touch screen computer use to minimum unless using wireless technology or the device is placed at eye level.

Laptops are convenient to use during meetings, travel, and telework due to their size and portability. However, laptops are not the best for posture as this article has hopefully pointed out. The quickest and best fix for using your laptop is to place the laptop at eye level and use a detachable keyboard and mouse.

Good Posture, Now What?

Now that you are on your way to maintaining good posture, keep in mind it takes about three weeks to get used to any new changes. As mentioned earlier, we are creatures of habit since we are controlled by the central nervous system. As you begin to adjust to your new posture, you may feel sore and achy, but after about three weeks, reverting to your old posture will begin to feel uncomfortable.

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Take a Break and Change Positions

Mini breaks are needed throughout the day to decrease eye fatigue, prevent physical and mental fatigue, and decrease risk of tissue breakdown.[4] Every hour, get up, move around, and take a five minute break to stretch or do an exercise before resuming work. Work-break cycles are just as integral a part of performance and health as workstation setup and posture.

Conclusion

As the article points out, posture plays an important role in overall health and well-being. The key to trying to decrease risk of developing a musculoskeletal disorder is getting assistance or treatment when you are feeling fatigued, especially prior to onset of pain. Posture is not only important when sitting at your desk and working on the computer, but when you perform activities of daily living or extracurricular activities.

Physical therapists are trained and well-versed in how the human body moves and are specialists in soft tissue dysfunction and treatment. They play an important role in recognizing poor posture and work with patients to correct their posture. If you have any concerns about how your workstation is set up and believe it could be contributing to your pain, talk to your physical therapist. He or she may refer you to a physical therapist that is a certified ergonomic assessment specialist to conduct an onsite assessment of your workstation at your office, and make additional recommendations of equipment that will allow you to use good posture.

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EMG and Spine Treatment

Neil Chatterjee, M.D.

Electrodiagnostic medicine encompasses both the nerve conduction study (NCS) and the electromyography (EMG). The information gathered by these studies is often useful in determining appropriate diagnoses and treatment options such as medical and surgical therapy. Electrodiagnostic testing provides critical information regarding the neuromuscular system and investigates the type of nerve fiber (e.g., motor, sensory, or a combination of both) involved. In addition, these studies assist in identifying whether lesions are acute or chronic to possibly determine the longevity of nerve damage.

These studies are unique in that they must be conducted in an individual manner after a thorough neurologic evaluation and differential diagnosis.[1] After the physician obtains the patient history and conducts the physical examination, the nerve conduction study is performed. In most cases, the EMG is performed after the NCS, as the information gathered during the nerve conduction study aides in the appropriate selection of muscles to be tested in the needle portion of the examination.[1]

Figure 1. Electrodiagnostic testing. *Image courtesy of Cadwell Laboratories, Inc.*

Anatomy

Ventral and dorsal nerves attach 31 pairs of spinal nerve roots: 8 cervical, 12 thoracic, 5 lumbar, 5 sacral and 1 coccygeal. Cells in the anterior and lateral gray column of the spinal cord comprise the majority of axons that are contained in the ventral roots. In contrast, the axons contained in the dorsal roots come from the spinal or dorsal root ganglia (DRG). The union of the DRG and ventral roots makes up the mixed spinal nerves.[2]

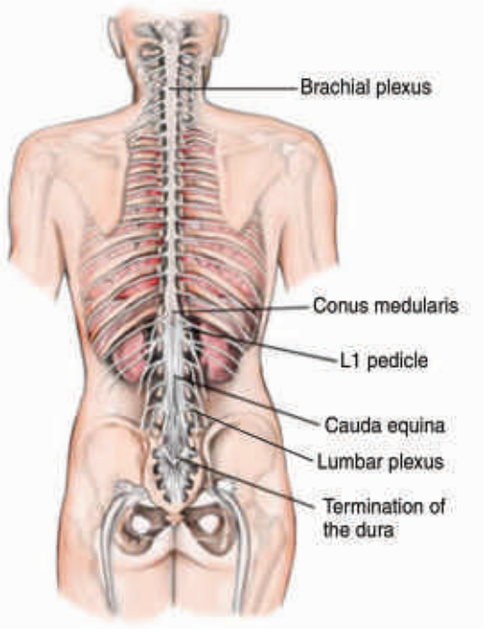

Figure 2. Spinal nerves exiting at all levels of the spinal cord. *Image courtesy of Medtronic.*

A *myotome* refers to a group of muscles that are innervated by a specific nerve root. The area of skin receiving sensory innervation from a single nerve root is a *dermatome*.[2] *Efferent* (motor) nerves deliver nerve signals away from the brain to other parts of the body such as muscles. Conversely, *afferent* (sensory) nerves deliver signals to the central nervous system.

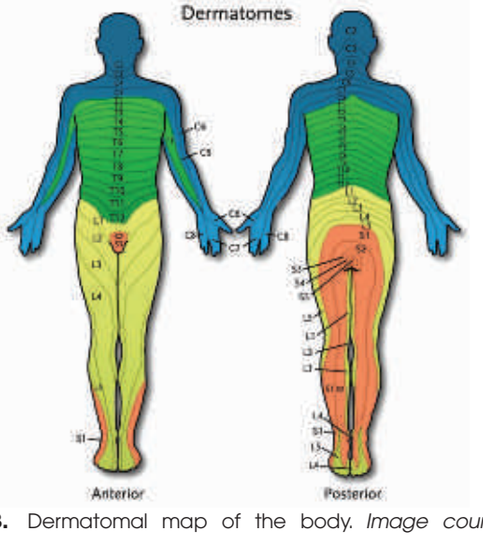

Figure 3. Dermatomal map of the body. *Image courtesy of Medtronic.*

N. Chatterjee/Journal of the Spinal Research Foundation 6 (2011) 40–44

Nerve Conduction Study (NCS)

The NCS measures the responses obtained by activating certain nerves in the upper and lower limbs. The choice of nerves to stimulate is contingent upon the presenting signs and symptoms. Peripheral nerves are typically easily stimulated to produce an action potential after an electrical pulse is applied to the skin.[1] The nerves are stimulated along the pathway as the electrical response of the nerve is recorded.[3] By analyzing the morphology of the nerve waveform, amplitude, latency, and conduction velocity, the physician conducting the exam can gather pertinent information.[4] Motor and sensory nerves can be evaluated. Motor nerve conduction is assessed by stimulating the motor nerve and evaluating the electrical signal from the corresponding muscle. Sensory nerve conduction is measured by stimulating a sensory nerve to produce a signal. The results of these studies offer distinct information about the potential pathology.[1]

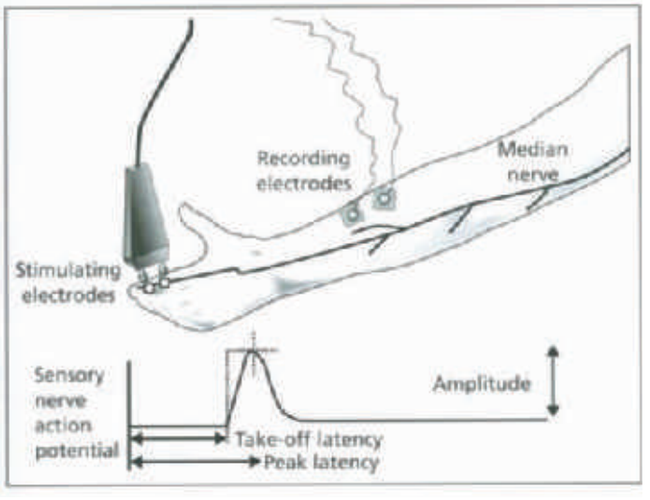

Figure 4. This is the basic setup for a sensory nerve conduction study. The machine gives a tracing of the sensory nerve action potential (SNAP). The amplitude and latency can easily be measured. *Image was published in Easy EMG, Weiss et al., page 3, Copyright Elsevier 2004.*

Electromyography (EMG)

Electomyographic testing involves inserting needles into the muscles innervated by the particular nerves. Most muscles can be tested by the EMG, however, there needs to be a balance between the number of muscles that are required for diagnosis and patient comfort.[1] EMG allows the physician to assess muscle contraction by analyzing the motor unit, which is the anterior horn cell, the axon, as well as the muscle fibers innervated by the particular motor neuron.[3] Motor unit action potential characteristics, including the amplitude, duration, and recruitment are observed by the electromyographer. Insertional activity is the electrical tracing produced in electromyography as a result of the insertion of the needle electrode. The motor unit is then evaluated by using distribution and size information as well as by analyzing the recruitment pattern.[3]

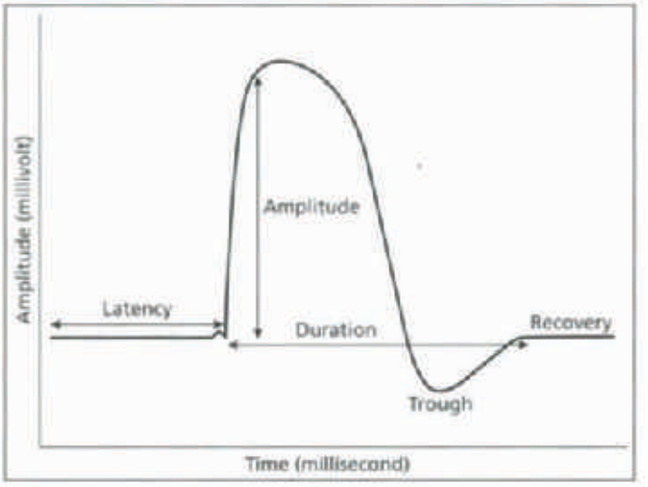

Figure 5. Example of an action potential. The different aspects of the wave are analyzed. *Image was published in Easy EMG, Weiss et al., page 20, Copyright Elsevier 2004.*

Prior to activation of a muscle fiber, the nerve action potential must travel across the neuromuscular junction, where the neuron initiates muscle contraction. The nerve fiber endings connect to motor endplates, where the muscle responds to acetylcholine, a neurotransmitter found in the peripheral nervous system that allows muscle activation.[5] Voltage-gated calcium channels become activated, resulting in an influx of calcium, which allows the release of acetylcholine.[1] Acetylcholine in turn transmits an impulse across this junction, allowing muscle contraction.[5]

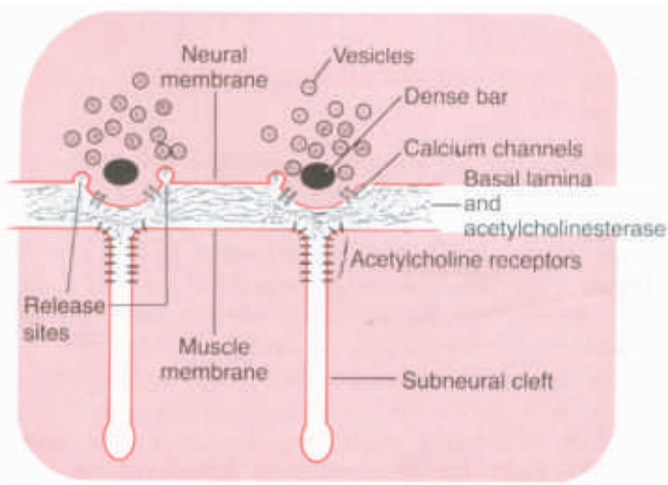

Figure 6. Release of acetylcholine from synaptic vesicles at the neural membrane of the neuromuscular junction. *Image was published in Textbook of Medical Physiology, 9th ed., Guyton and Hall, p.88, Copyright W.B. Saunders, 1996.*

The needle examination is used to evaluate the following: insertional activity, spontaneous activity, morphology, and size and recruitment.[4] Normal muscle at rest is electrically silent, with brief insertional activity.[3] Abnormal spontaneous activity is one of the most pertinent aspects of the needle EMG. The form of spontaneous activity can provide information on the diagnosis, the severity, and the chronicity of the lesion.[1]

Electrodiagnostic imaging can provide essential information regarding the underlying nerve disorder, such as entrapment neuropathies, carpal tunnel syndrome, peroneal neuropathies, polyneuropathies, brachial, and lumbar plexopathies. Neuromuscular disorders such as myasthenia gravis, Lambert Eaton syndrome, and botulism, as well as certain myopathies, can also be diagnosed using these studies.

Radiculopathy

Radiculopathy is one of the most common reasons for referrals for electrodiagnostic testing.[1] Radiculopathy may be caused by nerve root compression in the cervical, thoracic, or lumbar spine, but most commonly occurs in the cervical and lumbar spine.[1,2] Radiculopathy is a condition in which the nerve root is irritated and may present as weakness, pain, or numbness along the course of the nerve.[7] Radiculopathy typically presents with pain and numbness in a distribution of the particular nerve root involved. The etiology of radiculopathy includes mechanical compression by osteoarthritis, disc herniation, and thickening of surrounding ligaments. Less common causes include tumor or infection of the spine.

Frequently, radiculopathy exists in absence of an actual mass such as a disc herniation, abscess, or tumor. In these cases, infiltration secondary to cancer, granulomatous tissue, or infection are possible causes. Infarction of the nerve root seen in vasculitic neuropathy is often the cause of radiculopathy. These cases demonstrate the importance of electrodiagnostic testing, as they occur in the absence of any abnormal imaging studies, such as MRI or CAT scan.[1] The diagnosis of radiculopathy

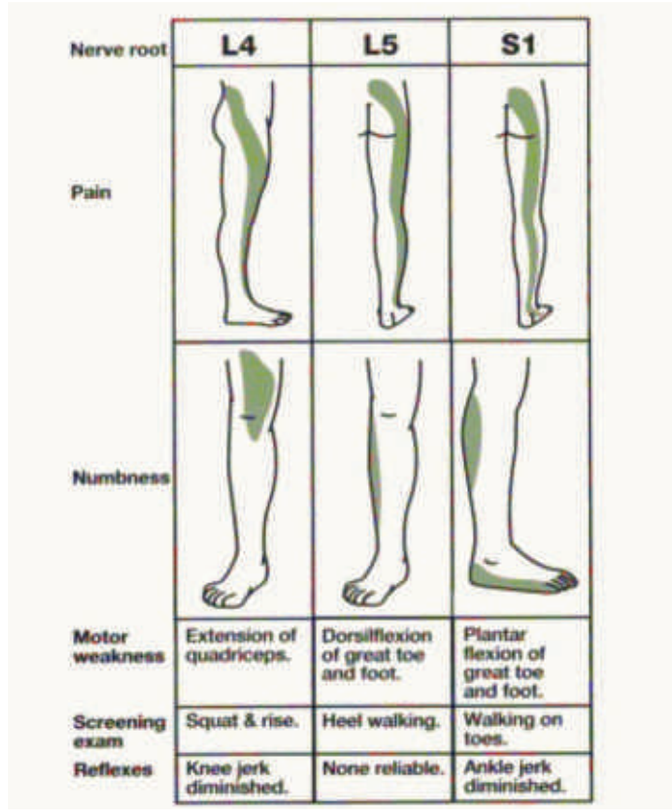

Figure 7. Testing for lumbar nerve root compromise. *Image was published by U.S. Department of Health and Human Services in 1994 (since 2000, the Agency for Healthcare Research and Quality).*

is mainly based on physical examination, clinical history, and advanced imaging studies, such as an MRI or CAT scan. Electrodiagnostic testing can localize the nerve root lesion, confirm the diagnosis, and assist with prognostication.[8] Distinguishing whether a nerve root injury is present and further localizing the exact root can be difficult, but essential for patient management.[9] Electrodiagnostic testing can aide in achieving a correct diagnosis to, in turn, allow for implementation of appropriate treatments. It is essential to reiterate that the needle EMG and NCS do not identify the etiology of the radiculopathy, but rather identifies the presence of axonal loss and confirms the presence, chronicity and localization of a radiculopathy.[8]

Differential Diagnosis of Radiculopathy

It is important for the clinician to rule out diagnoses with presentations very similar to radiculopathy. For instance, patients with C5 and C6 radiculopathy have pain in a very similar distribution as those with carpal tunnel syndrome. Plexopathy and entrapment neuropathy can also present similarly. Patients who present with foot-drop have weakness in their dorsiflexor muscles and are unable to elevate their foot. Foot-drop can be secondary to a peroneal neuropathy in the lower extremity, but can also be from sciatic neuropathy, L5 radiculopathy, or lumbosacral plexopathy. Using electrodiagnostic testing, objective data can be obtained to diagnose and prognosticate the proper condition.[1]

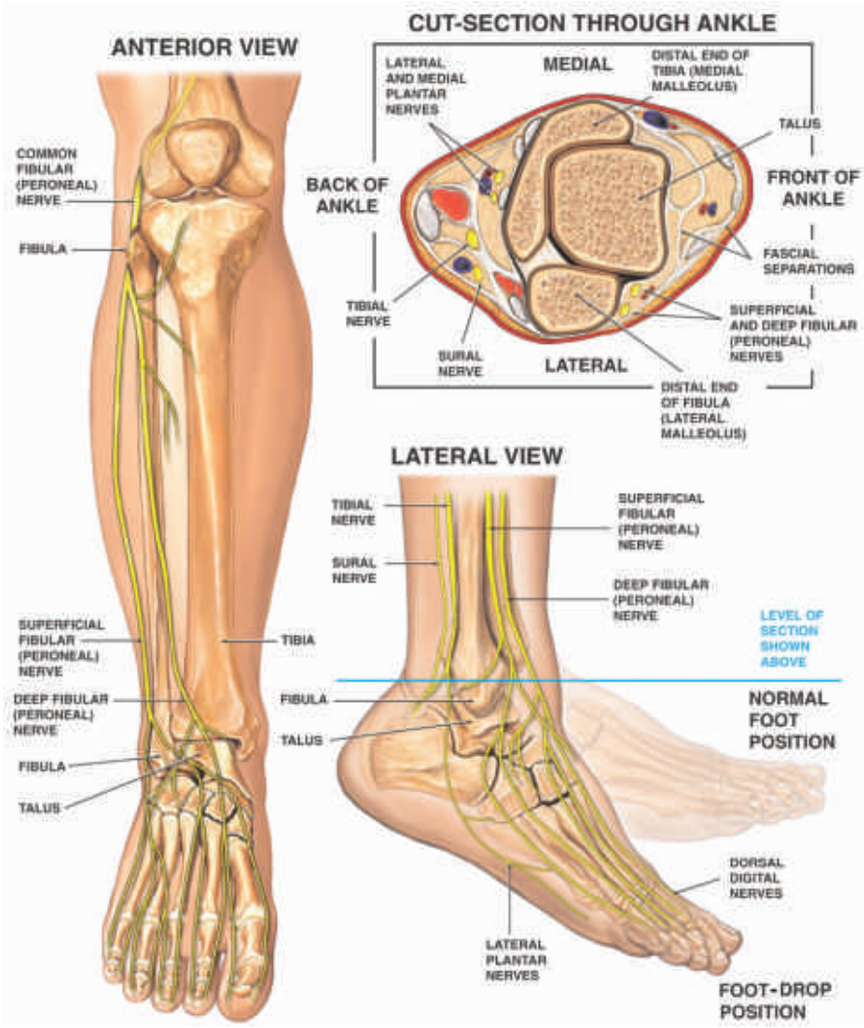

Figure 8. *Medical Illustration, Copyright 2011, Nucleus Medical Media, All rights reserved.*

Neuropathy: general term to mean disease or injury of a nerve. Neuropathy may be further specified according to the location of the nerve disease/injury.

Radiculopathy: injury or disease of a ***spinal*** nerve ***root***.

Plexopathy: Injury or disease of the nerve ***plexus***: the set of nerves after they leave the spinal cord at the level of the neck (brachial plexus) or the lower back (lumbosacral plexus).

Entrapment: compression of ***peripheral*** nerves (away from the spinal cord). Carpal tunnel syndrome is the most common entrapment neuropathy.

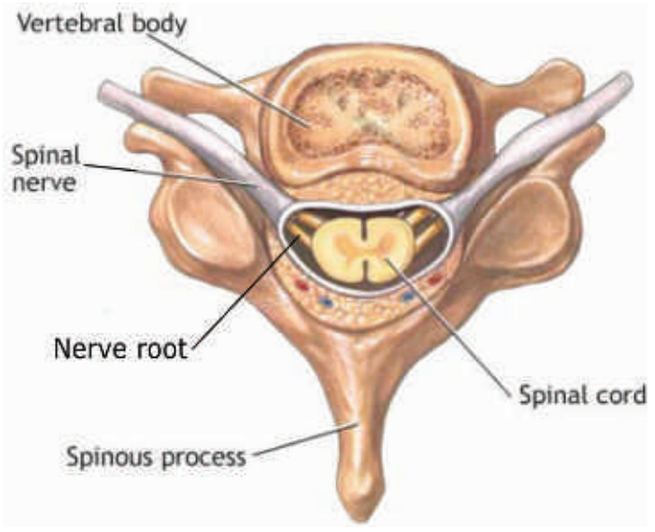

Figure 9. *Image courtesy of Medtronic.*

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Neil Chatterjee, M.D.

Dr. Chatterjee is a board certified, fellowship-trained physician who specializes in the non-operative treatment of spine, joint, neuropathic, and muscle pain. He has expertise in encompassing the treatment of cervical, thoracic, and lumbar spine pain in addition to myofascial pain, pelvic dysfunction, painful neuropathies, lower back pain during pregnancy, adolescent sport and spine injuries, and fibromyalgia. Dr. Chatterjee works with each of his patients to reach the goal of alleviating their pain, restoring and maximizing function, and improving their overall quality of life. He utilizes various techniques in the diagnosis and treatment of neuropathic, visceral, musculoskeletal, and spine pain. He begins with a conservative approach, including pharmacologic treatment, overseeing proper exercise and manual treatment, and if indicated, proceeds to more advanced techniques, including spinal interventions and peripheral joint injections. Dr. Chatterjee is trained in performing diagnostic and therapeutic spine injections under fluoroscopy, electrodiagnostic testing, and medical acupuncture. A major focus in his treatment approach is education of his patients on methods of preventing degenerative disc disease through smoking cessation, weight loss, proper nutrition, and exercise strategies. He also led several lectures on pain management. Dr. Chatterjee is a Fellow of the American Academy of Physical Medicine and Rehabilitation and an active member of the International Spine Intervention Society and the American Academy of Pain Medicine.

Botulinum Toxin Injections: Emerging Uses in Non-Surgical Spine Treatmentand Pain Management

Michael S. Cicchetti, M.D.

Botulinum toxin has been used for medical purposes in humans since the early 1980s when it was first used to treat strabismus, a condition commonly seen in children that involves "crossing of the eyes" secondary to failure of the extraoccular muscles to work together synchronously.[1] Since then, botulinum toxins have gained worldwide recognition for their beneficial effects in a myriad of medical conditions. However, growing popularity in Hollywood and cosmetic medicine, given the toxin's ability to soften wrinkles, has obscured some of the major medical advances that have been realized in recent years.

Clostridium botulinum, the bacteria that produces the toxin was first isolated in Belgium in 1895. It is commonly found in soils worldwide, and the deleterious effects of poisoning from the toxin were first recorded in Europe in 1735. Once consumed, the toxin is absorbed by the intestines and goes into the bloodstream. From there, it moves quickly to attack the nervous system resulting in paralysis, vomiting, nausea, blurred vision, and difficulty swallowing. The toxin was named after the Latin word for sausage, "botulus," because it was first associated with sausage poisoning. The syndrome, described above, is known today as "Botulism" and is associated with food poisonings (mostly home-canned foods) or exposure of open wounds to contaminated soils.

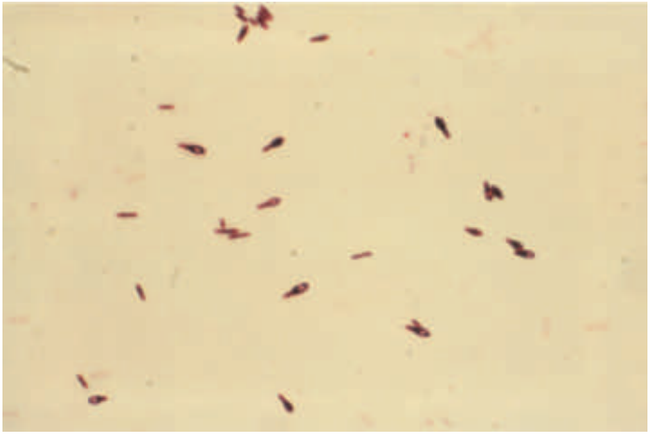

Figure 1. Picture of the bacterium, Clostridium botulinum, with spore formation. The bacteria is anaerobic, rod-shaped, and produces a potent neurotoxin. *Figure courtesy of CDC/Dr. George Lombard.*

Clostridium botulinum produces 7 similar, yet seriologically distinct, neurotoxins (Labeled A, B, C1, C2, D, E, F, G). Types A and B are the only serotypes currently used for medicinal purposes, and types C and D only cause symptoms in animals. Different production and purification methods are utilized to make the toxin safe for medical use. Commercially available type A neurotoxin is the most widely available serotype. It consists of a type A neurotoxin core molecule surrounded by proteins of various molecular weights.

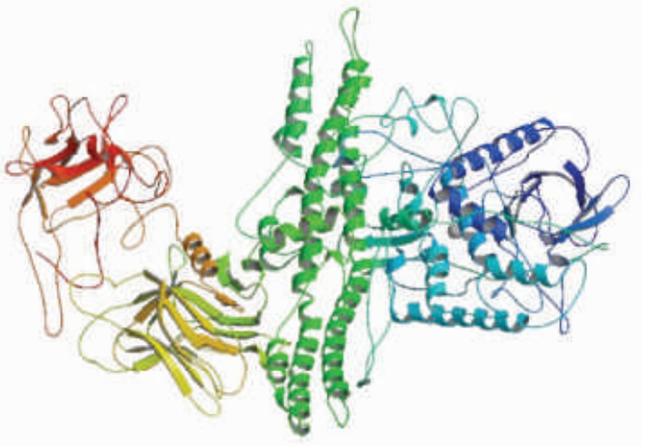

Figure 2. Molecular structure of type A botulinum toxin. A 150 kD toxin core molecule is surrounded by a 130 kD NTNH protein and HA proteins of various molecular weights to yielding a total molecular weight of 900 kD. kD = kiloDalton. One kiloDalton (kD) is equal to approximately the weight of one thousand hydrogen atoms, and is equivalent to 1.66×10^{-21} grams. This unit is used to express the size of proteins.

Botulinum toxins block the release of acetylcholine at the presynaptic neuromuscular junction, causing a chemical denervation and relaxation of the muscle. It is also thought that botulinum toxins block the release of certain neurogenic inflammatory mediators (e.g. substance P, glutamate, CGRP, etc.) in afferent pain fibers. This implies that there is a separate, indirect method (independent of its action on muscle) for botulinum toxins to work in patients with chronic pain.

Currently, there are four separate brands of botulinum toxins available in the United States (Botox, Dysport, Xeomin and Myobloc). Three of them are Type A and Myobloc is Type B. OnabotulinumtoxinA,

Botox	Dysport	Xeomin	Myobloc
Type A OnabotulinumtoxinA Allergan USA	Type A Abobotulinumtoxin A Ipsen Biopharm UK	Type A IncobotulinumtoxinA Merz Pharmaceuticals Germany	Type B RimabotulinumtoxinB Solstice Neurosciences USA
FDA Approvals: Cervical Dystonia, Chronic Migraine, Blepharospasm, Strabismus, Axillary Hyperhydrosis, UE Spasticity, Glabellar Lines (Botox Cosmetic)	FDA Approvals: Cervical Dystonia, Glabellar Lines	FDA Approvals: Cervical Dystonia, Blepharospasm	FDA Approvals: Cervical Dystonia
Reconstitution with sterile saline	Reconstitution with sterile saline	Reconstitution with sterile saline	Comes already prepared in solution
100 and 200 units	300 units	50 and 100 units	2500, 5,000 and 10,000 units

Figure 3. Comparison of different commercially available brands of Botulinum toxins available in the United States.

more commonly known as Botox, has been used in the U.S. much longer than any other brand of toxin. As a result, it has more supporting research and more FDA approvals to its credit.

There are numerous medical conditions that are being treated today with botulinum toxins. The remainder of this article will focus mostly on both FDA-approved, as well as off-label use, of Botox in the spine and pain management. A few comparative studies exist that compare different botulinum toxins, but no one has shown superiority of one type of botulinum toxin over another. Use of a particular type A toxin and/or use of a different serotype (type B, Myobloc) is subject to a physician's experience, their preferences, side effects, and specific indications for use.

Cervical Dystonia

Cervical dystonia (CD) is the most common form of focal dystonia and the most frequently misdiagnosed movement disorder. CD patients classically present with simultaneous and sustained contraction of both agonist and antagonist muscles of the neck. Based on head posture and positioning, CD can be described as torticollis (neck rotation), anterocollis (head-forward flexion or pulled forward), retrocollis (head-posterior extension or pulled backward), or laterocollis (head tilt or lateral flexion).[4] Combinations of the above postures are also common. Greater than 75% of patients will report neck pain associated with abnormal head and neck posturing.

Prevalence of the disease is about 9 to 30 cases per 100,000, and prevalence may differ between ethnic groups. More women than men are affected (1.3 to 2-fold) and mean age of onset is around 40 years, although symptoms can occur at any time in life. Furthermore, 12% may have a familial history of dystonia, and 5% to 16% have a history of a prior injury (e.g. whiplash).

Figure 4. Classic descriptions of abnormal head postures associated with cervical dystonia. A combination of postures is most common in the clinical setting. *Figure courtesy of the National Spasmodic Torticollis Association.*

Most cases of cervical dystonia are described as idiopathic and the etiology of the disease process is not well understood. Most theories involve pathogenesis at both the peripheral and central nervous system level. The efficacy of oral drug therapy is limited and many different medication trials have failed to show significant success in controlling the symptoms of CD.

Multiple randomized, placebo-controlled clinical trials support the efficacy and safety of commercially available botulinum toxins for the treatment of cervical dystonia, with up to 90% of the patients with CD benefiting from treatment.[3,14] Superiority of type A vs. type B has not been demonstrated, but there does appear to be a higher incidence of dry mouth with botulinum toxin type B.[15] Today, injection treatment with botulinum toxins is considered a first line treatment for cervical dystonia in adult patients to reduce the severity of abnormal head position and neck pain.[3,5,6,7] Injection of Botox and other botulinum toxins around the neck is usually accompanied by needle EMG (electromyography) guidance to assure the correct muscles are being targeted. The individual muscles to be injected are determined by the presentation of head posturing, location of pain, palpation of hypertonic/hypertrophic muscle, and activity of the muscle on EMG.

Most patients with cervical dystonia will require repeated injections about every three months based upon response. Unfortunately, there is no known cure for cervical dystonia, but there are reports of remission in 9 to 20% of patients.[16]

Chronic Migraine

Migraine is a well-known disorder in our society, with most people having some experience with it (either personal experience or knowing someone who suffers from it). Migraine can be debilitating and is responsible for billions of dollars annually in lost wages and time off work.[17]

Typical migraine headaches occur more commonly in women, frequently are unilateral (but can occur bilaterally), and are pulsating in nature.[18] Symptoms include, but are not limited to, nausea, vomiting, photophobia (increased sensitivity to light), and phonophobia (increased sensitivity to sound), and typically worsen with physical activity.[18,19] Approximately one-third of people who suffer from migraine headaches perceive an aura (unusual visual, olfactory, or other sensory experiences that are a sign that migraine will soon occur).[20]

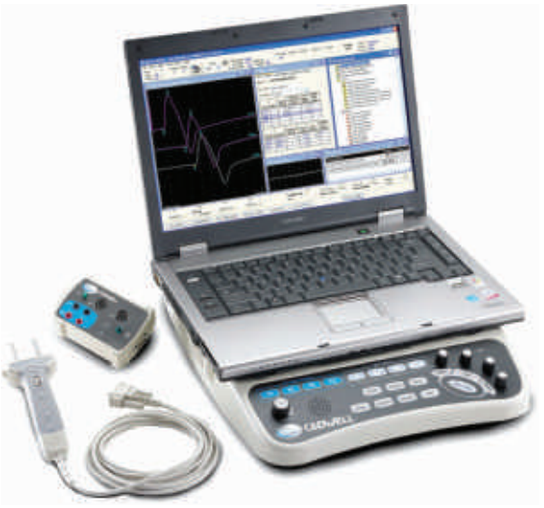

Figure 5. EMG machine. *Figure courtesy of Cadwell Laboratories, Inc.*

Initial treatment for migraine is with analgesics for the headache, an antiemetic for the nausea, and avoidance of triggering conditions. There are also medications to help abort or lessen the effects of migraine once they occur (e.g. triptans and ergotamines). Several classes of medicines are typically prescribed for prophylaxis treatment in patients with frequent recurrence of migraine (beta blockers, calcium channel blockers, anticonvulsants, and tricyclic antidepressants).

Current hypotheses regarding the pathogenesis of migraine are beyond the scope of this paper. However, it is now accepted that migraine headaches are related to persistent alterations in intracerebral neurovascular function, including sensitization of sensory trigeminal pathways, and enhanced excitability of the central nervous system.[21]

Episodic migraine is defined as headaches occurring on 14 or fewer days per month. Similarly, chronic migraine is now clinically defined as those with persistent migraine headache (and related symptoms) occurring >15 days per month with headaches lasting at least four hours a day.[18] The burden of disease associated with chronic migraine has a huge impact on society. Chronic migraineurs have 2.8-fold higher acute prescription medication costs than episodic migraineurs, and they are significantly more likely than episodic migraineurs to visit the emergency room, their primary care physician, a neurologist, or a headache specialist. Furthermore, direct and indirect health care costs are 341% higher per patient for chronic migraineurs than for episodic migraineurs.[17]

Botox has not been proven effective for those with episodic migraine. However, the results of two large, randomized, double-blinded, placebo-controlled studies that were carried out across multiple sites in the U.S. and Europe did show significant benefit in patients with chronic migraine.[22–23] These studies led the FDA to approve Botox for treatment of patients with chronic migraine in October 2010. The combined results of the studies showed that treatment with Botox resulted in significant reductions from baseline in the number of headache and migraine days, cumulative hours of headache on headache days, and frequency of moderate/severe headache days, which in turn reduced the burden of illness in adults with disabling chronic migraine. Few treatment-related adverse events were reported.

The recommended injection paradigm for chronic migraine is based on the phase 2 trials that led to the larger phase 3 trials mentioned above. A total of 155 units of Botox was administered as 31 fixed-site, fixed-dose injections across 7 specific head/neck muscle areas.[24–25] The injection sites follow the distribution and areas innervated by the trigeminal sensory system. Botox is hypothesized to decrease hypersensitization of the central nervous system, which is thought to be a predominant component in the development of migraine.

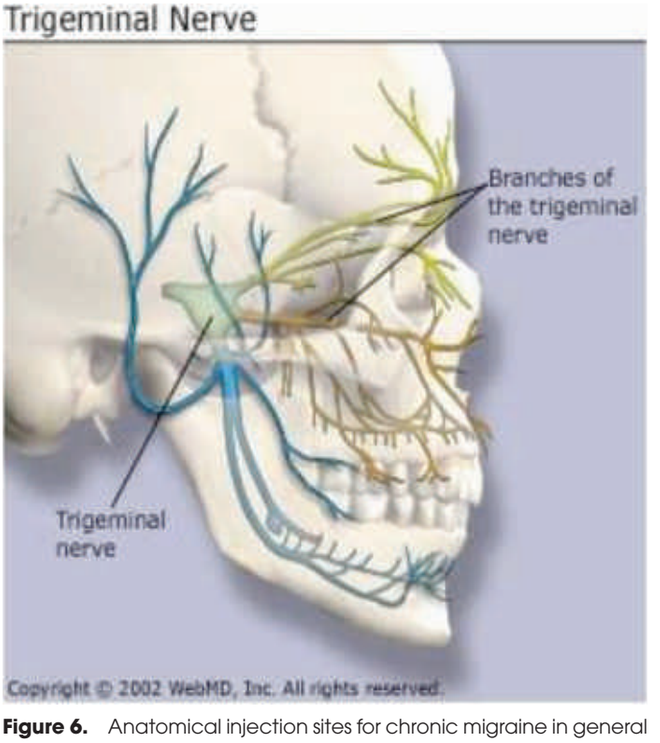

Figure 6. Anatomical injection sites for chronic migraine in general follow the distribution and areas innervated by the Trigeminal Sensory System. *Image courtesy of WebMD, Inc.*

Chronic Tension Headache and Myofascial Pain

Tension-type headache is the most prevalent of all the primary headache disorders and is associated with significant costs to both headache sufferers and society as a whole.[26,27] Symptoms are typically mild-to-moderate, have a pressing and/or tightening quality (bilaterally)

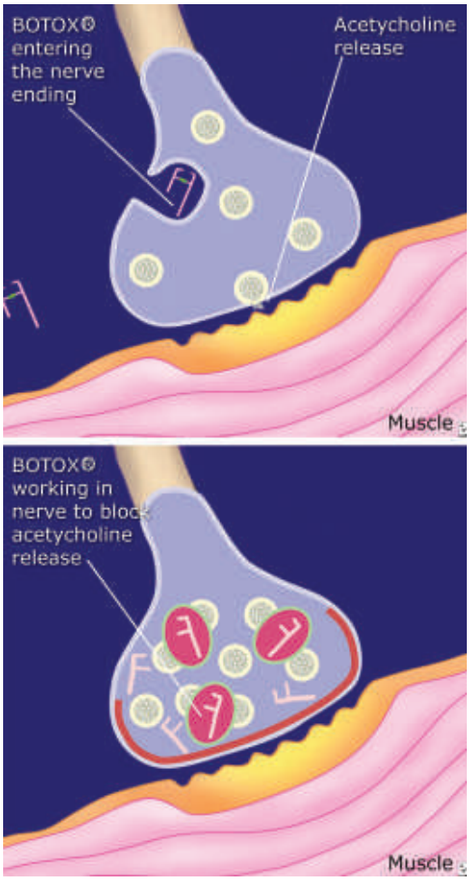

Figure 7. Botulinum toxin mechanism of action at neuromuscular junction. *Image reprinted with permission from Medscape.com, 2011. Available at: http://emedicine.medscape.com/article/89095-overview.*

that is nonpulsatile, and are not aggravated by routine physical activity.[18,19] Headache-related symptoms are likely secondary to myofascial and musculoskeletal abnormalities of the neck and shoulders.[29]

Myofascial pain syndrome (MFPS) is classically described as a muscular pain produced by trigger points that cause motor, sensory, and autonomic dysfunction. Myofascial trigger points are recognized as hyperirritable spots in skeletal muscles that are associated with a hypersensitive palpable nodule or taut band (e.g. knot). They can occur in just about any muscle in the body, but are much more commonly found in muscles that are overused or serve as axial stabilizers (e.g. trapezius, posterior cervical and lumbar muscles).[28]

Historically, management of tension headache and myofascial pain includes stretching exercises, massage, ice, myofasical release, dry needling, local anesthetic injections, and pharmacotherapy with muscle relaxers, anti-inflammatories, and analgesics.[29]

Multiple investigators have looked at botulinum toxin injections as a potential treatment for chronic tension headache sufferers given the toxin's known direct affect on muscle. Unfortunately, the results of these studies have been mixed.

Smuts et al. conducted a randomized, double-blind, placebo-controlled trial using Botox on 37 patients with chronic tension-type headache.[30] Headache diaries and chronic pain index scores were measured over a 4-month study period. Patients treated with Botox showed an improvement in headache severity over the 4-month study period with 13 out of 22 patients showing 25%, 50%, or greater than 50% improvement in headache scores, compared to only 2 out of 15 patients in the placebo group ($p = 0.001$). There was also a statistically significant improvement in the number of headache-free days in the group that received Botox. Likewise, Silberstein et al. observed that patients who received Botox were statistically more likely to receive more than a 50% decrease in the number of tension headache days vs. placebo at 90 days post-injection ($p \leq 0.024$).[31]

Mixed reviews for the efficacy of Botox in chronic tension headache were reported by Harden et al. Twenty three patients were randomized to receive Botox or a placebo. The Botox group reported greater reductions in headache frequency during the first part of the study, but these effects dissipated by week 12. Reductions in headache intensity over time did not differ significantly between groups, and there were no differences on any of the secondary outcome measures.[32] Finally, no benefit of using Botox in chronic tension-type headache was shown by Padberg et al. and Schmitt et al.[33,34]

A 2007 review of botulinum toxin type A for the prophylactic treatment of chronic daily headache (which includes migraine headache, tension headache, hemicrania continua, and new onset daily persistent headache) stated that no sufficient evidence for suc-

cessful treatment can be obtained from randomized, double-blind, placebo-controlled trials.[35] However, this review was published before the two studies emerged proving the effective use of Botox in chronic migraine. It is likely that some subgroups of patients with chronic tension headache will benefit from long-term treatment with Botox, however, definitive data is still lacking.

There is also evidence both for and against using Botox in patients with myofascial pain. Porta performed a randomized trial comparing the effects of Botox with the steroid methylprednisolone in 40 patients suffering from chronic myofascial pain. He reported that the results indicate the superior efficacy of Botox over conventional steroid treatment in patients suffering from MFPS when combined with appropriate physiotherapy.[36] Wheeler also found benefit of using Botox over a placebo in patients with cervicothoracic MFPS.[37] However, the results were not statistically significant.

On the other hand, Ferrante et al. showed that there was no benefit of using Botox when compared with placebo in patients with cervicothoracic myofascial pain.[38] Furthermore, two separate reviews state that the current evidence does not support the use of Botox injections for treatment of trigger points in MFPS, and that there are no statistically significant differences between trigger point injections with Botox vs. local anesthetic.[39,40]

Based upon the evidence presented above, physicians should discuss with patients that positive results from using Botox in tension headache and chronic MFPS cannot be predicted. However, the safety profile of Botox in these patient populations is more than acceptable, and if patients have tried and failed a wide variety of other traditional treatments, a trial of Botox injections (or another botulinum toxin) may be justified.

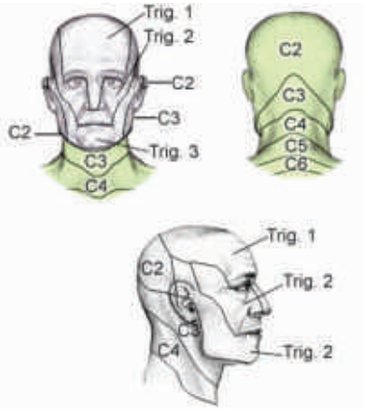

Figure 8. Dermatomal map of the head. *Image reprinted with permission from Medscape.com, 2011. Available at: http://emedicine.medscape.com/article/1878388-overview.*

Chronic Low Back Pain

Nearly 90% of adults experience back pain at some point in their lives.[41] This number is probably closer to 100% as the remaining 10% likely forgot about a time when they had back pain. Approximately 70% to 90% of cases of acute low back pain will resolve within 5 weeks.[42] Based on different reports, estimates of the total economic impact of treating low back pain are in the billions (25–50 billion dollars per year).[43] Only a small percentage of patients (approximately 7%) will go on to develop chronic, unremitting pain and disability, however this group accounts disproportionately for the costs associated with low back pain (75% of all the costs and 85% of all the disability days).[44,45]

A vast number of specialties treat patients with chronic low back pain and approaches to treatment will vary widely between specialties. There are literally hundreds of different treatment approaches mentioned in the literature, ranging from physical therapy, chiropractic, osteopathic, pharmacologic, homeopathic, acupuncture, interventional and surgical, that practitioners employ when treating pain and dysfunction associated with low back pain. For those patients who don't respond to traditional treatments, there is increasing evidence that a trial of Botox injections may be indicated to relieve pain and improve function.

Several prospective trials have indicated that Botox is beneficial in patients with chronic low back pain.[46,47,48] Foster et al. performed a randomized, double-blind, placebo controlled study on 31 consecutive patients with chronic low back pain.[49] At 3 weeks, 11 of the 15 patients who received Botox had greater than 50% relief (73.3%), while only 4 of the 16 in the saline group (25%). At 8 weeks, 9 of the 15 (60%) in the Botox group and 2 of the 16 (12.5%) in the saline group had relief ($p = 0.009$). No patients in any of the studies had significant adverse affects from treatment.

Also, a favorable initial response was reported to predict subsequent responsiveness in one of the prospective trials.[46]

Piriformis Syndrome

The piriformis muscle originates from the inner surface of the sacrum, exits the pelvis through the greater sciatic foramen, and inserts onto the posterior facet of the greater trochanter.[28] Its primary function is to externally rotate the thigh (i.e. turn the thigh outward). Many structures exit the greater sciatic foramen along with the piriformis muscle. It is theorized that enlargement of the muscle, spasm of the muscle, and/or anomalous locations of the sciatic nerve can cause symptoms. Classic symptoms of piriformis syndrome involve buttock and lower extremity pain associated with numbness, tingling, and/or weakness. Furthermore, Travel and Simmons refer to the piriformis muscle as a "double devil" because it causes as much distress by nerve entrapment as it does by projecting pain from trigger points.[28] The symptoms seen in patients with piriformis syndrome are very similar to more commonly observed pain syndromes often seen in clinical practice (e.g. disc herniation and/or stenosis associated with irritation of a lower lumbar nerve root). Because the symptoms are so similar in all three of these scenarios, it is imperative that the clinician first rule out all possible causes of spine pathology as a cause of sciatica before pursuing treatment for piriformis syndrome.

Typical treatments for piriformis syndrome may include a combination of physical/manual therapy, chiropractic manipulation, massage, anti-inflammatories, analgesics, and trigger point injections. When appropriate conservative treatment fails, a trial of botulinum toxin injections may be indicated to decrease muscle spasm, pain, and improve function.

Using a double-blind, crossover design, Childers showed a statistically significant decrease ($p < 0.05$) in visual analog pain scores when 100 units of Botox was injected into symptomatic piriformis muscles using fluoroscopic/EMG guidance.[51] Similarly, Fishman also established efficacy of Botox as an adjunct to physical therapy in patients with piriformis syndrome.[52] Patients experienced more relief in pain than patients receiving lidocaine with steroid ($p < 0.05$) and placebo ($p < 0.001$). In addition, he showed that H-reflex prolongation >1.86 msec (3 standard deviations) of the mean on standard nerve conduction study is a clinical indication of piriformis syndrome.

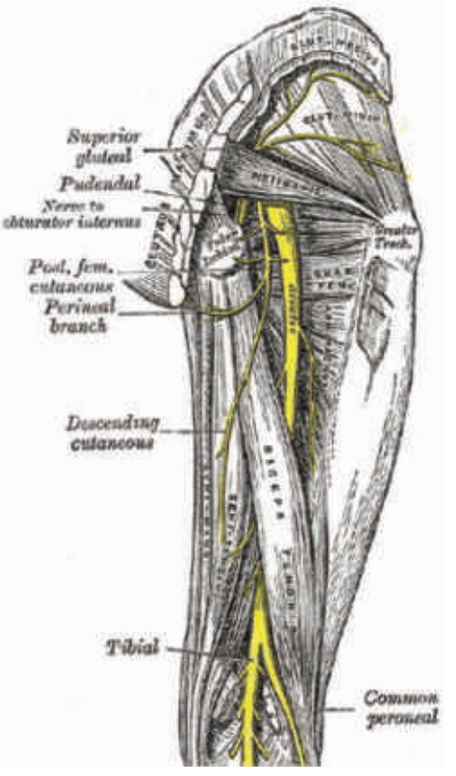

Figure 9. Image showing the normal course of the sciatic nerve anterior to the piriformis muscle. The piriformis muscle originates on the undersurface of the sacrum, exits through the greater sciatic foramen and inserts onto the posterior facet of the greater trochanter. *Image courtesy of Bartleby.com from Henry Gray's Anatomy of the Human Body.*

Chronic Neuropathic Pain

Injury to either our central or peripheral nervous system can result in pain. The experience of peripheral nervous system pain is unique from that of musculoskeletal or visceral pain. Peripheral neurogenic pain is usually localized to a dermatome or cutaneous nerve field, and the frequency of pain may be constant or intermittent. Classic descriptors include deep aching, cramping, superficial burning or stinging, numbness, electrical shock, and pins and needle-like pain.

The cause may be inflammatory, ischemic (decreased blood flow), traumatic, mechanical, or infectious.

Depending on the cause of nerve pain, different treatments are offered. If a nerve root is being irritated or mechanically "pinched" in the spine, physical therapy may help to establish normal nerve gliding and blood flow, or the offending disc or bone may need to be removed surgically to restore nerve health and function. If the nerve is inflamed, oral anti-inflammatories or a local injection of steroid/local anesthetic may be prescribed. Also, several classes of medications have successfully been shown to reduce the perception of nerve-related pain by interacting with different receptors and ion channels at the peripheral and central nervous system level (e.g. Gabapentin, Lyrica, Cymbalta, and tricyclic antidepressants).

For recalcitrant, focal, nerve-related pain that has not responded to traditional therapies, a trial of botulinum toxin injections may provide significant relief of chronic pain and suffering. It is theorized that the analgesic effects of botulinum toxins are independent of its action on muscle tone, possibly acting directly on neurogenic inflammation.[52]

Case reports have been published that show Botox's effectiveness in treating chronic neuropathic pain conditions associated with post-herpetic neuralgia (a painful complication after an attack of shingles), multiple sclerosis, cervical radiculopathy, and HIV-neuropathy.[53,54,55] Ranoux randomized 29 patients with chronic neuropathic pain to receive Botox or placebo (saline) injections subdermally in the focal region of pain. Most of these patients had traumatic peripheral nerve injuries and/or post-surgical nerve injury. The results showed that Botox was superior to the placebo, and that Botox may induce direct analgesic effects in patients, independent of its effects on muscle tone.

Potential Side Effects and Interchangeability of Botulinum Toxins

Botulinum toxin injections have been shown to be safe and well tolerated in many clinical settings. Side effects depend on the part of the body injected, dose and dilution used, the condition being treated, and co-existing illnesses. Figure 10 outlines some of the most common side effects (and frequency of occurrence) experienced in patients receiving type A botulinum injections for cervical dystonia and migraine headache. Also, all botulinum toxins carry a black box warning stating that their effects may spread outside the area of injection, may occur hours to weeks after injection, and may last for weeks to months. Swallowing and breathing difficulties can be life threatening and there have been reports of death.[3,5,6,7]

The lack of interchangeabilty between botulinum toxins make it so that units of one botulinum toxin cannot be compared or converted into units of another toxin.[3,5,6,7]

Also, there are no significant differences in side effects between separate type A formulations. However, type B (Myobloc) has consistently shown a higher incidence of dry mouth and dysphagia when compared with the type A serotype.[57]

Injections are not advised in patients who have an active infection (either locally or systemically).[3,5,6,7] Patients that are known to have prior hypersensitivity and/or allergic reactions to botulinum toxins or who have pre-existing neuromuscular disorders (e.g. Amyotrophic Lateral Sclerosis, Myasthenia Gravis, or Lambert-Eaton Syndrome) should be cautioned.[3,5,6,7] Botulinum toxins have also been associated with an increased incidence in difficulty swallowing and/or breathing (esp. when used to treat patients with cervical dystonia). Patients with a known history of dysphagia or breathing problems should be monitored closely.[3,5,6,7] Finally, the pharmacokinetics of botulinum toxin in pregnant and nursing mothers has not been firmly established.[3,5,6,7] Thus, the benefit of injections and the risk of harm to the fetus or newborn must be discussed with these patients, and the decision to proceed with injections should only occur if the benefit to the mother outweighs the risk to the child.

Cervical Dystonia	Chronic Migraine
Dysphagia	Neck Pain
Upper Repiratory Infection	Headache
Neck Pain	
Headache	
Increased Cough	
Flu-like Syndrome	

Figure 10. Most commonly observed adverse reactions (≥ 5% and > placebo) in controlled studies.

Other Barriers to Treatment with Botulinum Toxins

Botulinum toxin injections are expensive, and financial restrictions weigh heavily on one's decision to seek treatment for off-label, non-FDA approved indications. Also, treatments generally have to be repeated at 3-month intervals (sometimes longer) because the effects of the toxin are not permanent. Many times, the physician may be able to work with an insurance company to receive special approval. However, this process can be arduous and is notoriously unpredictable. Allergan has a patient assistance program that provides Botox at no charge to financially eligible patients. Those who may qualify include patients who are uninsured or underinsured. Eligibility requirements and application forms can be found online.[58]

Summary

A wise person once said, "Pain is inevitable, but suffering is optional." For those patients who suffer from chronic pain, this article has outlined some of the emerging uses of botulinum toxin injections that have proven useful in a non-surgical spine and pain management setting. Generally speaking, botulinum toxins are viewed as being very safe, and are provided as secondary or adjunctive treatments after more traditional measures have failed. The exception to this is in the case of cervical dystonia, where botulinum toxin injections are now considered a first line treatment.

Further research regarding the continued use of botulinum toxins is still needed, and in time, more FDA approvals should emerge among all the commercially available toxins. The increased visibility of multiple toxins in clinical use will also hopefully provide much needed competition between manufacturers that may lead to decreased costs of treatment.

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Dr. Cicchetti specializes in functional restoration of the whole patient with particular expertise involving the spine and neuromusculoskeletal systems. His treatment approaches include detailed dynamic assessment of how the body moves and how the patient interacts with their environment. He is skilled in physical therapy assessment, osteopathic assessment, sports medicine, exercise prescription, and knowledge of alternative modalities to treat spine, nerve and musculoskeletal pain. In addition, Dr. Cicchetti is trained in electrodiagnostic testing, fluoroscopic spine injections, diagnostic and interventional musculoskeletal ultrasound, and in the use of botulinum toxin injections to treat recalcitrant painful muscle spasm. He is an active member of the AAPMR, American College of Sports Medicine, North American Spine Society, International Spine Intervention Society, American Institute of Ultrasound in Medicine (AIUM), and American Association of Neuromuscular and Electrodiagnostic Medicine (AANEM).

A Brief Overview and Update of Myofascial Pain Syndrome and Myofascial Trigger Points*

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Introduction

This paper discusses the definition of a common, but incompletely understood syndrome associated with soft tissue pain, referred to as myofascial pain syndrome (MPS). It begins with a description of the syndrome and its frequently associated finding, the myofascial trigger point (MTrP). The paper describes current published data about biochemical, mechanical, and physical properties of the MTrPs and the surrounding tissue.

There are no proven models explaining the cause of MPS or MTrPs, and the pathophysiology of both is conjectural at this point. Therefore, the majority of the discussion will center around descriptions of the syndrome, hypotheses, and data supporting the etiology based on existing literature and approaches toward treatment of MTrPs and symptom control of myofascial pain.

Description of Myofascial Pain Syndrome and Myofascial Trigger Points

Myofascial pain syndrome (MPS) is a descriptive term used to define an acute or chronic soft tissue musculoskeletal pain condition. It is characterized by sensory, motor, and autonomic findings associated with myofascial trigger points (MTrPs).[1,2] The findings may be local to MTrPs or may be distant, with a referred pain pattern. It often involves the neck and back[1] and has a high prevalence in primary care settings. MPS was diagnosed in 21% of the patients seen in a general orthopedic clinic and 30% of the patients seen at an internal medicine group practice.[3] Myofascial pain is poorly understood and frequently not diagnosed.[4] It is the leading cause of job-related disability and the second leading cause of disability in the US, costing Americans more than $50 billion each year.[5]

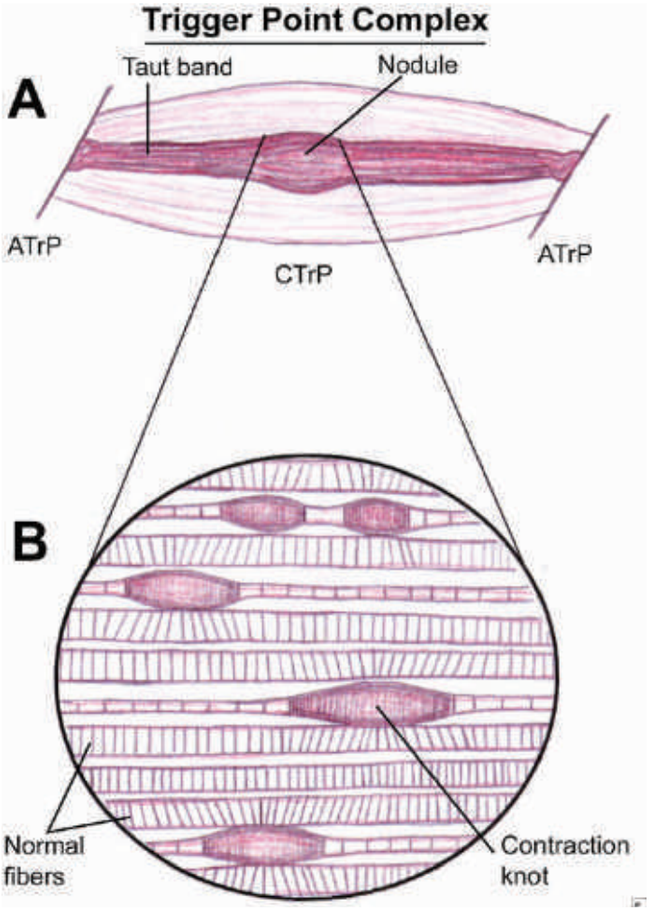

Figure 1. *Image reprinted with permission from Medscape.com, 2011. Available at http://emedicine.com/article/89095-overview.*

Chronic soft tissue pain, of which MPS may be an example, is a pathologic state with a spectrum of clinical signs and symptoms. The experience of pain is a multidimensional process that may include sensory components and perceptions that may result in aversive behaviors, all of which involve activation of different areas of the central and peripheral nervous system. Its origin may be secondary to tissue damage in which there is a lowering of pH and release of histamines and bradykinin locally. C fibre response may be up-regulated peripherally by serotonin, prostaglandins, thromboxane, and leukotrienes as a result of tissue hy-

This research was supported in part by the Intramural Research Program, National Institutes of Health (NIH), and the Clinical Center and Office of the Director, NIH, and by a grant 1R01-AR057348 from the National Institutes of Arthritis and Musculoskeletal and Skin Diseases at the NIH.

poxia and trauma. Substance P may also be released peripherally with resultant increase in peripheral vasodilation and further sensitization of the C fibre's peripheral ending. This may stimulate small, non-myelinated C fibres, generating an electrical impulse which travels to the dorsal horn of the spinal cord. Even chemical products of tissue breakdown may sometimes enter the neuron and be transported centrally to exert an effect at the dorsal horn synapse areas of the central and peripheral nervous system. The condition resulting from the upregulation is ***peripheral sensitization***. By comparison, the International Association for the Study of Pain has defined ***central sensitization*** as: "Increased responsiveness of nociceptive neurons in the central nervous system to their normal or sub-threshold afferent input" and is thought to occur at the dorsal horn.[6] Both central and peripheral sensitization are likely to occur in chronic pain patients. These types of sensitization involve nociception, a phenomenon which results from an actual or potential tissue damaging event transduced and encoded by nociceptors. Nociceptors are sensory receptors that are capable of transducing and encoding noxious stimuli. Persistent symptoms may be the result of peripheral sensitization of nociception. In addition, central sensitization, modulation, and structural modification also play an important role. Signs of peripheral and central sensitization are allodynia (pain due to a stimulus that does not normally provoke pain) and hyperalgesia (an increased response to a stimulus that is normally not painful).

MTrPs are hard, palpable, discrete, localized nodules located within taut bands of skeletal muscle, which are painful on compression. MTrPs can be either active (A-MTrP) or latent (L-MTrP).[1] An A-MTrP is associated with spontaneous pain (pain is present without palpation). This spontaneous pain can be at the site of the MTrP or remote from it. However, firm palpation of the A-MTrP increases pain locally and usually reproduces the subject's remote pain.[7] A L-MTrP is not associated with spontaneous pain, although pain can often be elicited in an asymptomatic subject by a mechanical stimulus such as finger pressure over it.[8] A visible local twitch response (LTR) can be elicited during mechanical stimulation of the MTrP.[1] The LTR is a transient, rapid contraction of a taut band of muscle fibers and is characteristic of MTrPs. A significant number of asymptomatic adults (such as 45% of healthy American Air Force personnel)[9] have L-MTrPs. In someone with a spontaneous pain complaint, thorough palpation of the myofascial tissue is required to identify and differentiate a A-MTrP from a L-MTrP. Pain elicited by palpation of a L-MTrP in a symptomatic subject is qualitatively different from the subject's pain complaint.

MTrPs are highly prevalent in selected populations: 85–93% of patients with chronic pain disorders presenting to specialty pain management centers have MPS.[10] A-MTrPs were the primary source of pain in 74% of 96 patients with musculoskeletal pain seen by a neurologist in a community pain medical center[11] and in 85% of 283 patients consecutively admitted to a comprehensive pain center.[12] MTrPs have been associated with tension-

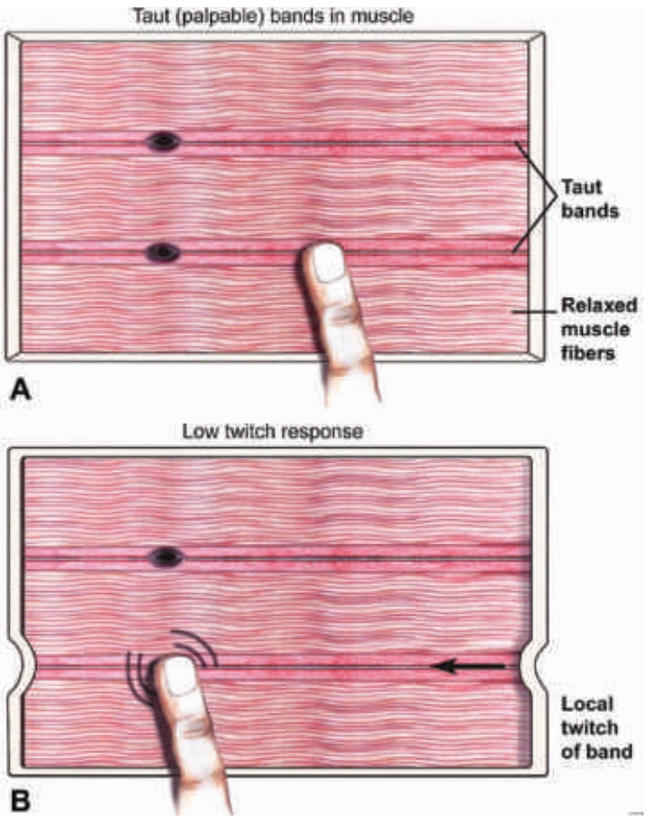

Figure 2. *Image reprinted with permission from Medscape.com, 2011. Available at http://emedicine.com/article/89095-overview.*

type headaches,[14] neck and low back pain,[12] and pelvic pain.[1] A study of 110 adults with migraine headaches showed that 94% of the patients reported migrainous pain with manual stimulation of cervical or temporal MTrPs compared to 29% of controls.[14] MTrPs have been associated with numerous other pain conditions including radiculopathies, joint dysfunction, disc pathology, tendinitis, craniomandibular dysfunction, migraines, carpal tunnel syndrome, whiplash-associated disorders, spinal dysfunction, post-herpetic neuralgia, and complex regional pain syndrome.[1,15]

The Unique Neurobiology of Muscle Pain

Muscle pain has a very unique neurobiology which helps explain its clinical presentation. In contrast to cutaneous pain, muscle pain causes an aching, cramping pain that is difficult to localize and is often referred to deep and distant somatic tissues. Muscle pain activates unique cortical structures in the central nervous system, particularly those which are associated with the affective or emotional components of pain. Muscle pain is inhibited more strongly by descending pain-modulating pathways, and activation of muscle nociceptors is much more effective at inducing maladaptive neuroplastic changes in dorsal horn neurons.[16] These neuroplastic changes are important harbingers of a chronic pain syndrome.

Central sensitization is a hallmark in the transition from normal to aberrant pain perception—i.e., when the central nervous system (CNS) experience of pain outlasts the noxious stimulus coming from the periphery. Peripheral sensitization of group IV afferents in the muscle is especially effective at driving central sensitization. In animal models of pain, nociceptive input from skeletal muscle is much more effective at inducing neuroplastic changes in the spinal cord than noxious input from the skin.[17]

Continuous activation of muscle nociceptors increases the "afferent drive," that is, the impulses per second bombarding dorsal horn neurons in the spinal cord. This may lead to changes in function and connectivity of sensory dorsal horn neurons via central sensitization.[18] This process can spread to adjacent neurons, leading to structural changes and maladaptive neuroplastic alterations in the central nervous system. For example, there may be loss of inhibitory neurons at segmental levels affected by the persistent noxious input.[19] The clinical consequences are

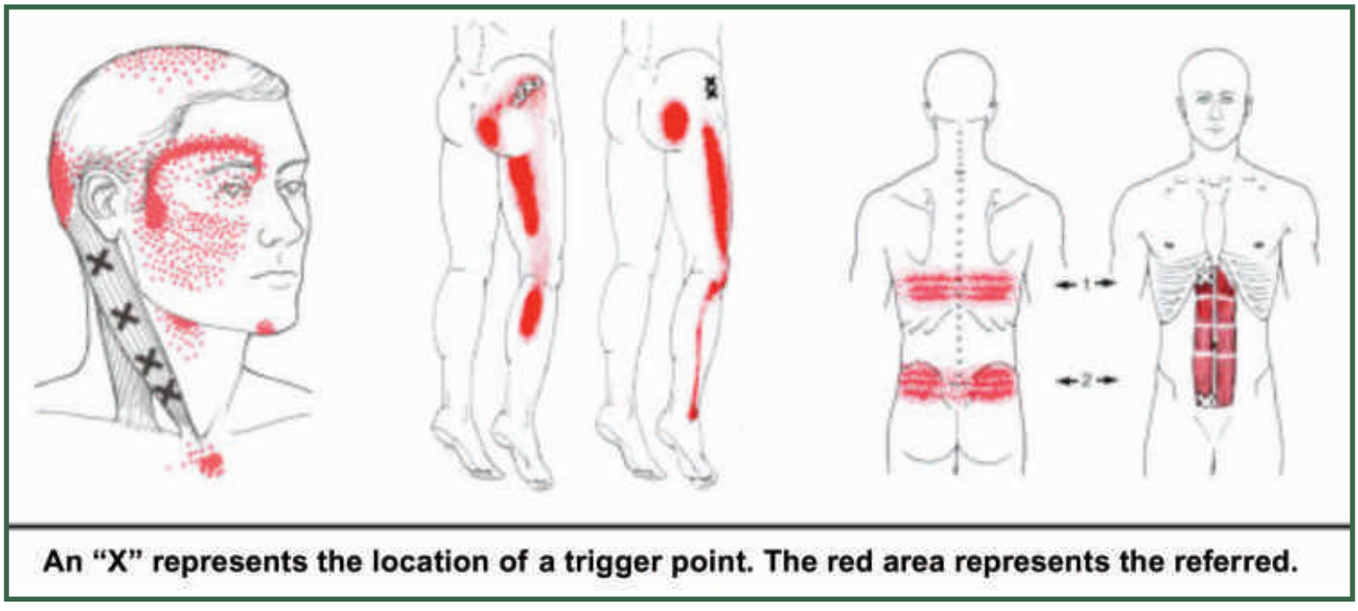

Figure 3. *Image courtesy of www.MyoRehab.net.*

allodynia (pain in response to a normally non-painful stimulus), hyperalgesia (increased sensitivity to pain), and expansion of the receptive field of pain. These clinical signs of central sensitization, which result in an intensified pain experience, are very distressing to patients.

There is a biochemical basis to the development of peripheral and central sensitization in muscle pain. For example, sensitizing agents released in muscle may up-regulate or increase the activity of receptor molecules on the nociceptor terminal. Continuous activation of muscle nociceptors leads to the co-release of substance P and glutamate at the pre-synaptic terminals of the dorsal horn.[20] This can eventually lead to maximal opening of calcium-permeable ion channels, which hyperexcites nociceptive neurons and induces apoptosis of inhibitory neurons. Moreover, prolonged noxious input may lead to long-term changes in gene expression, somatosensory processing, and synaptic connections in the spinal cord and other higher structures.[21] In addition, previously silent synapses may become effective. These mechanisms of peripheral and central sensitization lower the activation threshold of afferent nerves and their central terminals, allowing them to fire even in response to daily innocuous stimuli. Consequently, even non-noxious stimuli such as light pressure and muscle movement can cause pain.

Biochemical and Tissue Properties of MTrPs

Although the specific pathophysiological basis of MTrP development and symptomatology is unknown, several promising lines of scientific study (i.e. histological, neurophysiological, biochemical, and somatosensory) have revealed objective abnormalities.[22–27] It has been observed that MTrPs tend to occur most frequently in Type 1 muscle fibers. Slow motor units are always stiffer than fast units, although fast units can produce more force. Traumatic muscle fiber injury during sustained sub-maximal level exertions could lead to the development of an MTrP. Acute muscle overload can occur with direct impact and lifting injuries. The Cinderella Hypothesis[28] postulates that during low-level static continuous muscle contractions, smaller (type 1) muscle fibers are the first to be recruited and the last to be de-recruited and use only a fraction of motor units available (Henneman's "size principle"). As a result, these "Cinderella" fibers are continuously activated and metabolically overloaded. Accordingly, sub-maximal muscle exertions (e.g., contraction of trapezius muscle during postural maintenance), may cause possible damage to the sarcomere assembly and disturbance of Ca^{2+} homeostasis—features believed to be precursors to the formation of MTrPs and the onset of MPS.

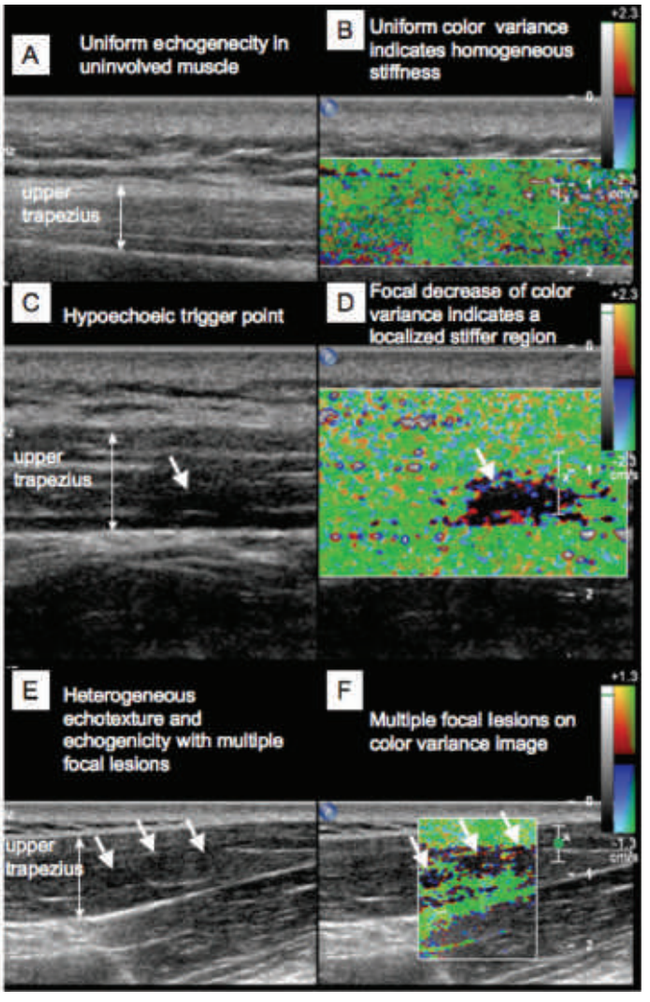

Figure 4. Simultaneous 2D grayscale and color variance imaging (A and B) of normal upper trapezius muscle. The normal muscle appears isoechoic and has uniform color variance (C and D). Muscle with a palpable MTrP. A hypoechoic region and a well-defined focal decrease of color variance indicating a localized stiffer region is visible (E and F). Muscle with a palpable MTrP. Multiple hypoechoic regions and multiple focal nodules are visible. (Sikdar, 2009).

Typical motor abnormalities seen in people with A- and L-MTrPs may be associated with motor weakness and stiffness as a result of restricted range of motion.[29–30] The contribution of the MTrP to this tissue stiffness is currently a very active area for investigations. In the last decade, a new modality for tissue characterization termed Elasticity Imaging (EI) or elastography has emerged. EI is based on generating a stress in the tissue using various static or dynamic means and measuring resulting strain by ultrasound or MRI. There are an increasing number of publications on elastography that have been applied to most organ systems. Magnetic resonance elastography (MRE), which uses a modified gradient echo pulse sequence to image the propagation of induced vibration shear waves, can be used to measure the viscoelastic properties of skeletal muscle. Recently, one study utilized MRE to show that the shear wave propagation pattern in the taut band in the upper trapezius was different compared to palpably normal muscle.[31–32] This study did not specifically identify MTrPs within the taut band but did not exclude this as a possibility. Our group has shown that ultrasound elastography can be used for imaging MTrPs and that muscle surrounding MTrPs appears stiffer on ultrasound scanning.[33]

MTrPs in histological studies in animals are localized contractions of sarcomeres into knots or nodules with disruption of normal fiber structures. Similar morphology can be induced by locally blocking AChE. These resulting lesions have been hypothesized to be similar to MTrPs.[1] One study shows evidence of muscle spindles,[34] while the other shows contraction knots and abnormal muscle fiber contracture.[15] However, biopsy evidence from human studies is very limited.

A key aspect of the Integrated Hypothesis is that muscle fiber contracture at MTrPs can cause capillary constriction, decreasing perfusion and leading to tissue hypoxia. A study of tissue oxygenation in MTrPs using a customized oxygen sensor indicated a focal region of hypoxia[35] at the center of the palpable nodule and a surrounding region of hyperoxia. Histological evidence suggests that MTrPs are sites of tissue distress. Inflammation, hemodynamic stress, and hypoxia, and tissue distress may lead to vascular remodeling[35] in the neighborhood of MTrPs. One investigation has demonstrated that circulatory disturbances secondary to increased intramuscular pressure may also lead to the development of myalgia.[36] The Integrated Hypothesis does not suggest that trauma may be a plausible explanation for the pathophysiology. Nonetheless, small muscle tears, due to persistent contraction has not been ruled out as a contributor to the pathophysiology.

The biochemical conditions associated with this hypothesis asserts that the primary dysfunction is an abnormal increase in the production and release of

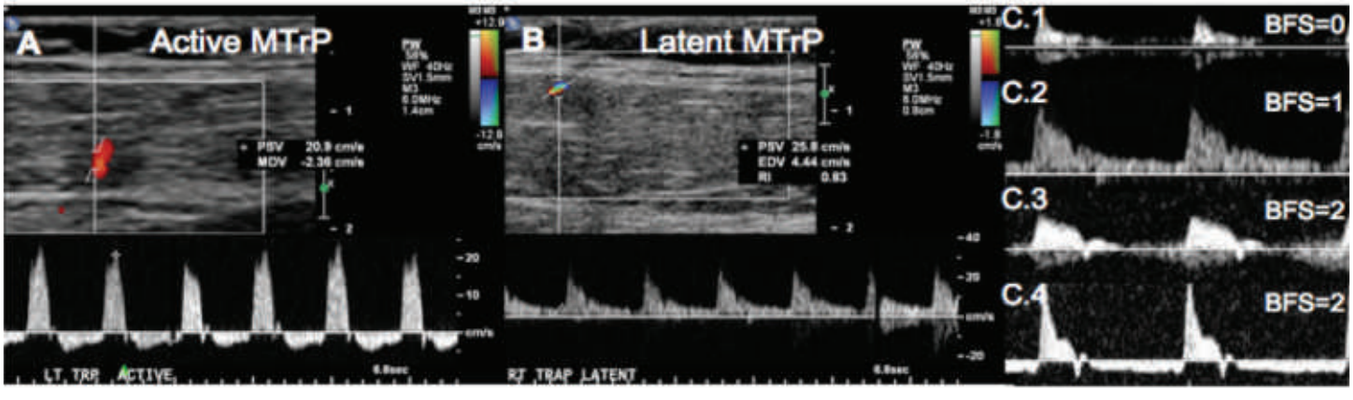

Figure 5. (A) Subject with an active MTrP visible as a hypoechoeic region on the grayscale image (arrow), and an artery running through the MTrP visible on color Doppler. (B) High-resistance blood flow waveform with reverse diastolic flow in the artery through the a-MTrP. (C) The same subject had a latent MTrP on the contralateral side with an artery running through it, which showed no reverse diastolic flow. (Sikdar, 2009).

acetylcholine (ACh) packets from the motor nerve terminal under resting conditions. This sustained depolarization causes the continuous release and inadequate uptake of calcium ions from the local sarcoplasmic reticulum, producing sustained sarcomere contraction and an increase in energy demand. The sustained muscle fiber shortening (contracture) compresses local blood vessels, which reduces the nutrient and oxygen supplies and leads to a local energy crisis. Sensitizing substances, including substance P (SP) and calcitonin gene-related peptide (CGRP) are released, which can interact with autonomic and sensory (some nociceptive) nerves traversing that region. Subsequent release of neurovasoactive substances (bradykinin, prostaglandins, histamine) could, in turn, contribute to excessive ACh release from the nerve terminal, completing a self-sustaining vicious cycle.[1,37]

The vascular environment of trigger points has received some attention from our group, and preliminarily findings have been reported.[33] The results of the blood flow measures, using ultrasound Doppler techniques, significantly associate blood flow disturbances with the pathophysiology of MTrPs.[33]

The observed waveforms of arteries in the neighborhood of MTrPs showed high-resistance blood flow with retrograde diastolic flow in the region of the A-MTrPs. This differed from the blood flow from the surrounding tissue of the L-MTrPs and normal uninvolved myofascial tissue. We believe that an increase in vascular resistance in A-MTrPs is consistent with blood vessel compression due to sustained contracture at or near the trigger point, or there may be vessel constriction due to oxidative stress or hypoxia. The blood vessel compression may be sufficient or one of a number of contributing factors that lead to local hypoperfusion or hypoxia. Ischemic tissue is often associated with pain, tenderness, and nodularity of an A-MTrP. The retrograde diastolic flow suggests a substantial vascular volume upstream of the constriction, where the blood accumulates in systole and is emptied retrograde in diastole since the antegrade path is obstructed. This is consistent with vascular remodeling in the neighborhood of active MTrPs.

The transformation of a tender nodule into a myofascial pain syndrome is poorly understood. However, local muscle pain is known to be associated

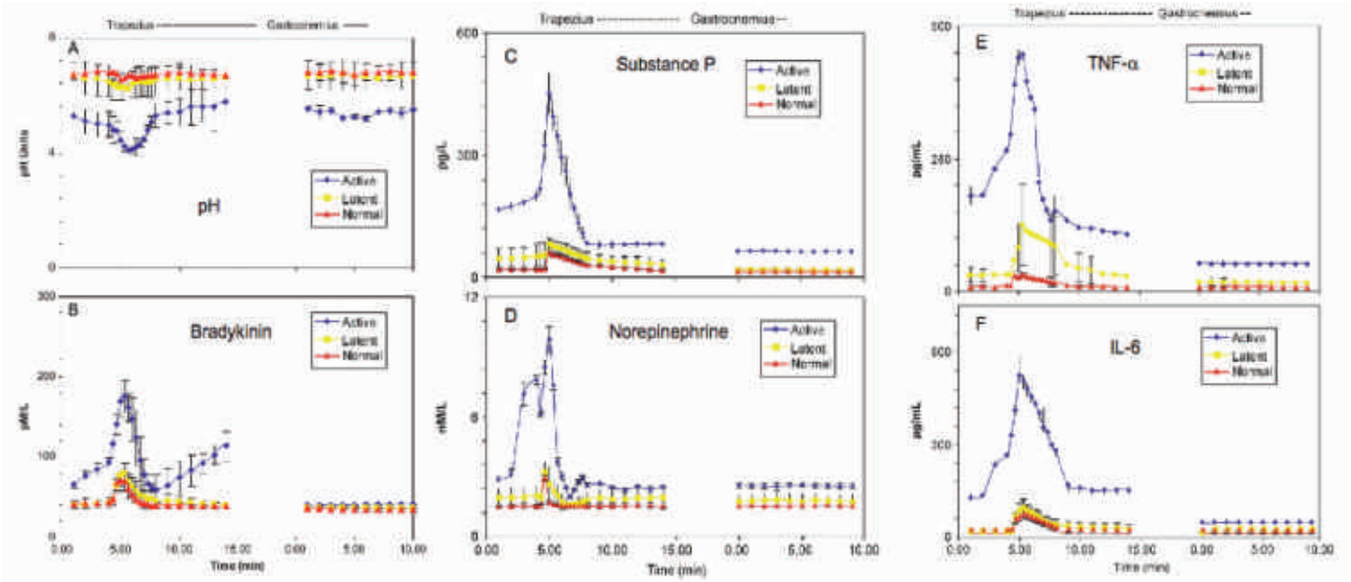

Figure 6. Biochemical analyte levels in active, latent and normal trigger points in the upper trapezius and at control locations in gastrocnemius. (Shah, et al, 2005).

with the activation of muscle nociceptors by a variety of endogenous substances including neuropeptides, arachidonic acid derivatives, and inflammatory mediators, among others.[38] Recent biochemical studies by our investigative group (39, 27) using a microdialysis technique confirmed that patients with A-MTrPs in the upper trapezius have significantly elevated levels of protons, bradykinin, pro-inflammatory cytokines (tumor necrosis factor (TNF)-α, interleukin(IL) 1-β, interleukin(IL)-6, interleukin(IL)-8), neuropeptides (CGRP, substance P), and catecholamines (serotonin, and nor-epinephrine) within the local milieu of the A-MTrP compared to those with a L-MTrP or normal tissue.

We did not assay for possible contributors of the arachadonic pathway in prior work nor did we assay for cellular changes. The reduced oxygen levels in A-MTrP, and increased metabolic demand results in a local energy shortage and a local shortage of ATP.[39] Under normal physiologic circumstances, ATP at presynaptic membranes of the motor neuron inhibits the release of acetylcholine (ACh). Decrease in ATP leads to increased ACh release and prolonged muscle contraction. Moreover, insufficient ATP at the motor endplate results in a failure of the calcium pump, increased levels of sarcoplasmic Ca^{2+}, and a Ca^{2+}–induced Ca^{2+} release from the sarcoplasmic reticulum, which further reinforces sarcomere contractures.

Calcitonin gene-related peptide can enhance the release of ACh from the motor endplate and simultaneously decrease the effectiveness of acetylcholinesterase (AChE) in the synaptic cleft, which decreases the removal of AChE.[40,41] Calcitonin gene-related peptide also upregulates the ACh-receptors (AChR) at the muscle and thereby creates more docking stations for ACh. Miniature endplate activity depends on the state of the AChR and on the local concentration of ACh, which is the result of ACh-release, reuptake, and breakdown by AChE. In summary, increased concentrations of CGRP lead to a release of more ACh, and increase the impact of ACh by reducing AChE effectiveness and increasing AChR efficiency. Miniature endplate potential frequency is increased as a result of greater ACh effect. The observed lowered pH has several implications as well. Not only does a lower pH enhance the release of CGRP, it also contributes to a further down-regulation of AChE. The multiple chemicals and lowered pH found in active MTrPs can contribute to the chronic nature of MTrPs, enhance the segmental spread of nociceptive input into the dorsal horn of the spinal cord.

Treatment

Current approaches for pain relief of MPS include pharmacological and non-pharmacological interventions. Anti-inflammatory, analgesic, and narcotic medications have been used for symptomatic control. Non-pharmacological interventions have been used for decades among a broad based group of investigators.[1,2] Specifically, these have included manual therapies, massage, spray and stretch techniques, among others.[42,43] A recent publication has shown the effectiveness of manual therapies in a controlled, blinded, single-assessor controlled trial.[44]

A frequent practice, which has approached acceptance as "standard of practice", is the use of soft tissue needling. This technique involves the use of a small, 30 gauge needle, and the infiltration of a small amount of anesthetic and/or steroid, or the use of needling without injection and massage therapy. The lack of objective clinical outcome measures has been a barrier for critically evaluating the efficacy of these therapeutic methods. All of these factors have led to a lack of consensus on myofascial pain as a clinical entity and have contributed to the uncertainty about the pathogenesis and pathophysiology of trigger points. Therefore, there is a need to develop objective, repeatable, and reliable diagnostic tests for evaluation and treatment outcome measures for MTrPs. Such measures can be used to properly diagnose and understand the natural history of MTrPs and to determine the underlying mechanisms and relevance to the development and resolution of myofascial pain.

Gerwin and Dommerholt have written extensively on the evaluation of several treatment options includ-

ing trigger point injections and dry needling. Local anesthetics (e.g. procaine, lidocaine, bupivacaine), isotonic saline, nonsteroidal anti-inflammatories, bee venom, and botulinum toxin have all been studied as potential injectables. Studies have found that 0.25% lidocaine is an effective therapy while nonsteroidal anti-inflammatory medication, steroids, and vitamin B_{12} are not as effective.[45] Although serotonin antagonists are not available in some locations throughout the world, tropisetron, a serotonin antagonist, has been found to be more effective than lidocaine in two German studies.[46,47]

Advancing a needle into a trigger point that does not have a lumen is a technique called dry needling. Evidence was first introduced[48] about the use of dry needling techniques as a clinical approach by Lewit in 1979.[49] It was demonstrated to produce analgesia in 87% of subjects, many of whom had lasting effects. The mechanism by which this was thought to work was a mechanical stimulation. Clinicians using this technique have attempted to produce a twitch response, which is an involuntary spinal cord reflex of the muscle fibers in the taut band. This twitch can be seen, palpated, and recorded on an oscilloscope if performed using electromyography. Our group has demonstrated that there is a rapid change in the biochemical milieu following the twitch and that this change restores the surrounding biochemical (cytokines, neuropeptides, and catecholamines) to a profile that is consistent with that of L-MTRPs and normal tissue.[27] In particular, there is a significant drop in substance P and calcitonin gene-related peptide, providing evidence supporting the role of these two substances in MPS.[27]

The additional information about biochemicals in the surrounding milieu of the MTrP does not provide adequate information to establish a mechanism by which dry needling works to relieve pain. The proposed mechanism involving stimulation of Aδ sensory afferent fibers are has not been supported by all biochemical research findings.[39] Hormones, neuropeptides, and cytokines other than those reported by our team[39] may play an important role in pain initiation and persistence, such as opioids and oxytocin, but these have not been conclusively demonstrated.

Despite the absence of a proven mechanism by which dry needling works, and despite the lack of a mechanism explaining the development of TrPs, clinical practice and some clinical trial evidence[44,48] have generated evidence for the use of this approach in an ever expanding group of practitioners.

Summary

The published data on the prevalence and clinical presentation and impact on function of MPS and MTrPs indicate that this syndrome is of concern to patients and practitioners. However, there also a common physical finding in asymptomatic individuals. This dichotomy challenges and behooves pain management practitioners to learn how to distinguish active from latent MTrPs. Making this distinction is critical in order to accurately identify and treat a myofascial component of pain.

Fortunately, advances in the field have enabled us to better describe and physically characterize the trigger point and its surrounding milieu using imaging, in particular ultrasound, and microanalytic approaches. These are likely to serve as objective, reliable, and sensitive measures for diagnosis and for measuring treatment efficacy. Future research will be needed to identify the pathophysiology and etiology of the syndrome, enabling us to target treatments toward prevention, early intervention, and effective treatments.

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Dr. Gerber, Professor of Global and Community Health and Director of the Center for Study of Chronic Illness and Disability, is responsible for developing a research program to help describe the mechanisms by which disease produces disability and explore treatments that can prevent or reduce disabilities and restore function. Dr. Gerber served as Chief of the Rehabilitation Medicine Department (RMD) in the Clinical Center of National Institutes of Health (NIH), a position held from 1975–2005. In this capacity, she assured the quality of Physical Medicine & Rehabilitation (PM&R) service for all referred NIH patients with impairments & disability. She is board certified in internal medicine, rheumatology, and PM&R. Much of her clinical research interest has been centered on measuring and treating impairments and disability in patients with musculoskeletal deficits; in particular, children with osteogenesis imperfecta, and persons with rheumatoid arthritis and cancer. Dr. Gerber now serves on the Board of Governors of the Academy of Physical Medicine and Rehabilitation. Dr. Gerber has authored/co-authored 90 peer reviewed, published manuscripts and 45 Chapters in major textbooks (Internal Medicine, Rheumatology, Cancer, Rehabilitation et al.)

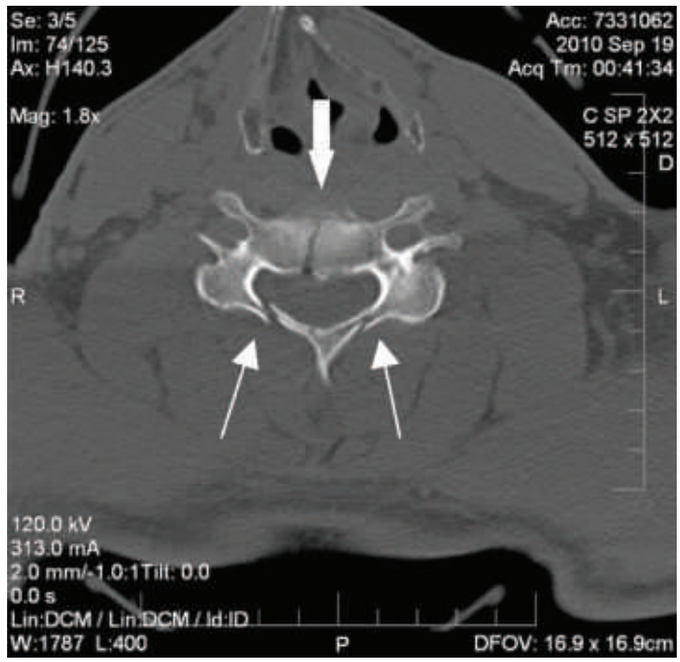
Halo Brace

Brian R. Subach, M.D., F.A.C.S.
Diana DeWolfe, PA-C, M.A.C.S.

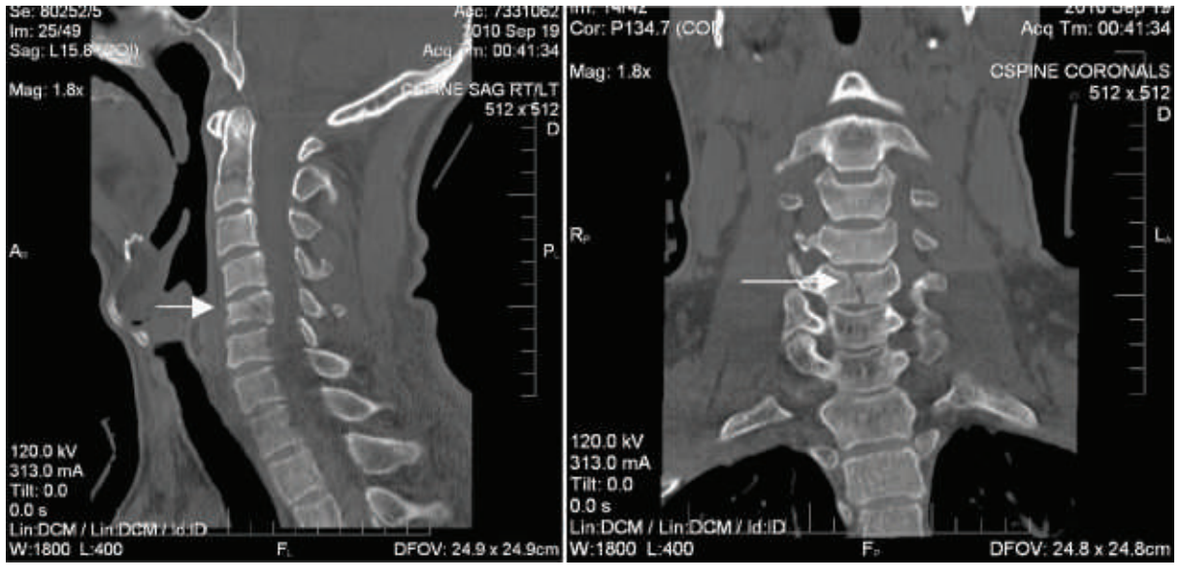
This 41-year old gentleman arrived at our office wearing a Halo-vest device and complaining of neck pain with pain and numbness into both arms. He described his traumatic neck injury of three weeks earlier. He went to play volleyball at the beach, but a hurricane off-shore caused the beach to flood, ending the volleyball game. He and several friends began body surfing in the shallow water when he struck his head on a sandbar. He recalled neck pain and the immediate inability to use his arms or legs.

Mechanism of hyper-flexion injury (Courtesy Medtronic).

Unable to lift his face out of the water, he nearly drowned. Fortunately, his friends recognized that there was a problem and rescued him. Emergency medical services arrived, placed him in spinal immobilization (cervical collar, spine board), and flew him immediately to a university medical center. Once there, he was evaluated by several physicians and diagnosed with a spinal cord injury due to a burst fracture of the C5 (fifth cervical) vertebrae.[1]



Axial CT scan showing burst fracture of the vertebral body (thick arrow) and both lamina of C5 (thin arrows).

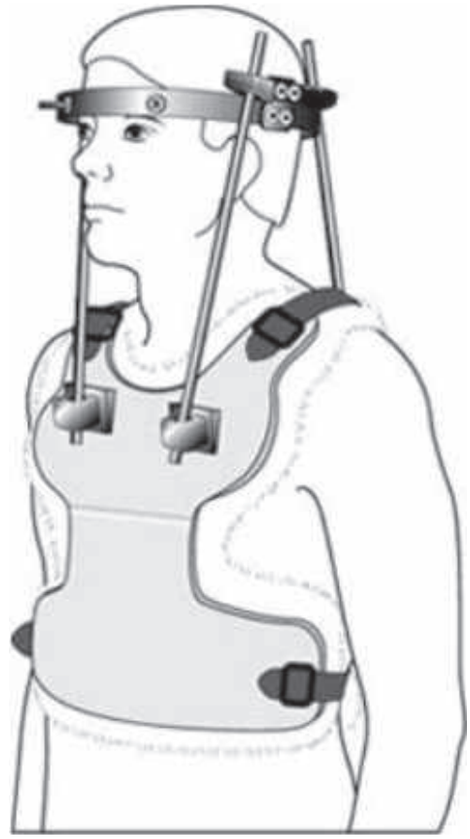


Sagittal (left) and coronal (right) CT reconstructions of the cervical spine showing a widened and flattened C5 vertebral body (left image, arrow) with a vertical fracture line (right image, arrow).

CASE REPORT

He was advised to avoid surgery and instead was placed in a Halo-vest device to stabilize his fracture.[2] The ring portion of this device has four screws which are inserted into the skull, and four vertical rods holding the ring to a padded plastic vest. Prior to discharge from the hospital, he started physical therapy and began recovering the use of his arms and legs. At discharge, he was walking, but was left with residual weakness in the arms and numbness throughout the torso and extremities.

On physical examination, he demonstrated brisk reflexes in the upper extremities bilaterally (left greater than right) with significant weakness in the left arm, compared to the right. Wasting of the muscles and decreased sensation was noted in the left arm as well. The placement of the Halo was noted to cause a fixed tilt of his neck to the left.



The Halo-vest. (Courtesy of UWHC).

Medical Management

The decision making process in this treatment is based upon two concepts: spinal balance and risk to the spinal cord. Spinal balance refers to the normal alignment of the neck when viewed from the front and side on x-rays. Spinal cord injuries are classified as either complete (absence of movement and sensation below the level of the spinal cord damage), or incomplete (some preservation of movement or function below the level of spinal cord injury). A third concern in many situations is the presence of other systemic injuries. For example, in the case of a cervical fracture with chest wall trauma (broken rib), a Halo-vest would be a painful treatment option due to direct pressure on the chest wall and would clearly limit the ability for the patient to take deep breaths, increasing the risk of pneumonia. In such situations, surgery may be a better solution for a cervical fracture rather than the Halo immobilization. On the other hand, surgery may not be appropriate for a patient with a severe closed-head injury. Often the blood pressure changes that occur with general anesthesia could lead to a stroke or hemorrhage in the brain in a person who has coexisting trauma.

In this specific patient's case, the spinal trauma team on call for the emergency room made their assessment. His cervical burst fracture was causing a forward flexed posture, known as kyphosis, with continued compression of the spinal cord. He was placed in a Halo-vest to immobilize him for a period of three months. Halo-vest immobilization essentially places four pins into the outer surface of the skull. The pins secure to a circular ring which is affixed to the head. The ring is then connected to a plastic "clamshell" (a plastic jacket placed circumferentially around the torso) via four vertical rods. In essence, the Halo-vest bridges head to torso while externally stabilizing the fractured neck. Typically, a minimum three-month period is advised. The Halo-vest does little to correct posture and does absolutely nothing to decompress the spinal cord.

There are numerous reports of the adverse effects associated with this type of immobilization.[3–6] Such complications include infection of the scalp from the skull pins, possibly leading to bony infection of the skull,[7] pressure sores from the vest itself, pneumonia, swallowing difficulties,[8] nonunion of the fracture, loss

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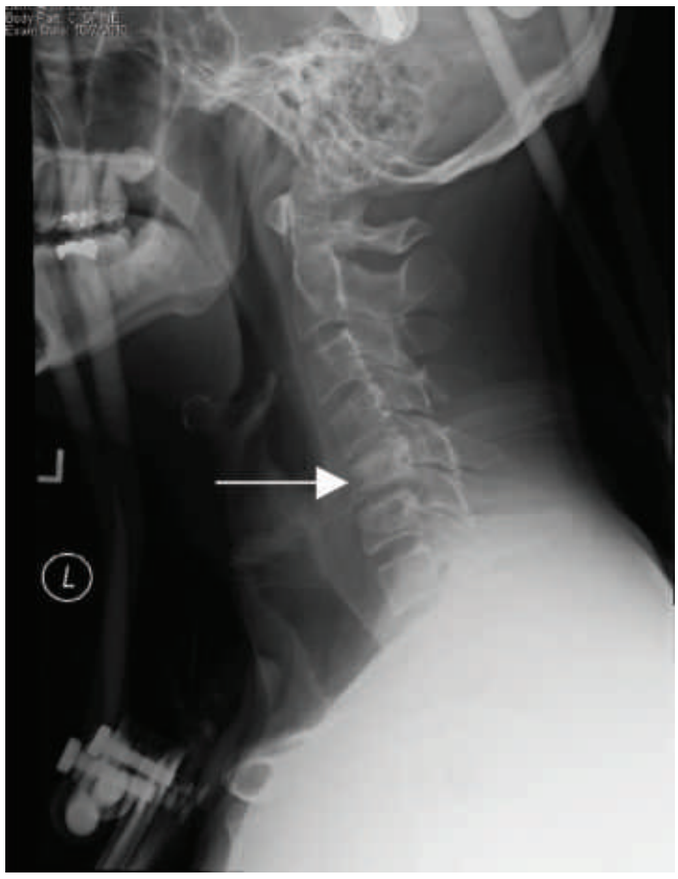
of spinal alignment, and the hygienic difficulties associated with wearing a device around the torso for three months without the ability to remove it for bathing.

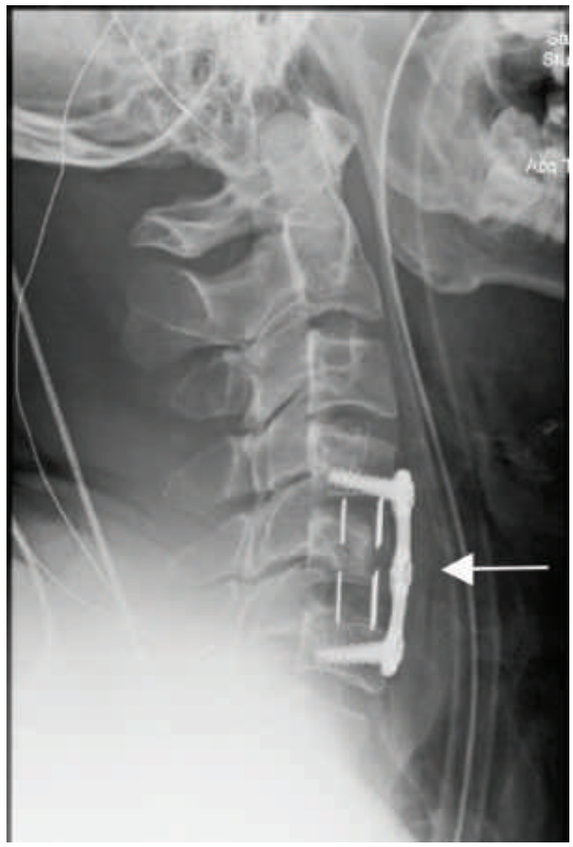
When the patient arrived in our office, he had been in the Halo-vest for approximately four weeks. He continued to have symptoms of numbness and weakness in his arms. His legs were stronger than his arms, but his balance was clearly poor. On MRI and x-ray imaging studies, his cervical posture was bent forward secondary to the fracture and the C5 burst fracture was clearly causing continued compression of the spinal cord. We discussed a more efficient treatment plan:[9] removal of the Halo-vest followed by removal of the broken cervical vertebra, decompression of the spinal cord, reconstruction of the vertebral bodies with a polymer implant, and fixation of a plate across the front of his spine.[10]

Under general anesthesia, the patient underwent a three-hour procedure to reconstruct his spine and remove the Halo. Throughout the operation, he was monitored with Evoked Potential Testing (electrodes in the scalp sending electrical shocks to electrodes placed in the arms and legs) to keep an eye on his spinal cord function during surgery. He awoke from the surgery moving his arms and legs, with only a soft collar on his neck for comfort.

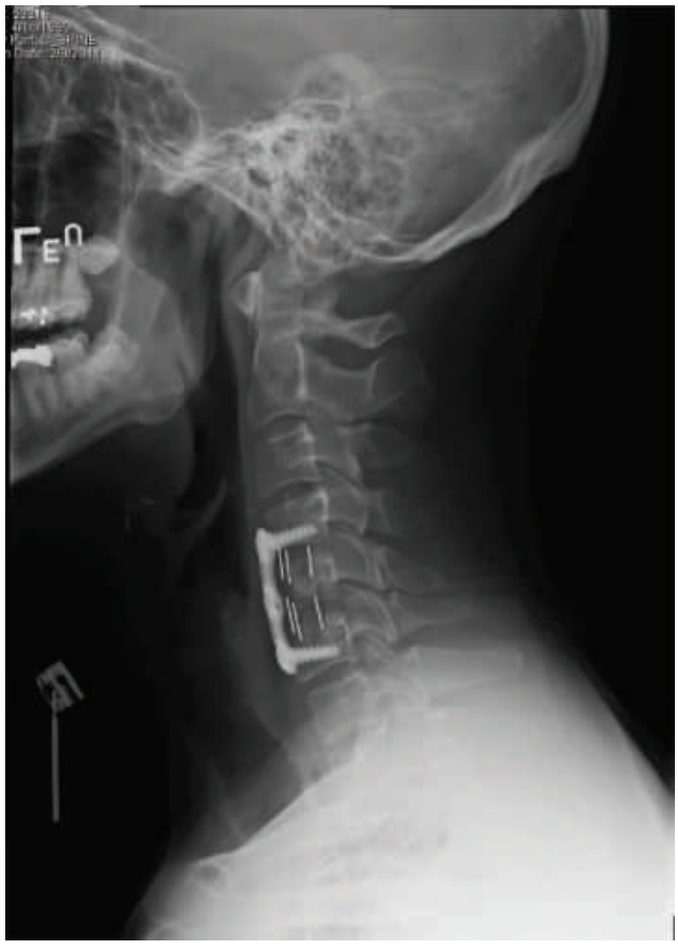
The reason for presenting this case report is to discuss the decision-making process in treating cervical spinal fractures, which are extremely common in the United States. The important elements of the decision-making are: the extent of injury, coexisting traumatic injuries, spinal alignment, spinal balance, and compression of the spinal cord. In this case, surgery seemed the better option.



Pre-operative x-ray showing kyphosis (forward curvature) of the neck at the level of the fracture.



Lateral X-ray taken during the surgery, showing a spacer replacing the fractured C5 vertebrae (dashed lines) and plate-screw fixation along the front of the spine (arrow).



Lateral X-ray 2-weeks after surgery showing corrected alignment of the neck.

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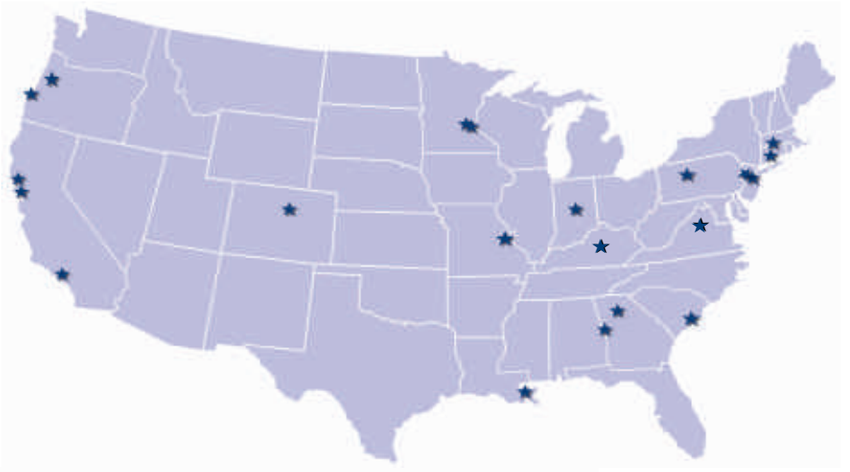
The Spinal Research Foundation has named 23 Regional Research Affiliates across the country that share one core mission: improving spinal health care for the future. These centers offer the best quality spinal health care while focusing on research programs designed to advance spinal treatments and techniques.



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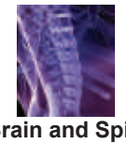

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Neck and Back Pain Affects Millions

The Spinal Research Foundation has made remarkable progress in scientific research associated with neck and back pain. Located in Reston, Virginia, the Foundation collects data relative to patients' treatments and outcomes and has embarked on projects designed to better understand the biochemistry of neuropathic pain and develop new drug and surgical regimens to address it. The Foundation continues to expand its research efforts, partnering with other research institutions to further the advancement of spine related research. The Spinal Research Foundation has been involved in numerous studies:

- ***The use of novel perioperative drug therapy to improve surgical outcomes.***
- ***The evaluation of medical devices for the relief of back pain.***
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- ***The development of non-operative techniques to resolve disabling neck and back pain.***
- ***Investigating the use of BMP (Bone Morphogenetic Protein) in minimally invasive spinal surgery to minimize post-operative pain and dysfunction.***
- ***The development of cervical and lumbar disc replacement technologies.***
- ***The development of disc regeneration technology through the use of stem cells derived from the bone marrow.***
- ***The investigation of lactic acid polymers to prevent fibroblast in-growth in surgical wounds.***
- ***A nation-wide multi-center prospective spine treatment outcomes study.***

The Spinal Research Foundation is a non-profit organization dedicated to improving spinal health care through research, education, and patient advocacy. The Foundation collaborates with spinal research centers of excellence around the nation to prove the success of traditional approaches, as well as develop new techniques and technologies. These results are shared with both the medical profession and the general public to improve the overall quality and understanding of optimal spinal health care.

More than 85% of the population will suffer from severe neck and/or low back pain during their lifetime. Eight percent of these people develop chronic pain, which means that at any given time, 25 million people in the United States are directly affected by this condition and many more indirectly. Techniques to cure, manage, and prevent this limiting and disabling condition need to be developed. Educating the public, health care providers, and insurance providers is the first step in advancing spinal health care.

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