Low Back Disorders

Effective: February 13, 2020

CONTRIBUTORS TO THE LOW BACK DISORDERS GUIDELINE
Editor-in-Chief:
Kurt T. Hegmann, MD, MPH, FACOEM, FACP

Evidence-based Practice Spine Panel Chair:
Russell Travis, MD

Evidence-based Practice Spine Panel Members:
Roger M. Belcourt, MD, MPH, FACOEM
Ronald Donelson, MD, MS
Marjorie Eskay-Auerbach, MD, JD
Jill Galper, PT, MEd
Michael Goertz, MD, MP
Scott Haldeman, MD, DC, PhD, FRCP(C), FAAN, FCCS
Paul D. Hooper, DC, MPH, MS
James E. Lessenger, MD, FACOEM
Tom Mayer, MD
Kathryn L. Mueller, MD, MPH, FACOEM
Donald R. Murphy, DC
William G. Tellin, DC, DABCO
Michael S. Weiss, MD, MPH, FACOEM, FAAPMR, FAANEM

Panel Consultant:
Cameron W. MacDonald, PT, DPT, GCS, OCS, FAAOMPT

These panel members represent expertise in neurology, neurosurgery, neurophysiology, occupational medicine, orthopedic surgery, pain medicine, physical medicine and rehabilitation, chiropractic medicine, family practice, and physical therapy. As required for quality guidelines – Institute of Medicine's (IOM's) Standards for Developing Trustworthy Clinical Practice Guidelines and Appraisal of Guidelines for Research and Evaluation (AGREE) – a detailed application process captured conflicts of interest. The above Panel has none to declare relevant to this guideline.

Methodology Committee Consultant:
Jeffrey S. Harris, MD, MPH, MBA, FACOEM

Research Conducted By:
Kurt T. Hegmann, MD, MPH, FACOEM, FACP
Jeremy J. Biggs, MD, MSPH
Matthew A. Hughes, MD, MPH
Matthew S. Thiese, PhD, MSPH
Ulrike Ott, PhD, MSPH
Emilee Eden, BS, MPH
Harkomal Kaur, BS
Jenna K. Lindsey, BS
Michael L. Northrup, BS
Skyler D. Walker, BS
Chapman B. Cox
Vivian Nguyen
Jenny Dang
Weijun Yu, BM, BA, MS
Atim Effiong, MPH
Kristine Hegmann, MSPH, CIC
Alzina Koric, MPP
Brenden Ronna, BS
Austen J. Knudsen
Pranjal A. Muthe
Leslie MC Echeverria, BS
Jeremiah L. Dortch, BS
Ninoska De Jesus, BS
Zackary C. Arnold, BS
Kylee F. Tokita, BS
Katherine A. Schwei, MPH
Deborah G. Passey, MS
Holly Uphold, PhD
Evelyn Graham
Louise Juliet

Specialty Society and Society Representative Listing:
ACOEM acknowledges the following organizations and their representatives who served as reviewers of the Low Back Disorders Guideline. Their contributions are greatly appreciated. By listing the following individuals or organizations, it does not infer that these individuals or organizations support or endorse the low back treatment guidelines developed by ACOEM. Additional organizations wish to remain anonymous.

American Academy of Family Physicians
David O'Gurek, MD

American Academy of Neurology
J.D. Bartleson, MD

American Academy of Orthopaedic Surgeons
Ryan Carter Cassidy, MD

American Academy of Physical Medicine & Rehabilitation

American Association of Neurological Surgeons/Congress of Neurological Surgeons Section on Disorders of the Spine and Peripheral Nerves
John E. O'Toole, MD, MS

American Board of Independent Medical Examiners
Mohammed Ranavaya, MD, JD, MS, FRCPI

American Chiropractic Association
Michele Maiers, DC, MPH, PhD

American College of Emergency Physicians
Joshua Broder, MD
Stephen V. Cantrill, MD, FACEP

The American Occupational Therapy Association, Inc.
Jeff Snodgrass, PhD, MPH, OTR

American Physical Therapy Association

American Psychological Association
Daniel Bruns, PsyD FAPA

California Orthopaedic Association

Society for Acupuncture Research
Vitaly Napadow, PhD
Claudia M. Witt, MD, MBA

Other Reviewers:
Steven Hwang, MD
Howard A. King, MD
Steven Mandel, MD

Introduction

The Low Back Disorders treatment guideline is designed to provide health care providers who are the primary target users of this guideline with evidence-based guidance on the treatment of working-age adults with low back disorders whether acute, subacute, chronic, or post-operative. While the primary patient population target is working adults, it is recognized the principles may apply more comprehensively. This guideline does not address several broad categories including congenital disorders or malignancies. It also does not address specific intra-operative procedures.

Objectives of this guideline include evaluations of baseline evaluation, diagnostic tests and imaging, physical activity, return to work, medications, physical therapy, cryotherapy, heat therapies, electrical therapies, manipulation, acupuncture, injections, operative procedures, and rehabilitation. Comparative effectiveness is addressed where available. This guideline does not address comprehensive psychological and behavioral aspects of pain management as those are addressed in the ACOEM Chronic Pain guideline. It is recognized that there are differences in workers' compensation systems.(1) There also are regional differences in treatment approaches.(2-4) The Evidence-based Practice Spine Panel and the Research Team have complete editorial independence from the American College of Occupational and Environmental Medicine and Reed Group which have not influenced the guidelines. The literature is routinely monitored and searched at least annually for evidence that would overturn this guidance. The guideline is planned to be comprehensively updated at least every five years, or more frequently should evidence require it. The health questions for acute, subacute, chronic, and post-operative low back disorders addressed by this guideline include:

- What evidence supports the initial assessment and diagnostic approach?
- What red flags signify serious underlying condition(s)?
- What diagnostic approaches and special studies identify clinical pathology?
- What initial treatment approaches have evidence of efficacy?
- What is the evidence of work-relatedness for various diagnoses?
- What modified duty and activity prescriptions and limitations are effective and recommended?
- When is return to work status recommended?
- When initial treatment options fail, what evidence supports other interventions?
- When, and for what conditions are injections and other invasive procedures recommended?
- When, and for what conditions is surgery recommended?
- Which surgeries are recommended for which conditions?
- What management options are recommended for delayed recovery?

A detailed methodology document used for guideline development including evidence selection, scoring, incorporation of cost considerations,(5, 6) and formulation of recommendations is available online as a full-length document(7) and also summarized.(8, 9) All evidence in the prior low back disorders guidelines garnered from 7 databases was included in this guideline (Medline, EBM Online, Cochrane, TRIP, CINAHL, EMBASE, PEDro). Additionally, new comprehensive searches for evidence were performed with both Pubmed and Google Scholar up through 2018 to help assure complete capture. There was no limit on year of publication. Search terms are listed with each table of evidence. Guidance is developed with sufficient detail to facilitate assessment of compliance(5) and auditing/monitoring.(6) Alternative options to manage conditions are provided.

This guideline has undergone extensive external peer review. The only AGREE(6) and IOM criteria(5) not adhered to is incorporation of the views of the target population. Neither patients with low back pain nor other affected patient groups were involved. In accordance with the IOM's Trustworthy Guidelines, detailed records are kept, including responses to external peer reviewers.(5)

Impact

It is estimated that 60 to 80% of the general population will experience an episode of low back pain (LBP) during their lifetime.(10, 11) The annual prevalence rate is between 25 and 60%.(12) LBP recurrence rates reportedly range from 24 to 80%.(13, 14) Low back disorders are the most frequent problems presented to health care providers. Back injuries are among the most common causes of reported occupational disorders with an incidence rate of 20 per 10,000 full-time workers and an average of 7 days away from work per injury.(15) In addition, low back disorders are disproportionately expensive, accounting for 10 to 33% of workers' compensation costs.(16-18) Occupationally related back pain has a national direct annual cost of $10.8 billion (US). However, this estimate is overly conservative as it does not include the indirect cost to employers who must rehire and retrain replacement workers, the loss of productivity, reduced quality work, administrative costs, and losses to the patient and patient's family (including productivity at home). Finally, it does not take into account those workers who do not file for disability, but nonetheless experience the effects of LBP.(19)

Overview

Recommendations on assessing and treating adults with low back problems are presented herein. Topics include the initial assessment and diagnosis of patients with acute, subacute, and chronic radicular and non-radicular low back disorders, identification of red flags that may suggest the presence of a serious underlying medical condition, initial clinical and mechanical evaluation, management, diagnostic considerations and special studies to identify clinical pathology, work-relatedness, modified duty and activity, and return to work, as well as further management considerations including delayed recovery. In accordance with the most common classification, LBP is categorized as acute (<1 month duration), subacute (1 to 3 months duration), and chronic (>3 months duration).[i]

Algorithms for patient management are included. This guideline's master algorithm schematizes how practitioners may manage acute, subacute, or chronic low back disorders. The text, tables, and numbered algorithms expand upon the master algorithm.

As there are few studies that primarily evaluated patients with work-related back disorders,[ii] studies that include broader populations of adults were necessarily used to develop the recommendations. In addition, most studies that focus on pharmaceuticals, appliances, and specific devices are industry-sponsored. In certain areas, this may have made little difference as the comparisons were between the medication and placebo and the results may be consistent and considerable. However, in other studies, the comparison groups may have been suboptimally treated (e.g., with low-dose of ibuprofen) and produced a bias in favor of the medication or device. In addition, industry-sponsored studies have been shown to frequently have better results and lower complication rates than studies conducted by independent investigators.(20-22) There are several widely used highly remunerative injections and

---

[i]When a study used a different classification, those articles were grouped into one or more of these three categories for purposes of uniformity.

[ii]Many studies do not describe the work status of the patients included. Many other studies excluded those with workers' compensation claims.

invasive procedures with sparse studies without significant replication. These are also concerning for potential biased reporting. High-quality studies of physical modalities and delayed recovery are methodologically challenging and thus scant. They commonly suffer from methodological weaknesses (e.g., unblinded, multiple co-interventions, non-standardized techniques) that necessarily limit the strength of conclusions.

Summary of Recommendations and Evidence

The following is a summary of many of this guideline's recommendations:

- The initial assessment of patients with low back problems focuses on detecting indications of potentially serious disease, termed "red flags" (i.e., fever or major trauma).
- In the absence of red flags, the focus should begin and remain on functional recovery.
- At the first visit, the patient should be assured that LBP is normal, has an excellent prognosis and, in all but rare cases, is not debilitating on a long-term basis. Patients with elevated fear avoidance beliefs may require additional instructions and interventions to be reassured of this prognosis. Those reassurances are thought to reduce the probability of the patient developing chronic pain syndrome.
- To avoid undue back symptoms and debilitation from inactivity, some activity or job modification may be helpful in the acute period. However, bed rest is not recommended for essentially all LBP and radiculopathy patients other than those with unstable fractures or cauda equina syndrome with pending neurological catastrophe. Maintaining ordinary activity as much as possible leads to the most rapid recovery.
- Patients should be encouraged to return to work as soon as possible as evidence suggests this leads to the best outcomes. This process may be facilitated with temporary modified (or alternative) duty particularly if job demands exceed patient capabilities. Full-duty work is a reasonable option for patients with low physical job demands and/or the ability to control such demands (e.g., alternate their posture) as well as for those with less severe presentations.
- An early mechanical evaluation using repeated end-range test movements to determine the presence or absence of a directional preference and pain centralization has been shown to guide directional exercise treatments that are associated with better outcomes.
- Appropriate adjustment of physical activity if needed, an exercise prescription, non-prescription medication or an appropriately selected nonsteroidal anti-inflammatory drug (NSAID), and the use of thermal modalities such as heat and/or cryotherapies may be helpful in relieving discomfort.
- In the absence of red flags, imaging and other tests are not recommended in the first 4 to 6 weeks of low back symptoms as they are highly unlikely to result in a meaningful change in clinical management.
- "Abnormal" findings on x-rays, magnetic resonance images, and other diagnostic tests are so common they *are normal by age 40*. Studies, if repeated today, would likely reduce that age for normal findings as obesity is associated with degenerative findings on imaging studies.(23-25) Bulging discs also continue to increase after age 40, and by age 60 will be encountered in 70 to 80% of patients. This requires that a careful history and physical examination be conducted in order to correlate historical, clinical,(26) and imaging findings prior to assigning the finding on imaging to a patient's symptoms. It is recommended that those providers unable to make those correlations, and thus properly educate patients about these complex issues, should defer ordering imaging studies to a qualified consultant in musculoskeletal disorders. Without proper education on prevalence, treatment, and prognosis, patients may become focused on "fixing" their abnormality (which may be

a completely normal finding) and thus iatrogenically increase their risk of developing chronic pain and needless debility.

- Among the modes of exercise, aerobic exercise has the best evidence of efficacy, whether for acute, subacute, or chronic LBP patients.
- Non-specific stretching is not recommended as it is not helpful for treatment of LBP. However, specific types of stretching exercises appear helpful (e.g., directional and slump stretching). Strengthening exercises, including lumbar stabilization exercises, are recommended, but not until the acute period of LBP has sufficiently subsided.
- Many invasive and noninvasive therapies are intended to cure or manage LBP, but no quality evidence exists that they accomplish this as successfully as therapies that focus on restoring functional ability without focusing on pain. In those cases, the traditional medical model of "curing" the patient does not work well. Instead, patients should be aware that returning to normal activities most often aids functional recovery.
- Patients should be encouraged to accept responsibility for managing their recovery rather than expecting the provider to provide an easy "cure." This process promotes the use of activity and function rather than pain as a guide, making the treatment goal of return to occupational and non-occupational activities more obvious.
- If symptoms persist without improvement, further evaluation is recommended.
- Patients with evidence of specific nerve root compromise confirmed by appropriate imaging studies may be expected to potentially benefit from surgery.
- Quality evidence indicates that patient outcomes are not adversely affected by delaying non-emergent surgery for weeks or a few months and continued conservative care is encouraged in patients with stable or improving deficits who desire to avoid surgery. However, patients with either moderate to severe neurological deficits that are not improving or trending to improvement at 4 to 6 weeks may benefit from earlier surgical intervention. Those with progressive neurological deficit(s) are believed to have indications for immediate surgery. Those with severe deficits that do not rapidly improve are also candidates for earlier testing and referrals.
- Nonphysical factors (such as psychiatric, psychosocial, environment including non-workplace and workplace, or socioeconomic problems) should be investigated and addressed, especially in cases of delayed recovery or delayed return to work.

Basic Principles and Definitions

Active Therapy: The term "active therapy" generally involves the patient taking an active role in the treatment of their LBP using various modalities. Active therapeutic exercises include aerobic activity, muscle reconditioning (light-weight lifting or resistance training), directional exercises, and active physiotherapy.(27) Active therapy may also include psychological, social, and educational components in conjunction with therapeutic exercises.(28)

Acute, Subacute, and Chronic Low Back Pain: Acute, subacute, and chronic LBP are categorized as less than 1 month, 1 to 3 months, and greater than 3 months duration, respectively (29).[iii]

Adjacent Segment Disease: This theory postulates that if there is disease in one spinal segment, it increases the probability of disease in the neighboring segment. It is most commonly used to indicate

---

[iii] This document uses these definitions regardless of whether other definitions were used at the onset of chronic LBP (e.g., a minority of studies use a 6-month duration for chronic pain).

the probability of a disc problem in the segment adjacent to a fused or otherwise operated segment, although surgery is not inevitably indicated.

Aggressive Exercise Therapy: This therapy typically concentrates on cardiovascular training and strengthening of muscles to improve back function.(29-31) Aggressive exercise therapy is a primary treatment for chronic LBP and after various back surgeries, and is frequently initiated in the course of treating subacute LBP.

Ankylosing Spondylitis: Spondylitis is a chronic, inflammatory, rheumatic condition of the sacroiliac (SI) joints and the spine. As the condition advances, it may cause fusion of the vertebrae and SI joints (ankylosis). Spondylitis can affect other body tissues.

Bulging Intervertebral Disc: The intervertebral disc is a fibrocartilaginous material. Its primary function is to allow slight movement between each individual spinal segment and significant ranges of motion when all segments are considered together as one functional unit. A disc also acts as a shock absorber for the spine and is composed of an annulus fibrosis (a broad circumferential ligamentous structure) surrounding the nucleus pulposus (a gel-like substance). A bulging intervertebral disc involves an assessment that the degree of natural disc bulging is larger than is typical at a given level. "Protrusion" is a term sometimes used to describe a bulging disc, particularly in radiological literature. Such bulging may be described as focal, diffuse, central, and/or lateral. A key distinction is that there is no rupture of the nucleus pulposus through the annulus. Disc bulging increases as the day progresses (approximately 20% diurnal volume variation) and disc bulging is also magnified if an MRI is performed in a standing position. Other than relatively unusual situations (e.g., large lateral bulging into a narrowed neuroforaminal space or large central bulging into a narrowed spinal canal), bulging is thought to be asymptomatic.(32)

Centralization: Centralization is a pattern of pain response elicited and reported by patients during a form of lumbar assessment using repeated end-range movements in one direction of testing and various postures, most often end-range positioning. When pain referred or radiating away from the spine retreats back toward or to the midline in response to a single direction of sustained or repeated positional spinal testing, that pain is "centralizing" or has "centralized."

Chemonucleolysis: Chemonucleolysis is the process of injecting chymopapain (or other enzyme) into the intervertebral disc to dissolve the gelatinous intradiscal material. The disc then shrinks in size. This procedure is less invasive then back surgery, but is currently largely unavailable in the U.S. due in part to adverse effects.

Chronic Non-specific Low Back Pain: LBP lasting longer than 3 months (12 weeks) is defined in this document as "chronic." Chronic LBP is labeled as "non-specific" when it is deemed to be not attributable to a recognized, known specific pathology.(29) The majority of chronic LBP is non-specific.(13, 33) Included in this category are terms used to attempt to describe these patients with specificity that includes purportedly "specific" terms such as degenerative disc disease, "discogenic" back pain, "black disc disease," micro instability, lumbar spondylosis, facet syndrome, piriformis syndrome, sacroiliac joint syndrome, and myofascial pain. There is no scientific consensus that the pain-generating structure can be reliably identified in these pain syndromes. There are specific treatments used to target these patients, but most are not supported by evidence from high-quality randomized controlled trials (RCTs). As the placebo or control populations used in many studies included throughout this document routinely

improve, one cannot infer that improvement in pain with such treatment is quality evidence in support of a mechanistic theory.

Degeneration of the Disc: Degeneration of the disc is the changes to the vertebral discs and may be a natural consequence of aging. It is sometimes used synonymously with the term "spondylosis." As it is typically a consequence of aging and thus is not a disease, it is nevertheless sometimes erroneously referred to as "Degenerative disc disease (DDD)." Degeneration of the disc may also lead to spinal stenosis (a narrowing of the spinal canal) that may place pressure on the spinal cord and other nerves.(34) It is generally considered to be a normal process of aging and is generally thought to be asymptomatic unless neurological impingement results.

Derangement: A non-specific term purportedly a painful displacement within the spine often used by those performing manipulation. A derangement is considered by some proponents to be "reducible" when a directional preference and pain centralization are elicited during a mechanical evaluation using repeated end-range test movements. May be used as an equivalent though less specific term to displaced intervertebral disc contents.

Delayed Recovery: Delayed recovery is an increase in the timeframe prior to returning to work or usual activities compared with the length of time expected based on average expectations, severity of the disorder, and treatments provided.

Directional Preference: The single direction of repeated end-range spinal bending or positioning tests that causes an individual's pain to centralize, abolish, or both. Midline-only pain cannot centralize (it is already central) but may have a directional preference where a single direction of end-range bending or positioning reduces or eliminates that midline pain.

Extrusion: See Herniated Intervertebral Disc below.

Facetectomy: Facet joints of the vertebrae (also called zygapophysial joints) are synovial fluid lubricated joints posterolaterally located on each side of the posterior (back) of the spine. The joint is formed where each side of the vertebrae overlap one another. A facetectomy is the removal of the bone that forms these joints.

Failed Back Surgery Syndrome: Failed back surgery syndrome (FBSS) is an ill-defined term sometimes used to label a heterogeneous set of conditions with suboptimal post-surgical results including chronic pain and persistent or recurrent disability. While indicating that surgery failed to achieve pre-operative goals, there are patients who do improve with either time or subsequent treatment. As negative terms may foster debility and impede recovery, this term is discouraged (LBP or chronic LBP are preferable diagnoses). However, because the term is used in the scientific literature, it is discussed in this document.

Foraminotomy: The intervertebral foramina are the normal gaps through the bone between the vertebrae through which a spinal nerve root exits the spinal canal. A foraminotomy is the removal of part of the bone around the intervertebral foramina to increase the size of this passage.

Functional Capacity Evaluation: A functional capacity evaluation (FCE) is a comprehensive battery of performance-based tests to determine an individual's ability to work and conduct activities of daily living.(35) An FCE may be done to identify an individual's ability to perform specific tasks associated with

a job (job-specific FCE), or his or her ability to perform physical activities associated with any job (general FCE). The term "capacity" used in FCE may be misleading, as an FCE generally measures performance and effort rather than capacity.

Functional Improvement (especially Objective Evidence): Evaluation of the patient prior to the initiation of treatment should include documentation regarding objective physical findings and current functional abilities both at home and at work. This should include a clear statement regarding what objective or functional goals are to be achieved through the use of treatment if anything other than full functional recovery occurs. These measures should be tracked during treatment and evidence of progress towards meeting these functional goals should be sought. Examples of documentation supporting improved function would be increased physical capabilities including job specific activities, return to work, return from off-duty-status to modified duty, performance of exercise goals, participation in progressive physical therapy, and other activities of daily living. Validated tool(s), such as the Modified Oswestry Questionnaire and Roland-Morris Disability Questionnaire may also help track progress, although they are subjective. Objectively measured improvements in strength or aerobic capacity may be physical examination correlates of improved function.

Functional Restoration: Functional restoration is a blend of various techniques and programs (both physical and psychosocial), rather than one specific set of active exercises, processes or therapies. The basic principle for all of these individually tailored programs is to help LBP patients cope with pain and return to the functional status required for their daily needs and work activities.(36) The term functional restoration program frequently refers to a full-day multidisciplinary, medically-directed program typically lasting from 3 to 6 weeks, employing an interdisciplinary team often consisting of therapists, psychologists, case managers, and nurses.(37)

Herniated Intervertebral Disc: A herniated intervertebral disc involves a defect in the annulus fibrosis with rupture of the nucleus pulposus out through that opening. A herniated disc may exert direct mechanical pressure and/or chemically irritate a nerve root, causing pain (see Table 2 for tests to help determine if a patient has a herniated intervertebral disc). Herniated discs are often asymptomatic.

Laminectomy: The lamina is the thin bony area of the vertebrae that covers each of the two posterolateral aspects of the spinal canal. Laminectomy is the complete removal of one lamina to expose or access the spinal canal.

Laminotomy: A laminotomy is the partial removal of the lamina to expose or access the spinal canal.

McGill Pain Questionnaire: The McGill Pain Questionnaire (MPQ) attempts to quantify pain, describing pain not solely in terms of intensity, but also in terms of sensory, affective, and evaluative qualities. It was intended to provide a way of identifying differences among different methods of relieving pain.(38-41)

Oswestry Disability Index: The Oswestry Disability Index (ODI) is a subjective tool intended to measure functional disability by evaluating a patient's perceived limitations in performing activities of daily living. There are 10 questions related to pain and disability. The "score" is presented as a percentage (0 to 100) – 0% represents no pain or disability while 100% represents total disability.(42, 43) However, the test is not standardized and is frequently modified, making interpretations difficult.(44, 45)

Passive Modality: Passive modalities refer to various types of treatment that usually involve administration of some form of applied stimulus rather than active therapy (see Active Therapy). Forms

of passive modalities include massage, hydrotherapy (e.g., whirlpools, hot tubs, spas, etc.), ultrasound, and hot/cold compresses.

Percutaneous Discectomy: Percutaneous means "through the skin." In the case of surgery, it typically means a smaller incision than a traditional "open" procedure and consequently there is less access to the total disc or extruded portion(s). Discectomy is the surgical removal of an intervertebral disc. Thus, a percutaneous discectomy is the removal of a spinal disc via a small incision through the skin with the hope that the remaining aspects collapse like a balloon.

Physical or Occupational Therapy: The term "physical therapy" is used in ACOEM's *Guidelines* generically to mean physical medicine, therapeutic and rehabilitative evaluations and procedures. Much research uses this term generically. This rehabilitative therapy may be performed by or under the direction of trained and licensed individuals such as physical therapists, occupational therapists, exercise physiologists, chiropracters, athletic trainers, and physicians. Jurisdictions may differ on the qualifications for licensure to perform these interventions. These *Guidelines* are not meant to restrict physical therapy to being performed only by physical therapists.

Protrusion: See Bulging Intervertebral Disc.

Radicular Pain Syndrome: Pain in the extremities (arms, hands, legs, and feet) that is caused by an associated nerve root being affected in or near the spine. Pain is usually substantially worse in the extremity than in the spine and some have only radiating pain in the extremity. An example of this syndrome is lumbar radiculopathy from a disc herniation, most typically resulting in sciatica (usually either an L5 or S1, less often L4, nerve root impingement with pain radiating down the lower extremity in those specific nerve root distributions). Radiculopathy may result in numbness or paresthesias in the corresponding dermatome, muscle weakness in the corresponding myotome, and/or loss of muscle stretch reflex corresponding to the affected root level (see Table 4).

Roland-Morris Disability Questionnaire: The Roland-Morris Disability Questionnaire is a self-administered disability measure consisting of 24 items abstracted from the Sickness Impact Profile. The items represent a variety of activities with which individuals with low back pain may have difficulty. However, the test is not standardized and is frequently modified, making interpretations difficult. (44, 45)

Sciatica: A clinical presentation of pain in the distribution of the sciatic nerve. While most commonly attributed to one, or rarely multiple, impinged L4, L5 or S1 nerve roots, there are many other potential causes (e.g., other musculoskeletal, tumors etc).(46-48)

Slump Stretching: Stretching by rounding the neck and back and flexing the hip to 90° with knee extension (ankle neutral or slightly dorsiflexed).

Spinal Motion Segment: The spinal motion segment is made up of two adjacent vertebrae, the intervertebral disc between them, connecting ligaments, and their two facet joints. The connections of these bones and discs constitute the functional unit of the spine. Spinal motion is the ability of the spine, as a whole, to flex in multiple directions. A spinal motion segment is the range of motion for one joint segment between two adjacent vertebrae. When two or more vertebrae are completely fused together, surgically or otherwise, the spinal motion of these two segments is eliminated and the overall range of motion for the entire spine decreases.

Spinal Stenosis: Spinal stenosis is anatomic narrowing of the spinal canal. It may or may not be accompanied by neurological impingement of the spinal cord and/or spinal nerves. When neurological impingement occurs in the lumbar segment of the spine, symptoms may include low back and lower extremity pain that is termed "neurogenic claudication," i.e., pain with walking. This condition is most often degenerative, although it may be congenital or acquired after significant trauma resulting in spondylolisthesis. Most commonly, spinal stenosis involves a combination of factors that may include facet joint osteoarthrosis with osteophytes, intervertebral disc space narrowing, hypertrophy of the ligamentum flavum and other ligamentous structures, and/or congenital narrowing of the spinal canal.

Spondylolisthesis: Spondylolisthesis is the abnormal alignment of one vertebra in relation to the adjacent vertebral body usually measured in millimeters of displacement between the posterior aspects of the two vertebral bodies. While most commonly degenerative, it may also be acquired from major trauma. Isthmic spondylolisthesis is a developmental defect. When congenital, it is a non-union of the pars. It also is believed to relatively rarely occur as a non-union of a stress fracture that occurs in childhood such as relatively rare circumstances such as football linemen and female gymnasts. It rarely progresses once skeletal maturity is attained. It is frequently asymptomatic, but it may be rendered symptomatic by adult trauma. Degenerative spondylolisthesis has a different pathophysiology. It occurs as the facet joints and adjacent disc lose their stabilizing ability due to degenerative changes (e.g., facet joint osteoarthrosis and degenerative disc space narrowing), typically in those over age 60. The degree of spondylolisthesis tends to increase with age-related changes, especially as the degree of disc space narrowing advances. It is usually thought to be asymptomatic unless there is neurological impingement (e.g., accompanying spinal stenosis).

Spondylosis: Lumbar spondylosis is the degeneration of the lumbar vertebral discs. It is sometimes used synonymously with the term "degeneration of the disc." This affects the spinal facets as well as the disc. Lumbar spondylosis may also lead to spinal stenosis (see above) that may place pressure on the spinal cord and other nerves.(34) Spondylosis is generally considered to be a normal process of aging and is thought to be asymptomatic unless neurological impingement results.

Spondylolysis: A term sometimes used to refer to non-union of a pars defect and/or pars fracture (see also spondylolisthesis above).

Visual Analog Scale: The Visual Analog Scale (VAS) attempts to measure a patient's level of subjective pain with a 0 to 100 scale. In research and some clinical settings, this is commonly obtained with a horizontal line that is 10cm long with verbal scale anchors of "no pain" to "worst pain" that a patient marks and can then be measured in millimeters to give a VAS (e.g., 45mm = 4.5). Most commonly, a 0 to 0 verbal rating scale is used clinically as a surrogate without being a true VAS.

Initial Assessment

Most LBP has no definable pathophysiological abnormality. Accordingly, the initial assessment has a somewhat unusual emphasis on "ruling out" serious underlying conditions (e.g., kidney stone, infection, cancer, fracture). If there are no serious underlying conditions, the emphasis typically shifts to ruling out discrete anatomic causes (e.g., a pinched nerve) before allowing the generic diagnosis of "low back pain."

Thorough medical and work histories and a focused physical examination (see General Approach to Initial Assessment and Documentation Guideline) are sufficient for the initial assessment of a patient

with potentially work-related low back symptoms. Findings of the medical history and physical examination may alert the examiner to other pathology (e.g., not of low back origin) that can present as low back disorders. In this assessment, certain findings, referred to as red flags, raise suspicion of serious underlying medical conditions (see Table 1). The absence of red flags and conditions rules out the need for special studies, referral, or inpatient care during the first 4 to 6 weeks. During this time, spontaneous recovery is expected, provided any associated workplace factors are mitigated.(29)

There also are psychological red flags that should be evaluated, such as PTSD, suicidality, hallucinations or intoxication, which have been called primary risk factors,(49) and have been reviewed elsewhere.(50) Suicidality though is a potentially fatal complication, which makes it a more severe complication than cauda equina.

Red Flags

Potentially serious disorders are referred to as "red flags." These include acute fractures, acute dislocations infection, tumor, progressive neurologic deficit, or cauda equina syndrome.

Table 1. Red Flags for Potentially Serious Low Back Conditions

Disorder	Medical History	Physical Examination/Diagnostic Testing
SPINAL DISORDERS		
Fracture	Major trauma, such as vehicular accident or fall from height Minor trauma or supra-maximal lifting in older or potentially osteoporotic patients	Percussion tenderness over specific spinous processes Careful neurological examination for signs of neurological compromise
Tumor and Neoplasia	Severe localized pain over specific spinal processes History of cancer Age >50 years Constitutional symptoms, such as recent unexplained weight loss or fatigue Pain that worsens when patient is supine Pain at night or at rest	Pallor, reduced blood pressure, diffuse weakness Tenderness over spinous process and percussion tenderness Decreased range of motion due to protective muscle spasm History of sciatica for detection of cancer[†] ▪ Sciatica sensitivity = 58 to 93% ▪ Sciatica specificity = 78% History of paresthesia for detection of cancer[†] ▪ Paresthesia sensitivity = 58% Plain radiography for detection of cancer[‡] ▪ Radiography sensitivity = 60% ▪ Radiography specificity = 90 to 99.5% Magnetic resonance imaging (MRI) for detection of cancer[‡] ▪ MRI sensitivity = 83 to 93% ▪ MRI specificity = 90 to 97% Radionuclide scanning for detection of cancer[‡] ▪ Planer imaging sensitivity = 74 to 98% ▪ Planer imaging specificity = 64 to 81%

		• SPECT sensitivity = 87 to 93% • SPECT specificity = 91 to 93%
Infection	Risk factors for spinal infection: recent bacterial infection (e.g., urinary tract infection); IV drug abuse; diabetes mellitus; or immune suppression (due to corticosteroids, transplant, or HIV) Constitutional symptoms, such as recent fever, chills, or unexplained weight loss	Tenderness over spinous processes Decreased range of motion Vital signs consistent with systemic infection (late): • Tachycardia • Tachypnea • Hypotension • Elevated temperature • Pelvic or abdominal mass or tenderness • High white blood cell count • Elevated erythrocyte sedimentation rate Plain radiography for detection of infection[‡] • Radiography sensitivity = 82% • Radiography specificity = 57% Magnetic resonance imaging (MRI) for detection of infection[‡] • MRI sensitivity = 96% • MRI specificity = 92% Radionuclide scanning for detection of infection[‡] • Radionuclide scanning sensitivity = 90% • Radionuclide scanning specificity = 78%
Cauda Equina Syndrome/ Saddle Anesthesia	Direct blow or fall with axial loading Perianal/perineal sensory loss Recent onset of bladder dysfunction, such as urinary retention, increased frequency, or overflow incontinence Bowel dysfunction or incontinence Severe or progressive neurologic deficit in lower extremities, usually involving multiple myotomes and dermatomes	Unexpected laxity of bladder* or anal sphincter Major motor weakness in hamstrings (knee flexion weakness); ankle plantar flexors, evertors, and dorsiflexors (foot drop). May have more proximal myotomal weakness if higher cord level(s) affected. Spastic (thoracic) or flaccid (lumbar) paraparesis Increased (thoracic) or decreased (lumbar) reflexes
Progressive Neurologic Deficit	Severe low back pain Progressive numbness or weakness	Significant and progressive myotomal motor weakness Significant and increased sensory loss – in anatomical distribution Radicular signs
EXTRASPINAL DISORDERS		
Dissecting Abdominal Aortic Aneurysm	Excruciating low back pain History of atherosclerotic disease or multiple cardiovascular risk factors History of hypertension	Pulsatile midline abdominal mass Absent or variable pulses Asymmetric blood pressure Bruits
Renal Colic	Excruciating pain from costovertebral angle to groin, testis, or labia History of urolithiasis	Possible tenderness at costovertebral angle

	Hematuria	
Retrocecal Appendicitis	Right lower quadrant abdominal pain and/or right low back pain Constipation Subacute onset without inciting event Nausea and vomiting variably present	Low-grade fever May have tender right lower quadrant Pain on rectal examination in right lower quadrant
Pelvic Inflammatory Disease	Vaginal discharge Pelvic pain Prior episode	Uterine tenderness Tender over right and/or left lower quadrants Cervical discharge
Urinary Tract Infection	Dysuria History of urinary tract infections	Fever Suprapubic tenderness Smelly or cloudy urine

Adapted from: [†]van den Hoogen HM, et al. 1995; [‡] Jarvik JG, Deyo RA 2002;*Bigos S, et al. 1994.
SPECT = single-photon emission computed tomography

Absence of Red Flags

Absent red flags, low back disorders can usually be classified into one of two working categories:

- **Non-specific disorders** including benign, self-limited disorders with unclear etiology, such as regional or non-specific LBP. This includes the majority of LBP patients' problems, generally more than 95% of those with acute LBP.
- **Specific disorders**, including potentially degenerative disorders such as herniated discs (see Table 2), spinal stenosis, other neurological impingements, and facet joint osteoarthrosis.

There may be overlap between these two categories.

Table 2. History and Physical Examination Findings with Reported Sensitivity and Specificity Estimates for Common Specific Spine Disorders

Disorder	Medical History	Physical Examination/Diagnostic Testing
Ankylosing spondylitis[‡†]	Onset usually <35 years of age Male gender at higher risk Reduced lateral mobility Pressure in the sacral or lumbar spine No relief from pain by lying down Three (3) months low back pain Stiffness in the morning Relief of pain with exercise Chronic onset	HLA B27 testing to detect ankylosing spondylitis - Sensitivity = 95% - Specificity = 85% Plain radiography for detection of ankylosing spondylitis[‡] - Radiography sensitivity = 26 to 45% - Radiography specificity = 100% Magnetic resonance imaging (MRI) for detection of ankylosing spondylitis[‡] - MRI sensitivity = 56% Radionuclide scanning for detection of ankylosing spondylitis[‡] - Radionuclide scanning sensitivity = 26% - Radionuclide scanning specificity = 100%
Herniated Disc[‡£]	Sciatica/radicular pain Dermatomal distribution	History of sciatica for detection of a herniated disc[‡£] - Sensitivity = 85 to 99% - Specificity = 6 to 88%

	Myotomal distribution	Ipsilateral straight-leg raising for detection of a herniated disc[‡]
	Low back pain	▪ Sensitivity = 80%
		▪ Specificity = 40%
		Crossed straight-leg raising for detection of a herniated disc[‡£]
		▪ Sensitivity = 23 to 25%
		▪ Specificity = 90 to 100%
		Ankle dorsiflexion weakness for detection of a herniated disc[‡]
		▪ Sensitivity = 35%
		▪ Specificity = 70%
		Great toe extensor weakness for detection of a herniated disc[‡]
		▪ Sensitivity = 50%
		▪ Specificity = 70%
		Impaired ankle reflex for detection of a herniated disc[‡£]
		▪ Sensitivity = 48 to 50%
		▪ Specificity = 60 to 89%
		Ankle plantar flexion weakness for detection of a herniated disc[‡]
		▪ Sensitivity = 6%
		▪ Specificity = 95%

*Adapted from: [‡]Jarvik JG, Deyo RA 2002; [†]van den Hoogen HM, et al. 1995; [£]Vroomen PC, et al. 1999.

Low Back Pain (LBP)

More than 95% of patients have no identifiable cause for acute LBP. Most with chronic LBP also have no clearly identifiable cause. Symptoms are pain, usually without radiation, although some patients have radiation into the buttocks or thigh. Pain that is solely or mostly in a thigh and calf generally, but not always, signifies radiculopathy, particularly when the radicular pain in the extremity substantially exceeds that in the back or is the sole symptom. LBP patients generally have no tingling, numbness, or muscle weakness other than weakness associated with pain-producing activities. Some practitioners refer to these LBP patients as having incurred "sprains" and/or "strains"; however, these labels are not appropriate. A sprain is a disrupted ligament and a strain is a myotendinous junction disruption. Both imply knowledge of the anatomic cause of LBP and a forceful mechanism of injury when the former is untrue for LBP patients and the latter may or may not be true. Use of those terms also confuses the proper use of those diagnoses elsewhere in the body, becomes problematic in determination of work-relatedness, and misdirects patients on the value of activity for early functional recovery. Low back "strain" and "sprain" are included in non-specific low back pain.

Radicular Pain Syndromes

Radicular pain denotes pain that is in a specific neurological distribution, nearly always involving only one nerve root. Symptoms typically include some combination of extremity pain, tingling and numbness, and muscle weakness (in the appropriate myotomal distribution). Corresponding signs, including sensory loss, muscle weakness, and a diminished reflex all in the distribution of that same nerve root may be present. Sciatica denotes pain in the sciatic nerve distribution and may be caused by many abnormalities, although it most commonly denotes impingement of either the L5 or S1 nerve roots as those are most frequently affected.(46-48) It less commonly may involve the L4 or other nerve roots as the sciatic nerve also has components from L4 to S3. The most common cause of sciatica is radiculopathy and the diagnosis of radiculopathy is generally not complex in moderate to severely

affected individuals. It becomes more difficult with milder cases, as symptoms and examination findings may be less pronounced or some of the findings may be absent.

There are multiple possible causes of radicular pain. Most commonly, at least in the occupational setting, pain is due to a herniated intervertebral disc. This involves a rupture in the fibrous annulus fibrosis and protrusion or extrusion of nucleus pulposus material.(32, 51) A combination of a physical displacement of the nucleus pulposus along with a purported chemical reaction to this material with consequent swelling in the acute phase appears responsible for the development of the symptoms of neurological compromise. Other possible causes of radicular pain include a significant laterally bulging (but not herniated) disc into a narrowed canal that is sufficient to impinge the nerve root. It is also possible for a severe degenerative arthritic process to accumulate substantial osteophytic growths around the facet joint and/or intervertebral disc space and cause radicular symptoms.

Zygapophysial (Facet) Joint Degenerative Joint Disease
Facet joints are small, synovial fluid filled, synovium lined, ligamentously encapsulated joints that are in alignment along the posterior aspect of the spinal column. They are in many ways similar to nearly all other joints (the main exceptions are the intervertebral discs). Facet joints are prone towards the same maladies that affect other joints, including osteoarthrosis (degenerative joint disease), gout,(52) psoriatic arthritis, and many other arthritides. There appears to be a propensity towards facet joint osteoarthrosis in those with other osteoarthrosis elsewhere in the body, sometimes referred to as "systemic osteoarthrosis."

The determination of facet joint osteoarthrosis is relatively straightforward. The disorder becomes nearly universal with increasing age.(53) Roentgenograms, particularly facet joint (or rotated) views for the lumbar spine and lateral views for the cervical spine, will show evidence of degenerative findings (i.e., sclerosis, joint space narrowing, and cyst formation). However, the diagnosis of pain arising from such degenerative facet joints is quite controversial compared with arthritis in peripheral joints. This is primarily due to a combination of the universal appearance of facet joint arthrosis with age, variable findings with facet joint blocks and injections, and especially the lack of an undisputed gold standard (see also facet joint injections and blocks).(53-55) Osteoarthrosis in the spine and disc space narrowing are extremely common (so common that many radiologists do not record these abnormal findings, especially when more mild, on x-rays as they are "normal" for age). It appears to be largely asymptomatic.(56-58) In those with multiple levels affected, there often is not pain at all of those levels. As LBP is so common and the overwhelming anatomic cause of LBP is unknown,(13) it follows that attempting to diagnose the pain as related to a specific structure such as the facet joints is quite challenging.

Important diagnostic limitations also include that diagnostic blocks are often accomplished involving intra-articular injection(s) of anesthetic agents. This cannot be directly related to the value of neurotomies.(59) Other limitations include single diagnostic blocks versus multiple blocks and the use of corticosteroids. Problems with diagnostic blocks of the dorsal root rami include: 1) the ability to anesthetize the joint; 2) the specificity to not anesthetize adjacent neural structures; and 3) the likelihood ratio of a single diagnostic block.(59)

Although not necessarily related to facet joint disease, chronic LBP patients may develop segmental rigidity (SR) at one or more lower lumbar joints, generally thought to be due to a combination of tissue scarring, chronic immobility and muscle splinting. The location is commonly in the lower half of the

lumbar spine, particularly above, below or bracketing a fusion or other prior lower lumbar surgical site. Segmental rigidity is initially noted on lateral bend motion, generally effects 1 to 2 levels, and may be asymmetric. Treatment involves a trial of *exercise only*, performed frequently to mobilize rigid facet joints after prolonged activity. If unsuccessful, the combination of facet injections and frequently-performed exercise may result in improvement of joint mobility, setting the stage for improved rehabilitative gains by decreasing pain and facilitating strengthening exercise.(60, 61)

Sacroiliac Joints
Sacroiliac joints (SIJs) are diarthrodial synovial joints at the lumbosacral junction. Nociceptors in the SIJ are reported to have a higher threshold than those within the lumbar facet joints, but lower than the anterior portions of lumbar discs, and may be a potential cause of pain. The joint is most prominently involved in ankylosing spondylitis, in which the joint may become obliterated, as well as Reiter's syndrome and psoriatic arthritis. Its role in other back pain is somewhat controversial, due in part to the lack of normal joint motion beyond a few degrees, the joint's close proximity to the L4-L5 and L5-S1 areas and consequent frequent tenderness in the surrounding structures. Physical examination maneuvers reportedly have poor ability to confirm a diagnosis of SI joint involvement.(62) These challenges make unequivocal definition of the SI joint as the problematic source of pain difficult, and in many cases, impossible.

A study evaluating pain diagrams in responders versus non-responders to double diagnostic fluoroscopically guided intra-articular sacroiliac joint block suggested subtle, but potentially significant differences in the pain diagrams to help guide diagnosis.(63) Those findings were a closer proximity to pain over the SI joint versus pain more distally in the lower buttocks in the non-responders. Another study compared the diagnostic accuracy of a multi-test regimen of 5 sacroiliac joint pain provocation tests with fluoroscopically controlled double SIJ blocks using a short- and long-acting local anesthetic in order to reduce the exposure of patients to unnecessary invasive SIJ procedures, for 60 patients with chronic LBP.(64) The study was designed to determine the relevance of a multi-test regimen of SIJ provocation tests. Application of this regimen was found to be useful in reducing unnecessary intra-articular SIJ block in the early stage of clinical decision making. "When three or more provocation tests are positive, the probability is between 65% and 93% that the pain is related to the SIJ, in which case confirming SIJ blocks are required." When fewer than three provocation tests were positive, "the probability is between 72% and 99% that the SIJ is unlikely to be the source of pain."(64)

The International Association for the Study of Pain (IASP) has proposed diagnostic criteria for SIJ pain of: 1) pain in the SIJ region; 2) stressing the joint in clinical tests selective for the joint to reproduce the pain; and 3) selectively infiltrating the symptomatic joint with local anesthetic to completely relieve the pain.(65) However, while prevalence rates are estimated at 2 to 26.6%, false-positive rates are estimated at 20 to 22%. A systematic review of clinical tests of SIJ concluded that "there is no evidence to support the inclusion of mobility and pain provocation tests for the SIJ in clinical practice."(66) Estimates from local anesthetic blocks of the SIJ(s) are that these joints may be responsible for 10 to 26.6% of chronic LBP cases.(67) The joint can be anesthetized using a fluoroscopic guided or unguided injection of a local anesthetic or steroid.

Estimates vary regarding the rate that the SI joint may contribute to LBP. A small case series of patients with chronic pain after successful fusion surgery performed anesthetic blocks found a 35% rate of positive blocks in this population (at least 75% pain relief), inferring that the SIJ may be partially related

to FBBS.(68) Another case series attributed the cause to the SI joint in 32% and another 29% were felt to be a possible cause.(69) Standard anteroposterior radiographs are thought to be sufficient for most purposes, rather that needing SIJ views in cases of reactive arthritides.(70) Therapies have been developed to attempt to address these joints including injections of glucocorticoids, radiofrequency neurotomy, physical therapy, manipulation, orthotics, mobilization, cryoneurolysis, neuroaugmentation, and surgery.(71)

Clinical Syndromes

The inability of conventional clinical testing and advanced imaging to reliably identify an anatomic pain source for most LBP has stimulated considerable research focused on reliably identifying and validating clinical syndromes or subgroups based on clusters of clinical examination findings. If homogeneous syndromes are validated, this may enable more effective individualized care than a less specific approach towards all non-specific LBP.

One syndrome with perhaps more support than others is "directional preference." A directional preference is often identifiable in a patient's history and examination. Directional preference patients typically describe a history of episodic and intermittent LBP with a directional theme as to what positions, movements and activities commence or worsen their pain and what improves or stops their pain. A presumptive pain generator's directional preference is that single direction of repeated end-range spinal bending tests or static positioning that causes the pain to "centralize," abolish, or both. Pain "centralization" is a pattern of pain response whereby pain referred or radiating away from the spine retreats back toward or to the midline in response to a single direction of sustained or repeated end-range spinal testing. Midline-only LBP cannot centralize because it is already central but it often has a directional preference where a single direction of testing will eliminate that midline pain. After pain centralization or elimination, the pain typically remains improved until or unless the patient moves excessively in the opposite direction of that preferred. Avoidance of moving in a direction that aggravates the pain should be minimized or avoided during the early phase of treatment to speed recovery.

The unique purpose of these end-range tests, performed in weight-bearing and recumbency, is to load the spine in different bending directions. The most common lumbar directional preference is extension, yet smaller numbers of pain-generators benefit from other directions of loading: lateral, rotational or flexion movements. Those with an extension directional preference typically worsen with lumbar flexion and improve with extension or simply restoring their lordosis.

This syndrome has been referred to as a "reducible derangement" or a "directional preference syndrome." Its two characteristic clinical findings (directional preference and pain centralization) are identified with strong interexaminer reliability (Kappa = 0.9, 0.823, 0.7, % agreement: 88 to 100%),(72-74) with training.(75)

The prevalence of this directional preference syndrome is reportedly high: 70-89% of acute(76-79) and 40 to 50% in chronic LBP.(80-83) It is commonly elicited in axial LBP, referred, as well as radicular pain.(84-86) There is also suggestive evidence of a concomitant psychosocial benefit by teaching and empowerment with the knowledge and skills to effectively self-treat.(87)

Medical History and Physical Examination

A focused and detailed medical history and physical examination are necessary to assess the patient's medical condition and specific low back disorder. This section will review the medical history including the questions that should be asked. This diagnostic approach also needs tailoring to the specific patient, particularly as factors such as the patient's age, past medical history, underlying medical conditions, significant injury history and genetic predilections all probablistically adjust the diagnostic approach by altering the probabilities for and against specific diagnoses. For example, increasing age is associated with far higher probabilities for degenerative conditions such as spondylolisthesis and is simultaneously associated with reduced ranges of motion in normal individuals that must be incorporated in the diagnostic approach.

It is also important to understand the context of the appearance of the patient in the clinic. Patients with back disorders generally initiate treatment due to pain, which is often attributed to an ostensible injury. However, one should not assume that complaints of acute pain are directly attributable to pathophysiology.(65) Pain is known to be associated with sensory, affective, cognitive, social, and other processes.(88-91) The pain sensory system itself is organized into two parts, often called first and second pain. A-delta nerve fibers conduct first pain via the neospinalthalamic tract to the somatosensory cortex, and provide information about pain location and quality. In contrast, unmyelinated C fibers conduct second pain via the paleospinalthalamic tract, and provide information about pain intensity. Second pain is more closely associated with emotion and memory neural systems than it is with sensory systems.(65, 88-100)

As a patient's condition transitions through the acute, subacute and chronic phases, the central nervous system is reorganized. The temporal summation of second pain produces a sensitization or "windup" of the spinal cord,(100) and the connections between the brain regions involved in pain perception, emotion, arousal, and judgment are changed by persistent pain.(95) These changes cause the CNS's "pain neuromatrix" to become sensitized to pain.(88-91) This CNS reorganization is also associated with changes in the volume of brain areas,(94) decreased gray matter in the prefrontal cortex,(94) and the brain appearing to age more rapidly.(93) As pain continues over time, the CNS remodels itself so that pain becomes less closely associated with sensation, and more closely associated with arousal, emotion, memory and beliefs.(96, 97) Because of these CNS processes, one should be aware that as the patient enters the subacute phase, it becomes increasingly important to consider the psychosocial context of the disorder being treated, including the patient's social circumstances, arousal level, emotional state, and beliefs about the disorder. However, behavioral complications and physiological changes associated with chronicity and central sensitization may also be present in the acute phase, and within hours of the initial injury.(99)

Medical History

Asking the patient open-ended questions, such as those listed below, allows gauging the need for further discussion or specific inquiries to obtain more detailed information.

1. What are your symptoms?
   - Do you have pain or stiffness?
   - Do you have numbness or tingling?
   - For traumatic injuries: Was the area deformed? Did you lose any blood or have an open wound?
   - Is the discomfort located primarily in your low back? In your leg?

- Do you have pain or other symptoms elsewhere? (Patients who present with a primarily with lower extremity pain may well have radiculopathy from a lumbar disc herniation or other lumbar pathology. Hip pain may present as back pain and vice versa. Hip pathology may affect the back.)
- Have you lost control of your bowel or bladder? Are you soiling your undergarments?
- Do you have fever, night sweats, or weight loss?
- When did your symptoms begin? Have you ever had symptoms like this before?
- Are your symptoms constant or intermittent? What makes the problem worse or better?
- What is the day pattern to your pain? Are you better first getting out of bed in the morning, during the morning, mid-day, evening, or while asleep? Worse as the day progresses? Do you have a problem sleeping? What position is most comfortable? Is there any pain with cough, sneezing, deep breathing, or laughing?
- How long can you sit, stand, walk, and bend?
- Can you lift? How much weight (use items such as gallons of milk, groceries, etc., as examples)?

2. How did your condition develop?

Past:

- Have you had similar episodes previously?
- Have you had previous testing or treatment? With whom?

Cause:

- What do you think caused the problem?
- How do you think it is related to work?
- Did your symptoms begin gradually or suddenly? Did you notice the pain the day after the event?
- Did you slip, trip, or fall?
- Were you doing anything at the time your symptoms began? (It is important to obtain all information necessary to document the biomechanical forces of injury.)

Job:

- What are your specific job duties?
- How long do you spend performing each duty on a daily basis?
- Do you have assistance of other people or lifting devices?

Off-work Activities:

- What other activities (hobbies, workouts, sports) do you engage in? At home or elsewhere?
- Any heavy lifting? How? How often?
- Any physically demanding activities requiring awkward postures, prolonged sitting or standing?

3. How do these symptoms limit you?
- What activities of daily living are limited? Are there specific challenges in your home environment (e.g., steep steps)?
- How long have your activities been limited? More than 4 weeks?
- Have your symptoms changed? How?

4. Do you have other medical problems?

5. What are your expectations regarding your return to work and disability from this health problem?

6. What are your concerns about the potential for further injury to your low back as you recover?

7. What is your job? What do you do on the job? How do you like your job? Your supervisor and coworkers? What is your relationship with your co-workers and supervisor and how do they treat you?

8. What do you hope to accomplish during this visit?

Determining whether or not there is lumbosacral nerve root compromise (and if so, the level of compromise) is important. Symptoms correlating with specific myotomal levels of compression and possible motor weakness are shown in Table 3.

Table 3. Symptoms of Lumbar Nerve Root Compromise

Root Level	Pain or Paresthesia	Motor Weakness
L1	Back, radiating to upper anterior thigh and groin	Hip flexion
L2	Back, radiating to anterior mid-thigh	Hip flexion and adduction, knee extension
L3	Back, radiating to anterior thigh and inner knee	Hip flexion and adduction, knee extension
L4	Back, radiating to lateral thigh, front and medial leg, and medial foot	Hip adduction, knee extension, foot inversion, foot dorsiflexion
L5	Back, radiating to lateral leg and dorsal foot (especially first web space)	Hip abduction, foot and great toe extension. Resisted extensor hallucis longus is considered the best of these as it is an L5 function.
S1	Back, radiating to back of thigh and lateral leg and foot	Knee flexion, plantar flexion. Plantar flexion is the best of these as it is purely an S1 function. It may be tested with repeated toe raises, particularly when there is a suspicion of radiculopathy, but weakness is not obvious on manual testing.

Physical Examination

The objective of the physical examination of the lumbosacral spine is to demonstrate those physical abnormalities that sort out the possible disease entities causing pain that were elicited during the medical history. Abnormalities of the lumbosacral spine may be discovered while the spine is static or during motion. Unless the tests are done in an orderly fashion, important observations may be missed. Therefore, it is helpful to evaluate the patient in a series of positions that test the function of musculoskeletal and neurologic structures of the lumbosacral spine.

The examination begins as soon as the provider introduces him or herself to the patient. The overall initial impression is a critical metric of functional status. Then, vital signs, such as an elevated temperature, may suggest the presence of an infection or neoplasm. Tachycardia may be a sympathetic nervous system response to the patient's pain or it may be anxiety related. For those undergoing more advanced testing for chronic pain, tachycardia may be relevant as indicating potential psychological disturbance, and illicit medication use. Physical examination tests show poor diagnostic performance when used to identify lumbar disc herniation.(101) It is estimated that 99% of patients with serious spinal pathology can be examined with a history and physical examination focusing on the L4, L5 and S1 nerve root distributions.(102)

There are three primary distributions for back pain:
1. Those localized to the back musculoskeletal system (e.g., most commonly LBP of unknown anatomic cause or muscles, tendons, ligaments, or nerves).
2. Those referred to the back (e.g., from internal organs such as kidney, uterus, or abdominal aneurysm).

3. Those referred to the extremities in a dermatomal or myotomal distribution and likely include neurogenic involvement.

Guided by the medical history, the physical examination includes:

- General observation, including changes in positions, stance,
- Gait while walking an extended distance, typically in the hallway, and changes in gait with distance walked,
- Regional examination of the spine,
- Examination of organ systems related to appropriate differential diagnosis,
- Neurologic screening,
- Testing for lumbosacral nerve root tension, and
- Monitoring pain behavior during range of motion and while seated as a clue to the problem's origin.

The completely objective parts of the low back examination are circumferential measurements for atrophy or findings of fasciculations. All other findings require the patient's cooperation, although reflexes are generally much more objective than subjective.

A. Observation and Regional Back Examination

The most important aspect of the examination is observation. This includes observing changes in position, stance, and gait. The examiner should ask the patient to walk down the hallway so there is sufficient distance over which to observe the gait as well as changes in the gait over some duration. In the process, the ease with which the patient stands should be carefully observed. The patient should be observed over at least 20 feet of ambulation. The examiner should observe whether the back is kept in a maintained flexed posture, erect, stiff, or if the lumbosacral spine is moved in the process. Gait fluidity should be carefully observed. How the patient turns around to return to the examination room is also of interest. Back pain usually decreases the mobility of the lumbar spine and produces restriction of normal spinal movement. The back is stiff, as if frozen in one position. Patients with LBP generally walk in a stiff, guarded fashion depending mainly on hip movement and lateral spine flexion rather than using a normal gait involving a more complete range of active spinal movements. This observation may provide some objectivity to the severity of the patient's problems and also provide a rapid assessment of subsequent progress. Thus, observation of gait is generally the most helpful aspect of the LBP physical examination.

The disrobed, but modestly covered, patient is examined standing. The spine is viewed from behind, laterally, and anteriorly for alignment. The levels of the shoulders and any lateral spinal curves (scoliosis), if present, should be noted. The patient should be positioned with his or her head centered over the feet and eyes level. It is wise to also have the shoulders and knees level so any discrepancy will not be due to a weight shift. Therefore, any deviation of the spine from the vertical is compensated by an opposite deviation elsewhere in the spine. The spine is compensated if the first thoracic vertebra is centered over the sacrum. Then, the posterior superior iliac spines, which should be of equal height, should be viewed. The gluteal folds and knee joints should be at an equal height. In the absence of foot or ankle deformity, the feet should be in normal alignment. The patient with lumbar muscle spasm on forward flexion may demonstrate a list to one side – a compensatory scoliosis, with loss of normal spinal contours. Movement of the sacroiliac joint may be examined with the patient standing. The examiner places one thumb on the posterior superior iliac spine and the other on the sacral spine. The patient flexes the ipsilateral hip. Normally, the iliac spine moves downward. Upward motion is indicative of a fixed sacroiliac joint.

The patient should be positioned anteriorly – head straight with shoulders level. The highest points on the flanks or iliac wings should be of equal height. There should be no or very little tilt to the pelvis. Anatomic structures in the lower extremities (patellae, malleoli) should be of approximately equal height and aligned appropriately, although minor leg length discrepancy with typically slightly longer left legs has been reported.(103) The patient should squat in place. This maneuver tests general muscle strength and the integrity of function of the joints from the hips to the feet in the lower extremity. With the patient in the standing position, the range of motion of the lumbosacral spine in forward flexion, extension, lateral bending (side flexion), and rotation is observed. The normal range of motion (ROM) is 40 to 60° for forward flexion, 25° for extension, 15 to 25° for lateral bending, and 3 to 18° for rotation. Inquiries regarding which of these positions produced pain, if any, are also of interest and are used therapeutically.

Spinal motion is important in terms of symmetry and rhythm. The absolute range of motion is not of major diagnostic significance because of wide individual variance. The statement is frequently made that the patient bends forward and reaches to within 6 inches of the floor or 12 inches of the floor or places his or her palms to the floor. The important part of the observation of the patient as he or she bends toward the floor is the quality of spinal flexion in terms of the smooth reversal of the normal lumbar lordosis as the spine flexes forward. This is termed lumbosacral rhythm, and when abnormal (patient keeps his or her lumbar lordosis and bends from the hips) it is theorized to signify local back disease. Although limitation of spine flexion is of limited diagnostic value, the improvement of spine flexion is a means to monitor response to therapy of an individual patient.

Forward flexion of the spine is a segmental motion, with bending occurring at each functional unit (a functional unit comprising two adjacent vertebrae along with their interposed disc). These units also contain the ligaments, nerves, and facet joints of the two adjacent vertebrae. The most movement occurs at the lumbosacral L5 to S1 and L4 to L5 levels. As a result, most of the damage and most symptoms relate to these two functional units. In forward bending, each unit flexes about 8 to 10°. This means that the entire lumbar spine has only 45° of excursion, and as a patient reaches to touch the ground the rest of the motion comes from the pelvis rotating through the hip joints.

When a patient with an injury to one of the functional units attempts to bend forward, his or her flexion may be inhibited by protective muscle spasm. The lumbar spine may not have the normal curve in the erect position nor is there any reversal of the sway of the back on attempting to bend forward. As the patient attempts to touch the floor, almost all of the motion occurs at the hip joints.

Although this inability to flex the lumbar spine can be due to injury, it also may be voluntary if the patient is either afraid or does not wish to bend forward. Consequently, this restriction is not necessarily indicative of an injury. Flexion from an upright position should be compared with similar movement while the patient is distracted. If the patient lies on his or her abdomen with a pillow under the ankles and the head and shoulders resting on the bed, this removes the hamstring tension and the back is not being extended. Therefore, palpation of the back in the absence of spasm reveals a relaxed or flaccid muscle.

Flexion is relative and its limitation may be an indication of poor conditioning. The patient's perceived stiffness may actually represent little loss of flexibility in respect to a pre-injury state. If the protective spasm is unilateral owing to injury of the tissues on one side of the spine, a compensatory scoliosis develops. The spine is tilted to one side because of one-sided muscle spasm. It frequently will increase

with forward flexion. Disc herniation can also cause a scoliosis by irritating nerves on one side of the spine.

Measurement of the distance from the floor to the patient's fingertips is an inexact measurement of lumbar flexion. However, the measurement is a useful way to follow the response of patients to therapy. Improvement in forward flexion will be manifested as a decrease in finger-to-floor distance whether the improvement is from decreased muscle spasm, increased hip motion, or decreased hamstring tightness.

After the patient has fully flexed, it is helpful to observe how an erect posture is regained. How this maneuver is performed reflects past habits as well as the constraints of any tissue injury. Patients with back pain tend to resume the erect position with a fixed lordosis and without any spine movement. The pelvis with the help of knee and hip flexion does it all. The ability to bend sideways in lateral flexion often has no major diagnostic significance. However, pain that increases with flexion to the ipsilateral side may be related to an articular disease or a disc protrusion lateral to the nerve root. If pain is increased with flexion to the contralateral side, the lesion may be articular, muscular (muscles are stretched), or a disc protrusion medial to the nerve root.

Hyperextension can cause pain by changing several anatomic relationships. Arching the back and increasing the lordosis forces the facet joints together, narrows the foramen through which the nerves exit the spine, and compresses the disc posteriorly. A combination of these three factors can create pressure on the nerves as they leave the spine and cause back pain, leg pain, or both. Rotation may be examined in the standing position, but care must be given to stabilize the pelvis to eliminate accessory motion of the hips. Rotation may be examined more accurately in the seated position. Hips and pelvis are stabilized with seating, limiting rotating motion of the spine.

The strength and stamina of the back and leg muscles can be tested by repeated active movement, especially flexion and extension of the lumbosacral spine. The patient should perform 10 toe raises on both feet and 10 more on each foot separately. Repeat testing causes fatigue which accentuates differences in strength in the lower extremities. The strength of the examiner's arms may be less than that of the patient's legs. By using the patient's own weight, instead of the examiner's strength, differences of strength between the legs are discovered. The patient may also be asked to walk on the heels to test for strength of the dorsiflexors of the foot. These muscles are also tested with the patient in the seated position.

The examiner should palpate the lumbosacral spine when the patient is both standing and sitting, and during testing of motions. It is helpful to palpate both groups of paraspinous muscles simultaneously to discern differences of firmness or tenderness in the muscle bodies. Muscles become more prominent as they contract with spasm. Observation may demonstrate this muscle prominence on one side of the midline of the spine. Localized areas of muscle tenderness, which may be a reflection of a trigger point for referred pain to other areas of the lumbosacral spine, should be identified. Unfortunately, even slight asymmetric stances will tend to produce relatively large differences in muscle texture and an appearance of asymmetric spasm even if such is not present, thus careful attention to position is important.

In addition to the soft tissue, bony structures should be palpated. The spinous processes are covered by ligamentous structures, not muscle, and are easily palpated. Localized tenderness suggests the presence of an isolated process, such as an infection, tumor, or fracture affecting that vertebral body. Localized tenderness over multiple spinous processes is also considered a sign of amplification.

Palpation of the lumbar spine should include the midline, paraspinous areas and out laterally. Palpation in the sciatic notch and along the sciatic nerve should also be performed. The levels of tenderness should be recorded and the presence or absence of widespread tenderness noted. The latter includes those who have tenderness that is present beyond the immediate paraspinous area of a few vertebral segments.

The patient should be examined in the seated position with feet on the floor. The strength of the dorsiflexors of the foot may be measured by the examiner maintaining steady downward pressure on the dorsum of the foot. The patient generates uniform resistance to pressure that is overcome in a smooth fashion. Patients may demonstrate give-way weakness, which is manifested by either resisted pressure for a few seconds and then suddenly release the muscle or demonstrate a stepwise release of the muscle resulting in a cogwheel effect. Causes of give-way weakness frequently include submaximal efforts, but can be due to other causes including pain, misunderstanding of directions, and attempting to help the examiner. The probability of feigning rises if the directions are repeated and give-way weakness remains. Testing ankle dorsiflexion bilaterally and simultaneously may help identify a mechanism for observed give-way weakness.

The patient should also be asked to bend forward over the examining table, allowing his or her weight to rest on the abdomen. This position flattens the lumbar lordosis and tilts the sacrum, allowing examination of the inferior portion of the sacroiliac joint, ischial tuberosities, and sciatic notch. Palpation over these anatomic structures may elicit pain. Patients with inflammatory processes of the sacroiliac joints (ankylosing spondylitis) are among those who experience increased pain with percussion over the sacroiliac joints.

Assessment of the neurologic status of the patient is important in the overall back evaluation. The history is the most critical feature and guides the degree to which the neurological testing must be performed. A positive neurologic finding will give objectivity to the patient's symptoms. Most of the neurological examination is performed with the patient seated with the legs dangling. Each nerve root must be examined. Abnormalities of motor, sensory, and reflex function are tested. It is worthwhile to review the anatomy of the nerve roots in order to better understand abnormalities discovered during the neurologic examination.

Each nerve root as it leaves the spinal canal through the neural foramen is enclosed within a sleeve that contains spinal fluid and small blood vessels about and within the nerve. This sac, referred to as the dural sleeve, provides nourishment to a particular nerve root. Any compression and/or traction on the dura will compress its contents and encroach upon the nerve and its blood supply. Secondary to compression, pain is produced along the course of the peripheral nerve and is accompanied by dysesthesias, motor weakness, and decreased reflex function associated with the affected nerve root. The goal of many of the maneuvers done during this phase of the examination is to increase nerve compression to uncover neurologic dysfunction. Of the possible neurologic abnormalities, true muscle weakness is the most reliable indicator of persistent nerve compression with loss of nerve conduction. Sensory changes are subjective, take significant time to document, and require the full cooperation and attention of the patient, but in certain circumstances may be helpful (e.g., lack of expected improvement with efficacious treatments, diagnostic uncertainty). Reflex changes may be lost in a previous episode of nerve root compression. Reflexes may not return even with recovery of sensory and motor function. With age, reflexes diminish and are more difficult to elicit even without any prior history

of nerve compression. However, the loss of reflexes is symmetric. Patients who lose reflexes in both lower extremities on the basis of compression may have spinal stenosis or a large central herniation of a disc.

In addition to nerve root lesions, upper motor neuron and peripheral nerve disease cause abnormalities that may be discovered during the neurologic examination. With upper motor neuron lesions, the fine control of muscles is lost while the trophic effects of the peripheral nerves remain intact. Muscle strength is diminished, but in a different pattern from lower motor neuron weakness. Patients develop spasticity of muscles (tonic contractions) and hyperreflexia. Patients also develop a positive Babinski reflex (extension of the large toe and spreading of the other toes with stroking of the sole of the foot). Ankle clonus, an involuntary rhythmic plantar flexion contraction/relaxation induced after rapid dorsiflexion of the ankle, may also suggest upper motor neuron compression. Peripheral nerve injuries may cause sensory and/or motor abnormalities, depending on the damaged nerve. Peripheral nerves receive nerve fibers from a number of nerve root levels.

Lying supine on the examining table is an excellent position for testing the status of the nerve roots and peripheral nerves. The classic test of sciatic nerve (L4, L5, S1) irritation is the straight leg raising test, the purpose of which is to stretch the dura. The more useful straight leg raising test is done by raising the leg with the knee extended. When the sciatic nerve is stretched and its nerve roots and corresponding dural attachments are inflamed, the patient will experience pain along its anatomic course to the lower leg, ankle, and foot. Symptoms should not be produced in the lower leg until the leg is raised to 30 to 35°. Until that elevation, there is no relevant movement of the nerve within the dura. Between 50 and 60 to 70° tension is applied to the dura and nerve roots. The rate of deformation of the roots diminishes as the angle increases. Symptoms produced at elevations above 70° are thought to more likely represent joint or muscle-related pain.

The patient with a positive straight leg raising test (Lasègue sign) will have pain that radiates from the posterior thigh to the lower leg (below the knee). To confirm the presence of nerve irritability, the raised leg should be lowered until the pain is relieved. At that position, the foot is dorsiflexed, which will cause a recurrence of pain as a result of stretching of the posterior tibial branch of the sciatic nerve. Pain with dorsiflexion of the foot with hip flexion is commonly referred to as Bragard's test. It is critical that the straight leg raising tests be noted as positive only with replication of true radicular symptoms. Mere LBP from these signs is not indicative of neurological compromise and is frequently incorrectly recorded in clinical practices. Due to the frequency of these errors, it is best to note that the positive test produced radicular pain to, for example, the calf.

A bilateral straight leg raising test may also detect sciatic nerve irritation. The test is performed in the supine position by raising both legs by the ankles with knees extended. Raising both legs simultaneously tilts the pelvis upward, diminishing some of the tethering of the sciatic nerve. Therefore, the legs may be raised to a greater angle before radicular pain appears. Pain that occurs before 70° of motion is caused by stress on the sacroiliac joints. Above 70° of motion, pain is related to a lesion in the lumbar spine. When the examination reveals a psychogenic cause of pain, a bilateral straight leg raising test is routinely painful at a lower elevation than a unilateral test.

Observing the patient's stance and gait is useful to guide the regional low back examination. Incoordination or abnormal use of the extremities may suggest the need for specific neurologic testing. Severe guarding of low-back motion in all planes may add credence to a suspected diagnosis of spinal or

intrathecal infection, tumor, or fracture. However, because of the marked variation among patients with symptoms and those without, range-of-motion measurements of the low back are of limited value.

Vertebral point tenderness to palpation over spinous process(es), when associated with other signs or symptoms, is suggestive but not specific for spinal fracture or infection. Palpable soft-tissue tenderness by itself is an even less specific and less reliable finding. Waddell's signs are useful for assessing symptoms.(104)

B. Neurologic Screening

The neurologic examination focuses on a few tests that reveal evidence of nerve root impairment, peripheral neuropathy, or spinal cord dysfunction. Most symptomatic herniated discs in the lumbar spine involve the L5 nerve root (exiting between the L4 and L5 vertebral bodies) or the S1 nerve root (exiting between the L5 vertebral body and the sacrum (regarding S1)). The clinical features of lumbosacral nerve root compression are summarized in Table 4.

1. Testing for Muscle Strength

There are no specific muscle tests for the L1 to L3 nerve roots. The iliopsoas, the main flexor of the hip, is innervated by L1, L2, and L3, and is tested by asking the patient to flex the hip against resistance. The L4 nerve root can best be tested by evaluating the strength of ankle inversion and the strength of the quadriceps (knee extension against resistance). However, the quadriceps are also innervated by L2 and L3. The L5 nerve root when compromised may cause weakness of the great toe extensor on the affected side. In severe cases, the ankle dorsiflexors also may be weak and if so, the patient will have foot drop during gait. The S1 root generally supplies the plantar flexors of the foot and ankle, but motor weakness in the foot is harder to detect due to the bulk and normal strength of these muscles (gastrocnemius, soleus). The recommended test to detect S1 root compromise is repeated toe raises, generally a set of 10 on each side. Hamstring weakness may also be detected by this test.

Table 4. Physical Examination Correlates of Lumbosacral Nerve Root Dysfunction

Root Level	Sensory Deficit	Motor Weakness	Reflex
L1	Upper anterior thigh below inguinal ligament to groin	Hip flexion – Iliopsoas	**Cremaster**
L2	Anterior mid-thigh – Level of L2-3 posterior	Hip flexion and adduction; occasional knee extension	**Cremaster**
L3	Lower anterior thigh and inner knee	Hip flexion and adduction; knee extension	Knee jerk*
L4	Back, radiating to lateral thigh and front and medial leg	Hip adduction; knee extension; foot dorsiflexion	Knee jerk*
L5	Back, radiating to lateral leg and dorsal and lateral foot	Foot and great toe extension; hip abduction	Medial hamstring
S1	Back, radiating to back of thigh and lateral leg and foot	Knee flexion; plantar flexion	Ankle jerk

*Note: patellar reflex diminishment is somewhat difficult to detect as the quadriceps are innervated by 3 nerve roots, thus detecting an asymmetric reflex is generally not present unless marked compromise of L4 or multiple nerve root involvement is present.

2. Circumferential Measurements

Muscle atrophy can be detected by bilateral circumferential measurements of the leg and thigh. This should be performed and recorded with specificity, e.g., with a tape measure and at identical levels of the leg and thigh such as 15cm below the inferior poles of the patellae in a seated position). Differences of less than 2 centimeters in measurement of the two limbs at the same level can be a normal variation, especially if the lesser measurement is on the non-dominant side. Symmetric muscle bulk and strength are expected unless the patient has a relatively long-standing neurologic impairment or disorder of the lower extremity muscle or joint.

3. Reflexes

Loss of or decrease in the ankle jerk reflex compared to the other side suggests interruption of the reflex arc, as may be found in S1 nerve root compromise such as L5-S1 disc herniation. For the other nerve root level commonly involved, L5 (L4-L5 disc), there is no reflex change except for the medial hamstring reflex or the posterior tibial tendon reflex, which is difficult to elicit. Patellar reflexes are rarely abnormal in radiculopathy patients due to the multiple myotomal innervations of the quadriceps. When abnormal, consider the L4 nerve root (L3-L4 disc).

4. Sensory Examination

Sensory examination for nerve root compromise in the low back includes pinprick and light-touch testing. In general, the dorsal foot (especially the first web space), ankle, and leg areas are correlated with the L5 root, and the lateral foot is correlated with the S1 root. It is important to remember the subjective nature of sensory testing and the influence that past examinations may have on a patient with a history of back problems. Light pinprick should not elicit a painful response. If it does, ask the patient if this replicates his or her typical LBP and if the pain is superficial or deep. If the pain *is* typical, or if it is described as deep, this suggests a non-organic basis for the pain.

5. Physical Examination Tests

To be most successful, the treatment of LBP must be based upon a correct diagnosis. For a variety of reasons, a patient's response on any single test may not be reflective of the presence of identifiable underlying pathology. When ambiguity or inconsistency in test results prompts a concern regarding the correct diagnosis or the appropriate treatment approach, corroborative testing may be recommended. A number of tests are employed to distinguish between physiologic and nonphysiologic responses. These are commonly called "Waddell signs,"(104) and were originally described in the chronic LBP patient. These signs have subsequently been expanded as relevant to the evaluation of acute LBP patients.(105, 106)

Waddell recognized five categories of physical examination findings that suggest major psychosocial factors are present in addition to whatever residual physical injury or illness may still be present. These signs are not thought to usually represent malingering or other conscious manipulation to deceive.(107) Patients with signs in two of the categories may require consideration of the role of psychosocial factors in their presentations, and those with signs in three or four of the categories should receive increased scrutiny. However, there is literature suggesting that just one sign portends a worse prognosis in acute LBP patients.(105, 108) Waddell's categories are tenderness, simulation, distraction, regional, and pain behaviors:

- *Tenderness* is considered positive for non-organic signs when there is widespread, superficial, non-anatomic discomfort generally found more than 2cm lateral to the spine.
- *Simulation* is assessed by two tests – axial loading and rotation simulation. **Axial loading** can be performed while the patient stands by the examiner who pushes down with a few pounds of force on the patient's superior scalp. This places no significant stress on the lumbar spine and should not change the patient's pain. If the patient reports that this gentle pressure increases the back pain intensity, or causes the pain to radiate to additional places, this is a non-organic finding. A modification is to have the patient put his or her own hands on the superior scalp and apply the downward or axial force. This modification would prevent the patient from attempting the illogical claim that he or she was injured by the physical examination, although it would be predicted to be

less sensitive. The other test is **rotation simulation**. While the patient is standing, the examiner holds the patient's wrists so that the wrists and forearms remain in contact with the patient's thighs. In this position, the examiner rotates the whole person (no significant spinal motion occurs) while asking if the pain changes. The non-organic pain response is when the patient perceives the twisting of the back as intensifying the existing pain or causing the pain to radiate to a new place.

▪ *Distraction* is assessed by the straight leg raising test performed in two different positions. The straight leg raising test is meant to detect irritation of the lumbar nerve roots by mechanically pulling on the sciatic nerve, and thus the root, as it goes around the posterior hip. Straight-leg raising should be tested in both the seated position (when the patient is unaware of the relevance to the back) and the supine position (when the patient is aware of this testing). When the patient is sitting, he or she should extend and flex the knee while being asked if there is any knee pain. The knee should then be left fully extended and the patient asked if passive toe motion changes the back or leg pain. If a true radicular component is present, the patient should not easily tolerate full extension of the knee with dorsiflexion of the ankle in the sitting position – the typical response of a true positive straight leg raise test would be instead for the patient to lean back and complain of radiating pain. If there is no such response in the seated position, but there is a positive lying straight leg raise with at least a 40° difference between the seated and recumbent straight leg raising tests, a non-organic basis for the pain is suggested. This is one of the non-organic signs. These tests are subjective and can be confusing if the patient is simply having generalized pain that is increased by raising the leg. Results of the test may be influenced by repeated examinations in patients with a recurrent history of back problems (a learned fear that since leg raising has hurt in past exam, the current exam will also be painful). A negative test is generally a good prognostic sign. A positive test for lumbar nerve root irritation generally produces pain that radiates below the knee and that follows a precise radicular distribution consistent with the nerve root involved. Crossed straight-leg raises are the most highly specific test of sciatic nerve tension.

▪ *Regional* includes assessment of non-physiologic weakness and sensory deficits. Non-organic weakness is typically widespread involving more than one myotome and not fitting with imaging/electrodiagnostic findings. True neurologic weakness still permits constant sustained muscle contractions, while non-organic weakness is typically a sudden "give way" pattern or a "cog-wheel" pattern.

▪ *Pain behaviors* is a fifth category. There are concerns that this category is potentially affected by observer bias and patient culture. However, there is literature to support some pain behaviors as reliable signs that psychosocial issues are distorting the patient presentation(109, 110) and do not necessarily imply malingering.(111-113)

C. Early Disability Prevention and Management Issues
As an example of the biopsychosocial model, initial patient management should include alertness to the presence or development of physical and psychosocial factors that can be barriers to recovery and, if not addressed, are thought to increase the probability of the development of delayed recovery or chronic pain.(114-119) Initial "yellow" flags drawing attention to these potential issues include excessive verbal attention to symptoms or physical features, inquiries about permanent impairments during an initial presentation, prior history of disability or impairment, family members with acquired disabilities, a history of mental health disorders, histories of substance abuse, an apparent overreaction on examination, and presence of other non-organic physical examination signs. Besides the issues noted above, some additional yellow flags include early signs of medication dependence, disproportionate

inactivity, fear avoidance, compliance/attendance problems, resistance to transitional work options, and provider shopping. See also the [Cornerstones of Disability Prevention and Management](#) guideline.

Management of the patient at this stage of treatment necessitates overcoming these identified barriers in order to facilitate functional recovery and patient autonomy. Avoidance of therapies that are not resulting in functional recovery or that foster treatment dependence should be terminated. In contrast to the "watch and wait" philosophy, it is increasingly recognized that better outcomes are associated with maintaining work status or early return to work and avoiding or resolving disability at the earliest possible time. These concepts recognize that chronicity of disability is the overriding barrier to ultimate benefit for the injured worker. For example, the provider should consider early discontinuation of ineffective treatment and avoidance of interventional procedures of questionable significant functional benefit. For more difficult cases, referral for psychosocial evaluation and/or single-or-interdisciplinary treatment options with a proven record of success may be needed. For providers familiar with these management concepts, early referral (including after the first visit) to a provider well versed in the conservative management of LBP is recommended upon the discovery of these signs.

Indications For Further Workup

Physical examination evidence of severe neurologic compromise that correlates with the medical history and test results may suggest a need for immediate evaluation and/or referral for definitive treatment. The examination may further reinforce or reduce suspicions of tumor, infection, fracture, or dislocation. A history of tumor, infection, abdominal aneurysm, or other related serious conditions, together with positive findings on examination, warrants further investigation or referral. A medical history that suggests pathology originating somewhere other than in the lumbosacral area may warrant examination of the knee, hip, abdomen, pelvis, or other areas.

Associated Factors, Risk Factors and Work-Relatedness

Most acute LBP is best modeled as a relatively sudden onset of pain in the context of a multifactorial disorder other than specific acute significant trauma (substantial slip, trip, or fall). The minority who sustained a significant traumatic event have workers' compensation claims that are largely non-controversial. As a method for determination of work-relatedness is already discussed detail in the Guideline on Work-Relatedness, this guideline will only briefly review back-specific issues.

Most patients either do not recall a specific event or recall an apparently trivial event even when job tasks are highly physical. Regardless of whether there was an obvious inciting event or not, the documentation of any initial event(s) along with the patient's job tasks is required and highly helpful for the patient's claim under most workers' compensation jurisdictional requirements. However, a prospective study addressing whether minor trauma causes significant permanent back pain showed that minor trauma is rarely the cause of serious low back illness, and when minor trauma and serious back pain are associated, it is when the back pain episode is potentially compensable.(120-122)

Recurrence of LBP is not uncommon and recurrences require adequate documentation of the inciting events if any. Physicians should distinguish between a temporary exacerbation of symptoms and a permanent aggravation of a back condition. Jurisdictions differ in defining permanent aggravations.(1) If an underlying, pre-existing condition is thought to be significantly aggravated or "flares up" in a worker at work, the purported aggravating event(s), prior medical course, prior extent of pain, and activity

limitations should be recorded. At subsequent follow-up appointments, the extent of pain and activity limitation after the aggravation should be tracked. Restoration to the prior activity level is the goal. When that level has been reached, in many jurisdictions the effects of the aggravation or exacerbation are said to have ceased, and a permanent aggravation has not occurred. At that point, "cure" of the aggravation has been accomplished. This also requires that the treating physician have an understanding of both the true risk factors for back pain and as well as the work the patient performs to adequately capture and evaluate this information. Specific descriptions of work-duty activities, weights, sizes, and the frequencies of objects lifted are all helpful. Although frequently too generic for usability, it is recommended that a job description be nevertheless obtained from employer, if possible, to attempt to assist the practitioner with understanding the patient's job demands and duties.

Associated Factors and Risk Factors for Non-specific Low Back Pain
There are many non-occupational factors that have been associated with LBP. The most consistent and strongest is a prior history of LBP, which is one of the factors also confirmed in prospective studies.(123-135) Aging has been associated with LBP in some studies,(136-139) but many do not support a relationship with non-specific LBP in contrast with degenerative spine conditions. Instead, aging has been consistently associated with degenerative back disorders.(12, 24, 140, 141) Additional reported risk factors for LBP include: smoking,(132, 137, 142-144) obesity(126, 132, 133, 136, 137, 139, 142-161) height,(160) high triglycerides,(162) hypertension,(144) genetic factors,(53, 141, 163, 164) poor general health,(114, 165) poor sleep,(132, 142, 166) pain-related fear,(114, 134) prolonged driving,(132) deconditioning,(167) and physical inactivity or lack of exercise.(132, 142, 144, 168) A pattern of increased risk associated with cardiovascular risk factors and cardiovascular risk factor scores has been observed.(144) A U-shaped relationship between physical activity and risk of LBP has been reported in two epidemiological studies.(169, 170)

A number of physical factors are reported to be associated with LBP, although most of the evidence is from retrospective studies without measured job factors. Yet, recent data from a prospective cohort study with measured job physical factors have supported high lifting forces, as measured by the Cumulative Lifting Index, as associated with increased risk of LBP.(124, 125, 128) Cross sectional studies have reported mostly unconfirmed associations between LBP and heavy physical work (particularly heavy awkward of heavy lifting),(131, 132, 137, 142, 148, 165, 171-178) lifting weights above shoulder level,(176) carrying,(139, 177) trunk in a bent or twisted posture,(134, 139, 142) prolonged or highly repeated bending, inability to change posture regularly,(134, 179) standing and walking,(180) frequent reaching, or forceful pushing or pulling,(176, 181) kneeling(176) or squatting.(176) Housework was shown to be a risk factor in a prospective cohort study.(124, 128) Prolonged sitting and whole body vibration(140, 142, 182-184) are also suggested by some to be contributors. Work with scaffolding is a reported association.(165) These activities are not exclusive to job functions and should be reviewed as they pertain to non-occupational activities as well. Unaccustomed physically-demanding work (or sports or hobbies), another probable risk factor, is under recognized and may be fairly potent.

Until recently, prospective data supporting work-relatedness of LBP were limited. Recent data suggest increased risk of LBP as assessed by the Cumulative Lifting Index that was derived from the Revised National Institute for Occupational Safety and Health (NIOSH) Lifting Equation.(124, 125, 128, 185) Yet, support for degenerative disorders remains unsubstantiated.

Reduced lifting programs have been found to be successful at reducing risk of LBP in settings of manual patient transfers,(186-191) but not in most other settings. Programs have been ineffective for stress

management, shoe inserts, insoles, back supports.(192) Lifting advice and training also do not appear effective.(193)

It has also been theorized that these "stressors" do not cause back disorders. Rather, when a back disorder arises in an individual who does heavy physical work, the work is then more difficult to accomplish and the individual is more likely to file a workers' compensation claim. This is compared to the sedentary worker who develops back pain and may continue to perform work though more carefully (reporting bias).(194, 195)

Psychosocial factors, both occupational and non-occupational, also have been reportedly associated with back disorders.(196) These include task enjoyment, monotony,(176) mental stress,(142, 176) work stress,(137) job dissatisfaction,(124, 197) life dissatisfaction,(142) high demand/low control,(165, 166) low supervisor support,(166) low co-worker support,(166) and social isolation.(132) Psychiatric symptoms such as anxiety, depression,(124, 128, 131, 198) low energy,(132) emotional problems,(132) and somatization all are apparent risk factors. Providers with high fear avoidant beliefs also may contribute by prescribing more sick leave, bed rest, and less return to normal function.(199, 200) Many cases of LBP in the general population are idiopathic and the mechanism of LBP has not yet been elucidated.

Associations with Degenerative Spine Conditions including Sciatica
There are no quality studies of degenerative spine conditions including radiculopathy, and thus no true job physical risk factors are known. There is a poor correlation between LBP and degenerative findings on imaging studies,(12) as well as between LBP and MRI findings of disc protrusion, nerve root displacement or compression, disc degeneration, and high intensity zone.(58) The prevalence of nerve root contact is 11 to 23% and for displacement and/or compression 2 to 5%. Overall prevalence of disc degeneration in asymptomatic people is 54%, with a strong relationship with age.(58) Prevalence of HIZ or anular tear overall is 28 to 56%.(201)

Risk factors for degenerative back conditions that include spinal stenosis are not well defined compared with those for non-specific LBP. Nutrient vessels disappear to the disc, requiring diffusion.(202) This may provide a mechanistic explanation for cardiovascular disease risk factor impacts, particularly on degenerative spine disorders.(144) Degenerative disc changes have been well linked with inheritance,(53, 141, 163, 164, 203-206) and genetic influences on the outcomes of spine surgery have also been reported.(207, 208) Available epidemiological studies suggest the risk factors for degenerative conditions include aging,(12, 24, 140) male gender,(24, 209-211) obesity,(24) heredity,(12) and systemic arthrosis.(212) Reported risks for spondylolysis include increasing age and male gender.(24) Risks for degenerative spondylolisthesis include age and female gender.(24) Risks for facet joint arthritis are increasing age and obesity.(24) A trend towards greater spinal stenosis in those with a BMI >30 has been reported,(24) but that study is likely underpowered. There are no quality ergonomic-epidemiological studies reported for degenerative spine conditions and job physical factors.

There are no proven risk factors for radiculopathy as it is a relatively rare event and quality epidemiological studies have not been reported. However, heavy lifting and activities that substantially increase the intradiscal pressures are theorized factors. Prolonged whole-body vibration such as prolonged driving is a reported, but disputed factor.(182) Aside from age, smoking appears to be a factor. Spondylolisthesis is most often degenerative in nature. There are acute trauma-related cases in which causal analysis is straight forward and centers on whether the inciting trauma was in the context of work and that the magnitude of the event was sufficient to truly be an acute traumatic event.

There are no quality epidemiological studies that support the theory that degenerative spondylolisthesis, spinal stenosis, degenerative facet disease, or sciatica/radiculopathy are occupational conditions. However, there is a biomechanical theory that physical factors may contribute through degenerative disease in the discs with resulting theoretically altered biomechanical forces in the facets resulting in or accelerating degenerative facet osteoarthrosis. Yet, there also is evidence that these conditions may have a genetic basis.(213, 214)

Follow-up Visits

It is recommended that patients with potentially work-related low back disorders should follow-up every 3 to 5 days with a health care provider who can offer subsequent assessments and counseling regarding advancing activity levels, avoiding static positions or inactivity, medication use, anticipated favorable prognosis, and other concerns [**Recommended Insufficient Evidence (I)**]. Interactive sessions may assist involving the patient fully in his or her recovery. If the patient has returned to work, these interactions may be conducted on site or by telephone to avoid interfering with work activities. Subsequent follow-up can occur when there is need for: 1) altered treatment; 2) release to modified, increased, or full duty; or 3) after appreciable healing or recovery can be expected. Typically, this will be no later than 1 week into the acute pain period. At the other extreme, in the stable chronic LBP setting, follow-up may be infrequent, such as every 6 months.

Special Studies and Diagnostic and Treatment Considerations……………..

Detailed discussion of various imaging studies follows this section. Lumbar spine x-rays are not recommended in patients with LBP in the absence of red flags for serious spinal pathology within the first 4 to 6 weeks. Among patients with evidence of radiculopathy, imaging in the acute pain setting is also not recommended as the natural history is for such problems to resolve with conservative care. Table 5 provides a general comparison of the abilities of different techniques to identify physiologic insult and define anatomic defects. An imaging study may be appropriate for a patient whose limitations due to consistent symptoms have persisted for 1 month or more to further evaluate the possibility of potentially serious pathology such as a tumor.

Table 5. Ability of Various Techniques to Identify and Define Low Back Pathology and Sequela

Technique	Low Back Pain	Disc Herniation/ Protrusion	Cauda Equina Syndrome	Spinal Stenosis	Post-laminectomy Syndrome
History	++++	+++	+++	+++	+++
Physical examination	++	+++	++++	++	++
Laboratory studies	0	0	0	0	0
Imaging studies Radiography[1]	0	+	+	+	+
Computerized tomography (CT)[1,2]	0	+++	+++	+++	++
Magnetic resonance imaging (MRI)[1,2]	0	++++	++++	+++	++++
Electromyography (EMG), sensory evoked potentials (SEPs)[3]	0	+++	0 / +	++	+

[1]Risk of complications (e.g., infection, radiation) highest for myeloCT, second highest for myelography, and relatively less for bone scan, radiography, and CT.
[2]False-positive results in up to 30% of people over age 30 who do not have symptoms and may be over 50% in those over age 40.
[3]EMG is generally unhelpful in the first month of symptoms other than to document prior disease or injury status.

Diagnostic Testing and Other Testing

Diagnostic tests can be categorized into three broad categories: 1) anatomical; 2) functional; and 3) physiological. Anatomical tests help to define anatomy and include roentgenograms, magnetic resonance imaging (MRI), bone scans, computerized tomography (CT), and myelograms. Functional tests include those that assess voluntary lifting or pushing or pulling capacities. Physiological tests include electromyography and thermography. Tests such as discography attempt to bridge the gap between two of these testing domains and are organizationally included in this document in one domain. In considering which test to order, it is important to be able to address two key questions:

1. What is the specific question to be addressed?
2. What will be done with the results?

The first question must be clearly addressed and the second must result in an unequivocal answer used for a decision point with the results having a significant probability of altering the clinical management. Otherwise, the test is almost never indicated.

The operant characteristics of the test being ordered are critical to the proper interpretation of the results. For example, lumbosacral spine MRIs are more likely to be "abnormal" by age 40 in normal individuals (show normal aging changes), and herniated discs are not infrequently found in screening studies of asymptomatic teenagers. The pre-test probability of disease, determined by a careful clinical evaluation is critical to address the probability that the abnormality identified on the image is actually causing the individual's symptoms. At present, there is not one type of imaging method that shows a clear advantage over others. Generally, MRI is superior for imaging soft tissue including intervertebral disc herniations.

There are many additional diagnostic tests possible for the evaluation of LBP and spinal conditions. In the absence of moderate- to high-quality studies, other tests are **Not Recommended, Insufficient Evidence (I)**.(9)

Functional Capacity Evaluations

Functional capacity evaluations (FCEs) consist of a comprehensive battery of performance-based tests to attempt to determine an individual's ability for work and activities of daily living.(35, 118, 215-236) The goals of FCEs include:

- determine individual's readiness to work after injury or illness at Maximum Medical Improvement (MMI),
- assist with goal-setting and treatment planning for rehabilitation or to monitor the progress of a patient in a rehabilitation program,
- estimate potential vocational status and provide a foundation for effective vocational rehabilitation,
- provide information to assist in disability determinations,
- provide information for hiring decisions (post-offer or fit-for-duty testing),
- assess the extent of disability in litigation cases, and
- provide information regarding a patient's level of effort and consistency of performance.

1. *Recommendation: Functional Capacity Evaluations for Chronic Disabling Low Back Pain*

Functional capacity evaluations (FCEs) are a recommended option for evaluation of disabling chronic LBP where the information may be helpful to attempt to objectify worker capability, function, motivation, and effort vis-à-vis either a specific job or general job requirements. There are circumstances where a patient is not progressing as anticipated at 6 to 8 weeks and an FCE can evaluate functional status and patient performance in order to match performance to specific job demands, particularly in instances where those demands are medium to heavy. If a provider is comfortable describing work ability without an FCE, there is no requirement to do this testing. Recordings of observation for signs of mismatch between effort and self-reported abilities may be particularly helpful.

Harms – Medicalization, worsening of LBP with testing; may have misleading results that understate capabilities.

Benefits – Assess functional abilities and may facilitate greater confidence in return to work.

> *Strength of Evidence* **– Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Moderate

2. *Recommendation: Functional Capacity Evaluations for Chronic Stable Low Back Pain or Post-Operative Recovery*

There is no recommendation for or against the use of functional capacity evaluations for chronic stable low back pain or after completion of post-operative recovery among those able to return to work.

> *Strength of Evidence* **– No Recommendation, Insufficient Evidence (I)**
> *Level of Confidence* – Low

3. *Recommendation: Functional Capacity Evaluations for Acute Low Back Pain, Acute or Subacute Radicular Syndromes, or Post-Operative Back Pain*

Functional capacity evaluations are not recommended for evaluation of acute low back pain, acute or subacute radicular syndromes, or post-surgical back pain problems within the first 12 weeks of the post-operative period.

> *Strength of Evidence* **– Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – High

Rationale for Recommendations
FCEs are one of the few means to attempt to objectify limitations and are frequently used in workers' compensation systems, particularly as the correlation between pain ratings and functional abilities appears weak.(237-243) Yet, obtaining objective data regarding spine problems is somewhat more challenging than for extremity-related impairments due to the degree of reliance on the patient's subjective willingness to exert or sustain major activities (e.g., standing, walking, sitting) that are critical for job performance. Because their reliability and validity have not been proven, FCEs should be utilized to evaluate work ability about what a patient was willing to do on a given day. They should not be used to override the judgment about the work ability of a patient with a back problem.

Many commercial FCE models are available. There is research regarding inter-and intra-rater reliability for some of the models (complete discussion is beyond the scope of this guideline). The validity of FCEs, particularly predictive validity, is more difficult to determine, since factors other than physical performance may affect return to work.(217, 244) An FCE may be done for one or more reasons, including identifying an individual's ability to perform specific job tasks associated with a job (job-

specific FCE) and physical activities associated with any job (general FCE), or to assist in the objectification of the degree(s) of impairment(s). The type of FCE needed, and any other issues the FCE evaluator needs to address, should be specified when requesting a FCE.

The term "capacity" used in FCE may be misleading, since an FCE generally measures an individual's voluntary performance rather than his or her capacity. Physical performance is affected by psychosocial as well as physical factors. The extent of an individual's performance should be evaluated as part of the FCE process through analysis of his or her level of physical effort (based on physiological and biomechanical changes during activity) and consistency of performance. Perhaps more importantly, the objective findings identified in the musculoskeletal evaluation should correlate with any identified functional deficits. The individual's performance level, especially as it relates to stated levels of performance, should be discussed in the FCE report. A properly performed and well-reported FCE will highlight such discrepancies. This is particularly important in low back evaluations where there may be greater degrees of impairments at stake and where there are somewhat fewer metrics available than for the distal upper extremity.

FCE test components may vary depending on the model used, but most contain the following:
- Patient interview including:
  - Informed consent
  - Injury/illness and medical history
  - Current symptoms, activities and stated limitations
  - Pain ratings/disability questionnaires
- Musculoskeletal examination (e.g., including Waddell's non-organic signs)
- Observations throughout the session (e.g., demonstrated sitting tolerance, pain modifying behaviors)
- Material handling tests (lifting, carrying, pushing, pulling)
- Movement tests (walking, crouching, kneeling, reaching, etc.)
- Positional tolerance tests
- Dexterity/hand function
- Static strength (varies among models)
- Aerobic fitness (usually submaximal test-also variable among models)
- Job specific activities as relevant
- Reliability of client reporting (e.g., non-organic signs, pain questionnaires, placebo tests, etc.)
- Physical effort testing (e.g., Jamar Dynamometer maximum voluntary effort, bell curve analysis, rapid exchange grip, competitive test performance, heart rate, observation of clinical inconsistencies, etc.)

FCE test length may vary between FCE models, although most 1-day FCEs are completed in 3 to 4 hours. Two-day tests, where the patient is seen on 2 consecutive days, may be recommended when there are problems with fatigue (e.g., chronic fatigue syndrome), delayed onset of symptoms, unusually complex job demands to simulate, and questions about symptom validity. Test length for 2-day tests is generally 3 to 4 hours on the first day, and 2 to 3 hours on the second day.

Interpretation of FCE results is complicated in that it is a measure of voluntary performance. Before beginning testing, the patient is counseled to avoid doing anything to knowingly reinjure him or herself.

Thus "fear avoidance" may cause testing to seriously underestimate actual ability and result in a report that the patient had "self-limited performance due to pain," suggesting a low pain tolerance, when in reality the patient was doing what he or she was instructed.

The best studies on the ability of FCEs to predict safe re-entry to the workplace following rehabilitation of work-related back pain/injury suggest that FCEs are not able to predict safe return to work (concurrent validity).(218, 245, 246) In a prospective cohort study of 1,438 consecutive work-related back patients, all underwent a FCE prior to return to work. In the control group, the FCE was used to write return-to-work guidelines, while in the study group it was ignored and the worker was returned usually to full duty. Ignoring the FCE improved outcome.(247)

Evidence for Use of Functional Capacity Evaluations (FCEs)
We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: functional capacity evaluations, FCE, chronic low back pain, postoperative recovery, acute low back pain, acute radicular pain, subacute radicular pain, postoperative back pain, diagnostic, sensitivity, specificity, predictive value, efficiency, and efficacy to find 781 articles. Of the 781 articles, we reviewed 10 and included five articles.

ROENTGENOGRAMS (X-RAYS)
X-rays are commonly utilized for evaluation of LBP, particularly that which is chronic, persistent and accompanied by red flags or trauma.(248, 249) Similar to most diagnostic studies, MRI is usually considered the gold standard comparison.

1. *Recommendation: X-ray for Acute Non-specific Low Back Pain*
   Routine x-ray is moderately not recommended for acute non-specific low back pain.

   > *Strength of Evidence* – **Moderately Not Recommended, Evidence (B)**
   > *Level of Confidence* – High

2. *Recommendation: X-ray for Acute Low Back Pain with Red Flags or Subacute or Chronic Low Back Pain*
   X-ray is recommended for acute low back pain with red flags for fracture or serious systemic illness, subacute low back pain that is not improving or chronic low back pain as an option to rule out other possible conditions.

   Indications – Option to rule out other possible conditions.

   Frequency/Duration – Obtaining x-rays once is generally sufficient. For patients with chronic LBP, it may be reasonable to obtain a second set of x-rays years later to re-evaluate the patient's condition, particularly if symptoms change.
   Harms – Medicalization or worsening of otherwise benign back condition; radiation exposure.
   Benefits – Diagnosis of a fracture or otherwise latent medical condition(s).

   > *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
   > *Level of Confidence* – High

3. *Recommendation: X-ray for Spondylolisthesis*
   Flexion and extension views are recommended for evaluating symptomatic spondylolisthesis in which there is consideration for surgery or other invasive treatment or occasionally in the setting of trauma.

Indications – Chronic severe mechanical pain suspected to be due to instability.

Frequency/Duration – Flexion and extension views are generally needed no more than every few years. However, after surgical intervention, flexion/extension views may be used to attempt to assess extent of successful fusion.

Harms – Medicalization or worsening of otherwise benign back condition. Radiation exposure.

Benefits – Diagnosis of significant spondylolisthesis that is able to be surgically improved.

> *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Moderate

Rationale for Recommendations
Standard film views are generally an anterior-posterior (AP) film, a lateral film, and on occasion, a coned or focused view of the L5-S1 joint. Routine inclusion of oblique views has been discouraged except in specific circumstances, such as an evaluation of trauma where the AP and lateral views fail to show a fracture but there remains significant concern that a fracture did occur.(250) Oblique views are also needed if there is reason to evaluate a pars defect. If an MRI is used as imaging, plain x-ray may not be needed.

Flexion and extension films are occasionally used to evaluate spinal instability, particularly in the setting of degenerative spondylolisthesis and fractures. The criteria generally accepted for this purpose are to measure whether there is 5mm or more of movement of one vertebral body in relation to an adjacent vertebral body, or whether the angular motion measured on radiographs at a disc given level exceeds 20° for the L1-L2 level through the L4-L5 level, or exceeds 25° for the L5-S1 level.(251) Depending on the translation forward or backwards, referred to as anterolisthesis or retrolisthesis.

X-ray is unnecessary for the routine management of LBP outside of the setting of red flags.(252-255) When red flag(s) are present, x-rays at the first visit are usually recommended to assist in ruling out these possible conditions (e.g., fracture, neoplasias, infection, etc.). Without red flags, there also is concern for medicalization and catastrophization of the case by obtaining x-rays.(256) Even when red flags are suspected, judgment is recommended and it should not be mandatory to order an x-ray in all cases (e.g., significant typical LBP in the course of a manual patient transfer in a patient with a remote history of cancer). In the event that there is LBP without any improvement over 4 to 6 weeks, x-rays may be recommended to rule out other possible problems. Those with subacute LBP that is not improving or chronic LBP should generally have x-rays at least once for purposes of ruling out other conditions. X-rays are non-invasive, moderately costly, and have a low risk of adverse effects, other than their considerable exposure to ionizing radiation. Thus, x-rays are recommended for select situations. The radiation dosage from common medical tests is available from the Australian Radiation Protection and Nuclear Safety Agency at **https://www.arpansa.gov.au/understanding-radiation/what-is-radiation/ionising-radiation/x-ray**, and further reviewed in scientific literature.(257, 258)

Evidence for the Use of Roentgenograms (X-ray)
There are 5 moderate-quality studies incorporated into this analysis.(253-255, 259) There is 1 low-quality studies in Appendix 1.(260)

We searched PubMed, Ebsco, Cochrane Review and Google Scholar with limits between 2008 and 2013. We used the following search terms: X-rays, roentgenograms, radiography, acute low back pain, subacute low back pain, chronic low back pain, spondylolisthesis, low back pain, diagnostic, sensitivity,

specificity, negative predictive value, positive predictive value, efficiency, and efficacy to find 258 articles in PubMed, 548 in EBSCO, 11 on Cochrane Review, and 173,720 on google scholar, for a total of 174, 537. From the 174, 537 articles, we reviewed 11 articles, and included 9 in the draft (5 RCTs, 3 reviews, 1 cross sectional study).

MAGNETIC RESONANCE IMAGING (MRI)

Magnetic resonance imaging (MRI) has been widely used to evaluate the lumbar spine, particularly soft-tissues such as the intervertebral discs.(248, 261-271) This discussion will cover the three types of MRI – open, closed, and standing or weight-bearing.

Several terms are used to describe disc abnormalities and five different terms are used to describe a change in disc shape that can potentially cause radicular symptoms (bulge, protrusion, extrusion, sequestration, and herniation). There are multiple "definitions" of these terms, which creates confusion, but a consensus conference has provided definitions that may facilitate communication.(32)

Table 6. Terms Used to Describe Disc Abnormalities/Change in Disc Shape

Term	Definition
Normal	Does not reach beyond the borders of adjacent vertebral bodies.
Bulging	A circumferential symmetric extension of the disc beyond the vertebral border.
Herniation	Localized displacement of disc material beyond the limits of the intervertebral disc space. Disc material may be nucleus, cartilage, fragmented apophyseal bone, anular tissue, or any combination thereof. The term "localized" contrasts to "generalized," the latter arbitrarily defined as >50% (180°) of the periphery of the disc. Localized displacement in the axial (horizontal) plane can be "focal," signifying <25% of the disc circumference, or "broad-based," meaning between 25 and 50% of the disc circumference. Presence of disc tissue "circumferentially" (50-100%) beyond the edges of the ring apophyses may be called "bulging" and is not considered a form of herniation. Herniated discs may take the form of protrusion or extrusion, based on the shape of the displaced material.
Protrusion	Present if the greatest distance, in any plane, between the edges of the disc material beyond the disc space is less than the distance between the edges of the base in the same plane. In the cranio-caudal direction, the length of the base by definition cannot exceed the height of the intervertebral space.
Extrusion	Present when, in at least one plane, any one distance between the edges of the disc material beyond the disc space is greater than the distance between the edges of the base or when no continuity exists between the disc material beyond the disc space and that within the disc space. Extrusion may be further specified as sequestration if the displaced disc material has completely lost any continuity with the parent disc.
Sequestration	A herniated disc fragment that is detached and separated from the disc. It may or may not appear to have migrated cephalad or caudally.
Migration	Signifies displacement of disc material away from the site of extrusion, regardless of whether sequestrated or not. Because posteriorly displaced disc material is often constrained by the posterior longitudinal ligament, images may portray a disc displacement as a protrusion on axial sections and an extrusion on sagittal sections, in which cases the displacement should be considered an extrusion.
Intravertebral Herniations	Herniated discs in the cranio-caudal (vertical) direction through a break in the vertebral body endplate.

Adapted from Fardon DF, Milette PC. Nomenclature and classification of lumbar disc pathology: recommendations of the Combined Task Forces of the North American Spine Society, American Society of Spine Radiology, and American Society of Neuroradiology. *Spine*. 2001;26(5):E93-113.

1. *Recommendation: MRI for Diagnosing Red Flag Conditions*
   MRI is recommended for patients with acute low back pain during the first 6 weeks if they have demonstrated progressive neurologic deficit, cauda equina syndrome, significant trauma with no improvement in atypical symptoms, a history of neoplasia (cancer), persistent fever plus elevated

erythrocyte sedimentation rate without other infectious source, or atypical presentation (e.g., clinical picture suggests multiple nerve root involvement).

Harms – Medicalization or worsening of otherwise benign back condition.

Benefits – Diagnosis of a surgically treatable condition or otherwise latent medical condition(s).

> *Strength of Evidence –* **Recommended, Insufficient Evidence (I)**
> *Level of Confidence –* High

2. *Recommendation: Early MRI for Diagnosing Radicular Syndrome*
MRI is moderately not recommended for acute radicular pain syndromes in the first 6 weeks unless the problems are severe and not trending towards improvement and both the patient and the clinician are willing to consider prompt surgical treatment, assuming the MRI confirms ongoing nerve root compression. Repeat MRI imaging without significant clinical deterioration in symptoms and/or signs is also not recommended.

> *Strength of Evidence –* **Moderately Not Recommended, Evidence (B)**
> *Level of Confidence –* Moderate

3. *Recommendation: MRI for Diagnosing Subacute and Chronic Radicular Syndromes*
MRI is moderately recommended for patients with subacute or chronic radicular pain syndromes lasting at least 4 to 6 weeks in whom the symptoms are not trending towards improvement if both the patient and clinician are considering prompt surgical treatment, assuming the MRI confirms a nerve root compression consistent with clinical examination. In cases where an epidural glucocorticosteroid injection is being considered for temporary relief of acute or subacute radiculopathy, MRI at 3 to 4 weeks (before the epidural steroid injection) may be reasonable. It is recommended to administer with and without contrast in post-operative settings when there are concerns about recurrent disc problems (see Epidural Glucocorticosteroid Injections).

Harms – Medicalization or worsening of otherwise benign back condition.

Benefits – Diagnosis of a surgically treatable condition or otherwise latent medical condition(s).

> *Strength of Evidence –* **Moderately Recommended, Evidence (B)**
> *Level of Confidence –* High

4. *Recommendation: MRI for Diagnosing Select Chronic LBP*
MRI is recommended as an option for the evaluation of select chronic LBP patients in order to rule out concurrent pathology unrelated to injury. This option is not recommended before 3 months and only after other treatment modalities (including NSAIDs, aerobic exercise, and directional preference exercises) have failed.

Harms – Medicalization or worsening of otherwise benign back condition.

Benefits – Diagnosis of a surgically treatable condition or otherwise latent medical condition(s).

> *Strength of Evidence –* **Recommended, Insufficient Evidence (I)**
> *Level of Confidence –* Moderate

5. *Recommendation: Standing or Weight-bearing MRI for Back or Radicular Pain Syndrome Conditions*
Standing or weight-bearing MRI is not recommended for back or radicular pain syndrome conditions as, in the absence of studies demonstrating improved patient outcomes, this technology is experimental.

Strength of Evidence – **Not Recommended, Insufficient Evidence (I)**
Level of Confidence – Moderate

Rationale for Recommendation: Closed MRIs
MRI has been evaluated in quality studies. The sensitivity and specificity of MRI or CT are difficult to define as they require a "gold standard" that is difficult to define in back pain since the final diagnosis often is based on the same imaging modality being tested; therefore, these clinical studies may be prone to incorporation bias, artificially inflating the sensitivity and specificity with some assuming MRI has 100% sensitivity and specificity. Most cases of LBP and radicular pain syndromes spontaneously resolve and require no imaging. Disc degeneration, disc bulging and herniation, and endplate changes are widely prevalent in asymptomatic people on MRI(121, 201, 272-289) have been shown to either not correlate, or correlate poorly with symptoms,(121, 201, 278-280, 282, 284, 289-291) suggesting that MRI is not useful for the vast majority of patients.(292) In a 17-year follow-up study, patients with LBP at age 20 who had degenerative changes on MRI have greater risk for more severe degenerative changes. However, there was almost no correlation with clinical outcomes and no increased risk of surgery.(293) Early imaging likely results in higher overall costs and increased morbidity through the performance of some unnecessary procedures and/or surgeries.

Despite disc degeneration, bulging, herniations, and endplate changes that are widely prevalent on MRI in asymptomatic people, MRI is still considered the gold standard in diagnostic imaging for defining anatomy because it typically has the greater ability to distinguish soft tissues of any test currently available.(261-265, 267-269, 271) While computerized tomography (CT) remains an important analytical tool especially for evaluating bony or calcified spinal structures, there is less need for CT at the current time as MRI has greater soft tissue resolution. In patients of reproductive age, MRI may be preferable for the diagnosis of disc herniation, as CT involves considerable ionizing radiation. An evaluation of the association between the rates of advanced spinal imaging and spine surgery across geographic areas concluded that a significant proportion of the variation in rates of spine surgery can be explained by differences in the rates of advanced spinal imaging. "Improved consensus on the use and interpretation of advanced spinal imaging studies could have an important effect on variation in spine surgery rates."

In the absence of red flags suggesting fracture or serious systemic illness, imaging before 6 weeks produces no clear benefits. MRI is either non- or minimally-invasive and has few adverse effects, but is costly. In the absence of red flag symptoms and/or signs, MRI is not recommended to reassure patients that no serious injury or disease is present.(294) MRI is not recommended for evaluation of acute, subacute, or nearly all chronic LBP cases. MRI is indicated for discrete, potentially surgically treatable disorders such as radiculopathy, spondylolisthesis, and spinal stenosis.

Radicular pain syndrome patients should not have MRI within the first 6 weeks, except in rare cases for which early emergent/urgent surgery is proposed. Patients presenting with single nerve root neurological deficit, including an absent deep tendon reflex, should not have early MRI, as their condition usually resolves spontaneously, thus the test does not alter the course of treatment. Those who have a documented presentation that then objectively deteriorates (particularly a significant increase in weakness, an increased loss of sensation, compared with the prior examination, cauda equina syndrome, history of cancer with symptoms suggesting atypical radicular presentation) do have an indication for early imaging with MRI. It is strongly recommended that those ordering MRIs should be

well aware of the tremendously high prevalence of abnormalities, which are essentially "false positives" in otherwise normal people (285).

Patients should be *a priori* informed that their MRI is highly unlikely to be "normal" as few have a normal MRI. A patient handout describing the prevalence of "abnormal findings" on lumbar MRI of asymptomatic individuals is helpful. Providers lacking the time or knowledge to explain these facts to patients should avoid ordering MRIs. The discovery of degenerative changes or clinically irrelevant disc herniations in many may cause them to focus on the need to "fix" MRI changes that are actually normal for their age or are asymptomatic findings. This may also become a rationale for avoiding participation in the therapeutic activities that promote functional recovery. In addition, lack of understanding of the strengths, indications, and limitations of a technology preclude adequate clinical interpretation of the results. In those cases, consultation with a provider experienced in treating musculoskeletal disorders may be recommended.

Rationale for Recommendation:Open MRIs
Open MRIs have gained in popularity. However, they have lower resolution without lower costs and are not recommended other than when the patient's weight exceeds the closed MRI unit's specifications, or suffers from claustrophobia that is not sufficiently alleviated with a pre-procedure low-dose anxiolytic.

Rationale for Recommendation: Standing ("Upright" or "Positional") MRIs
Standing MRI units are designed to evaluate the discs and spine under usual conditions of axial loading and can be used in other positions. Magnets are typically weaker than conventional MRI, resulting in lower resolution ("fuzzier images"). These units have unsurprisingly revealed a modestly greater prevalence of disc bulging with the spine loaded.(295, 296) There are studies demonstrating higher prevalence rates of disc herniations with upright-sitting examinations and an overall estimation of superiority for detections of spine abnormalities. These findings have not been shown to improve patient outcomes.(297) Another study of asymptomatic volunteers demonstrated a 41% prevalence rate for disc bulges.(298) There is a case report of positive findings where a closed MRI did not show neurological impingement.(299) One study noted that the information gained in addition to that from standard MRIs is limited.(300) Another comparative study in multiple positions concluded that positional MRIs more frequently demonstrate minor neural compromise than conventional MRI and that positional pain differences are related to position-dependent changes in foraminal size.(301) There are currently no quality studies to recommend standing MRI for uses outside of research settings, and interpretation of normal findings of increased disc bulging with standing are unclear.

Table 7. Change in MR Findings at 6-week Follow-up

Change in MR Findings at 6-week Follow-up		
Finding	No. of Patients with LBP	No. of Patients with Radiculopathy

Degenerative disc disease		
Normal at Baseline		
Unchanged	41 (91.1)	22 (84.6)
New herniation	4 (8.9)	4 (15.4)
Herniation at baseline		
Unchanged	46 (69.6)	25 (54.3)
New and/or enlarged	10 (15.2)	5 (10.9)
Reduced or gone	10 (15.2)	16 (34.8)
Nerve root compression		
Normal at baseline		
Unchanged	74 (91.4)	37 (97.4)
New compression	7 (8.6)	1 (2.6)
Compression at baseline		
Unchanged	21 (70.0)	18 (52.9)
New and/or worse	4 (13.3)	6 (17.7)
Reduced or gone	5 (16.7)	10 (29.4)
No 6-week MR imaging	39	24

Note: Data in parentheses are percentages.

Modic MT, Obuchoski NA, Ross JS, et al. Acute low back pain and radiculopathy: MR imaging findings and their prognostic role and effect on outcome. *Radiology*. 2005;237:597-604. Reprinted with permission from the Radiological Society of North America.

Evidence for the Use of Magnetic Resonance Imaging (MRI)
There are 8 high-quality(121, 263, 268, 290, 302-305) and 30 moderate-quality(261, 262, 265, 267, 271, 278, 284, 287, 292, 294, 306-325) studies incorporated into this analysis (see also Cervical and Thoracic Spine Disorders Guideline for additional studies). There is 1 low-quality study(259) and 2 other studies(326, 327) in Appendix 1. It is important to note that the sensitivity and specificity of CT or MRI are difficult to define as they require a "gold standard" that is difficult to define in back pain since the final diagnosis often is based on the same imaging modality being tested; therefore, these clinical studies may be prone to incorporation bias, artificially inflating the sensitivity and specificity with some assuming MRI has 100% sensitivity and specificity.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar with limits on publication dates from 2008-present. We used the following terms: magnetic resonance imaging, MRI, acute low back pain, subacute low back pain, chronic low back pain, diagnostic testing, sensitivity, specificity, positive predictive value, negative predictive value, efficacy, efficiency, and low back pain to find 58,060 articles. Of the 58,060 articles, we reviewed 20 articles (11 original articles, 4 review articles, and 5 new RCTs) and an addition 18 articles from references and 20 articles were included.

MRI for Evaluation of Non-specific Chronic Low Back Pain
See Cervical and Thoracic Spine Disorders Guideline.

Table 8. Findings of Lumbar MRI

Finding	Percentage
Normal disc signal	42%
Normal disc height	45%
Annular fissure	7%
Bulging disc	14%
Disc contact with nerve root	8%
Displacement of nerve root	2%

End plate changes	0.5%
Anterolisthesis	3%

Adapted from Kjaer P, Leboeuf-Yde C, Sorensen JS, Bendix T. 2005 and "Lumbar disc nomenclature: Version 2.0" Spine vol 19 n0 24 E1448-E14665 (280, 328)

A review of LBP found the following prevalence of "abnormalities" on MRI in asymptomatic individuals:

Table 9. Abnormalities on MRI in Asymptomatic Individuals

Finding	Number of Studies	Prevalence of Finding
Herniated disc	5	22-40%
Bulging disc	5	24-81%
Degenerative disc	4	46-93%
Stenosis	3	1-21%
Annular tear	3	14-56%

Adapted from Deyo RA, Weinstein JN. 2001.

COMPUTERIZED TOMOGRAPHY (CT)

Computerized tomography (CT) is primarily used today to define fractures not visible on plain x-rays or to image when MRI is unavailable or contraindicated.(329) CT was the main imaging study for defining spinal anatomy prior to the advent of MRI. Due to the greater soft tissue contrast of MRIs, there is less current need for CT.(248, 330)

1. *Recommendation: Routine CT for Acute, Subacute, or Chronic Non-specific Low Back Pain or Radicular Pain Syndromes*
   Routine CT is not recommended for acute, subacute, or chronic non-specific low back pain, or for radicular pain syndromes.

   Strength of Evidence – **Not Recommended, Evidence (C)**
   Level of Confidence – High

2. *Recommendation: CT for Patients with Acute or Subacute Radicular Pain Syndrome*
   CT is recommended for patients with acute or subacute radicular pain syndrome who failed to improve within 4 to 6 weeks and if there is consideration for an epidural glucocorticoid injection or surgical discectomy (see Epidural Steroid Injection). If there is strong consideration for surgery, then CT myelography should be considered instead of CT alone (see below).

   Indications – Patients with an indication for MRI who cannot complete the MRI due to contraindications such as implanted metallic-ferrous device or significant claustrophobia.

   Frequency/Duration – Obtaining serial CT exams is not recommended, although if there has been a significant worsening in the patient's history of examination, repeat imaging may be recommended.

   Harms – Medicalization or worsening of otherwise benign back condition. Radiation exposure.

Benefits – Diagnosis of a fracture or otherwise latent medical condition(s).
   Strength of Evidence – **Recommended, Evidence (C)**
   Level of Confidence – Moderate

Rationale for Recommendations
CT is equivalent to MRI for many typical spine imaging purposes. The sensitivity and specificity of CT or MRI are difficult to define as they require a "gold standard" that is difficult to define in back pain since the final diagnosis often is based on the same imaging modality being tested; therefore, these clinical studies may be prone to incorporation bias, artificially inflating the sensitivity and specificity with some assuming MRI has 100% sensitivity and specificity. CT is also widely thought to be sufficient to evaluate most patients with suspected disc herniations even though it is not as successful for soft tissue imaging.(331-333) CT is most useful to evaluate the spine in patients with contraindications for MRI (most typically an implanted metallic-ferrous device). CT is somewhat less costly than MRI. There also may be situations in which MRI is so distant geographically that CT is the most practical option. Contraindications for MRI that may necessitate CT include any implantable ferrous or metallic device and claustrophobia to an extent that even open MRI is infeasible or unavailable. CT myelography has limited uses, however, if there is a contraindication to MRI and surgery is considered moderate to high probability, then CT myelography is a consideration instead of CT followed by another CT with myelography. CT and MRI are both options for consideration before invasive procedures (e.g., acute severe radiculopathy with consideration of epidural glucocorticoid injection or surgery). CT is not invasive (minimally invasive when contrast is needed), has low potential adverse effects, but is costly.

Evidence for the Use of Computerized Tomography (CT)
There are 4 high-(334-337) and 4 moderate-quality(338-341) incorporated into this analysis. Please note that older generation machines were used in older studies rendering the results difficult to interpret in today's world.

We searched PubMed, EBSCO, Cochrane Review and Google Scholar with limits between 2008 and 2013. We used the following search terms: Computerized Tomography, CT scan, acute low back pain, subacute low back pain, chronic low back pain, acute radicular pain, subacute radicular pain, low back pain, radicular pain, diagnostic, sensitivity, specificity, negative predictive value, positive predictive value, efficiency, and efficacy to find 103 articles in PubMed, 413 in EBSCO, 1 on Cochrane Review, and 13,004 on Google Scholar, for a total of 13,521. From the 13,521 articles, we reviewed 12 articles, and included 6 in the draft (1 RCTs, 1 cross-sectional study, 1 case study, and 3 reviews).

MYELOGRAPHY (INCLUDING CT MYELOGRAPHY AND MRI MYELOGRAPHY)
Myelography is the injection of a radiocontrast media into the thecal sac with subsequent imaging. Historically, myelography with standard roentgenograms was the most common method to diagnose herniated discs, spinal stenosis, or other forms of neurological compromise.(342-345) It was subsequently paired with CT (CT myelography) or rarely MRI (MRI myelography). However, it has been almost completely replaced by MRI that produces superior resolution of images. Consequently, there may be little use for myelography,(346) though many spine surgeons use CT myelography to help with surgical decision-making in cases in which MRI is equivocal or not possible.

Recommendation: Myelography in Uncommon Situations

Myelography, including CT myelography, is recommended only in uncommon specific situations (e.g., contraindications for MRI such as implanted metal that preclude MRI, equivocal findings of disc herniation on MRI suspected of being false positives, spinal stenosis, and/or a post-surgical situation that requires myelography). MRI is preferred in most post-operative settings to distinguish, e.g., residual or recurrent disc problems.

Harms – Headache; rare infections or cord compromise; medicalization or worsening of otherwise benign back condition; radiation exposure.

Benefits – Diagnosis of significant neurological impingement that is able to be surgically improved.

      Strength of Evidence – **Recommended, Insufficient Evidence (I)**
      Level of Confidence – High

Rationale for Recommendation
The primary use of CT myelography today is for those with contraindications for MRI, such as implanted ferrous metal. Quality literature correlating surgical discectomy outcomes with CT myelogram results in cases with equivocal MRIs is sparse. However, MRI may well have false-positives for disc herniation, and CT myelograms may then confirm the "disc" seen on MRI is actually an osteophyte without nerve root compression. CT myelography is still considered by many spine surgeons to be the gold standard test for spinal stenosis. However, there are no recent quality studies to document this belief, rather there are small case series reporting continuing uses in evaluating neurological compromise based on positional changes.(347, 348) Myelogram/CT is not useful in determining a recurrent disc herniation. One cannot distinguish between normal post-operative fibrosis and a disc on CT/myelogram. This can only be reliably determined with MRI with and without contrast. Post-operative fibrosis with granulation tissue will enhance, whereas a disc with no vascularity does not enhance.

Myelography is substantially invasive compared with other imaging procedures because it involves a lumbar puncture.(349, 350) As such, a post-procedure headache is not uncommon and procedures (e.g., blood patching) are required when headaches are severe. Myelography is costly. It has been almost entirely replaced by MRI and other imaging procedures.(346) Myelography (as well as CT myelography and MRI myelography) is recommended only on a limited basis (see above) and is otherwise not recommended as the first diagnostic study for the diagnosis of lumbar nerve root compromise. Plain CT is not an adequate substitute for most patients meeting the above indications.

Evidence for the Use of Myelography
There are 2 high-(302, 303) and 2 moderate-quality(351, 352) incorporated into this analysis. There is 1 low-quality study in Appendix 1.(353)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: myelography, acute low back pain, subacute low back pain, chronic low back pain, and low back pain to find 1443 articles. Of the 1443 articles, we reviewed 5 articles and included 5 articles (5 epidemiological).

BONE SCANS
Bone scans involve intravenous administration of a radioactive tracer medication that is preferentially concentrated in areas of metabolic activity in bone. The radioactivity is then converted into skeletal images. Bone scans show increased radioactive uptake and are most commonly used for evaluating many types of metastases,(354, 355{Szot, 2014 #8863) infection, inflammatory arthropathies, occult fractures,(356-358) or other significant bone trauma.(359)

Recommendation: Bone Scanning for Low Back Pain
Bone scanning is not recommended for routine use in diagnosing low back pain. However, it has select use including for suspected metastases, occult fractures, and infectious complications. May help to distinguish acute versus old fractures.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – High

Rationale for Recommendation
Bone scanning is not used for evaluation of most LBP. However, it is a good diagnostic test for specific situations, including evaluations of suspected metastases, infected bone (osteomyelitis), inflammatory arthropathies, and trauma (fractures). Perhaps the most common use of bone scans for evaluating LBP is imaging of sacroiliac joints (one study reported that a combination of a quantitative bone scan and an HLA-B27 measurement were superior to MRI and CT scans for assessing sacroiliitis).(360) Bone scanning is minimally invasive, has no adverse effects aside from radiation exposure, but is costly. The combination of a bone scan and HLA-B27 is occasionally required when attempting to differentiate LBP that is occupational from ankylosing spondylitis, particularly in young males. Aside from specific indications which involve a minority of LBP patients, the routine use of bone scanning is not recommended in LBP patients.

Evidence for the Use of Bone Scanning
There are no quality studies evaluating bone scans for diagnosis of typical occupational LBP patients. Reported sensitivity and specificity were not satisfactory for evaluating chronic LBP patients and the population studied was felt to be too small to develop normative values.(361)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar with limits on publication dates from 2008-2013. We used the following terms: bone scans, acute low back pain, subacute low back pain chronic low back pain, diagnostic testing, sensitivity, specificity, positive predictive value, negative predictive value, efficacy, efficiency, and low back pain to find 69,215 articles. Of the 69,125 articles we reviewed zero articles and included zero articles.

SINGLE PROTON EMISSION COMPUTED TOMOGRAPHY (SPECT)
Single proton emission computed tomography (SPECT) is a 3-dimensional imaging technique. For evaluation of LBP issues it has been primarily used for the diagnosis of inflammatory arthropathies, particularly spondyloarthropathies such as ankylosing spondylitic affecting the SI joints and other structures which are difficult to image.(362-369)

Recommendation: SPECT for Low Back Pain and Related Disorders
SPECT is not recommended for the evaluation of patients with low back pain and related disorders.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

Rationale for Recommendation
There is no quality evidence with patient-related outcomes that SPECT is helpful in improving care of acute, subacute, or chronic LBP, or radicular pain syndromes or other LBP-related conditions. However, one study found SPECT helpful in evaluating patients with inflammatory arthropathies, particularly if there are concerns about the SI joints.(370) Some data suggest SPECT may outperform bone scanning.

Additional studies are needed to determine if SPECT adds something to the diagnosis, treatment and outcomes beyond that obtained by a careful history, physical examination, plain x-rays, and clinical impression before it can be recommended for evaluating facet arthropathies.

Evidence for the Use of Single Proton Emission Computed Tomography (SPECT)
There is 1 high-(371) and 4 moderate-quality(372-375) studies incorporated into this analysis.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: Back, SPECT, work, low, pain, diagnostic, acute, subacute, sensitivity, specificity, positive, negative predictive, value, efficiency, efficacy, and chronic to find 263,834 articles. Of the 263,834 articles, we reviewed six articles and included six articles.

ELECTROMYOGRAPHY
Electromyography (EMG) is a physiological test that assesses the function of the motor unit (including the neuron's anterior horn cell, its axon, the neuromuscular junctions, and muscle fibers it supplies).(376, 377) It differs from surface EMG which is discussed below. EMG technically refers to the needle electromyogram and the term "EMG" is usually misused as a euphemism for an electrodiagnostic exam that includes both needle EMG and peripheral nerve conduction testing. Among spine patients, EMG has been used primarily to evaluate radiculopathy.(378)

1. *Recommendation: EMG with Leg Symptoms*
   Electrodiagnostic studies, which must include needle EMG, are recommended where a CT or MRI is equivocal and there is ongoing pain that raise questions about whether there may be a neurological compromise that may be identifiable (i.e., leg symptoms consistent with radiculopathy, spinal stenosis, peripheral neuropathy, etc.). Also, may be helpful for evaluation of chronicity and/or aggravation of a pre-existing problem.

   Indications – Failure to resolve or plateau of suspected radicular pain without resolution after waiting 4 to 6 weeks (to provide for sufficient time to develop EMG abnormalities as well as time for conservative treatment to resolve the problems), equivocal imaging findings such as CT or MRI, and suspicion by history and physical examination that a neurologic condition other than radiculopathy may be present instead of, or in addition to radiculopathy.
   Harms – Medicalization or worsening of otherwise benign back condition; pain; hematoma, or misinterpretation if not done by an appropriately trained person.
   Benefits – Diagnosis of neurological compromise.
   > *Strength of Evidence* – **Moderately Recommended, Evidence (B)**
   > *Level of Confidence* – High

2. *Recommendation: EMG without Leg Symptoms*
   Electrodiagnostic studies are not recommended for patients with acute, subacute, or chronic back pain who do not have significant leg pain or numbness.

   > *Strength of Evidence* – **Not Recommended, Evidence (C)**
   > *Level of Confidence* – Moderate

Rationale for Recommendations
Needle EMG may help determine if radiculopathy and/or spinal stenosis is present and can help address acuity.(379) EMG requires full knowledge of the anatomy and precise innervation of each muscle to

properly perform and interpret the test results. Needle EMG also requires the skills of an experienced physician who can reliably spot abnormal motor potentials and recruitment patterns. Nerve conduction studies are usually normal in radiculopathy (except for motor nerve amplitude loss in muscles innervated by the involved nerve root in more severe radiculopathy and H-wave studies for unilateral S1 radiculopathy). Nerve conduction studies rule out other causes for lower limb symptoms (generalized peripheral neuropathy, peroneal compression neuropathy at the proximal fibular, etc.) that can mimic sciatica.

An abnormal EMG that persists after anatomic resorption of the herniation and that correlates with the patient's symptoms is generally considered proof the symptoms are due to radiculopathy. Thus, the EMG study documents that management for chronic neuropathic pain appears appropriate.

As imaging studies (especially CT and MRI) have progressed, the need for EMG has declined. However, EMG remains helpful in certain situations. These include ongoing pain suspected to be of neurological origin, but without clear neurological compromise on imaging study. EMG can then be used to attempt to rule in/out a physiologically important neurological compromise. An abnormal study confirming radiculopathy permits a diagnosis of neuropathic pain (helping with pain management decisions). This test should not be performed in the first month unless there is a desire to document pre-existing neurological compromise, as it requires time (generally at least 3 weeks) to develop the needle EMG abnormalities. EMG is minimally invasive, and has no long-term adverse effects (although it is somewhat painful), and it is costly. To result in reliable measures, it must be performed by a practitioner well skilled in the appropriate anatomy and testing procedures. Post-operative changes may persist in normal individuals without clinical significance, thus also requiring careful interpretation.

Evidence for the Use of Electromyography

We searched PubMed, EBSCO, Google Scholar, and Cochrane Review with limits on publication dates from 2011-2012 and then an updated search was conducted using PubMed for publications between 1/1/2013 and 11/15/2017. We used the following search terms: electromyography, EMG, surface EMG, intramuscular EMG, acute low back pain, subacute low back pain, chronic low back pain, diagnostic testing, sensitivity, specificity, positive predictive value, negative predictive value, efficacy, efficiency, and low back pain to find 10,054 articles. Of the 10,054 articles, we reviewed and included 7 articles (7 randomized controlled trials and 0 systematic reviews).

SURFACE ELECTROMYOGRAPHY

Surface electromyography (sEMG) has been used to diagnose LBP(380-396) and involves the recording of summated muscle electrical activity by skin electrodes (such as those used in an electrocardiogram or EKG). Unlike traditional needle EMG (see above), no needle is used to explore specific portions of specific muscles for motor unit potentials.

Surface EMG has also been used for many neuropathies, myopathies, myotonic dystrophy, Duchenne muscular dystrophy, Becker muscular dystrophy, spinal muscular atrophy, hereditary motor and sensory neuropathy, amyotrophic lateral sclerosis, McArdle's disease, postpoliomyelitis, familial hypokalemic periodic paralysis, limb girdle dystrophy, Steinert disease, and Charcot-Marie-Tooth.(397-413) These disorders are beyond the scope of this guideline.

Recommendation: Surface EMG for Diagnosing Low Back Pain

Surface EMG is not recommended to diagnose low back pain.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – High

Rationale for Recommendation
There are no quality studies demonstrating that the use of surface EMG results in improved diagnosis or evaluation of patients with LBP. Available studies have methodological weaknesses, including poor descriptions of patients, small sample sizes, types of machine, electrode placement, and analysis of the output making outcomes difficult to compare across studies.(380, 387, 391, 395, 414)

Surface EMG primarily measures the muscle activity of the nearest muscle group and over a wide geographic area rather than measuring deep and/or individual muscles,(404, 415) although some research suggests it may be possible to obtain measurements from deeper muscles.(416) Surface EMG is highly sensitive to the placement of the electrode, as well as quite sensitive to changes in posture. Thus it is technically demanding to obtain valid and reliable data. Common uses of sEMG are in research laboratory studies (e.g., physiology, kinesiology, gait analysis, ergonomics) and small scale-ergonomics studies in employment settings. Research studies of sEMG have suggested some differences between normal and chronic LBP patients and in pre- and post-intervention populations.(380, 381, 384, 388-391, 395, 396) A meaningful application to the clinical setting resulting in improved outcomes is not as clear.

The American Association of Neuromuscular and Electrodiagnostic Medicine's position is that there are no clinical indications for the use of sEMG in the diagnosis and treatment of disorders of nerve and muscle, although potential future uses are possible.(400, 417) Surface EMG is not invasive, has few adverse events, is moderately costly, but has a lack of quality evidence of benefits for the clinical evaluation or treatment of back disorders and thus is not recommended.

Evidence for the Use of Surface Electromyography
There are 4 moderate-quality studies incorporated into this analysis.(395, 418-420) There are 2 low-quality studies(380, 421) and 19 other studies in Appendix 1.(393, 397-399, 401, 403, 405, 407-411, 414, 422-427)
We searched PubMed, EBSCO, and Cochrane Review without limits on publication dates. We used the following search terms: Surface Electromyography, low back pain, Diagnostic, Sensitivity, Post-operative to find 170 articles. Of the 170 articles we reviewed 28 articles and included 24 articles.

ULTRASOUND (DIAGNOSTIC)
There are two uses for ultrasound technology – one is therapeutic and is discussed in the heat therapies section, and the other is for diagnostic purposes. Ultrasound projects high-frequency sound waves through tissue and records the echoes through a 2-dimensional imaging system. Ultrasound is seldom used for diagnostic purposes in the spine other than for unusual specific purposes such as detection and guided drainage of superficial abscesses.(428-434)

Recommendation: Ultrasound for Diagnosing Low Back Pain
Diagnostic ultrasound is not recommended for diagnosing low back pain.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – High

Rationale for Recommendation

Ultrasound has not been shown to result in improved patient outcomes or diagnoses other than minor applications. Ultrasound has been used to train patients to preferentially activate their transverse abdominis muscle.(435) However, altered long-term outcomes in a sizable patient population have not been shown. Ultrasound is not invasive, does not have adverse effects, and is moderately costly. There are other imaging techniques which are currently shown to be useful for diagnosis in patients with LBP. For most imaging purposes, CT and MRI are superior.

Evidence for the Use of Ultrasound
There is 1 high-(430) and 1 moderate-quality(436) study incorporated into this analysis. There is 1 low-quality study in Appendix 1.(437)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: Back, ultrasound, work, low, pain, diagnostic, acute, subacute, sensitivity, specificity, positive, negative predictive, value, efficiency, efficacy, and chronic to find 1,383,441 articles. Of the 1,383,441 articles, we reviewed one article, found an additional four articles from the reference list and included three articles.

THERMOGRAPHY
Thermography is a diagnostic test that has been used to assess LBP and radicular pain syndromes and other conditions.(438) This involves measuring skin surface temperature through infrared scanning. For the purposes of spinal assessments, these measurements involve particular attention to the lower extremities and over the lower spine.

Recommendation: Thermography for Diagnosing Acute, Subacute, or Chronic Low Back Pain or Radicular Pain
Thermography is not recommended for diagnosing acute, subacute, or chronic low back pain, or radicular pain.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Moderate

Rationale for Recommendation
There are no studies documenting meaningful impacts of thermography on improving outcomes of LBP patients. Studies have inferred that there are differences in thermal imaging, and thus blood supply, among patients with LBP, lumbar radicular syndromes, and sacroiliitis. There are both positive(439) and negative studies(440, 441) for asymmetry for LBP. Studies have been positive for lumbar radicular syndromes,(442, 443) while others have been negative(442, 444, 445) including one moderate-quality study that evaluated 55 lumbosacral radiculopathy patients and 37 controls with 5 blinded readers interpreting thermograms and calculated a positive predictive value of thermography for the diagnosis of radiculopathy at less than 50%, concluding that "thermography has little or no utility in the diagnosis of lumbosacral radiculopathy."(446) Studies have also failed to find associations with tender points.(447) Other diagnostic tests have been shown to be effective in the evaluation of acute, subacute, and chronic LBP. The added expense of thermography has not been shown to positively influence patient management. As it is not specific for musculoskeletal disorders, it has been shown to have poor specificity for LBP and back-related conditions. It is not invasive, has little potential for adverse effects, but is costly. Thus, there is no convincing evidence that thermography is an effective test for assessing LBP.

Evidence for the Use of Thermography
There are no quality studies regarding the use of thermography. There are 2 low-quality studies in Appendix 1.(439, 448)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: Back, thermography, work, low, pain, diagnostic, acute, subacute, sensitivity, specificity, positive, negative predictive, value, efficiency, efficacy, and chronic to find 74,025 articles. Of the 74,025 articles, we reviewed two articles and included two articles.

FLUOROSCOPY

Fluoroscopy is live (real-time) x-ray imaging which can define abnormalities that may be visualized on movement, but that are not apparent on static films. It has been used for evaluation of LBP.

Recommendation: Fluoroscopy for Evaluating Acute, Subacute, or Chronic Low Back Pain
Fluoroscopy is not recommended for evaluating acute, subacute, or chronic low back pain.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Moderate

Rationale for Recommendation
The main use for fluoroscopy is to guide procedures (e.g., facet injections, radiofrequency procedures, etc.) that are discussed individually elsewhere. While this test was previously used to image the spine, it has been largely supplanted by other studies. Because continual x-ray exposure is needed to obtain the images, exposure to radiation is far higher with this procedure than with static x-rays. Fluoroscopy is not invasive, has low risk of adverse effects, but is costly and involves considerable radiation exposure. There are no evidence-based indications for fluoroscopy outside of its use in the performance of specific diagnostic tests or procedures and other infrequent indications.

Evidence for the Use of Fluoroscopy
There are no recent quality studies of the value of fluoroscopy in the evaluation of LBP or radicular pain syndromes or other back-related conditions.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: fluoroscopy, sensitivity, specificity, acute low back pain, subacute low back pain, chronic low back pain, and low back pain to find 3,299 articles. Of the 3,299 articles, we reviewed 1 article and included zero articles.

VIDEOFLUOROSCOPY

Videofluoroscopy involves recording a videotape of fluoroscopic images of the spine that has been used for diagnostic purposes. Videofluoroscopy has been used for evaluation of LBP, particularly searching for possible spinal instability. After evidence interpreted as consistent with instability is found, surgery is typically proposed. A dynamic spinal motional analysis system for videofluoroscopy has been developed to reduce the tedious and time-consuming aspects of videofluoroscopy.(449)

Recommendation: Videofluoroscopy for the Assessment of Acute, Subacute, or Chronic Low Back Pain
Videofluoroscopy is not recommended for the assessment of acute, subacute, or chronic low back pain.

Strength of Evidence – **Not Recommended, Insufficient Evidence (I)**
Level of Confidence – Moderate

Rationale for Recommendation
There are no studies demonstrating improved clinical outcomes attributable to videofluoroscopy. There are no validated criteria for the utilization of videofluoroscopy to evaluate lumbar spine conditions. Other diagnostic tests have been shown to be effective in the evaluation of acute, subacute, and chronic LBP. One pilot study of videofluoroscopy suggested some differences between young healthy individuals and older individuals with spondylolisthesis.(450) However, there was no difference between young individuals and those with chronic LBP. Thus, as this study contains uncontrolled confounders, there are no quality studies evaluating videofluoroscopy for the evaluation of acute, subacute, or chronic LBP or radicular pain syndromes. The added expense of videofluoroscopy has not been shown to positively influence patient management. It is not invasive, has little potential for adverse effects, but is costly. It involves considerable radiation exposure. The clinical relevance of instability demonstrated via videofluoroscopy has not been established.

Evidence for Use of Videofluoroscopy
There are no quality studies regarding the use of videofluoroscopy. There are 2 low-quality studies in Appendix 1.(449, 451)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: videofluoroscopy, diagnostic, sensitivity, specificity, predictive value, efficiency, efficacy, acute low back pain, subacute low back pain, chronic low back pain, and low back pain to find 128 articles. Of the 128 articles, we reviewed 3 articles and included two articles (1 prospective case-series, 1 prospective case-control).

LUMBAR DISCOGRAPHY
Discography attempts to determine if chronic spinal pain is caused by disc pathology. Discography is usually used in patients with chronic spinal pain without significant leg pain, as MRI and/or CT provide adequate anatomic information for surgical decisions on decompressive surgery for patients with significant radiculopathy. Discography involves a needle that is inserted into the middle (nucleus) of a disc and x-ray dye is injected. Images are then made, usually both by plain x-ray and by computed tomography (CT).(452-457) Images are able to classify a disc as normal or as having varying degrees of degeneration.(458) Positive test results involve reproduction and/or augmentation of the patient's pain with the injection. This procedure is fairly painful and sedation is required.(454, 456, 459-461) The procedure has been variously modified to include injection of anesthetics sometimes followed by provocative physical activity such as lifting(462-464) and pressure measurements to attempt to improve its operant characteristics. Few quality studies have evaluated these modified procedures.

Recommendation: Discography for Assessing Acute, Subacute, or Chronic Low Back Pain or Radicular Pain Syndromes
Discography, either performed as a solitary test or when paired with imaging (e.g., MRI), is strongly not recommended for acute, subacute, or chronic low back pain or radicular pain syndromes.

Strength of Evidence – **Strongly Not Recommended, Evidence (A)**
Level of Confidence – High

Rationale for Recommendation

This test relies on a theory that discs with more severe degrees of degeneration are more likely to be painful on discography.(453, 456, 465) The test analyzes the pain responses of the sedated patient. If a patient does not experience pain on injection, that disc is considered as unlikely to be the source of chronic spinal pain.(454, 456) If a patient experiences pain that is mild or that is clearly different in location or character to his or her chronic pain, that disc is considered as unlikely to be the source of chronic spinal pain. However, if the patient experiences significant pain that is identical in location and character to the patient's chronic pain ("concordant pain"), proponents believe that discography has identified the pain-generating structure responsible for chronic spinal pain.(453, 456, 457, 465-468) It also follows that changes on MRI (e.g., Modic changes) should be more severe in those with positive discography, however, that has not been shown.(469)

Due in part to recognition that discography is not a highly accurate test in the lumbar, thoracic, or cervical spine,(459, 470-473) attempts have been made to modify the test to attempt to increase its accuracy, including measurement of pressures where pain occurs,(455, 465, 467) as well as injection of anesthetics.(456, 474, 475) Some studies have added measurement of the injection pressure (pressure in the disc at the time of pain production) as a test criterion. Those discs with pain provoked at less than 15 psi have been categorized as chemically sensitive, 15 to 50 psi are mechanically sensitive, and those over 50 psi are classified as not clinically significant.(476) Chemical sensitivity supposedly suggests the disc is degenerate, but not necessarily the pain-generating structure. High injection pressures may produce pain even in radiographically normal discs. Thus, concordant pain response at injection pressures of 15 to 25 psi has been sought as a criterion for determining the disc to be the pain-generating structure.

The technique of discography is not standardized. There is no validated definition of what constitutes a concordant painful response. There are no published intra-rater or inter-rater reliability studies on discography. The discussion of discography is important to the subsequent discussion of IDET, spinal fusion for degeneration of the disc, and artificial disc replacement, as many North American (but not European) surgeons continue to use discography results in surgical planning.(472) If discography can accurately identify a disc as the pain-generating structure, then surgical procedures on that disc should lead to patient improvement.(467, 477) If discography can produce pain, but cannot accurately identify that disc as the pain generating structure, then surgery on that disc is presumably unlikely to be helpful.(459, 470, 472)

Discography has been evaluated in quality studies (see also Cervical and Thoracic Spine Disorders Guideline). The highest quality study with at least 50 subjects suggests the test is unhelpful for evaluation of spine patients.(478) Currently, the estimated positive predictive value appears to be at or below 50%, which means the test is not helpful.(479) These studies have failed to find that discography reliably indicates what particular disc is the source of the patient's pain. Validity of those findings through improved operative successes is not present.(480) There are a number of studies comparing lumbar discography to other imaging studies such as MRI and CT myelography. These studies can describe how likely a given finding on imaging is to be associated with pain on injection, but cannot determine whether the pain response is a true-positive or a false-positive response. Thus, these studies are not capable of guiding surgical therapy. Studies on imaging have shown that most imaging findings do not correlate with an individual's pain status.(481) There are a number of studies that have assessed the rate of positive or painful responses in individuals without back pain. If the asymptomatic population has a high rate of painful responses to disc injection, a similar pain response (and the inevitable age-related

degeneration on imaging studies) can easily be interpreted as a positive discogram (false-positive). Since these were experimental subjects who did not have back pain, the pain could not be concordant with pain they did not have; however, the intensity of the pain response is such that it could easily be misinterpreted as a painful response (false-positive).

Discography is invasive and has adverse effects. The 0.1 to 0.2% rate of discitis (disc space infection) is low.(482, 483) Temporary complications include headache, nausea, and worsened back pain. Uncommon, but serious reported complications include meningitis, epidural abscess, arachnoiditis, intrathecal hematoma, intradural injection of contrast, retroperitoneal hematoma, cauda equina syndrome, and acute disc herniation.(454, 470, 475, 484-486) Some literature reporting longitudinal evaluations after discography of normal (or "control") discs suggests discography results in more rapid disc degeneration and an increased incidence of disc herniation.(487, 488) Discography requires that one or two normal discs be injected and be painless on injection, so that the disc that is painful during injection can be identified. If discography iatrogenically damages the normal control discs, and does not lead to improved treatment outcomes, then there is evidence that discography should not be performed. Discography results in a patient exposure to radiation of 1.5 to 4.0 rads.(250, 489) Discography is also costly and has not been found to provide information that has sufficient positive or negative predictive value to warrant its addition to the clinical examination or other testing currently under use. It is not currently recommended, although there are potential modifications to the procedure being further studied.

Evidence for the Use of Lumbar Discography
There are 2 high-(489-491) and 22 moderate-quality(82, 291, 462, 478, 479, 481, 492-507) studies incorporated into this analysis. A recent systematic review did not find high-quality evidence to support cervical discography and did not find studies that show discography could improve clinical outcomes in patients considering cervical surgery.(508)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar for articles published from 2008-present. We used the following search terms: lumbar discography, low back pain and diagnostic sensitivity to find 3,110 articles. Of the 3,110 articles, we reviewed 24 articles and included 21 articles.

MRI DISCOGRAPHY
MRI is sometimes paired with discography for evaluation of the intervertebral discs.(494-496, 498, 501)

Recommendation: MRI Discography for Evaluating Herniated Discs
MRI discography is not recommended for evaluating herniated discs.

> *Strength of Evidence* – **Not Recommended, Evidence (C)**
> *Level of Confidence* – Moderate

Rationale for Recommendation
There is no quality evidence supporting the use of discography combined with MRI to improve outcomes for herniated discs. MRI discography is invasive, has adverse effects, and is costly.

Evidence for the Use of MRI Discography
There are 5 moderate-quality studies incorporated into this analysis. (494-496, 498, 501) There is 1 other study in Appendix 1.(509)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: MRI discography, herniated disc, diagnostic, sensitivity, specificity, predictive value, efficiency, and efficacy to find 222 articles. Of the 222 articles, we reviewed 7 articles and included six articles (5 comparative studies, 1 prospective case-series).

DIAGNOSTIC FACET BLOCKS (INTRA-ARTICULAR AND NERVE BLOCKS)
See Injection Therapies.

MYELOSCOPY
Endoscopic examination of the epidural space is termed "myeloscopy." This procedure is minimally invasive and theoretically can be used solely for diagnostic purposes. It is most often performed in conjunction with adhesiolysis (see Adhesiolysis). The other method for performing adhesiolysis does not involve myeloscopy.(510-512)

Recommendation: Myeloscopy for Diagnosing Acute, Subacute, or Chronic Low Back Pain, Spinal Stenosis, Radicular Pain Syndromes, or Post-surgical Back Pain
Myeloscopy is not recommended for diagnosing acute, subacute, or chronic low back pain, spinal stenosis, radicular pain syndromes, or post-surgical back pain problems.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

Rationale for Recommendation
Currently, while there are studies suggesting different levels of neurological impingement are identified with myeloscopy, there are no quality controlled studies identifying the utility of this diagnostic procedure for improving long-term outcomes. A few reported studies have used this procedure in conjunction with adhesiolysis (see surgical treatments section of this Guideline). Myeloscopy has not been shown to be beneficial in large scale, medium- to long-term studies sufficient. (511, 512) It is invasive, has likely complications, and is costly. Well-designed multi-center studies are needed prior to recommending this procedure.

Evidence for the Use of Myeloscopy
There are 3 moderate-quality studies incorporated into this analysis.(513-515)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar with limits on publication dates from 2008 to 2014. We used the following search terms: myeloscopy, epiduroscopy, spinal endoscopy, acute low back pain, subacute low back pain, chronic low back pain, radicular pain, spinal stenosis, postsurgical back pain, diagnostic, sensitivity, specificity, efficiency, efficacy and predictive value to find 672 articles. Of the 672 articles, we reviewed 10 articles and included four articles (1 RCT, 2 prospective cohort, 1 review).

Initial Care. . . . .

Comfort is normally a patient's first concern. Activity levels, aerobic exercise and directional preference exercises (stretching in the direction that centralizes or abolishes the pain, see below) should be addressed. Nonprescription analgesics may provide sufficient pain relief for most patients with acute and subacute LBP. If treatment response is inadequate (i.e., if symptoms and activity limitations continue) or the provider judges the condition limitations to be more significant, prescribed

pharmaceuticals or physical methods can be added. Comorbid conditions, invasiveness, adverse effects, cost, and provider and patient preferences help guide the provider's choice of recommendations. Initial care and comfort items may include non-steroidal anti-inflammatory drugs (NSAIDs), acetaminophen, heat, cryotherapy, exercises, advice on activities, and manipulation. Education about LBP should begin at the first visit, including principles of fear avoidance belief training (FABT) for patients who appear to have elevated fear avoidance beliefs.

There is increasing belief that chronically impaired LBP patients begin a course towards disability at their first clinical encounter. As such, those who do not respond to appropriate treatment should have their treatment, compliance, and psychosocial factors assessed early. Additionally, those patients whose course ventures beyond the abilities of that healthcare provider should be referred to others with greater experience in evaluation and functional recovery of complex LBP patients.

The remainder of this document discusses evidence of efficacy for dozens of LBP interventions used for spinal conditions. This evidence and consequent guidance is further subdivided into acute, subacute, and chronic LBP, radicular pain syndromes, post-operative, and when evidence is available, other spinal conditions including spondylolisthesis, spinal stenosis, facet joint osteoarthrosis, and failed back surgery syndrome. A rigorous attempt has been made to ascertain evidence for radicular versus non-radicular pain in the development of this guideline. Unfortunately, the literature largely lacks specification of clear exclusionary criteria. Most trials did not report lower extremity symptoms and those that did nearly always reported percentages of subjects with "leg pain" without clarifying whether this was general lower extremity pain or anatomically consistent nerve root pain. A minority of such studies reported stratified analyses to detect if such patients responded differently. However, where identifiable radicular pain patients were included, these have been noted.

This guideline recommends interventions with quality evidence of proven efficacy. Known complication rates and safety profiles, if available, should always be utilized in decision making and were considered in developing this guideline. Besides those treatments reviewed herein, there are many additional theoretically potential treatments possible for the management of LBP and spinal conditions. In the absence of moderate- to high-quality studies,(9) other interventions are not recommended and are indicated as **Not Recommended, Insufficient Evidence (I).**

Activities and Activity Modification

There has been a major revision in the management of activity limitations in patients with LBP over the past 10 to 20 years. Previously, bed rest was prescribed. It is now widely recognized that prescription bed rest was ineffective (see following discussion), reinforced a belief that the injury was severe and contributed to delayed recovery in some cases. Patient management recommendations pertaining to occupational and non-occupational physical capabilities have advanced and there is now information available on posture, lumbar supports, and mattresses. There also has been much revision in the approaches for patient management regarding work restrictions, other activity limitations, and some information available on posture, mattresses, lumbar supports, and other appliances. The approach to exercise, or physical activity, has similarly advanced and has been significantly revised. Revisions have also been the result of the greater understanding that natural history shows that LBP is commonly a persistent or recurrent problem and "most workers do continue working or return to work while symptoms are still present: if nobody returned to work till they were 100% symptom free, only a minority would ever return to work."(516)

In general, activities causing a *significant* increase in low back symptoms should be reviewed with the patient and modifications advised when appropriate. Driving posture or duration, workstation design, lifting modifications, and other activities may require at least temporary modification. Usually these activities are obvious to the patient, yet, this is not always true. For example, patients may not realize the importance of monitoring symptoms and adjusting their positions or activities. It is now believed to be quite important to emphasize that a modest increase in pain does not represent or document damage. Instead, such symptoms may actually be beneficial to the patient to experience some short-term pain. For example, getting out of bed in the morning is frequently painful for acute LBP patient. Yet, it is beneficial to the patient's overall recovery to get out of bed and to maintain as nearly normal a functional status as possible (see Bed Rest, Exercise, and Fear Avoidance Belief Training). While the patient is recovering, activities that do not aggravate symptoms should be maintained and exercises to prevent debilitation due to inactivity should be advised. Aerobic exercise is highly beneficial as a cornerstone therapeutic management technique for acute, subacute, and chronic LBP (see Aerobic Exercise). The patient should be informed that such activities might temporarily increase symptoms.

WORK ACTIVITIES

Work activity modification is an important part of many treatment regimens. A patient's expectations regarding return-to-work status are often set prior to the first appointment,(517) thus education may be necessary to set realistic expectations and goals. Advice on how to avoid aggravating activities that at least temporarily increase pain includes a review of work duties to decide whether or not modifications can be accomplished without employer notification and to determine whether modified duty is appropriate and available. Making every attempt to maintain the patient at the maximal levels of activity, including work activities, is strongly recommended as there is evidence that not returning to work has detrimental effects on a patient's pain ratings and functionality.(518) No specific profession is recognized as singularly qualified to opine on job requirements and changes in job physical factors. Some occupational physicians by training and experience and by having visited the workplace in question will be qualified to recommend potential workplace modifications. Others who may also have the training and experience to assist with workplace assessments may include certified professional ergonomists, occupational therapists, physical therapists, certified safety professionals, or certified industrial hygienists. There are large differences in practice patterns and capabilities among these professionals (e.g., some measure job physical demands, some measure worker capabilities, some match these demands and capabilities, etc.), thus inquiries to ascertain the professional's experiences and capabilities are often necessary.

The analysis of work ability requires an assessment of "risk," "capacity," and "tolerance." Risk refers to what a patient can do but should not do, due to the substantial risk of significant harm, considering probability and severity of potential adverse events. Providers impose work restrictions based on estimates of risk. Capacity refers to what a patient is physically capable of doing as measured by concepts such as range of motion, exercise ability in metabolic equivalents (METs), etc. Providers describe work limitations (for example "can only exert to 6 METs due to prior myocardial infarction"). Tolerance for chronic symptoms such as back pain is the basis for a patient (not a provider) to decide whether the rewards of work are worth the cost of the symptoms. However, it is incumbent to inform the patient that in the chronic pain setting, the development of routine symptoms in the course of normal occupational activities (or exercise) is not believed to signify tissue damage. Details of this assessment methodology have been described.(519)

The first step in determining whether work activity modifications are required usually involves a discussion with the patient regarding whether he or she has control over his or her job tasks. In cases where the worker can obtain assistance from someone else to lift, and can alternate sitting and standing as needed, there may be no requirement to write any restrictions even if the pain is severe. In some situations, it may be advisable to confirm this report with the patient's supervisor or to write "activities as tolerated by pain" to signal to the supervisor that the person is under treatment, although again judgment is required as writing that phrase for a patient with perceptions of LBP equating serious injury may reinforce a detrimental injury mindset that contributes to further disability beyond that needed (see Fear Avoidance Belief Training). In such cases, specified limitations may be a better treatment strategy.

Work modifications should be tailored taking into account the three main factors: 1) job physical requirements; 2) severity of the problem; and 3) patient's understanding of his or her condition. A fourth factor, employer expectations, does not influence the writing of limtations, but does influence whether the limitations will be accepted and/or enacted. Sometimes it is necessary to write limitations or prescribe activity levels that are above what the patient feels he or she can do, particularly when the patient feels that bed rest or similar non-activity is advisable. In such cases, the provider should be careful to not overly restrict the patient as it is clearly not in his or her best interest. Education about LBP and the need to remain active should be provided.

Common limitations involve modifying the weight of objects lifted, frequency of lifts, and posture all while taking into account the patient's capabilities. For severe cases of acute LBP with or without radicular symptoms, frequent initial limitations for occupational and non-occupational activities include:
▪ no lifting over 10 pounds,
▪ no prolonged or repeated bending (flexion), and
▪ alternate sitting and standing as needed.

These work and home activity guidelines are generally reassessed every week in the acute phase with gradual increases in activity recommended so that patients with severe non-specific back pain evolve off modified duty, typically within a couple weeks, but nearly always within 6 to 12 weeks. The amount of weight handled can be progressively increased. An alternative is to return the patient at first to 1 to 2 hours a day on his or her prior full-duty job, with the remainder of the day spent at modified duty. The numbers of hours of full-duty work can be increased every 1 to 2 weeks.

However, individualization is often necessary and if the prior job physical tasks involved frequent lifting of more than 100 pounds, then restrictions at work guidance may reasonably be substantially greater, e.g., initial limitations of 25 pounds of lifting and carrying. The size of the object lifted is a major consideration as it requires a long horizontal distance between the hands and the spine, which necessitate high back forces to lift the object even if it weighs under 20 pounds. Twisting while lifting is thought to put significantly greater stress on the back. However, epidemiological evidence to support this theory is weak. Regardless, this is usually readily controlled by patient education as few jobs are structured to require simultaneous lifting and twisting. In some cases, preclusion of a specific lift may be necessary. The need to alternate positions frequently is clinically highly helpful. LBP patients tend to experience significant increases in pain when in one position for an extended period of time, and perhaps this is one reason why bed rest is counterproductive. Patients should be encouraged to change positions frequently, ideally prior to experiencing major increases in symptoms. Thus, restrictions that

state "sedentary work" are *not* appropriate for most LBP patients as they convey misinformation while also potentially increasing symptoms.

Some workplaces provide health care or physical therapy on-site, thus brief periods of recumbent time during the day may be possible. Physical therapy may also be provided on-site and this may further facilitate the rehabilitation process. While there is one report that modified duty policies were not effective in Norway,(520) there have been large savings realized in the U.S. from accommodation of modified ("light") duty.

It is best to communicate early in the treatment that limitations will be progressively reduced as the patient progresses. This should be communicated at each successive visit so that the patient is well advised in advance of the treatment plan. Tailoring of limitations in the context of radicular pain may also be necessary as some workers have specific intolerances (e.g., intolerance of sitting or prolonged driving).

The provider can make it clear to patients and employers that:
- even moderately heavy lifting, carrying, or working in awkward positions may aggravate symptoms of LBP or lumbosacral nerve root irritation, and
- any restrictions are intended to allow for spontaneous recovery or for time to build activity tolerance through exercise.

Every attempt to maintain the patient at maximal levels of activity, including work activities, should be made as it is in the patient's best short- and long-term interest. *Work activity limitations should be written whether the employer is perceived to have modified duty available or not. Written activity limitations guidance communicates the status of the patient and also gives the patient information on what he or she should or should not do at home.*

Activity Modification and Exercise . . . .

BED REST

Bed rest has long been used for the treatment of LBP,(521-536) particularly acute LBP. Use of bed rest is believed to have evolved from consideration of the pain experienced by those with acute LBP when engaged in activities such as getting out of bed, without consideration of whether there might be any adverse short- or long-term implications. Description of bed rest as a treatment implied that compliant patients were those that spent a greater proportion of time in bed, thus increasing the likelihood that they would get better sooner. Traditional teaching held that patients who did not get better with bed rest were either non-compliant or needed longer periods of bed rest.

1. *Recommendation: Bed Rest for Acute, Subacute, Chronic, Radicular Low Back Pain, or Stable Spinal Fractures*
   Bed rest is not recommended for the management of acute, subacute, chronic, radicular low back pain, or stable spinal fractures.

   Strength of Evidence – **Strongly Not Recommended, Evidence (A)** [Acute]
   Moderately Not Recommended, Evidence (B) [Subacute, Chronic]
   Not Recommended, Evidence (C) [Radicular]
   Not Recommended, Insufficient Evidence (I) [Stable Spinal Fractures]
   Level of Confidence – High

2. *Recommendation: Bed Rest for Unstable Spinal Fractures*
    Bed rest is recommended for unstable spinal fractures.
    Harms – Deconditioning, DVT risk.
    Benefits – Avoidance of catastrophic injury.

    > **Strength of Evidence –** Recommended, Insufficient Evidence (I)
    > *Level of Confidence* – High

3. *Recommendation: Bed Rest for Other Low Back Problems*
    Bed rest is not recommended for other low back problems.

    > *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
    > *Level of Confidence* – Moderate

4. *Recommendation: Specific Beds or Other Commercial Products for Prevention or Treatment of Acute, Subacute, or Chronic Low Back Pain*
    Specific beds or other commercial sleep products are not recommended for prevention or treatment of acute, subacute, or chronic low back pain.

    > *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
    > *Level of Confidence* – Moderate

Rationale for Recommendations

In 1986, bed rest was usually recommended for acute LBP.(523) Today, multiple quality studies demonstrate that bed rest of any duration is ineffective for LBP (see Evidence Table). Several trials have either included significant numbers of patients with radicular pain symptoms,(523, 525, 529, 530, 536) or specifically focused on patients with sciatica(527, 533) and failed to find evidence that bed rest had a favorable impact on outcomes among patients with either LBP or radicular pain syndromes.

Bed rest, while non-invasive, is costly (due to lost time), and can have documented adverse effects beyond those associated with deconditioning such as pulmonary emboli.(527) Studies document compliance to be poor, which likely results in underestimation of the magnitude of the adverse effects of bed rest. Bed rest is strongly not recommended as a treatment strategy for management of acute LBP. Evidence is modestly less strong but also suggests bed rest is ineffective for subacute and chronic LBP.

There are no quality studies evaluating the role of bed rest in the management of unstable spinal fractures or cauda equina syndrome, yet it is required for those conditions. There is consensus that these require bed rest or other marked activity limitations to prevent adverse events. Although bed rest is costly and has no documented benefits, the hazard of mobilization in this setting is theoretically catastrophic, thus this treatment strategy is recommended for unstable fractures. There is also no quality evidence regarding the use of bed rest or other activity limitations for the treatment of stable spinal fractures, such as transverse process fractures or compression fractures. In those settings, bed rest is costly, has no documented benefits, and is expected to be associated with higher morbidity, although it is non-invasive. Instead, gentle activity within tolerance is recommended.

There is no quality evidence that other back pain-related problems are successfully treated with bed rest, including spondylolisthesis, spondylolysis, spinal stenosis, facet related pain, or pain thought to be related to the sacroiliac joint. There also are likely adverse effects. Bed rest is costly, has no documented benefits, and is expected to be associated with higher morbidity, although it is non-invasive.

There is no quality evidence that specific commercial products (e.g., pillows, mattresses, etc.) have a role in the primary prevention or treatment of acute, subacute, or chronic LBP.

Evidence for the Use of Bed Rest
There are 11 moderate-quality RCTs incorporated into this analysis.(521, 523-525, 527-532, 536) There is 1 low-quality RCT in Appendix 1.(535)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following terms: bed rest, subacute low back pain, chronic low back pain, radicular pain syndromes (including 'sciatica'), Spinal stenosis, spinal fractures' sacroiliitis, and spondylolisthesis to find 9,972 articles. Of the 9,972 articles we reviewed 15 articles (11 original RCT, and 4 reviews) and all were included.

SITTING POSTURE
There are strong beliefs and little supportive quality evidence that lordotic postures are superior for LBP treatment and prevention.(537, 538)

Recommendation: Sitting Posture for Acute, Subacute or Chronic Low Back Pain, Radicular Pain or Post-operative Pain
Lordotic sitting posture is recommended for treatment of acute, subacute, or chronic LBP, radicular pain and post-operative pain.

> *Indications* – Acute, subacute, or chronic LBP.
> *Indications for Discontinuation* – Non-tolerance.
> *Harms* – Negligible.
> *Benefits* – Better sleep and potentially reduced pain.
>> *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
>> *Level of Confidence* – Low

Rationale for Recommendation
There are no quality trials that address sitting posture as a treatment for LBP. Yet, low-quality trials suggest efficacy, the intervention would help to maintain a typical lordotic posture, and the intervention is simple.(537, 538) A pillow or an existing feature of a motor vehicle seat is not invasive, has few adverse effects, is low cost and is recommended.

Evidence for the Use of Sitting Posture
There are 2 low-quality RCTs which reported on sitting postures to prevent or treat LBP in Appendix 1.(537, 538)

SLEEP POSTURE
Certain sleep postures have been sometimes thought of as superior. The controversy appears largely driven by a theory that a straight spine while sleeping is beneficial. This theory holds that specific sleep postures that maintain the nocturnal alignment of the spine will reduce LBP incidence, persistence, and/or severity. Recommendations include sleeping on the side, sleeping with a pillow between the legs, and use of brand-name pillows and mattresses (see Mattresses, Water Beds, and other Sleeping Surfaces section).

Recommendation: Sleep Posture Adjustment for Acute, Subacute or Chronic Low Back Pain
Sleep postures are recommended that are most comfortable for the patient. If a patient habitually chooses a particular sleep posture, it is reasonable to recommend altering posture to determine if there is reduction in pain or other symptoms.

> *Indications* – Acute, subacute, or chronic LBP that results in nocturnal awakening, particularly if not amenable to other treatments.
> *Indications for Discontinuation* – Non-tolerance.
> *Harms* – Negligible.
> *Benefits* – Better sleep and potentially reduced pain.
>
> > *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
> > *Level of Confidence* – Low

Rationale for Recommendation
Changing sleep posture is low cost and not invasive, although there is the potential for increased symptoms. Alteration of sleep posture may initially affect quality of sleep, which has been suggested to be a contributor to daytime pain. Thus, recommendations to change sleep posture should be given with appropriate counseling, because the theory may not be correct.

Evidence for the Use of Sleep Posture
There are no quality studies reported on sleep posture to prevent or treat LBP.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: sleep posture, subacute low back pain, chronic low back pain, post-operative, and post surgery, to find 0 articles in PubMed, 0 on EBSCO, 0 on Cochrane Review, and 10,737 in Google Scholar, for a total of 10,737 articles. No RCT's were found.

MATTRESSES, WATER BEDS, AND OTHER SLEEPING SURFACES
Sleep disturbance is common with LBP.(539) Dogma holds that a firm mattress is superior for LBP treatment and/or prevention.(540) Commercial advertisements also advocate brand-name mattresses allegedly to treat LBP.(541) The purpose for including a discussion about mattresses and sleeping surfaces in this section is not to involve providers in prescriptions of mattresses, but to make health care providers aware of the available evidence so that patients can make informed decisions.

1. *Recommendation: Mattresses for Treatment of Acute, Subacute, or Chronic Low Back Pain*
   There is no recommendation for or against the use of mattresses for treatment of acute, subacute, or chronic low back pain other than to raise provider awareness that the dogma to order patients to sleep on firm mattresses appears wrong. By analogy, sleeping on the floor may be incorrect as well.

   > *Strength of Evidence* – **No Recommendation, Insufficient Evidence (I)**

2. *Recommendation: Other Sleeping Surfaces for Treatment of Acute, Subacute, or Chronic Low Back Pain*
   There is no recommendation for or against the use of optimal sleeping surfaces (e.g., bedding, water beds, hammocks) for treatment of acute, subacute, or chronic low back pain. It is recommended that patients select mattresses, pillows, bedding, or other sleeping options that are most comfortable for them.
   Individuals with LBP may report better or worse pain and associated sleep quality with different sleeping

surfaces. In cases where there is pain sufficient to interfere with sleep, recommendations by the provider for the patient to explore the effect of different surfaces in the home is appropriate. This could include switching to a different mattress, sleeping on the floor with adequate padding, or using a recliner. Any recommendation in this regard should be preceded by adequate exploration of varied sleep positions/posture that could improve sleep quality. For instance, a recommendation to place a pillow between the knees in the side-lying position or a pillow under the knees in the supine position to alter lumbopelvic posture could be useful.

Strength of Evidence – **No Recommendation, Insufficient Evidence (I)**

Rationale for Recommendations
One quality study of chronic LBP patients reported a medium firm mattress was superior to a firm mattress,(542) but it neither discussed sleep position nor prior mattress firmness which may be important issues. Another trial suggested a waterbed or foam mattress is superior to a hard mattress.(543) Mattress selection is subjective and depends on many factors including personal habits and the weight/size of an individual. For these reasons, individuals must evaluate which mattress is best suited to provide some relief to their particular problem and it is not appropriate for providers to order mattresses or bedding for patients. However, providers should be aware that the dogma that a more firm mattress is superior to a less firm mattress currently appears wrong.

Evidence for the Use of Mattresses, Water Beds, and Other Sleeping Surfaces
There is 1 high-(542) and 1 moderate-quality(543) RCT incorporated into this analysis. There are no quality studies on water beds or sleeping on the floor. There are 2 low-quality RCTs in Appendix 1.(544, 545)

We searched PubMed, EBSCO, Google Scholar, and Cochrane Review with no limits on publication dates. The following search terms were used :"(beds OR other commercial sleep products OR Mattresses made of optimal sleeping surfaces OR bedding OR water beds OR hammocks) AND (sub-acute low back pain OR chronic low back pain)" to find 148 articles. Of those 148 articles, we reviewed 2 articles and included 2 articles (2 RCT, 0 reviews).

EXERCISES

For decades, exercises have been considered among the most important therapeutic options for the treatment and rehabilitation of LBP.(60, 61, 85, 546-589) While there are many ways to categorize and analyze exercise, this guideline evaluates exercise in three broad groupings: 1) aerobic exercise, 2) stretching and 3) strengthening. Additional subsequent sections include reviews of aquatic therapy, yoga, tai chi, and pilates.

ALL EXERCISE PRESCRIPTIONS
Recommendation: Exercise Prescriptions for Acute, Subacute, Chronic, Post-operative or Radicular Low Back Pain
An exercise prescription is moderately recommended for treatment of acute, subacute, chronic, post-operative and radicular low back pain.

Indications – All patients with LBP appear to benefit from an exercise prescription.

Frequency/Duration – If a supervised program is felt to be needed, recommended frequency is 1 to 3 sessions a week for up to 4 weeks as long as objective functional improvement and symptom reduction is occurring. If self-directed, daily exercise is recommended. An exercise prescription should address specific treatment goals and be time limited with transition to an independent exercise program as part

of a healthy lifestyle (no longer considered treatment). The purpose of supervised exercise therapy is symptom reduction, functional improvement, and educating the patient so that he or she can independently manage the program. Evaluation for an exercise prescription involves consideration of five critical components:

1. stage of (theoretical) tissue healing (acute, subacute, chronic),
2. severity of symptoms (mild, moderate, severe),
3. identification of the presence or absence of a directional preference
4. degree and type of deconditioning (flexibility, strength, aerobic, muscular endurance), and
5. psychosocial factors (e.g., medication dependence, fear-avoidance, secondary gain, mood disorders).

Harms – None reported in quality studies. Theoretical risk of myocardial infarction, angina and musculoskeletal injury in a severely deconditioned patient.
Benefits – Improvement in low back pain, improved cardiovascular fitness.
>*Strength of Evidence* – **Moderately Recommended, Evidence (B)**
>*Level of Confidence* – High

AEROBIC EXERCISES

1. *Recommendation: Aerobic Exercise for Treatment of Acute or Subacute Low Back Pain*
   Aerobic exercise is moderately recommended for treatment of acute and subacute low back pain.

   Indications – All patients with acute or subacute LBP appear to benefit from aerobic exercises.[iv]
   Frequency/Duration –For acute or subacute LBP patients, a graded walking program is generally desired, often using distance or time as minimum benchmarks – e.g., start with 10 to 15 minutes twice a day for 1 week, increase in 10 to 15 minute increments per week until ≥30 minutes walking a day is achieved. A reasonable eventual target for patients based on treatment of chronic LBP is walking at least 4 times a week at 60% of predicted maximum heart rate (220-age = predicted maximum heart rate).(590)

   Indications for Discontinuation – Development of angina pectoris, myocardial infarction or other intolerance. After LBP resolves, nearly all patients should be encouraged to maintain aerobic exercises on a long-term basis for prevention of LBP,(*192*, *591*) and to maintain cardiovascular fitness and optimal health.

   Harms – None reported in quality studies. Increased pain with onset of exercise. Theoretical risk of myocardial infarction and angina in a severely deconditioned patient.

   Benefits – Improvement in low back pain, improved cardiovascular fitness.

   >*Strength of Evidence* – **Moderately Recommended, Evidence (B)**
   >*Level of Confidence* – High

2. *Recommendation: Aerobic Exercise for Radicular Low Back Pain*

[iv]Those with significant cardiac disease, or significant potential for cardiovascular disease should be evaluated prior to institution of vigorous exercises. It is recommended that the American College of Sports Medicine's *Guidelines for Exercise Testing and Prescription*, 9th ed., be followed for health screening and risk stratification. This is rarely required in the acute LBP setting as the initial exertion levels are so low relative to prior activity levels.

Aerobic exercise is recommended for patients with radicular low back pain symptoms.

Indications – All radicular LBP patients.

Frequency/Duration – A graded walking program is generally desired, often using distance or time as minimum benchmarks – e.g., start with 10 to 50 feet depending largely on severity of the condition. Gradually increasing distance and duration of walking. A reasonable eventual target for the post-recovery period is based on treatment of chronic LBP and is walking at least 4 times a week at 60% of predicted maximum heart rate.(590)

Indications for Discontinuation – Development of angina pectoris, myocardial infarction or other intolerance. Nearly all patients should be encouraged to maintain aerobic exercises on a long-term basis for prevention of LBP and to maintain cardiovascular fitness and optimal health.

Harms – None reported in quality studies. Increased back pain may occur. Possible fall risk if moderate to severe weakness. Theoretical risk of myocardial infarction and angina in a severely deconditioned patient.
Benefits – Improvement in low back radicular pain, improved cardiovascular fitness.

> *Strength of Evidence* – **Recommended, Evidence (C)**
> *Level of Confidence* – Moderate

3. *Recommendation: Aerobic Exercise for Chronic Low Back Pain*

Aerobic exercise is strongly recommended for treatment of chronic low back pain.

Indications – All patients with chronic LBP. However, those with significant cardiac disease or significant potential for cardiovascular disease should be evaluated prior to instituting vigorous exercises, following the American College of Sports Medicine's *Guidelines for Exercise Testing and Prescription,* 9th ed.,(592) in regards to health screening and risk stratification.

Frequency/Duration – For patients with chronic LBP, walking at least 4 times a week at 60% of predicted maximum heart rate (220-age = maximum heart rate) is recommended.(590) Benchmarks were 20 minutes during Week 1, 30 minutes during Week 2, and 45 minutes after that point. Nearly all patients should be encouraged to maintain aerobic exercises on a long-term basis additionally to maintain optimal health.

Indications for Discontinuation – Intolerance (rarely occurs), development of other disorders.
Harms – None reported in quality studies. Increased back pain with exercise initiation common. Theoretical risk of myocardial infarction and angina in a severely deconditioned patient. Intolerance of weight bearing is severe lower extremity osteoarthrosis. Other musculoskeletal disorders possible (e.g., plantar heel pain).
Benefits – Improvement in LBP, improved cardiovascular fitness, improved health status.

> *Strength of Evidence* – **Strongly Recommended, Evidence (A)**
> *Level of Confidence* – High

4. *Recommendation: Aerobic Exercise for Post-operative Low Back Pain*

Aerobic exercise is recommended for patients with post-operative low back pain.

Indications – All post-operative LBP patients.

Frequency/Duration –A graded walking program is generally desired, often using distance or time as minimum benchmarks – e.g., start with 10 to 50 feet depending largely on severity of the operative procedure. Gradually increasing distance and duration of walking. A reasonable eventual target after the operative recovery period is based on treatment of chronic LBP and is walking at least 4 times a week at 60% of predicted maximum heart rate.(590)

Indications for Discontinuation – Development of angina pectoris, myocardial infarction or other intolerance. Nearly all patients should be encouraged to maintain aerobic exercises on a long-term basis for prevention of LBP and to maintain cardiovascular fitness and optimal health.

Harms – None reported in quality studies. Brief increased pain with onset of exercise. Theoretical risk of myocardial infarction and angina in a severely deconditioned patient.

Benefits – Improvement in LBP, improved cardiovascular fitness.

> *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – High

General Exercise Approach: Acute Low Back Pain
Directional exercises and aerobic exercise are recommended. Strengthening is delayed to late in the acute recovery stage or for subacute or chronic LBP as there is a potential for aggravation of LBP. Pain control modalities may be needed as a complement to exercise. The recommended frequency is 1 to 3 sessions a week for up to 4 weeks as long as objective functional improvement and symptom reduction are occurring.

General Exercise Approach: Acute Radicular Low Back Pain
The treatment strategy is the same as for acute LBP. However, movements that centralize LBP are recommended to guide exercise selection. Concentration on radicular symptoms is emphasized over axial pain. Rapid progression of radicular symptoms and objective signs may necessitate discontinuation of exercise, changing the exercise approach and consideration of further diagnostic testing.

General Exercise Approach: Subacute Low Back Pain
For patients with no prior treatment, the treatment plan is similar to non-specific LBP. The frequency is 1 to 3 sessions a week for 4 weeks as long as objective functional improvement and symptom reduction is occurring. For those who failed acute treatment, a trial of more intensive reconditioning that includes strengthening exercises is recommended. Particular attention should be paid to psychosocial factors that may impair compliance with exercise recommendations among those with subacute LBP, as it is believed possible to reduce risk for the LBP to become chronic. Providers should educate patients to help motivate, encourage, and facilitate recovery. The frequency is 2 to 5 sessions a week for 4 weeks as long as there is objective functional improvement, symptom reduction, patient compliance, and efficacy. Progress should be reassessed after 8 sessions. Visit frequency depends on work status, symptom severity, comorbidities, and functional status.

General Exercise Approach: Subacute Radicular Pain
Subacute radicular pain is treated similarly to subacute LBP unless there is rapid progression of radicular symptoms and objective signs. If this occurs, it may be necessary to consider further diagnostic testing.

General Exercise Approach: Post-operative Exercising

Post-operative progressive exercise programs should initially emphasize progressive aerobic exercises. Flexibility should begin after appropriate tissue healing, which may be prolonged in the case of fusion surgery and requires careful coordination with the treating surgeon. Strengthening is similarly begun after appropriate tissue healing. Treatment frequency of 1 to 3 sessions a week progressing to 2 to 4 sessions a week is recommended depending on patient compliance, objective functional improvement, and symptom reduction. Reassessment should occur after 10 sessions with continuation based on demonstration of functional improvement. The upper range is 20 sessions.

General Exercise Approach: Chronic Episodic Low Back Pain and Radicular Pain
For patients with mild symptoms or a flare-up of symptoms, the treatment focus is on education regarding home management and exercise. Individuals with mild symptoms and minimal functional limitations may receive a therapy evaluation and 1 follow-up visit to adjust the home therapy program. For individuals with moderate to severe flare-up with mild to severe disability, treatment should consist of a progressive exercise program first emphasizing flexibility and aerobic exercises and progressing to strengthening treatment frequency of 1 to 3 visits a week up to a maximum of 12 visits. Reassessment should occur after Visit 6, with continuation based on patient compliance, objective functional improvement, and symptom reduction. For patients with spinal stenosis, 1 to 3 visits a week up to a maximum of 12 visits to teach flexion exercises and aerobic exercises has evidence of efficacy comparable with surgery for many patients.(593)

General Exercise Approach: Chronic Low Back Pain and Radicular Pain
For patients with mild symptoms and minimal disability, treatment should consist of a therapy evaluation to instruct the patient in a home-based exercise program, with 1 to 2 follow-up visits. For patients whose prior treatment failed and who have moderate symptoms and some functional deficits but no previous exposure to exercise therapy, he or she should be treated the same as a patient with subacute symptoms (outlined above). If the patient failed prior exercise therapy, consider 6 additional exercise visits, or consider an interdisciplinary approach (see Chronic Pain Guideline for managing patients with severe chronic pain or disability).

Evidence for the Use of Aerobic Exercise
There are 18 moderate-quality studies incorporated into this analysis.(590, 593-609) There are 2 low-quality studies in Appendix 1.(610, 611)

We searched PubMed, Cochrane Review, Google Scholar and EBSCO with no limits on publication dates and with the following search terms "Aerobic exercise Sub-acute low back pain, chronic low back pain" to find 71144 articles. Of 71,144 articles, we reviewed 6 articles and included 16 articles. (Original studies 15 RCTs and 1 review).

DIRECTIONAL EXERCISE
Recommendation: Directional Exercises for Treatment of Acute, Subacute, Chronic, or Radicular Low Back Pain
Directional exercises are recommended for patients found to have directional preference (i.e., centralization or abolishment of pain in a direction).(612) For chronic pain, directional exercises are generally not the primary or sole exercise treatment as aerobic and strength deficits are usually present.

Indications – For acute, subacute, or chronic LBP, directional preference exercises are recommended.

Frequency/Duration – Exercise frequency is determined by the stage of recovery. They are initially performed every 2 hours (8 to 10 repetitions) to fully centralize and abolish the pain, along with posture modifications that also honor patients' directional preference and protect the patient from symptoms returning when not exercising. Once the pain is eliminated even for a short period of time, the same exercises and posture changes should continue proactively to attempt to prevent the pain from returning. Proactive exercise remains important in maintaining a pain-free status as the opposite direction of spinal movement and positioning are progressively re-introduced. The duration of this sequence is typically a few days or weeks.

Indications for Discontinuation – Directional exercises should be discontinued if there is worsening pain in the course of treatment or failure to improve.

Benefits – Often rapid elimination of the pain and earlier return to function.

Harms – None reported in quality studies. Theoretical risk of increased pain from over-stretching.

> *Strength of Evidence* –   Recommended, Evidence (C) [Acute]
> **Recommended, Insufficient Evidence (I)** [Chronic, Subacute, Radicular]
> *Level of Confidence* – Moderate

STRETCHING AND FLEXIBILITY

1. *Recommendation: Slump Stretching for Treatment of Acute, Subacute, or Chronic Low Back Pain*
   Slump stretching is recommended for those with acute, subacute, or chronic low back pain, but without directional preference (see Directional exercise above).
   Indications – For acute, subacute, or chronic LBP among patients without directional preference, stretching exercises are recommended. Generic stretching exercises are not recommended. Among those with directional preference, directional exercise is believed to be preferable to slump stretching.
   Frequency/Duration – Three to 5 times a day for acute LBP; 2 to 3 times a day for subacute or chronic LBP.
   Indications for Discontinuation – Resolution, worsening pain or failure to improve.
   Benefits – Improvement in low back pain.
   Harms – Increased pain especially short term, and particularly if stretch in a direction of worsening (see Directional Exercise). Theoretical risk of muscle strain from over-stretching.

   > *Strength of Evidence* – **Recommended, Evidence (C)** [Acute]
   > *Strength of Evidence* – **Recommended, Insufficient Evidence (I)** [Subacute, Chronic]
   > *Level of Confidence* – Low

2. *Recommendation: Aggressive Stretching for Treatment of Low Back Pain*
   Aggressive stretching is not recommended for treatment of low back pain.

   > *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
   > *Level of Confidence* – Moderate

3. *Recommendation: Stretching Exercises for Prevention of Low Back Pain*
   Stretching exercises as an isolated prescription or program for purposes of preventing low back pain are not recommended.

   > *Strength of Evidence* – **Not Recommended, Evidence (C)**
   > *Level of Confidence* – Low

4. *Recommendation: Stretching Exercises for Treatment of Chronic Low Back Pain*

Stretching exercises are not recommended for treatment of chronic low back pain in the absence of significant range of motion deficits. In select cases, stretching exercises may be added for self-treatment if needed.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

STRENGTHENING AND STABILIZATION EXERCISES

1. *Recommendation: Strengthening Exercises for Acute (Late Recovery), Subacute, Chronic, or Post-Operative Low Back Pain*
   Strengthening exercises are recommended for patients with acute (late recovery), subacute, chronic, or post-operative low back pain. Specific strengthening exercises, such as stabilization exercises, are helpful for the prevention and treatment (including post-operative treatment) of low back pain.(613-616)

   Indications – Nearly all LBP patients other than those with acute LBP that resolves rapidly or acute LBP in the acute treatment phase when strengthening could aggravate the pain. As evidence of efficacy of aerobic exercises appears greater (see above), these exercises should be added after aerobic exercises have already been instituted and additional treatment is needed or in situations where both are felt to be required. Exercises should be taught and then performed by the patient in a home exercise program. For those patients who do not improve, follow-up appointments to verify technique and compliance (by exercise log books) are recommended. Some patients, particularly those lacking motivation to be in a home exercise program or those with fear avoidant behaviors may benefit from a supervised exercise program, although strong questions about long-term compliance are apparent among such patients particularly with chronic LBP. More intensive programs with more intensive exercises and direct supervision with active coaching appear warranted for chronic LBP.

   Frequency/Duration – Home program frequency is 1 to 2 times a day for acute LBP, and 2 to 3 times a day for subacute or chronic LBP. Supervised treatment frequency and duration is dependent of symptom severity and acuity and the presence of comorbid conditions and yellow flags (see recommendations under General Exercise Approaches and Recommendation).

   Indications for Discontinuation – Indications to discontinue strengthening exercises include development of a strain in the course of treatment or failure to improve.
   Benefits – Improvement in LBP, improved strength and fitness.
   Harms – Increased pain, especially short-term; theoretical risk of musculoskeletal injury.

   > *Strength of Evidence* – **Recommended, Evidence (C)**
   > *Level of Confidence* – High

2. *Recommendation: Abdominal Strengthening Exercises for Treatment or Prevention of Low Back Pain*
   Abdominal strengthening exercises as a sole or central goal of a strengthening program are not recommended for treatment or prevention of low back pain.

   > *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
   > *Level of Confidence* – Low

3. *Recommendation: Fear Avoidance Belief Training During Rehabilitation*

Inclusion of fear avoidance belief training during the course of rehabilitation is recommended.
Benefits – Improvement in exercise and activity compliance,with resultant improved LBP and fitness.
Harms – None reported.

> *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Moderate

Rationale for Recommendations
General Summary of Exercise Issues
There is a large body of RCTs on exercise to treat LBP. However, the majority of studies combined different exercises. Others left exercise programmatic components unstructured and/or did not clearly describe the interventions. These limitations restrict the utilization of a substantial body of the literature for purposes of drawing evidence based conclusions regarding any single intervention. Still, there is a considerable, remaining body of evidence to draw evidence-based conclusions on the relative value of aerobic, stretching, and strengthening exercises.

There are two major patterns which are apparent in reviewing this body of evidence. First, aerobic exercise is uniformly beneficial and appears to be the most promising modality of exercise. The second pattern is that the more vigorous the strengthening exercises, the more benefit appears to be derived from those exercises. These are discussed in more detail below.

A common issue for all exercise programs is the propensity for individuals to not participate. Even in RCTs where motivation to participate may be higher than in a clinical population, participation rates are frequently suboptimal. Some trials defined compliance as meeting a benchmark of participation that was less than that prescribed (e.g., accomplishing exercises at least 3 times a week versus 5 times a week as prescribed). This raises questions about the value of higher degrees of compliance compared with lesser compliance rates. There is some evidence that results from those attending supervised programs are superior to performing unsupervised programs, yet other studies show a lack of improvement with supervised programs compared with home-based exercise programs. Those with chronic pain seem to do better in supervised programs and those with acute pain appear to do no better with supervised programs, perhaps reflecting the natural excellent prognosis for acute LBP.

Thus, treatment is by inference from treatment of chronic LBP patients. For most patients, a structured, progressive walking program is recommended. There has been some controversy about whether bicycling is helpful or harmful from a biomechanical perspective (lordosis) and the back muscles are less active with bicycling, thus it may be theoretically less appropriate except for lumbar stenosis where bicycling is usually superior to walking. For those patients who desire other aerobic exercises, there are no specific data, although there are indications of a direct correlation between benefit and the amount of aerobic activity that results in higher MET expenditure. Therefore, the activity that the patient will adhere to is believed to be the one most likely to be effective, given that compliance is a recognized problem. Theoretical benefits of aerobic exercise include improved aerobic capacity, improved blood flow, lower depression, higher pain thresholds and pain tolerance. These exercises include walking, running, bicycling and many other activities. Whether there is benefit from weight-bearing versus non-weight bearing aerobic exercises remains unclear. There is evidence that a treadmill is superior to upper extremity or bicycle ergometers in assessing aerobic capacity in chronic LBP patients.(617) However, an exercise test is not necessary to evaluate and treat the majority of LBP patients.

While many studies included some aerobic exercises as part of a battery of exercises, there are some studies that appear to either solely or largely rely upon significant durations of aerobic exercise for treatment of LBP.(27, 618-621) All of these studies show favorable benefits from aerobic exercises, including reductions in LBP measures and some functional outcomes such as lost time, disability scores, or measures of depression. Most used walking programs, others either used bicycles or simply encouraged aerobic activities. Aerobic exercise, particularly self-directed, is low cost, not invasive and has low potential for adverse effects. Available evidence suggests that aerobic exercises may be more efficacious than other types of exercise for treatment of LBP. Weak evidence suggests weight bearing exercise may be superior. There is no quality evidence to support aerobic exercise for patients with post-operative pain. This review assumes that other chronic pain conditions respond similarly to aerobic exercise.

Rationale for Recommendations: Aerobic
Theoretical benefits of aerobic exercise include improved aerobic capacity, improved blood flow, lower depression, and higher pain thresholds and pain tolerance. These exercises include walking, running, bicycling, and many other activities. Whether there is benefit from weight-bearing versus non-weight bearing aerobic exercises remains unclear. There is evidence that a treadmill is superior to upper extremity or bicycle ergometers in assessing aerobic capacity in chronic LBP patients.(617) However, an exercise test is not believed to be necessary for the evaluation and treatment of the vast majority of LBP patients. For most patients, a structured, progressive walking program on level ground or no incline on a treadmill is recommended. There has been some controversy about whether bicycling is helpful or harmful from a biomechanical perspective (lordosis) as the back muscles are less active with bicycling, thus it may be less appropriate other than for spinal stenosis. Yet, if bicycling is the preferred exercise for the patient, it is believed to be far superior to obtaining no aerobic exercise. For patients who desire other aerobic exercises, there are no specific data, although there are indications that infer that there is a direct correlation between benefit and the amount of aerobic activity that results in higher MET expenditure. Therefore, the activity that the patient will adhere to is believed to be the one most likely to be effective, given that compliance is a recognized problem.

Rationale for Recommendations: Stretching
Stretching exercises may be the most widely utilized of the three major exercise domains. Stretching exercises include active movements to improve joint mobility and centralize symptoms, and flexibility exercises to increase the length of a target muscle group. There is longstanding dogma that this is the most important of the exercise domains, e.g. "one of the main goals of therapeutic exercise in low back disorder is to maintain and promote normal flexibility."(622) Stretching exercises also have been utilized for both treatment as well as prevention, and are used in some manufacturing settings as part of an "ergonomics program" or injury prevention program.

Rationale for Recommendations: Directional Exercises
Directional exercises are used most commonly to "centralize" and abolish symptoms when it has been determined that a patient has a *directional preference,* whether for extension, flexion, lateral bending or axial rotation.(85, 550, 612, 623-627) "Directional preference" is defined as back pain that centralizes or decreases with movement in one direction (e.g., flexion or left bending resulting in relief of the buttocks pain and centralizing that pain to only central lumbosacral pain) and that increases with motion in the opposite direction (e.g., extension or right bending). Directional preference exercises are then prescribed to be performed in the direction which centralizes and abolishes the pain. It is believed

important to also modify sitting posture temporarily consistent with the directional preference identified during patient assessment.

Historically, the two most widely used directional programs of exercises are referred to as Williams flexion exercises and McKenzie exercises.(612, 628) However, the direction of McKenzie exercises for each patient varies, determined by the directional examination findings that reflect the mechanical characteristics of the pain-generator. Directional exercises as part of McKenzie care are entirely passive in the lumbar spine, with either the patient, or occasionally a provider, providing the remote or external force to achieve the required end-range positioning or repetitions. There are many additional stretching exercises and these all involve standing or recumbent positions.

There is one primary theory, and considerable evidence to support it, regarding why directional exercises are effective. The cause of axial and more proximal leg pain is uncertain, yet the axial and more proximal pain frequently responds to directional testing and exercises. Repeated flexion loading on a disc may theoretically cause posterior nuclear displacement into a fissure or even creates a protrusion.(629, 630) Changing to repeated extension loading has been suggested to reverse or reduce that displacement.(631) This is consistent with patients in whom a directional preference is elicited who so often centralize their referred or radiating pain and then recover rapidly and fully using directional exercises and posture modifications.

There are several theories proffered to support the use of stretching exercises for purposes of preventing LBP or other musculoskeletal disorders. These include providing more flexibility and warming up the muscles. These theories have weaknesses. Providing more flexibility does not change a sarcomere, does not increase strength, will result in the performance of a task at the same percentage of maximum voluntary contraction, and thus is unlikely to provide an increased margin of safety. Stretching exercises also are unlikely to substantially warm up muscles as the aerobic demands of such activities are so minor. Perhaps these exercises may be useful for highly strenuous or otherwise demanding tasks to improve focus on the task at hand and use a smooth lifting technique that lowers peak physical demands. Another concern is the potential for adverse effects in an otherwise asymptomatic population. Flexibility varies in the population, yet there is a social drive to achieve a theoretically standard normal range of motion. Overstretching is more likely in those normal individuals with less flexibility. Such overstretching may result in a true strain which is painful and slow to heal.

There is a lack of evidence that generic stretching exercises are of assistance in treating patients with acute LBP.(632) There is relatively weak evidence suggesting that specific exercises(85, 633) may be of assistance among those with subacute or chronic LBP.

In addition, flexibility exercises are frequently targeted at muscles that are shortened in length, which often include the piriformis, quadratus lumborum, hamstring, hip flexor, and iliotibial band groups. Stretching exercises actively performed by patients for purposes of treatment and rehabilitation of LBP are low cost when performed as a home exercise program, are not invasive, and have low potential for adverse effects. They may help alleviate the stiffness that occurs with LBP that is thought to contribute to increased pain.

There is one reported low-quality RCT of aggressive stretching exercises for the treatment of chronic "myofascial" LBP,(634) but no duplication of those results in the literature. Thus, there is no quality evidence base for aggressive stretching. There are concerns that over-stretching may result in additional injuries to patients. Aggressive stretching requires a health care provider for each session and thus costs are considerably greater than those for self-performed stretching exercises. While they were not invasive, there are concerns that the potential for harm outweighs the potential for benefit. There are many other interventions with evidence of efficacy.

Rationale for Recommendations: Strengthening

Strengthening exercises may be theoretically used for purposes of improving maximum strength. Such improved strength would result in the ability to perform the same task at a lower percentage of maximum voluntary contraction, which in theory improves the individual's margin of safety. The evidence to support the theory is not particularly strong. A caution is that in the process of strengthening, sustaining a strain is possible. Another issue is that long-term compliance is required, is extremely difficult to achieve for all but the most highly motivated individuals. Fear avoidance belief training and principles appear important in the management of patients with LBP (see Fear Avoidance Belief Training). Inclusion of these principles in the course of exercise training or supervision appears highly desirable. This would also strengthen the education of the patient about LBP that should be a message in unison with other members of the team treating the patient.

There are multiple, heterogeneous studies that have evaluated exercise programs that either largely consisted of, or heavily relied upon, strengthening.(614-616, 635-642) Generally, these studies have demonstrated benefits, yet not all have demonstrated efficacy. For example, one study among subacute LBP patients showed a cognitive program was superior to the exercise arm.(609) As there are no high-quality studies of strengthening exercises and the study designs employed do not generally allow for a conclusion of efficaciousness above that obtained with the natural history of LBP, there is at least some concern that the strengthening exercises may have relatively low magnitudes of benefits.

There has been a trend towards stabilization or "core" strengthening exercises over the past decade. Stabilization exercises attempt to develop improved muscle strength and endurance of muscles that surround the spinal column (such as multifidus and transverse abdominus). There is some support for this theory,(614) but there are no high-quality studies demonstrating that stabilization exercises are superior to other strengthening exercise regimens. As there is evidence that a home exercise program is as effective as a supervised program for treatment of chronic LBP,(643) a home-based exercise program may be particularly cost effective while presumably resulting in the same benefits as a supervised program.

Dogma holds that strengthening abdominal muscles will variously successfully treat LBP, are effective for primary prevention, or prevent recurrence of LBP. However, abdominal muscles (rectus, obliques) are not materially involved in lifting tasks as they flex rather than extend the back; still, some believe they support the spine without a clearly defined mechanism of action. There also is no quality evidence that strengthening abdominal muscles is effective for either treatment or primary, secondary, or tertiary prevention of LBP. Abdominal strengthening exercises have been labeled an ergonomic myth.(644) That said, many providers instruct LBP patients in the activation of abdominal, trunk, and hip extensor muscles for the purpose of stabilizing the pelvis during lifting and activities of daily living. Traditional abdominal strengthening exercises such as sit-ups are not utilized in these stabilization programs.

Unfortunately, despite a plethora of literature, the vast numbers of possible permutations and combinations of exercises impairs the ability to identify specific exercises that demonstrate particular benefit. Additionally, there is some preliminary evidence that patients with differing clinical presentations of LBP do not benefit equally from all types of therapeutics. Rather, some patients are more likely to benefit from stabilization exercises,(645) while others benefit from specific directional exercises.(85) There are many different types of exercise that have been assessed in many different settings with heterogeneous populations of patients. Outcomes used are similarly quite heterogeneous (e.g., pain, modified duty, lost time, or disability ratings). While applicable throughout the spinal literature, there also has been a recognized problem with a concentration on finding statistical significance instead of clinical importance in the literature on exercise.(646)

There are also different schools of thought with different rationale for various sequences and combinations of exercises. Taken in composite, the evidence of a beneficial effect of exercise for the treatment of LBP is moderately strong, but taken individually, the evidence for any one exercise is generally weak or absent. A systematic approach to research investigating exercises for the treatment of LBP is clearly needed. Exercises can be segregated into different categories, but for purposes of this discussion, the three broad categories or "domains" of exercise will be utilized – aerobic, stretching/flexibility, and strengthening/stabilization.

Evidence for the Use of Exercise
There are 2 high-(647, 648) and 107 moderate-quality RCTs (one with multiple reports) incorporated into this analysis (see evidence table below).(28, 85, 529, 549, 550, 565, 586, 597, 600, 601, 605, 609, 613-615, 619, 620, 622, 624, 632, 633, 635, 637-640, 643, 645, 649-724, 725{Ben Salah Frih, 2009 #8875, 726, 727) Most articles have mixed various forms of exercise, thus this summary evidence overview does not attempt to segregate the evidence into the three broad domains of exercise – aerobic, stretching/flexibility, and strengthening/stabilization. Instead, summaries of the quality evidence are provided and later reviewed for each of the three exercise domains. One study was scored high quality; however, while it had quality study design features, it also had significant problems with heterogeneity of treatments in both the interventions and controls. There is a plethora of moderate-quality studies. The studies below are organized based on the type of study, acuity, and score. There are 36 low-quality RCTs in Appendix 1.(60, 537, 538, 610, 611, 621, 634, 636, 641, 673, 728-753)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: stretching and flexibility exercises, strengthening, strengthening exercise, abdominal strengthening exercises, abdominal exercises, abdominal, home exercise, program, subacute low back pain, chronic low back pain, acute low back pain, clinical trial, randomized controlled trial or random, post-operative, postoperative or post-surgery, systematic reviews, or reviews, and population study, epidemiological study, or prospective cohort Of the 110,821 articles found and reviewed, we included 141 articles.

AQUATIC THERAPY (INCLUDING SWIMMING)
Aquatic therapy involves the performance of aerobic and/or flexibility and/or strengthening exercises in a pool to minimize the effects of gravity, particularly where reduced weight-bearing status is desirable.(754-760, 761{Bender, 2005 #8827, 762)

1. *Recommendation: Aquatic Therapy for Select Patients with Subacute or Chronic Low Back Pain*
   A trial of aquatic therapy is recommended for the treatment of subacute or chronic low back pain in select patients.
   Indications – If patient has subacute or chronic LBP and meets criteria for a referral for supervised exercise therapy and has co-morbidities (e.g., extreme obesity, significant degenerative joint disease, etc.) that preclude effective participation in a weight-bearing physical activity, then a trial of aquatic therapy is recommended for the treatment of subacute or chronic LBP.
   Frequency/Duration – Program should generally begin with 3 to 4 visits per week. Patient should have demonstrated evidence of functional improvement within the first 2 weeks to justify additional visits. Program should include up to 4 weeks of aquatic therapy with progression towards a land-based, self-directed physical activity or self-directed aquatic therapy program by 6 weeks.

Indications for Discontinuation – Non-tolerance, failure to progress, or reaching a 4 to 6 week timeframe.

Benefits – Ability to engage in exercise and rehabilitation when unable to sufficiently tolerate weight-bearing exercises in a traditional physical therapy program.

Harms – Aggravation of pain during rehabilitation among a minority of patients.

   Strength of Evidence – **Recommended, Evidence (C)** [Chronic]
                                    Recommended, Insufficient Evidence (I) [Subacute]

   Level of Confidence – Moderate

2. *Recommendation: Aquatic Therapy for Acute and All Other Subacute or Chronic Low Back Pain*
   Aquatic therapy is not recommended for all other subacute or chronic low back pain patients or for all acute low back pain, as other therapies are believed to be more efficacious.

   Strength of Evidence – **Not Recommended, Insufficient Evidence (I)**
   Level of Confidence – Moderate

Rationale for Recommendations
All quality studies address chronic LBP and none address efficacy for acute or subacute LBP. One moderate-quality trial found mostly comparable results with a land-based therapy program(763) while another reported modest efficacy compared with wait-listed controls.(764) One trial compared exercise plus spa therapy with physical therapy exercise plus passive modalities and found few differences between the groups combined treatment.(765) Two moderate-quality trials compared mineral water with tap water and suggested benefits; however, they may be culturally biased.(766, 767) Aerobic exercise is felt to be beneficial for the rehabilitation of acute, subacute, and chronic LBP. However, a few select patients are unable to tolerate those land-based therapies. Aquatic therapy is moderate cost, not invasive, and has little potential for adverse effects.

Evidence for Use of Aquatic Therapy
There are 7 moderate-quality RCTs incorporated into this analysis.(594, 597, 763-767) There is 1 low-quality RCT in Appendix 1.(755)

We searched PubMed, EBSCO, Cochrane Review, and Google scholar without the limits on publication dates. We used the following search terms "(Aquatic therapy) AND (subacute OR chronic low back pain)" & "(Aquatic therapy OR Swimming AND (subacute OR chronic low back pain)" to find 7,435 articles. We included 10 articles (9 RCTs, 1 review).We also used the following search terms: balneotherapy, fangotherapy, water massage, subacute back pain, chronic back pain, low back pain, and postoperative to find 728 articles. Of the 728 articles, we reviewed 7 articles and included 5 articles.

LUMBAR EXTENSION MACHINES
Lumbar extension machines are intended to address LBP through the development of muscle strength in specific muscle groups through specific exercises.(768-770)

Recommendation: Lumbar Extension Machines for Acute, Subacute, or Chronic Low Back Pain or Any Radicular Pain Syndrome
Lumbar extension machines to strengthen the lumbar spine are not recommended for acute, subacute, or chronic low back pain or for any radicular pain syndrome.

   Strength of Evidence – **Not Recommended, Insufficient Evidence (I)**
   Level of Confidence – Low

Rationale for Recommendation
There is one moderate quality study of lumbar extension machines, but it has significant methodological issues and does not clearly demonstrate their utility in the treatment of LBP;(703) there are a few studies of low quality.(771, 772) The one moderate-quality RCT is also of relatively lower quality and has major flaws. There is no moderate- or high-quality evidence that strengthening on these machines is more effective than other strengthening exercises or other low-tech, low-cost exercise interventions.

Evidence for the Use of the Lumbar Extension Machines
There is 1 moderate-quality RCT incorporated into this analysis.(703) There are 5 low-quality RCTs in Appendix 1.(750, 773-776)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following terms: lumbar extension machines, low back pain to find 211 articles. Of the 211 articles we reviewed 8 articles (6 original RCT's and 2 reviews).

YOGA, TAI CHI, and PILATES
Yoga and Tai Chi have been used for treatment of chronic LBP.(579, 777-779) Yoga for purposes of treating LBP has not been standardized, but tends to involve postures, stretches, breath control, and relaxation. Traditional yoga is different and involves rules for personal conduct, postures, breath control, sense withdrawal, concentration, meditation, and self-realization,(780, 781) and different versions are practiced (e.g., Ashtanga, Iyengar, Hatha). This review focuses on the exercise aspects of yoga and tai chi and does not endorse or support spiritual elements or specific religious beliefs.

1. *Recommendation: Yoga for Chronic Low Back Pain*
   Yoga is recommended for select, highly motivated patients with chronic low back pain.

   Indications – Chronic LBP patients who are motivated to try and adhere to a program of yoga.

   Indications for Discontinuation – Non-tolerance and/or non-compliance.
   Benefits – *Modest* reductions in pain.
   Harms – May reduce compliance with aerobic and strengthening exercises due to time commitment. One report of back strain.

   > *Strength of Evidence* – **Recommended, Evidence (C)**
   > *Level of Confidence* – Low

2. *Recommendation: Yoga for Acute or Subacute Low Back Pain*
   There is no recommendation for or against the use of yoga for the treatment of acute or subacute low back pain.

   > *Strength of Evidence* – **No Recommendation, Insufficient Evidence (I)**
   > *Level of Confidence* – Low

3. *Recommendation: Tai Chi for Chronic Low Back Pain*
   Tai Chi is recommended for select highly motivated patients with chronic low back pain.

   Indications – Chronic LBP patients who are motivated to try and adhere to a program of Tai Chi.

   Indications for Discontinuation – Non-tolerance and/or non-compliance.

   Benefits – Modest reductions in pain*.*

Harms – None reported. May reduce compliance with aerobic and strengthening exercises due to time commitment.

> *Strength of Evidence* – **Recommended, Evidence (C)**
> *Level of Confidence* – Low

4. *Recommendation: Tai Chi for Acute or Subacute Low Back Pain*
    There is no recommendation for or against the use of Tai Chi for the treatment of acute or subacute low back pain.

> *Strength of Evidence* – **No Recommendation, Insufficient Evidence (I)**
> *Level of Confidence* – Low

6. *Recommendation: Pilates for Chronic Low Back Pain*
    There is no recommendation for or against the use of Pilates for treatment of acute, subacute, chronic or post-operative back pain.

> *Strength of Evidence* – **No Recommendation, Insufficient Evidence (I)**
> *Level of Confidence* – Low

Rationale for Recommendations
All quality studies of yoga address chronic LBP and none address efficacy for acute or subacute LBP. Different types of yoga have been assessed. There are some small studies that are likely underpowered.(782-784) The sizable studies generally show efficacy compared with an educational book,(784, 785) usual care,(786) breathing exercises and relaxation,(787, 788) and self-directed medical care.(789) However, yoga was not found superior to stretching classes,(647) raising questions about whether yoga may be inferior to aerobic and strengthening exercise. Due to these weaknesses the recommendation is downgraded to "C" level evidence.(783, 785) Patient motivation, compliance and adherence must be high and there is much self-selection in the studies. Yoga is not invasive, has low potential for adverse effects, and is low cost (self-administered is very low cost). It is recommended for highly select and motivated patients.

Tai Chi has been assessed in one study and some evidence of efficacy is suggested. As Tai Chi is not invasive, has few adverse effects and is low cost, it is recommended for highly select and motivated patients.

The few studies on Pilates have poor compliance rates and other methodological challenges(704, 790) that limit conclusions and result in no recommendation.

Evidence for the Use of Yoga, Tai Chi, and Pilates
There are 2 high-(647, 785) and 9 moderate-quality(704, 781-784, 786, 789-791) RCTs incorporated into this analysis. There is 1 low-quality RCTs in Appendix 1.(792)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: yoga, hatha yoga, subacute low back pain and chronic low back pain to find 13,685 articles. Of the 13,685 articles we reviewed 17 articles and included 16 articles.

General Treatment Approach

Many patients, but particularly chronic LBP patients tend to receive excessive treatments that are either minimally or completely ineffective. The pattern of treatments appears to follow the practitioner's practice, experience and qualifications. Examples of such excesses include polypharmacy, excessive therapy, ongoing manipulation, recurring injections, and multiple surgical procedures. Instead, the following are **Recommended (I)** approaches (see also Algorithms).

It is **Recommended, Insufficient Evidence (I)** that patients receive one or at most two medications and assess the benefits. A lack of clear functional benefits suggests a need to either discontinue the medication, try a different medication after discontinuation of the ineffective medication(s) or try a different treatment approach.

Similarly, physical therapy, manipulation and other physical treatment methods are **Recommended, Insufficient Evidence (I)** to be tried for at most 5 to 6 appointments. A lack of clear functional improvement indicates the treatment should be changed markedly or stopped altogether.

Ongoing invasive pain procedures are also **Recommended, Insufficient Evidence (I)** to not be repeated without objective evidence of major functional improvements.

Medications…………………………………………………………………

NON-STEROIDAL ANTI-INFLAMMATORY DRUGS (NSAIDs) AND ACETAMINOPHEN

Nonsteroidal anti-inflammatory drugs (NSAIDs) have been widely used for treatment of painful back conditions, including acute LBP, subacute LBP, chronic LBP, radicular, and post-operative patients and other back disorders.(793-801)

1. *Recommendation: NSAIDs for Treatment of Acute, Subacute, Chronic, Radicular, or Post-operative Low Back Pain*
   NSAIDs are recommended for treatment of acute, subacute, chronic, radicular, or post-operative low back pain. Evidence is strong for acute LBP, chronic LBP, and radicular pain syndromes (**Evidence (A)**) and moderately strong for subacute and post-operative LBP (**Evidence (B)**). Acetaminophen is a reasonable alternative, although evidence indicates it is modestly less efficacious.

   Generally, generic ibuprofen, naproxen or other older generation NSAIDs are recommended as first-line medications. Second-line medications should generally include one of the other generic NSAIDs. While COX-2 selective agents generally have been recommended as either third- or fourth-line medications to use when there is a risk of gastrointestinal complications, proton pump inhibitors, high-dose misoprostol, and sucralfate are also gastro-protective. COX-2 selective agents may still be used for those with contraindications to other medications, especially those with a history of gastrointestinal bleeding or past history of peptic ulcer disease.

   Indications – For acute, subacute, chronic, radicular, or post-operative LBP, NSAIDs are recommended for treatment. Over-the-counter (OTC) agents may suffice and may be tried first.

   Frequency/Duration – In most acute LBP patients, scheduled dosage rather than as needed is generally preferable. As needed prescriptions may be reasonable for mild or moderate LBP. The NSAID should generally be scheduled, rather than as-needed for treatment of more severe LBP especially if there is consideration for adjunctive treatment with muscle relaxants, opioids, or other potentially impairing medications. Once the patient moves to a supportive long-term care plan for

chronic back pain, the patient may revert to selective use for "flare ups," with some patients also using NSAIDs to maintain work status and function.

Indications for Discontinuation – Resolution of LBP, lack of efficacy, or development of adverse effects that necessitate discontinuation.

Benefits – Modest reduction in low back pain disorders and earlier recovery.

Harms – Gastrointestinal bleeding, other bleeding, and possible delayed fracture healing. Possible elevated cardiovascular risks including myocardial infarction, especially for high-dose COX-2 inhibitors. Renal failure may occur particularly in the elderly or those with otherwise compromised function.

> *Strength of Evidence* – **Strongly Recommended, Evidence (A)** – acute and chronic LBP, radicular pain
>
> **Moderately Recommended, Evidence (B) –** subacute, post-operative
>
> *Level of Confidence* – High

2. *Recommendation: NSAIDs for Patients at Risk for GI Adverse Effects*
   Concomitant prescriptions of cytoprotective medications are recommended for patients treated with non-selective NSAIDs at substantially increased risk for gastrointestinal bleeding. There are four commonly used cytoprotective classes of drugs: misoprostol, sucralfate, double-dose histamine Type 2 receptor blockers (famotidine, ranitidine, cimetidine, etc.), and proton pump inhibitors (esomeprazole, lansoprazole, omeprazole, pantoprazole, rabeprazole).(802) There also are combination products of NSAIDs/misoprostol.

   Indications – For patients with a high-risk factor profile who also have indications for NSAIDs, cytoprotective medications should be considered, particularly if longer term treatment with non-selective COX inhibiting NSAIDs is contemplated. At-risk patients include those with a history of prior gastrointestinal bleeding, the elderly, diabetics, and cigarette smokers.

   Frequency/Duration – Frequency as recommended by manufacturer.

   Indications for Discontinuation – Intolerance, development of adverse effects, or discontinuation of NSAID.

   Benefits – Reduced risk of gastrointestinal bleeding when used with an NSAID.

   Harms – Misoprostol may cause diarrhea. Other medications typically well tolerated, although as with all medications, allergic intolerances have been reported.

   > *Strength of Evidence* – **Strongly Recommended, Evidence (A) –** Proton pump inhibitors, misoprostol
   >
   > **Moderately Recommended, Evidence (B) –** Sucralfate
   > **Recommended, Evidence (C) –** H2 blockers
   >
   > *Level of Confidence* – High

3. *Recommendation: NSAIDs for Patients at Risk for Cardiovascular Adverse Effects*
   It is recommended that patients with known cardiovascular disease or multiple risk factors for cardiovascular disease have the risks and benefits of NSAID therapy for pain discussed. Degree of risk is believed to be associated with degree of COX inhibition. Lower risk of myocardial infarction is believed to be associated with naproxen and ibuprofen. Diclofenac is believed to have intermediate risk. High dose celecoxib is believed to have higher risk for myocardial infarction.
   Benefit – Counter risk of adverse event.
   Harms – None.

Strength of Evidence – **Recommended, Insufficient Evidence (I)**
Level of Confidence – High

4. *Recommendation: Acetaminophen/Aspirin for Patients at Risk for Cardiovascular Events*
Acetaminophen or aspirin is strongly recommended as the first-line therapy for patients with high risk of cardiovascular events as these appear to be the safest.
Benefits – Addresses LBP without increased risk of cardiovascular event.
Harms – Less effective than NSAID. Aspirin also more prone towards gastrointestinal bleeding and other hemorrhage.

> *Strength of Evidence* – **Strongly Recommended, Evidence (A)**
> *Level of Confidence* – High

If needed, NSAIDs that are non-selective are preferred over COX-2 selective drugs. In patients receiving low-dose aspirin for primary or secondary cardiovascular disease prevention, to minimize the potential for the NSAID to counteract the beneficial effects of aspirin, the NSAID should be taken at least 30 minutes after or 8 hours before the daily aspirin.(803)

5. *Recommendation: Acetaminophen for Treatment of Low Back Pain*
Acetaminophen is recommended for treatment of low back pain with or without radicular symptoms, particularly for those with contraindications for NSAIDs.

Benefit – Addresses LBP among those unable to tolerate an NSAID.
Harms – Less effective than NSAID.
> *Strength of Evidence* **–** Recommended, Evidence (C)
> *Level of Confidence* – High

Rationale for Recommendations
There are many quality trials that NSAIDs improve pain and some report higher subjective functional status (see evidence table). Evidence is strong and nearly consistent among the high-quality studies for treatment of acute LBP,(804) chronic LBP,(805-807) and radicular pain.(808) Evidence is moderate for subacute and post-operative pain.(809-811) There is only one high-quality trial with negative results for NSAIDs compared with placebo.(812)

There are several classes of NSAIDs: 1) salicylates [aspirin, diflunisal, salicyl salicylate (salsalate)], 2) arylalkanoic acids (diclofenac, etodolac, ketorolac, nabumetone, sulindac, tolmetin), 3) 2-arylpropionic acids (ibuprofen, fenoprofen, ketoprofen, naproxen), 4) n-arylanthranilic acids (mefenamic acid), 5) oxicams (piroxicam, meloxicam), 6) COX-2 inhibitors (celecoxib, rofecoxib, etoricoxib), and 7) sulphonanilides (nimesulide). Acetaminophen is considered an analgesic that is not an anti-inflammatory agent. Acetaminophen blocks the activation of COX by another enzyme, peroxidase. Tissues with high levels of peroxidase (i.e., platelets and immune cells) are "resistant" to acetaminophen, but tissues with low levels of peroxidase (i.e., nerve and endothelial cells that participate in pain and fever) are "sensitive" to acetaminophen.(813)

There are two isoenzymes of cyclooxygenase, COX-1 and COX-2. NSAIDs are (non) selective to different degrees. COX-2 selective agents were designed to reduce inflammation while not increasing risks for gastrointestinal bleeding. It appears that certain COX-2 selective agents may increase the risk of cardiovascular events.

There is a dearth of trials comparing the various NSAIDs, and the doses used are at times submaximal in some of the comparative arms of the trials, raising major problems with direct comparability to help guide specific NSAID selection. As piroxicam is the only medication to have a trial showing lack of benefit compared with placebo,(814) and there is quality evidence that suggests it is inferior for management of lateral epicondylitis, piroxicam should generally be avoided as either a first-, second-line agent in the management of musculoskeletal disorders including LBP.(815-817) It appears that despite widespread usage, diclofenac does not have superiority for LBP, and as it may have increased risks for adverse cardiovascular events,(818) it generally should not be used as a first or second-line agent. Otherwise, evidence that one medication is superior to another is lacking.

Cardiovascular risks of NSAIDs are somewhat controversial.(803) Most studies have suggested elevated risks with high-dose rofecoxib, few have shown elevated risks with ibuprofen or naproxen, and there is some evidence for increasing risks with greater degrees of COX-2 inhibition.(818-825) The sequence of NSAIDs from lowest COX-2 to highest varies somewhat between studies but is reportedly: flurbiprofen, ketoprofen, fenoprofen, tolmetin, aspirin, oxaprozin, naproxen, indomethacin, ibuprofen, ketorolac, piroxicam, nabumetone, etodolac, celecoxib, meloxicam, mefenamic acid, diclofenac, rofecoxib and nimesulide.(826)

There are few quality studies of acetaminophen as a single agent. However, paracetamol, a close analog, has been studied more extensively and has some evidence of mild efficacy in most trials,(827) although a recent review concluded it lacks efficacy.(801) Most studies have used these agents, particularly paracetamol, as rescue agents in RCTs. The direct evidence of efficacy from the two available studies suggests paracetamol is not quite as successful at alleviating LBP as diflunisal,(828) mefenamic acid,(809) indomethacin,(809) or aspirin.(809) It also has relieved pain less successfully than the muscle relaxants orphenadrine(829) and parazolidin.(830) It is interesting that paracetamol appears more effective in combination with orphenadrine than as a single agent.(831) There is one trial suggesting it is more efficacious than physiotherapy and manipulation,(832) and worse than electroacupuncture.(833) Acetaminophen (4,000mg per day) was modestly superior to ibuprofen in the heat wrap study, but the trial's utilization of a relatively low ibuprofen dose of 1,200mg a day precludes a direct comparison.(834) Acetaminophen was worse than chlorzoxazone(835) and was inferior to diflunisal even when combined with codeine.(836) Thus, while the evidence suggests efficacy of acetaminophen and paracetamol, it appears these medications are modestly less efficacious than NSAIDs (although safer).

NSAIDs are not invasive, have low side effect profiles in a healthy working-age patient population, and when generic medications are used are low cost. The potential for NSAIDs to increase the risk of cardiovascular events needs to be carefully considered in high-risk patients and will likely require additional quality studies to fully address. There is substantial, quality evidence that COX-2 selective NSAIDs reduce the risk of adverse GI effects.(820, 837-840) Additionally, the four commonly used cytoprotective classes of drugs are proton pump inhibitors, misoprostol, sucralfate, and double-dose histamine-type 2 receptor blockers (see Hip and Groin Disorders Guideline for details).

Evidence for the Use of Non-steroidal Anti-inflammatory Drugs (NSAIDs) and Acetaminophen
There are 12 high-(804, 806-808, 812, 841-847) and 37 moderate-quality RCTs (one with two reports)(683, 805, 809-811, 814, 817, 828, 834, 848-872, 873{Le Roux, 1999 #8095, 874-876) incorporated into this analysis. There are 2 low-quality RCTs(877, 878) and 3 other studies(879-881) in Appendix 1.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following terms: NSAIDs, nonsteroidal anti-inflammatory drugs, aspirin, acetaminophen, diflunisal, salsalate, Ibuprofen, Dexibuprofen, Dexdetoprofen, Naproxen, Fenoprofen, Ketoprofen, Dexketoprofen, Flurbiprofen, Oxaprozin, Loxoprofen, Indomethacin, Tolmetin, Sulindac, Etodolac, Ketorolac, Diclofenac and, Nabumetone,

Piroxicam, Meloxicam, Tenoxicam, Droxicam, Lornoxicam, Isoxicam, Celecoxib, Etodolac , Etoricoxib , Firocoxib , Licofelone , Lornoxicam , Lumiracoxib , Meclofenamic acid , Mefenamic acid, Nimesulide, Parecoxib, Rofecoxib, Tolfenamic acid, Valdecoxib and low back pain to find 131,158 articles. Of the 131,158 articles we included 31 articles. We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following terms: acetaminophen, paracetamol, ibuprofen, and low back pain to find 122,114 articles. Of the 122,114 articles we reviewed 9 articles and all were included.

ANTIBIOTICS

Antibiotics have been used for treatment of LBP with Modic changes and bone edema.(882, 883)

1. *Recommendation: Antibiotics for Acute, Subacute, and Other Chronic or Radicular Low Back Pain, including Chronic Low Back Pain with Modic I Changes*
    There is no recommendation for antibiotics for treatment of chronic low back pain with Modic I changes lacking objective signs of infection.

    Strength of Evidence – No Recommendation, Insufficient Evidence (I)
    Level of Confidence – Low

Rationale for Recommendations
There are two quality studies of the efficacy of antibiotics for the narrow indication of chronic LBP with Modic changes (883) and these studies conflict regarding efficacy; thus there is no recommendation. Antibiotics are not invasive, have relatively low adverse effects, and are low to moderate cost for 100 days treatment. However, with conflicting evidence of efficacy, there is no recommendation for or against use of antibiotics for treatment of LBP absent evidence of infection.

Evidence for the Use of Antibiotics
A comprehensive literature search was conducted using PubMed, Scopus, CINAHL, Cochrane Library, and Google Scholar without date limits using the following terms: Antibiotics; antibacterial agents, anti-infective agents, low back pain, radicular pain syndrome, radiculopathy, nerve compression syndromes, sciatica, sciatic neuropathy, spinal stenosis, modic changes, controlled clinical trial, controlled trials, randomized controlled trial, randomized controlled trials, random allocation, random*, randomized, randomization, randomly; systematic, systematic review, retrospective, prospective studies; not pediatric, and not adolescents. We found and reviewed 212 articles in PubMed, 281 in Scopus, 281 in CINAHL, 5 in Cochrane Library, 496 in Google Scholar, and 2 from other sources[†]. We considered for inclusion 7 from PubMed, 2 from Scopus, 1 from CINAHL, 0 from Cochrane Library, 0 from Google Scholar, and 2 from other sources. Of the 12 articles considered for inclusion, 3 randomized trials and 5 systematic studies met the inclusion criteria.
[†] The results for databases are sorted by relevancy based on customized search term algorithms. Algorithms for each database determine relevancy. The first 100 articles are reviewed in each search, and if relevant literature appears in the first 100 articles, we review an additional 100 articles. If relevant articles appear in these additional 100 articles, we then review another 100. We continue this pattern of review until we review a batch of 100 articles that contains no relevant literature. When this happens then the remaining articles are not reviewed due to a lack of relevancy.

ANTI-DEPRESSANTS

Anti-depressants have been widely utilized for the treatment of chronic pain, including chronic LBP. This review addresses uses for LBP (see the Chronic Pain Guideline for a more detailed discussion). These

recommendations are segregated into whether the anti-depressant blockes norepinephrine or not (including dual serotonin-norepinephrine agents), as that appears to be the critical feature that produces efficacy for treatment of pain.

1. *Recommendation: Serotonin-Norepinephrine Reuptake Inhibitors (SNRIs) aka "Dual Action Agents," and Tricyclic Antidepressants (TCAs) for Acute, Subacute, and Chronic Low Back Pain*
Norepinephrine reuptake inhibitor anti-depressants (e.g., tricyclic anti-depressants – amitriptyline, imipramine, nortriptyline, desipramine, maprotiline, doxepin) and mixed serotonin norepinephrine reuptake inhibitors (e.g., duloxetine) are recommended for the treatment of acute, subacute, and chronic low back pain. This recommendation does not include "SSRIs."

Indications – Chronic LBP that is not fully resolved with NSAIDs and an exercise program. Some evidence of efficacy for acute and subacute LBP. There is some evidence of efficacy for LBP with radiation to proximal extremity, but distal radiation (i.e., sciatica) has not been clearly studied in quality studies. This intervention may be more helpful where there is insomnia (especially where habituating agents are not recommended), nocturnal sleep disruption, depression, dysthymia and anxiety.

Frequency/Duration – Generally prescribed at a low dose at night and gradually increased (e.g., amitriptyline 25mg QHS, increase by 25mg each week) until a sub-maximal or maximal dose is achieved, sufficient effects are achieved, or adverse effects occur. Most practitioners use lower doses, (e.g., amitriptyline 25 to 75mg a day to avoid adverse effects and necessity of blood level monitoring), as there is no evidence of increased pain relief at higher doses. Imipramine is less sedating, thus if there is carryover daytime sedation, it may be a better option. If the patient cannot sleep at night, amitriptyline is the recommended initial medication to prescribe.

Indications for Discontinuation – Resolution of pain, intolerance, or development of adverse effects.
Benefits – Modest improvements in LBP. May improve sleep quality.
Harms – Daytime somnolence, interference with work, dry mouth, cardiac risks, and other adverse effects.

> *Strength of Evidence* **–** Strongly Recommended, Evidence (A) **(Chronic)**
> *Strength of Evidence* – Recommended, Evidence (C) (Acute, Subacute)
> *Level of Confidence* – Moderate

2. *Recommendation: Serotonin-Norepinephrine Reuptake Inhibitors (SNRIs) aka "Dual Action Agents," and Tricyclic Antidepressants (TCAs) for Post-operative and Radicular Low Back Pain*
There is no recommendation for or against use of norepinephrine reuptake inhibitor anti-depressants (e.g., tricyclic anti-depressants – amitriptyline, imipramine, nortriptyline, desipramine, maprotiline, doxepin) and mixed serotonin norepinephrine reuptake inhibitors (e.g., duloxetine) for treatment of post-operative or radicular low back pain absent other indicators for treatment, as there is no quality evidence supporting their efficacy. They may be a reasonable option for select cases particularly with sleep disruption with concerns regarding habituating agents or inability to manage with NSAIDs or other agents. There is some evidence of efficacy for treatment of patients with proximal limb radiation.(899,906)

> *Strength of Evidence* – **No Recommendation, Insufficient Evidence (I)**

3. *Recommendation: SSRIs for Acute, Subacute, Pos-operative, Radicular and Chronic Low Back Pain*
Selective serotonin reuptake inhibitors (e.g., citalopram, escitalopram, fluoxetine, paroxetine, sertraline) are strongly not recommended for treatment of chronic low back pain. (They may be

effective for treatment of depression, dysthymia and other psychiatric conditions.) **They also are not recommended for treatment of acute, subacute, radicular or post-operative LBP.**

Strength of Evidence – **Strongly Not Recommended, Evidence (A)** (Chronic)
Strength of Evidence – **Not Recommended, Insufficient Evidence (I)** (Acute, subacute, radicular, post-operative LBP)
Level of Confidence – Moderate

Rationale for Recommendations
There are multiple placebo-controlled trials evaluating efficacy of anti-depressants for treatment of LBP, with nearly all studies evaluating chronic LBP (see evidence table). Some included patients with depression while some specifically sought to exclude those with depression. Effects appear to differ by class of agent.

Selective Serotonin-Reuptake Inhibitor Anti-depressants (SSRIs): Bupropion and Trazodone
There were four trials of anti-depressants that primarily inhibit serotonin reuptake for the treatment of chronic LBP. Two high-quality studies evaluated paroxetine 20mg or 30mg in the treatment of chronic LBP and neither found evidence of efficacy.(885, 886) One study enlisted patients with depression and found no benefit except a tendency toward increased use of analgesics while on paroxetine. The other study did not include patients with depression. One moderate-quality trial of trazodone (150mg a day) did not show benefit in any measure of pain or function among subjects with at least 1 year of LBP.(887) One moderate-quality crossover trial of bupropion (300mg a day for 16 weeks) among subjects with at least 6 months of LBP failed to find improvement in back pain or other measures of function.(888)

Norepinephrine-Reuptake Inhibitor Anti-depressants (Tricyclic Anti-depressants) and Dual Reuptake Inhibitors (SNRIs)
Six quality RCTs of tricyclic anti-depressants (TCAs) in the treatment of chronic LBP were found. Two moderate-quality studies evaluated imipramine. One compared 150mg nightly for 8 weeks with placebo for LBP of at least 6-weeks duration and found that those taking imipramine had significantly fewer limitations with work or activities.(889) A second study evaluated 75mg for 1 month and found non-significant improvements in pain scores.(890) A moderate-quality randomized crossover study evaluated the efficacy of varying doses of amitriptyline for 6 weeks for treatment of LBP (at least 1 year duration) and found subjective improvements, no change in activity level, and declines in analgesic usage of approximately 50% while on treatment.(891) One high-quality study of nortriptyline evaluated 100mg a day among primary care subjects with chronic LBP and found significant improvements in pain scores and borderline disability scores.(892) One high-quality study of maprotiline found it superior to either placebo or paroxetine for LBP.(885) Doxepin (over 200mg nightly) was evaluated in a moderate-quality study and found to improve pain scores.(893) There is limited evidence that TCAs result in modest reductions in pain ratings in the treatment of radicular pain compared with placebo. There is no quality evidence of an association between serum levels and pain relief, suggesting that doses less than those used for depression may be sufficient.(889, 892) Two trials with 3 reports have reported efficacy of duloxetine for treatment of chronic pain.(894-896)

One study specifically sought to treat those with sciatica and found no significant benefits from morphine, nortriptyline, or a combination compared with a control for radicular pain.(897) However, other studies have included some with radiating pain into an extremity. Thus, evidence for use of antidepressants for treatment of radicular pain is unclear.

Norepinephrine reuptake inhibitor anti-depressants are not invasive, have low to moderate dose-dependent adverse effects at low doses, and are not costly in their generic formulations. The degree to which depression or dysthymia is present may suggest earlier use of these medications. Discussions with mental health professionals

may be helpful, particularly when mental health conditions are more severe. Norepinephrine reuptake inhibiting anti-depressants are recommended for treatment of chronic LBP.

Evidence for the Use of Anti-depressants
There are 4 high-(885, 886, 892, 897) and 14 moderate-quality(887-891, 893-896, 898-902) RCTs or crossover trials incorporated into this analysis. There is 1 1ow-quality RCT with two reports in Appendix 1.(903, 904)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following terms: anti-depressants, antidepressants, Citalopram, Escitalopram, Paroxetine, Fluoxetine, Fluvoxamine, Sertraline, Desvenlafaxine, Duloxetine, Milnacipran, Tramadol, Sibutramine, Etoperidone, Lubazodone, Nefazodone, Trazodone, Jegguzine, Atomoxetine, Reboxetine, Viloxazine, Bupropion, Dexmethylphenidate, Methylphenidate, Amphetamine, Dextroamphetamine, Dextromethamphetamine, Lisdexamfetamine, Amitriptyline, Butriptyline, Clomipramine, Desipramine, Dosulepin, Doxepin, Imipramine, Iprindole, Lofepramine, Melitracen, Nortriptyline, Opipramol, Protriptyline, Trimipramine, Amoxapine, Maprotiline, Mianserin, Mirtazapine, Isocarboxazid, Moclobemide, Phenelzine, Pirlindole, Selegiline, Tranylcypromine, and low back pain to find 368,696 articles. Of the 368,696 articles we reviewed 8 articles and all were included. For Serotonin Reuptake Inhibitors- We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: serotonin reuptake inhibitors, paroxetine, bupropion, trazodone, duloxetine, chronic low back pain to find 62,545 articles. Of the 62,545 articles, we reviewed eight articles and included seven articles. For Norepinephrine reuptake inhibitors- We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: norepinephrine reuptake inhibitor antidepressants, tricyclic antidepressant, amitriptyline, imipramine, nortriptyline, maprotiline, doxepin, SNRI, chronic low back pain, radicular pain, and sciatica to find 24,991 articles. Of the 24,991 articles, we reviewed 21 articles, and included 21 articles (15 RCTs and 6 reviews).

ANTI-CONVULSANT AGENTS
Anti-convulsant agents have been utilized off-label for some chronic pain syndromes since the 1960s, prominently including neuropathic pain, chronic radicular syndromes and diabetic neuropathy.(905-910) Reported uses have expanded to include treatment of nociceptive pain, fibromyalgia, and non-specific pain syndromes. Gabapentin, a GABA analog, is an anti-convulsant originally approved by the U.S. Food and Drug Administration (FDA) for treating seizures, particularly in conjunction with other anti-convulsants. The FDA later approved its use as a treatment of neuropathic pain. The mechanism of action is unknown. It is believed to act directly on the central nervous system, although not at the GABA receptor. Pregabalin is also an anti-convulsant and is used to treat neuropathic pain (see Chronic Pain Guideline for more details).

1. *Recommendation: Anti-convulsants for Peri-operative Pain Management*
   Gabapentin or pregabalin are strongly recommended for peri-operative management of pain to reduce the need for opioids, particularly in patients with adverse effects from opioids.

   Indications – Peri-operative pain management. Caution is warranted if the patient is taking pre-operative opioids.

Frequency/Dose – Varying doses used. Highest quality studies suggest gabapentin 300mg,(911) 600mg,(912) 800mg,(913) and 1200mg(914) 1 to 2 hours pre-operatively. Two studies suggested re-dosing 12 hours post-op of either gabapentin or pregabalin.(915, 916)

Indications for Discontinuation – Resolution or intolerance. Careful monitoring of employed patients is indicated due in part to elevated risks for CNS-sedating adverse effects.

Benefits –Reduced opioid use, which may potentially speed recovery and produce better outcomes.

Harms – Drowsiness, dizziness and other CNS sedating effects are the most common adverse effects. Increased fatalities associated with opioids (2392).

> *Strength of Evidence* **–** Strongly Recommended, Evidence (A)
> *Level of Confidence* – High

2. *Recommendation: Anti-convulsants for Peri-operative Pain Management*
   There is no recommendation for or against the use of other anti-convulsant agents for peri-operative management of pain to reduce need for opioids, particularly in patients with adverse effects from opioids.

   > *Strength of Evidence* –**No Recommendation, Insufficient Evidence (I)**

3. *Recommendation: Topiramate for Chronic Low Back Pain*
   Topiramate is recommended for chronic non-neuropathic pain or low back pain among patients with depression or anxiety.

   Indications for Initiation – Chronic LBP patients with depression or anxiety. Failure of multiple other modalities including trials of different NSAIDs, aerobic exercise, specific stretching exercise, strengthening exercise, anti-depressants, and distractants.

   Frequency/Dose – This medication is initiated by gradually increasing the dose – beginning at 50mg and increasing by 50mg/day each week.(917) The most appropriate steady dose is unclear, but appears to be 300mg. Patients should be carefully monitored for the development of adverse events.

   Indications for Discontinuation – Resolution, development of adverse effects, lack of improvement, or failure to adhere to a functional restoration program. Careful monitoring of employed patients is indicated due in part to elevated risks for central nervous system- (CNS) sedating adverse effects.

   Benefits – Modest reductions in pain and may improve psychological profile. Potential to spare need for more impairing medications.

   Harms – Sedative effects are the highest risks especially in safety-sensitive or cognitively demanding positions.

   > *Strength of Evidence* **– Recommended, Evidence (C)**
   > *Level of Confidence* – Low

4. *Recommendation: Anti-convulsants for Acute, Subacute, or Chronic Low Back Pain*
   Other anti-convulsants, including gabapentin, are not recommended for acute, subacute, or chronic low back pain (918)**.**

   > *Strength of Evidence* **– Not Recommended, Evidence (C)**

5. *Recommendation: Anti-convulsants for Radicular Pain Syndromes*

Anti-convulsants, including gabapentin and pregabalin, are not recommended for chronic radicular pain syndromes (918-920) While there is evidence of efficacy for peripheral neuropathies (see Chronic Pain Guideline), the highest quality study of pregabalin for radicular pain was negative (2406).

>   *Strength of Evidence –* **Not Recommended, Evidence (C)**

6. *Recommendation: Gabapentin for Severe Neurogenic Claudication*
   Gabapentin is recommended for treatment of severe neurogenic claudication with limited walking distance.

   Indications – Severe neurogenic claudication from spinal stenosis or chronic radicular pain syndromes.

   Indications for Discontinuation – Resolution or intolerance. Careful monitoring of employed patients indicated due in part to elevated risks for CNS-sedating adverse effects. If gabapentin dose is reduced, discontinued, or substituted with an alternative medication, this is recommended to be done gradually over a minimum of 1 week (a longer period may be needed at the discretion of the prescriber).
   Benefits – Improved walking distance
   Harms – Drowsiness, dizziness and other CNS sedating effects are the most common adverse effects. Increased fatalities associated with opioids (2392).
   >   *Strength of Evidence –* **Recommended, Evidence (C)**
   >   *Level of Confidence –* Moderate

Rationale for Recommendations

There are a few quality studies evaluating other anti-epileptic medications for LBP and related disorders.(917, 921, 922) This class of medications has long been thought to be effective in treating neuropathic pain. However, that may not be correct,(917) as there appears no clear pattern to indicate that a single conclusion of efficacy for this class of medications for a group of disorders is accurate. Instead, treatments appear to require specification or individualization. There is quality evidence that topiramate is effective for treating chronic LBP,(917) thus an anti-epileptic has been shown to be effective for nociceptive pain instead of neuropathic pain.

The most commonly used medication in this class may be carbamazepine. However, as it has been available in a generic formulation, it has not been studied in large-, moderate-, or high-quality studies for purposes of treating chronic pain. There is however some evidence from both an experimental design,(921) as well as from inference from a chemically related compound, oxcarbazepine,(906) that it is useful for treatment of neuropathic pain. Thus, it presumably has some efficacy for treatment of chronic radicular pain syndromes.

Gabapentin and the closely related compound pregabalin have been evaluated in quality studies for treatment of multiple pain syndromes. However, results are not uniformly positive for all conditions (see Chronic Pain Guideline for other conditions). A meta-analysis failed to find statistical benefit of gabapentinioids for treatment of LBP and reported several adverse effects (918) One study analyzed neurogenic claudication and found significant improvements in distances walked.(923) Studies do not

clearly indicate whether the overall risk/benefit analysis favors use of gabapentin for treatment of LBP (other than perhaps pre-operatively) given that its use can be associated with moderately significant side effects, such as nausea (19%), dizziness (24%) and mentation problems.(918, 923, 924) Results for other spine conditions conflict.

Gabapentin has been shown to reduce post-operative pain and the need for opioids in patients undergoing back surgery (2407). Almost all of these studies except one,(913) showed efficacy, with one showing significant, dose-dependent reductions across a range of 4 different doses.(912) Thus, quality evidence documents that gabapentin reduces the need for post-operative opioids. It has not been shown effective for LBP. One study on chronic radicular pain is of short-term duration(920) and another 1 month study of pregabalin found little efficacy for treatment of chronic radicular symptoms.(919) Gabapentin has beneficial effects (distance walked) for patients with severe spinal stenosis.(923) Gabapentin and pregabalin are not invasive, have moderately significant side effects, and are moderately costly. Side effects are largely CNS-related and are of concern in employed populations. Gabapentin and pregabalin are not controlled substances, but do have psychoactive properties and therefore do carry slight risks of abuse.

Anti-epileptic agents may be reasonable fourth- or fifth-line treatments (e.g., after trials of different NSAIDs, aerobic exercise, other exercise) to attempt to treat chronic radicular symptoms. Physicians prescribing such agents in patients employed in safety-sensitive positions should be aware that such medications may raise concerns about fitness for duty due to the possibility of a seizure disorder. These drugs are not invasive, have some adverse effects, and may be moderately costly. There is no evidence for efficacy in chronic radicular pain syndromes, but these medications have been used for treatment, although not as first- or second-line treatments, as NSAIDs, muscle relaxants, aerobic exercise, other exercise, and manipulation are all likely more efficacious.

Evidence for the Use of Anti-convulsant Agents
We searched PubMed, EBSCO, Google Scholar, and Cochrane Review with limits on publication dates from 2011-2012 and then an updated search was conducted using PubMed for publications between 1/1/2013 and 11/15/2017. We used the following search terms: radicular pain syndrome, sciatica, carbamazepine, anti-convulsant agents, and neuropathic pain, randomized clinical trial or randomized controlled trial or random, systematic review or reviews, population study or epidemiological study or prospective cohort to find 2,022 articles. Of the 5,420 articles, we reviewed 20 articles and included 20 articles (16 randomized controlled trials and 4 systematic reviews.

BISPHOSPHONATES
Bisphosphonates reduce osteoclastic activity, resulting in net gain of bone mass. While more popularly used for treating and preventing osteoporosis, bisphosphonates have been used to treat CRPS.(925) (See Chronic Pain Guideline). They have been postulated to have analgesic properties.(926)

Recommendation: Bisphosphonates for Chronic Low Back Pain
Bisphosphonates are not recommended for patients with chronic low back pain.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Moderate

Rationale for Recommendation

There are no quality studies evaluating the use of bisphosphonates for chronic LBP. Bisphosphonates are either not invasive in oral formulations or are minimally invasive in parenteral administrations. They are moderate to high cost and have adverse effects that include gastritis, reflux esophagitis (can be severe and erosive causing stricture and achalasia), subtrochanteric hip fracture, and osteonecrosis of the jaw (uncommon). Based on the literature, their use is recommended for consideration as an option for CRPS in patients who have remained symptomatic despite other interventions (see Chronic Pain Guideline). However, since there is no evidence for LBP, they are not recommended.

Evidence for the Use of Bisphosphonates
There are no quality studies incorporated into this analysis.

We searched PubMed, EBSCO, Google Scholar, and Cochrane Review with no limits on publication dates. The search terms used included Bisphosphonates, chronic low back pain, Clinical trial, randomized controlled trial, random. Of those, we included none of the RCTs and reviews.

CALCITONIN
Calcitonin, the lesser known of the thyroid's two main hormones, is secreted by parafollicular cells, and is involved in increasing calcium uptake from the GI tract while also decreasing bone resorption. It is also thought to have anti-nociceptive effects that have not been well elucidated.(927)

Recommendation: Calcitonin for Chronic Low Back Pain
Calcitonin is not recommended for the treatment of chronic low back pain.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Moderate

Rationale for Recommendation
There is no evidence of efficacy. Calcitonin is minimally invasive, has relatively few adverse effects, and is moderately costly (see Chronic Pain Guideline). Adverse effects are relatively rare and include nausea, vomiting, decreased appetite, abdominal pain, injection site reactions, nasal symptoms, rhinitis, sinusitis, anaphylaxis, bronchospasm, hypersensitivity reactions, osteogenic sarcoma, and hypocalcemic tetany.

Evidence for the Use of Calcitonin
There are no quality studies incorporated into this analysis.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: Calcitonin, chronic, low, back, and pain to find 32,668 articles. Of the 32,668 articles, we reviewed zero articles and included zero articles.

COLCHICINE
Colchicine inhibits microtubule formation. Its primary use is to treat acute gout attacks. Because of its anti-inflammatory properties, it has been used to treat LBP. Thiocolchicoside is a muscle relaxant derived from colchicoside.

1. *Recommendation: Oral and IV Colchicine for Acute, Subacute, or Chronic Low Back Pain*
   Oral and IV colchicine are not recommended for treatment of acute, subacute, or chronic low back pain.

   > *Strength of Evidence* **–** Not Recommended, Insufficient Evidence (I)
   > *Level of Confidence* – Moderate

2. *Recommendation: Thiocolchicoside for Acute, Subacute, or Chronic Low Back Pain*

There is no recommendation for or against the use of thiocolchicoside for the treatment of acute, subacute, or chronic low back pain.

Strength of Evidence – No Recommendation, Insufficient Evidence (I)

Rationale for Recommendations
The results from studies of colchicine are conflicting and there is no clear evidence of lasting benefit.(928-930) Newer results with thiocolchicoside are more impressive,(931, 932) but need to be replicated by a different group. Intravenous or intramuscular colchicine is invasive, moderately expensive, has potentially serious adverse effects, and has not been shown to be superior to placebo. Oral colchicine is not invasive, has adverse effects, is not costly, but has not been shown to be superior to placebo.

Evidence for Use of Colchicine
There are 5 moderate-quality RCTs incorporated into this analysis.(928-932)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: Oral colchicine, colchicine, Thiocolchicoside, IV placebo, Oral TCC, tizanidine, subacute, low, back, pain, and chronic to find 20,676 articles. Of the 20,676 articles, we reviewed and included 5 articles.

KETAMINE
Ketamine is a strong NMDA receptor antagonist that is also a general anesthetic and has been used orally and intravenously to treat CRPS(933-935) and other neuropathic pain conditions (see Chronic Pain Guideline). Ketamine affects a number of receptors and inhibits serotonin and dopamine reuptake and has also been used as an adjunct to psychotherapy in alcohol and heroin addiction.(936)

Recommendation: Ketamine for Chronic Low Back Pain
Ketamine infusion is not recommended for treatment of chronic low back pain.

Strength of Evidence – **Not Recommended, Insufficient Evidence (I)**
Level of Confidence – High

Rationale for Recommendation
High-quality experimental studies show intravenous ketamine can lead to pain reductions in patients with chronic neuropathic pain; however, the pain reduction paralleled the length of the infusion with follow-up periods of 160 minutes or less. Adverse effects were considerable.(937, 938) Lower, oral doses have been associated with lightheadedness, dizziness, tiredness, headache, bad dreams, and sensory changes. Ketamine has high abuse potential and when used as a general anesthetic leads to direct myocardial and respiratory depression. Ketamine is invasive, has adverse effects (e.g., respiratory depression and hallucinations), and is moderate to high in cost. Other treatments have evidence of efficacy. Ketamine is not recommended for diagnostic or therapeutic use until clinical studies demonstrate efficacy.

Evidence for the Use of Ketamine
There are 2 high-(937, 938) and 3 moderate-quality(939-941) RCTs/ crossover trials incorporated into this analysis.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar with no limits on publication dates. We used the following terms: ketamine infusion, ketalar infusion, intravenous ketamine, intravenous ketalar, chronic low back pain and low back pain. This search found 1,100 articles, we reviewed 557 and included 5 articles.

KETANSERIN

Ketanserin is a selective S2 serotonergic antagonist that has been used to treat patients with CRPS (see Chronic Pain Guideline).

Recommendation: Ketanserin for Chronic Low Back Pain
Ketanserin is not recommended for treatment of chronic low back pain.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Moderate

Rationale for Recommendation
There are no quality studies evaluating ketanserin for treatment of chronic LBP (see Chronic Pain Guideline).

Evidence for the Use of Ketanserin
There are no quality studies incorporated into this analysis.

We search PubMed, EBSCO, Cochrane Review, Google scholar with no limits on publication dates. The search terms used were following chronic low back pain and ketanserin to find 1075 articles. Of 1075 articles, we reviewed none and included none.

LIDOCAINE PATCHES

Topical lidocaine patches have been increasingly used to treat numerous pain conditions ranging from LBP to carpal tunnel syndrome (CTS) to postherpetic neuralgia.(942, 943)

1. *Recommendation: Lidocaine Patches for Chronic Low Back Pain*
   Lidocaine patches are not recommended for treatment of chronic low back pain.

   > *Strength of Evidence* – **Not Recommended, Evidence (C)**
   > *Level of Confidence* – Moderate

2. *Recommendation: Lidocaine Patches for Acute, Subacute, Radicular, or Post-operative Low Back Pain*
   There is no recommendation for or against the use of lidocaine patches for treatment of acute, subacute, radicular, or post-operative low back pain.

   > *Strength of Evidence* – **No Recommendation, Insufficient Evidence (I)**

Rationale for Recommendations
There is one placebo-controlled quality trial for treatment of chronic LBP that failed to show superiority of the lidocaine patch.(944) For other potential indications, there are no quality studies. Lidocaine patches are not invasive and have a low adverse effect profile, although some patients may experience local reactions such as skin irritation, redness, pain, or sores. Lidocaine patches have moderate to high cost over time. Without quality evidence, there is no recommendation for indications. They are not recommended for treatment of chronic LBP.

Evidence for the Use of Lidocaine Patches

There is 1 high-(942) and 1 moderate-quality(944) RCT or crossover trial incorporated into this analysis.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: lidocaine patch, chronic low back pain, and postoperative to find 1,564 articles. Of the 1,564 articles, we reviewed 8 articles and included 8 (2 RCT).

NMDA RECEPTOR ANTAGONISTS (MK-801, Amantadine, Dextromethorphan, Memantine)
Numerous new compounds that specifically target mechanisms mediating neuropathic pain such as the N-methyl-D-aspartate (NMDA) receptor complex are currently used in clinical trials. These compounds include dextromethorphan, amantadine, and memantine.(945) Methadone is a mu agonist that also has affinity for the NMDA receptor. NMDA inhibitors purportedly help to prevent acute pain from progressing to chronic pain. These agents theoretically act by blocking receptors of neurotransmitters that are essential to long-term memories. They are thought to potentially help reduce opioid tolerance and may enhance opioid analgesia. Dextromethorphan is the most studied of these agents,(946) having been used to treat malignant,(947, 948) neuropathic,(949, 950) and chronic pain,(951, 952) and as an adjunct for peri-operative pain relief.(953) The utility of these agents has been limited by their significant adverse-effect profile, which includes lightheadedness, dizziness, tiredness, headache, nervous floating sensation, bad dreams, and sensory changes. Dextromethorphan, amantadine, and memantine are better tolerated with lower CNS adverse effects than ketamine possibly due to a lower affinity for the NMDA receptor which plays a role in both normal physiological functions as well as pathological pain processing.

Recommendation: NMDA Receptor/Antagonists for Chronic Low Back Pain
NMDA receptor/antagonists, including dextromethorphan, are not recommended for treatment of chronic low back pain.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Moderate

Rationale for Recommendations
There are no quality studies evaluating NMDA receptor/antagonists other than dextromethorphan (see Chronic Pain Guideline for these studies).

Evidence for the Use of NMDA Receptor/Antagonists
There are no quality studies incorporated into this analysis.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: NMDA receptor, chronic, low, back, pain, Ketamine, Dextromethorphan, NMDA receptor antagonist, MK-801, Amantadine, and Memantine to find 36,805 articles. Of the 36,805 articles, we reviewed zero articles and included 0 articles

OPIOIDS – Oral, Transdermal, and Parenteral (Includes Tramadol)
Opioids are addressed in a separate guideline. The treatment recommendations are summarized below (see Opioids Guideline for all supporting evidence).

Acute Pain (Up to 4 Weeks)
1. Recommendation: Routine Use of Opioids for Treatment of Non-Severe Acute Pain

Routine opioid use is strongly not recommended for treatment of non-severe acute pain (e.g., low back pain, sprains, or minor injury without signs of tissue damage).

Harms – May inadequately treat acute, severe pain.

Benefits – Faster recovery, less debility, reduced accidents risks, risks of dependency or addiction.

    Strength of Evidence – **Strongly Not Recommended, Evidence (A)**

    Level of Confidence – High

2. *Recommendation: Opioids for Treatment of Acute, Severe Pain*

Opioids are recommended for treatment of acute, severe pain (e.g., crush injuries, large burns, severe fractures, injury with significant tissue damage) uncontrolled by other agents and/or with functional deficits caused by pain. They also may be indicated at the initial visit for a brief course for anticipated pain accompanying severe injuries (i.e., failure of other treatment is not mandatory). A Schedule IV[v] opioid may be indicated if there is true allergy to NSAIDs and acetaminophen, other contraindication to an alternative medication, or insufficient pain relief with an alternative. Recommend to taper off opioid use in 1 to 2 weeks.

Indications **–** Patients should meet all of the following:

1) Severe injury with a clear rationale for use (objective functional limitations due to pain resulting from the medical problem, e.g., extensive trauma such as forearm crush injury, large burns, severe radiculopathy).[vi]

2) Other more efficacious treatments should have been instituted,[vii] and either: a) failed; and/or 2b) have reasonable expectations of the immediate need for an opioid to obtain sleep the evening after the injury.

3) Where available, prescription databases (usually referred to as Prescription Drug Monitoring Program (PDMP)) should be checked and not show evidence for conflicting opioid prescriptions from other providers or evidence of misreporting.[viii]

4) Non-opioid prescriptions (e.g., NSAIDs, acetaminophen) absent contraindication(s) should nearly always be the primary treatment and accompany an opioid prescription.

5) Low-dose opioids may be needed in the elderly who have greater susceptibility to the adverse risks of opioids. Those of lower body weight may also require lower opioid doses.

6) Dispensing quantities should be only what is needed to treat the pain. Short-acting opioids are recommended for treatment of acute pain. Long-acting opioids are not recommended.

7) Due to greater than 10-fold elevated risks of adverse effects and death, considerable caution is warranted among those using other sedating medications and substances including: i) benzodiazepines, ii) anti-histamines (H_1-blockers), and/or iii) illicit substances.(243, 954-956) Patients should not receive opioids if they use illicit substances unless there is objective evidence of significant trauma or moderate to severe injuries. Considerable caution is also warranted among those who are unemployed as the reported risks of death are also greater than 10-

---

[v]USA classifies controlled substances that includes a classification system, ranging from Class 1 to Class V corresponding to lower risks of abuse and dependence. Class I includes substances with a high potential for abuse and without a recognized medical use (e.g., heroin, marijuana, LSD). Class II includes most opiates, amphetamines and cocaine. Class III includes buprenorphine, dihydrocodeiene, hydrocodone/codeiene when compounded with an NSAID, Marinol. Class IV includes tramadol (in some states), carisoprodol, benzodiazepines, and long-activing barbiturates. Class V includes small amounts of codeine (e.g, 30mg, 60mg).

[vi]Other indications beyond the scope of this guideline include acute myocardial infarction or agitation interfering with acute trauma management.

[vii]Treatments to have tried generally include NSAIDs and acetaminophen. For LBP patients, additional considerations include muscle relaxants, progressive aerobic exercise, and directional exercise.

[viii]Exceptions such as acute, severe trauma should be documented.

fold.(243, 955) Due to elevated risk of death and adverse effects, caution is also warranted when considering prescribing an opioid for patients with any of the following characteristics: depression, anxiety, personality disorder, untreated sleep disorders, substance abuse history, current alcohol use or current tobacco use, attention deficit hyperactivity disorder (ADHD), post-traumatic stress disorder (PTSD), suicidal risk, impulse control problems, thought disorders, psychotropic medication use, chronic obstructive pulmonary disease (COPD), asthma, or recurrent pneumonia.(955, 957-978) Considerable caution is also warranted among those with other comorbidities such as chronic hepatitis and/or cirrhosis,(979) as well as coronary artery disease, dysrhythmias, cerebrovascular disease, orthostatic hypotension, asthma, recurrent pneumonia, thermoregulatory problems, advanced age (especially with mentation issues, fall risk, debility), osteopenia, osteoporosis, water retention, renal failure, severe obesity, testosterone deficiency, erectile dysfunction, abdominal pain, gastroparesis, constipation, prostatic hypertrophy, oligomenorrhea, pregnancy, human immunodeficiency virus (HIV), ineffective birth control, herpes, allodynia, dementia, cognitive dysfunction and impairment, gait problems, tremor, concentration problems, insomnia, coordination problems, and slow reaction time. There are considerable drug-drug interactions that have been reported (see Appendices 2-3 of Opioids Guideline).

Frequency/Duration – Generally, opioids should be prescribed at night or while not working.(980) Lowest effective, short-acting opioid doses are preferable as they tend to have the better safety profiles, less risk of escalation,(981) less risk of lost time from work,(982) and faster return to work.(983) Short-acting opioids are recommended for treatment of acute pain and long-acting opioids are not recommended. Recommend opioid use as required by pain, rather than in regularly scheduled dosing.

If parenteral administration is required, ketorolac has demonstrated superior efficacy compared with opioids for acute severe pain,(857, 868) although ketorolac's risk profile may limit use for some patients. Parenteral opioid administration outside of obvious acute trauma or surgical emergency conditions is almost never required, and requests for such treatment are clinically viewed as red flags for potential substance abuse.

Indications for Discontinuation – Resolution of pain, sufficient improvement in pain, intolerance or adverse effects, non-compliance, surreptitious medication use, consumption of medications or substances advised to not take concomitantly (e.g., sedating medications, alcohol, benzodiazepines), or use beyond 2 weeks.
Harms – Adverse effects are many (see section below on "Opioids Benefits and Harms").
Benefits – Improved short-term pain control.

> *Strength of Evidence* – **Recommended, Evidence (C)**
> *Level of Confidence* – High

3. *Recommendation: Screening Patients Prior to Initiation of Opioids*
Initial screening of patients is recommended with more detailed screening for: i) requiring continuation of opioids beyond 2 weeks for those with an acute severe injury, and ii) at consideration of initiation for severe pain but no objective evidence. Screening should include history(ies) of depression, anxiety, personality disorder, other psychiatric disorder, substance abuse, sedating medication use (e.g., anti-histamine/anti-H_1 blocker(955)), benzodiazepine use, opioid dependence, alcohol abuse, current tobacco use, other substance use history, COPD, PTSD, other psychotropic medications, (severe) obesity, cognitive impairment, balance problems/fall risk, osteoporosis, and renal failure (see Appendix 1 of Opioids Guideline). Those who screen positive, especially to multiple criteria, are recommended to: i) undergo greater scrutiny for appropriateness

of opioids (may include psychological evaluation), ii) consideration of consultation and examination(s) for complicating conditions and/or appropriateness of opioids, and iii) if opioids are prescribed, more frequent assessments for compliance, achievement of functional gains,(243, 984, 985) adverse effects, and symptoms and signs of aberrancy.

Harms – Negligible. If a consultation is needed, there are additional costs that are incurred.
Benefits – Improved identification of more appropriate candidates for opioids. Identification of patients at increased risk of adverse effects. In cases where a patient has an elevated, but potentially acceptable risk, the provider may be alerted to improve surveillance for complications and aberrant behaviors.

> *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – High

4. Recommendation: Opioid Dose Limits in Acute Pain
Dispense only that which is required. The maximum daily oral dose recommended for opioid-naïve, acute pain patients based on risk of overdose/death is 50mg morphine equivalent dose (MED) (986).[ix] In rare cases with documented functional improvement (see Appendix 1 of Opioids Guideline), higher doses may be considered, however, risks are substantially higher and greater monitoring is also recommended (see Subacute/Chronic Opioid recommendations below). Lower doses should be used for patients at higher risk of dependency, addiction and other adverse effects. Monitoring is also recommended and consultation may be considered for those patients on higher doses.

Harms – Theoretical potential to undertreat pain in some patients with increased pain sensitivity.
Benefits – Reduced risk for adverse physical and cognitive effects, dependency, addiction and opioid-related overdoses and deaths.

> *Strength of Evidence* – **Recommended, Evidence (C)**
> *Level of Confidence* – Moderate

Post-Operative Pain (Up to 4 Weeks) (After 4 weeks, see Subacute Pain)
Oral opioids are commonly prescribed after sinus surgery,(987) major noncardiac surgical procedures,(988) mastectomy and immediate breast reconstruction (IBR),(989, 990) coronary artery bypass graft surgery,(991) major abdominal surgery (abdominal laparoscopic, abdominal hysterectomy, bowel resection or radical hysterectomy),(992-995) orthopedic surgery,(996) and molar extraction.(997)

1. Recommendation: Limited Use of Opioids for Post-operative Pain
Limited use of opioids is recommended for post-operative pain management as adjunctive therapy to more effective treatments.

Indications – For post-operative pain management, a brief prescription of short-acting opioids as adjunct to more efficacious treatments (especially Cox-2 NSAIDs such as celecoxib, non-selective NSAIDs after risk of bleeding is no longer a concern).[x] A brief course of opioids is often needed for minor surgical procedures. However, minor wound laceration repairs often require no opioids. Evidence suggests perioperative pregabalin for 14 days and/or continuous femoral nerve catheter analgesia instead of solely using oral opioids

---

[ix]Statistical significance present for acute and chronic pain at and above 50mg per day of oral morphine equivalent dose.
[x]More efficaciouos treatments also include therapeutic exercises, e.g., progressive ambulation especially for moderate to extensive procedures (e.g., arthroplasty, fusion).

results in superior knee arthroplasty functional outcomes with less venous thromboses.(998) Additional considerations include:

1) Non-opioid prescriptions (e.g., NSAIDs, acetaminophen) should nearly always be the primary treatment and accompany an opioid prescription. Computerized programs may also assist in optimal management.(999)

2) The lowest effective dose of a short-acting opioid should be used,(981) as well as weaker opioids if possible.(982, 983)

3) Short-acting opioids are recommended for treatment of acute pain.

4) Dispensing should be only what is needed to treat the pain.[xi]

5) Long-acting opioids are not recommended.

6) Low-dose opioids may be needed in the elderly who have greater susceptibility to the adverse risks of opioids. Those of lower body weight may also require lower opioid doses.

7) Where available, prescription databases (usually referred to as Prescription Drug Monitoring Program (PDMP)) should be checked for other opioid prescriptions. Due to greater than 10-fold elevated risks of adverse effects and death, considerable caution is warranted among those using other sedating medications and substances including: i) benzodiazepines, ii) anti-histamines (H_1-blockers), and/or iii) illicit substances.(243, 954-956) Patients should not receive opioids if they use illicit substances unless there is objective evidence of significant trauma or moderate to severe injuries. Considerable caution is also warranted among those who are unemployed as the reported risks of death are also greater than 10-fold.(243, 955)

   Due to elevated risk of death and adverse effects, caution is also warranted when considering prescribing an opioid for patients with any of the following characteristics: depression, anxiety, personality disorder, ADHD, PTSD, suicidal risk, impulse control problems, thought disorders, psychotropic medication use, substance abuse history, current alcohol use or current tobacco use, untreated sleep disorders, COPD, asthma, or recurrent pneumonia.(955, 957-978) Considerable caution is also warranted among those with other comorbidities such as chronic hepatitis and/or cirrhosis,(979) as well as coronary artery disease, dysrhythmias, cerebrovascular disease, orthostatic hypotension, thermoregulatory problems, advanced age (especially with mentation issues, fall risk, debility), osteopenia, osteoporosis, water retention, renal failure, severe obesity, testosterone deficiency, erectile dysfunction, abdominal pain, gastroparesis, constipation, prostatic hypertrophy, oligomenorrhea, pregnancy, HIV, ineffective birth control, herpes, allodynia, dementia, cognitive dysfunction and impairment, gait problems, tremor, concentration problems, insomnia, coordination problems, and slow reaction time. There are considerable drug-drug interactions that have been reported (see Appendices 2-3 of Opioids Guideline).

   Inpatient management may moderate these recommendations provided there is careful monitoring, although these same management issues then apply post-discharge.

8) For patients taking opioids chronically prior to surgery, consultations with anesthesiology and/or pain management are generally needed as post-operative dosing may be very high and management is often quite challenging.

9) Ongoing prescriptions of opioids after the immediate post-operative period should generally be for patients who have undergone a major surgery or have other condition(s) necessitating opioids. Most patients should be making progress towards functional restoration, pain reduction and weaning off the

---

[xi]Generally, this should be sufficient to cover two weeks of treatment. Prescriptions of 90-day supplies in the post-operative setting are not recommended.

opioids. Patients who have not progressed should be carefully evaluated for physical complications or psychiatric comorbidity, adherence to active treatments, and pending development of addiction or dependency.

Frequency/Duration – For moderate and major surgeries, opioids are generally needed on a scheduled basis in the immediate post-operative period. Other post-operative situations may be sufficiently managed with an as needed opioid prescription schedule. Provision of opioids sufficient to participate in therapeutic exercise (e.g., progressive ambulation) and allow sleep may be needed. However, high dose use at night is not recommended due to respiratory depression and disruption of sleep architecture. Weaning should begin as soon as function is recovering and pain is subsiding. Subsequent weaning to as needed opioid use is recommended.

Indications for Discontinuation – Physician should discontinue the use of opioids based on sufficient recovery, expected resolution of pain, lack of efficacy, intolerance or adverse effects, non-compliance, surreptitious medication use, self-escalation of dose, or use beyond 3 to 5 days for minor procedures, and 2 to 3 weeks for moderate or less extensive procedures. Use for up to 3 months may occasionally be necessary during recovery from more extensive surgical procedures (e.g., spine fusion surgery). However, with rare exceptions, only nocturnal use is recommended in months 2 to 3 plus institution of management as discussed in the subacute/chronic guidelines below. For those requiring opioid use beyond 1 month, the subacute/chronic opioid use recommendations below apply.

Harms – Adverse effects are many (see section on "Opioids Benefits and Harms").

Benefits – Improved short-term, post-operative pain control. Some studies suggest this may modestly improve functional outcomes in the post-operative population.

> *Strength of Evidence* – **Recommended, Evidence (C)**
> *Level of Confidence* – High

2. Recommendation: Screening Patients Prior to Continuation of Opioids

Screening of patients is recommended for patients requiring continuation of opioids beyond the second post-operative week. Screening should include history(ies) of: depression, anxiety, personality disorder, pain disorder, other psychiatric disorder, substance abuse history, sedating medication use (e.g., anti-histamine/anti-H$_1$ blocker), benzodiazepine use, opioid dependence, alcohol abuse, current tobacco use, and other substance use history, COPD, PTSD, other psychotropic medications, (severe) obesity, cognitive impairment, balance problems/fall risk, osteoporosis, and renal failure (see Appendix 1 of Opioids Guideline). Those who screen positive, especially to multiple criteria, are recommended to: i) undergo greater scrutiny for appropriateness of opioids (e.g., may include psychological and/or pain evaluation), ii) compliance with active therapies (e.g., ambulation and other exercise after arthroplasty), iii) consider consultation examination(s) for complicating conditions and/or appropriateness of opioids, and iv) if ongoing opioids are prescribed, ensure more frequent assessments for treatment compliance, achievement of functional gains,(243, 984, 985) and symptoms and signs of aberrancy.

Harms – Negligible. If a consultation is needed, there are additional costs that are incurred.

Benefits – Identification of patients at increased risk of adverse effects. Improved identification of more appropriate and safe candidates for opioids compared with attempting post-operative pain control with non-opioids. This should reduce adverse effects. In cases where someone has elevated, but potentially acceptable risk, this may alert the provider to improve surveillance for complications and aberrant behaviors.

> *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – High

3. Recommendation: Opioid Dose Limits in Post-operative Pain

The maximum daily oral dose recommended for opioid-naïve, acute pain patients based on risk of overdose/death is 50mg morphine equivalent dose (MED) (986).[xii] Post-operative patients particularly require individualization due to factors such as the severity of the operative procedure, response to treatment(s) and variability in response. Higher doses beyond 50mg MED may be particularly needed for major surgeries in the first 2 post-operative weeks to achieve sufficient pain relief, however, greater caution and monitoring are warranted and reductions below 50mg MED at the earliest opportunity should be sought. Lower doses should be used for patients at higher risk of dependency, addiction and other adverse effects. In rare cases with documented functional improvement, ongoing use of higher doses may be considered, however, risks are substantially higher and greater monitoring is also recommended (see Subacute/Chronic Opioid recommendations below).

Harms – Theoretical potential to undertreat pain, which could modestly delay functional recovery.
Benefits – Reduced risk for adverse effects, dependency, addiction and opioid-related deaths.

> *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

Subacute (1-3 Months) and Chronic Pain (>3 Months)

1. *Recommendation: Routine Use of Opioids for Subacute and Chronic Non-malignant Pain*
   Opioid use is moderately not recommended for treatment of subacute and chronic non-malignant pain. Opioid prescription should be patient specific and limited to cases in which other treatments are insufficient and criteria for opioid use are met (see below).

   Harms – May inadequately treat severe subacute or chronic pain.
   Benefits – Less debility, fewer adverse effects, reduced accident risks, lower risks of dependency, addiction, overdoses, and deaths.
   > *Strength of Evidence* – **Moderately Not Recommended, Evidence (B)**
   > *Level of Confidence* – High

2. *Recommendation: Opioids for Treatment of Subacute or Chronic Severe Pain*
   The use of an opioid trial is recommended if other evidence-based approaches for functional restorative pain therapy have been used with inadequate improvement in function.(1000, 1001) **Opioids are then recommended for treatment of function impaired by subacute or chronic severe pain (e.g., inability to work due to any of the following: chronic severe radiculopathy, chronic severe peripheral neuropathies, complex regional pain syndrome (CRPS), and severe arthroses)**(984) **(See Appendix 1 of Opioids Guideline).**

   Indications **–** Patients should meet all of the following criteria:
   1) Reduced function is attributable to the pain. Pain or pain scales alone are insufficient reasons.(237, 238, 240-243, 984, 1002-1008)
   2) A severe disorder warranting potential opioid treatment is present [e.g., CRPS, severe radiculopathy, advanced degenerative joint disease (DJD)].(1003)
   3) Other more efficacious treatments have been documented to have failed.(1003) Other approaches that should have been first utilized include physical restorative approaches, behavioral interventions, self-applied modalities, non-opioid medications (including NSAIDs, acetaminophen, topical agents, norepinephrine adrenergic reuptake blocking antidepressants or dual reuptake inhibitors; also antiepileptic medications particularly for neuropathic pain) and functional restoration. For LBP patients,

---

[xii]Statistical significance present for acute and chronic pain at and above 50mg per day of morphine equivalent dose.

this also includes[xiii] fear avoidant belief training and ongoing progressive aerobic exercise, and strengthening exercises. For CRPS patients, this includes progressive strengthening exercise. For DJD, this includes NSAIDs, weight loss, aerobic and strengthening exercises.

4) An ongoing active exercise program is prescribed and complied with.

5) Non-opioid prescriptions (e.g., NSAIDs, acetaminophen) absent a contraindication should nearly always be the primary pain medication and accompany an opioid prescription. Other medications to consider include topical agents, norepinephrine adrenergic reuptake blocking antidepressants or dual reuptake inhibitors; also antiepileptic medications particularly for neuropathic pain).

6) The lowest effective dose should be used.(981) Weaker opioids should be used whenever possible.(982, 983) Meperidine is not recommended for chronic pain due to bioaccumulation and adverse effects.

7) Low-dose opioids may be needed in the elderly who have greater susceptibility to the adverse risks of opioids. Those of lower body weight may also require lower opioid doses.

8) Dispensing should be only what is needed to treat the pain.[xiv]

9) Extended-release/long-acting opioids are recommended to be used on a scheduled basis, rather than as needed.(1003) As needed opioids should generally be avoided for treatment of chronic pain, although limited use for an acute painful event (e.g., fracture, sprain) is reasonable. Sublingual fentanyl is not recommended for treatment of subacute or chronic pain. Caution is warranted with fentanyl patches due to unpredictable absorption.

10) Where available, prescription databases (usually referred to as Prescription Drug Monitoring Program (PDMP)) should be checked for conflicting opioid prescriptions from other providers or evidence of misreporting.

11) Due to greater than 10-fold elevated risks of adverse effects and death, considerable caution is warranted among those using other sedating medications and substances including: i) benzodiazepines, ii) anti-histamines (H_1-blockers), and/or iii) illicit substances.(243, 954-956) Patients should not receive opioids if they use illicit substances unless there is objective evidence of significant trauma or moderate to severe injuries. Considerable caution is also warranted among those who are unemployed as the reported risks of death are also greater than 10-fold.(243, 955)

Due to elevated risk of death and adverse effects, caution is also warranted when considering prescribing an opioid for patients with any of the following characteristics: depression, anxiety, personality disorder, untreated sleep disorders, substance abuse history, current alcohol use or current tobacco use, ADHD, PTSD, suicidal risk, impulse control problems, thought disorders, psychotropic medication use, COPD, asthma, recurrent pneumonia.(955, 957-978) Considerable caution is also warranted among those with other comorbidities such as chronic hepatitis and/or cirrhosis,(979) as well as coronary artery disease, dysrhythmias, cerebrovascular disease, orthostatic hypotension, asthma, recurrent pneumonia, thermoregulatory problems, advanced age (especially with mentation issues, fall risk, debility), osteopenia, osteoporosis, water retention, renal failure, severe obesity, testosterone deficiency, erectile dysfunction, abdominal pain, gastroparesis, constipation, prostatic hypertrophy, oligomenorrhea, pregnancy, HIV, ineffective birth control, herpes, allodynia, dementia, cognitive dysfunction and impairment, gait problems, tremor, concentration problems, insomnia, coordination problems, and slow reaction

---

[xiii]A previous trial of a muscle relaxant is generally recommended. However, if an opioid trial is contemplated, cessation of all depressant medications including muscle relaxants is advisable.

[xiv]Generally, this should be sufficient to cover one week of treatment at a time during the trial phase. If a trial is successful at improving function, prescriptions for up to 90-day supplies are recommended.

time. There are considerable drug-drug interactions that have been reported (see Appendices 2-3 of the Opioids Guideline).

Frequency/Duration – Opioids use is generally initiated as a "trial" to ascertain whether the selected opioid produces functional improvement (see Appendix 1 of Opioids Guideline). Opioid use is generally prescribed on a regular basis,(1009) at night or when not at work.(980) Only one opioid is recommended to be prescribed in a trial. More than one opioid should rarely be used. Lower opioid doses are preferable as they tend to have the better safety profiles, less risk of dose escalation,(981) less work loss,(982) and faster return to work.(983) Patients should have ongoing visits to monitor efficacy, adverse effects, compliance and surreptitious medication use. Opioid prescriptions should be shorter rather than longer duration.(1010)

Indications for Discontinuation – Opioids should be discontinued based on lack of functional benefit(1001) (see Appendix 1), resolution of pain, improvement to the point of not requiring opioids, intolerance or adverse effects, non-compliance, surreptitious medication use, medication misuse (including self-escalation and sharing medication), aberrant drug screening results, diversion, consumption of medications or substances advised to not take concomitantly (e.g., sedating medications, alcohol, benzodiazepines).

Harms – Adverse effects are many (see section on "Opioids Benefits and Harms"). May initiate path to opioid dependency.

Benefits – Improved short-term pain ratings. Theoretical potential to improve short-term function impaired by a painful condition.

> *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

3. Recommendation: Screening Patients Prior to Initiation of Opioids

Screening of patients is recommended prior to consideration of initiating a trial of opioids for treatment of subacute or chronic pain. Screening should include history(ies) of depression, anxiety, personality disorder and personality profile,(983, 1011, 1012) other psychiatric disorder, substance abuse history, sedating medication use (e.g., anti-histamine/anti-H$_1$ blocker),(975) benzodiazepine use, opioid dependence, alcohol abuse, current tobacco use, and other substance use history, COPD, PTSD, other psychotropic medications, (severe) obesity, cognitive impairment, balance problems/fall risk, osteoporosis, and renal failure (see Appendix 1 of Opioids Guideline). Those who screen positive, especially to multiple criteria, are recommended to: i) undergo greater scrutiny for appropriateness of opioids (may include psychological and/or psychiatric evaluation(s) to help assure opioids are not being used instead of appropriate mental health care); ii) consideration of consultation and examination(s) for complicating conditions and/or appropriateness of opioids; and iii) if opioids are prescribed, more frequent assessments for compliance, achievement of functional gains and symptoms and signs of aberrant use.

Harms – Negligible. If a consultation is needed, there are additional costs that are incurred.

Benefits – Identification of patients at increased risk of adverse effects. Improved identification of more appropriate and safe candidates for treatment with opioids. This should reduce adverse effects. In cases where someone has elevated, but potentially acceptable risk, this may alert the provider to improve surveillance for complications and aberrant behaviors.

> *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – High

4. Recommendation: Opioid Dose Limits in Subacute and Chronic Pain

The maximum daily oral dose recommended for subacute or chronic pain patients based on risk of overdose/death is 50 mg Morphine Equivalent Dose (MED).(961, 986) In rare cases with documented

functional improvements occurring with use above 50 mg MED, subsequent doses up to 100 mg may be considered, however, risks of death are much greater and more intensive monitoring is then also recommended. Lower doses should be considered in high risk patients. Caution appears warranted in all patients as there is evidence the risk of dose escalation is present even among patients enrolled in a "hold the line (Stable Dose) prescribing strategy" treatment arm.(1013)

For those whose daily consumption is more than 50 mg MED, greater monitoring is recommended to include: 1) at least monthly to not more than quarterly appointments with greater frequencies during trial, dose adjustments and with greater co-morbid risk factors and conditions; 2) at least semiannual attempts to wean below 50mg MED if not off the opioid; 3) at least semiannual documentation of persistence of functional benefit; 4) at least quarterly urine drug screening (see drug screening section); and 5) at least semiannual review of medications, particularly to assure no sedating medication use (e.g., benzodiazepine, sedating anti-histamines).

Harms – None in a short-term trial. For chronic pain patients, theoretical potential to undertreat pain and thus impair function. However, there is no quality literature currently available to support that position.

Benefits – Reduced risk for adverse effects, dependency, addiction, and opioid-related deaths.

> *Strength of Evidence* – **Recommended, Evidence (C)**
> *Level of Confidence* – High

5. Recommendation: Use of an Opioid Treatment Agreement (Opioid Contract, Doctor/Patient Agreement, Informed Consent)

The use of an opioid treatment agreement (opioid contract, doctor/patient agreement, or informed consent) is recommended to document patient understanding, acknowledgement of potential adverse effects, and agreement with the expectations of opioid use (see Appendix 1 of Opioids Guideline).(1000, 1014-1025) **If consent obtained, it is recommended appropriate family members be involved in this agreement.**

Harms – Negligible.

Benefits – Educates the patient and significant others that these medications are high risk, with numerous adverse effects. It allows for a more informed choice. It provides a framework for initiation of a trial, monitoring, treatment goals, compliance requirement, treatment expectations, and conditions for opioid cessation. It should reduce risk of adverse events and opioid-related deaths, although that remains unproven to date.

> *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Moderate

6. Recommendation: Urine Drug Screening

Baseline and random urine drug screening, qualitative and quantitative, is recommended for patients prescribed opioids for the treatment of subacute or chronic pain to evaluate presence or absence of the drug, its metabolites, and other substance(s) use. In certain situations, other screenings (e.g., hair particularly for information regarding remote use(1026-1031) **or blood (for acute toxicity) may be appropriate.**

Indications – All patients on opioids for subacute or chronic pain.

Frequency – Screening is recommended at baseline, randomly at least twice and up to 4 times a year and at termination. More intensive screening is recommended for those consuming more than 50mg MED

(see above). Federal guidelines recommend at least 8 tests a year among those utilizing opioid treatment programs.(1032) Screening should also be performed "for cause" (e.g., provider suspicion of substance misuse including over-sedating, drug intoxication, motor vehicle crash, other accidents and injuries, driving while intoxicated, premature prescription renewals, self-directed dose changes, lost or stolen prescriptions, using more than one provider for prescriptions, non-pain use of medication, using alcohol for pain treatment or excessive alcohol use, missed appointments, hoarding of medications, and selling medications). Standard urine drug/toxicology screening processes should be followed (consult a qualified medical review officer).(1032-1035) If there is an aberrant drug screen result (either positive for unexpected drugs or unexpected metabolites or unexpectedly negative results), there should be a careful evaluation of whether there is a plausible explanation (e.g., drug not tested, drug metabolite not tested, laboratory cutpoint and dosing interval would not capture the drug/metabolite, laboratory error). In the absence of a plausible explanation, those patients with aberrant test results should have the opioid discontinued or weaned.(1001)

Harms – No adverse clinical effects if properly interpreted.

Benefits – Identifies aberrant medication(s) and substance(s) use. Such uses are high-risk for opioid events including fatalities (see tables below). It provides objective evidence to cease an opioid trial or ongoing treatment. Identifies patients who may be diverting medication (those screening negative for prescribed medication).

> *Strength of Evidence* – **Recommended, Evidence (C)**
> *Level of Confidence* – High

Evidence for the Use of Opioids
See Opioids Guideline.

SKELETAL MUSCLE RELAXANTS
Skeletal muscle relaxants comprise a diverse set of pharmaceuticals designed to produce "muscle relaxation" through different mechanisms of action – generally considered to be effects on the central nervous system (CNS) and not directly on skeletal muscle.(1036, 1037) These medications are widely used to treat painful conditions, most prominently LBP.(646, 1038-1043)

1. *Recommendation: Muscle Relaxants for Mild to Moderate Acute, Subacute, or Chronic Low Back Pain*
   Muscle relaxants are not recommended for mild to moderate acute low back pain due to problems with adverse effects, or for chronic use in subacute or chronic low back pain (other than acute exacerbations).

   > *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
   > *Level of Confidence* – Moderate

2. *Recommendation: Muscle Relaxants for Moderate to Severe Acute Low Back Pain*
   Muscle relaxants (not including carisoprodol) are moderately recommended as a second-line treatment in moderate to severe acute low back pain that has not been adequately controlled by NSAIDs.

   Indications – Recommended for select cases of moderate to severe acute LBP. For most cases, these agents are not recommended as NSAIDs, progressive walking, and other exercises will be sufficient to control the symptoms. Generally, it is recommended that these agents be prescribed nocturnally initially and not during workdays or when patients plan to operate motor vehicles. Diazapam should generally be avoided. Caution should be used in prescribing skeletal muscle relaxants for those with

a history of depression, personality disorder, and/or substance addiction/abuse, including alcohol or tobacco. If a muscle relaxant is felt to be necessary in patients with those problems, cyclobenzaprine has a chemical structure resembling a tricyclic anti-depressant, and so addiction and abuse of this drug typically do not occur but may occur with other muscle relaxants.

Frequency/Dose – The initial dose should generally be in the evening, and not prior to starting a work shift, operating a motor vehicle, machinery or performing safety-sensitive work. Daytime use is acceptable in circumstances where there are minimal CNS-sedating effects and little concern about sedation compromising function or safety. There is no evidence of benefit from higher doses (e.g., cyclobenzaprine 10mg over 5mg).(1044) If significant daytime somnolence results, the medication may need to be discontinued, particularly if it interferes with performance of the aerobic exercise and other components of the rehabilitation plan. Another option is to decrease a dose of cyclobenzaprine by 50% to as little as 2.5mg.(1044)

Indications for Discontinuation – Resolution of pain, non-tolerance, significant sedating effects that carry over into the daytime, or other adverse effects.
Benefits – Modest reduction in acute LBP compared with placebo.
Harms – Sedation, daytime fatigue. Modest potential for abuse. Risk for safety including motor vehicle crash and other injuries.

> *Strength of Evidence* – **Moderately Recommended, Evidence (B)**
> *Level of Confidence* – Moderate

3. *Recommendation: Carisoprodol for Moderate to Severe Low Back Pain*
   Carisoprodol is not recommendended for moderate to severe acute low back pain that has not been adequately controlled by NSAIDs or for acute exacerbations of chronic pain, or acute post-surgical situations.
   > *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
   > *Level of Confidence* – Moderate

4. *Recommendation: Muscle Relaxants for Acute Radicular Pain, Acute Exacerbations of Chronic Pain, or Post-surgical Use*
   Muscle relaxants are recommended as second- or third-line agents for selective use to treat acute exacerbations of chronic pain, or acute post-surgical situations. However, other agents may be more efficacious for relieving radicular pain, e.g., NSAIDs.

   Indications – Moderate to severe acute worsening of pain and/or functional loss associated with worsening of LBP, radicular pain syndromes or post-surgical pain thought to be musculoskeletal in nature. Generally, muscle relaxants should be prescribed nocturnally initially and not during workdays or when patients plan on operating motor vehicles.

   Frequency/Dose – The initial dose should be in the evening. Daytime use is acceptable in circumstances where there are minimal CNS-sedating effects. If significant daytime somnolence results, then the medication may need to be discontinued, particularly if it interferes with the patient's performance of aerobic exercise or other components of the rehabilitation plan.

   Indications for Discontinuation – Resolution of pain, non-tolerance, significant sedating effects that carry over into the daytime, or other adverse effects.
   Benefits – Modest reduction in acute low back pain compared with placebo.

Harms – Sedation, daytime fatigue. Modest potential for abuse. Risk for safety including motor vehicle crash and other injuries.

> *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

5. *Recommendation: Muscle Relaxants for Chronic Low Back Pain*
   Muscle relaxants are not recommended for ongoing use for treatment of chronic low back pain, particularly without documented functional benefit.

   > *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
   > *Level of Confidence* – Low

Rationale for Recommendations
Skeletal muscle relaxants have been evaluated in quality studies although the outcomes comparing these agents to placebo may be overstated due to the unblinding that would be inherent in taking a drug with substantial CNS-sedating effects.(1038) Nevertheless, there is quality evidence that skeletal muscle relaxants modestly improve acute LBP, particularly for the first several days.(829, 1044-1048) The mechanism of action is unclear. However, the adverse-effect profile is concerning,(1049) and there are many adverse effects from these agents. Most concerning is the significant potential for CNS sedation which has typically ranged between 25 to 50%. There are some studies indicating that more than 50% of patients are affected by CNS sedation. Thus, prescriptions for skeletal muscle relaxants for daytime use should be carefully weighed against the need to drive vehicles, operate machinery, perform at heights, direct others, perform safety-sensitive work, or otherwise engage in occupations where mistakes in judgment may have serious consequences. Skeletal muscle relaxants also have a modest but significant potential for abuse(1043, 1050, 1051) and caution should be used when prescribing them for patients with a history of any substance abuse or dependence.(796, 1052) Some caution should be exerted with all of these agents when a patient has a history of substance abuse or requests specific medications.

Carisoprodol is more commonly abused because one of its active metabolites is meprobamate. There also is no evidence it is superior to any other muscle relaxant. Thus, it is not recommended as a first, second or third choice muscle relaxant. Use of this agent is recommended to be only under highly selective circumstances that would include having tried the other available muscle relaxants, as well as more effective and usual treatments such as progressive active exercise and NSAIDs.

There is little evidence of muscle relaxant efficacy for treatment of chronic LBP as the few available studies appear to have mostly evaluated acute exacerbations of chronic pain.(1046, 1053, 1054) Skeletal muscle relaxants have demonstrated efficacy in acute LBP, have significant adverse effects, and are low cost, especially if generic medications are prescribed. Thus, skeletal muscle relaxants are recommended for select management of moderate to severe acute LBP. They are not recommended for continuous management of subacute or chronic LBP although they may be recommended for brief management of acute exacerbations in the setting of chronic LBP.(1053-1055)

Diazepam appears inferior to skeletal muscle relaxants,(1056) has a higher incidence rate of adverse effects, and is addictive. Diazepam is not recommended for use as a skeletal muscle relaxant. Evidence suggests that carisoprodol is comparable to cyclobenzaprine in efficacy. However, cyclobenzaprine may have advantages of lower abuse potential and some chemical analogy to tricyclic anti-depressants.

Chlorzoxazone has been associated with hepatocellular toxicity. Chlormezanone has been implicated in Stevens-Johnson syndrome and toxic epidermal necrolysis.

Evidence for the Use of Skeletal Muscle Relaxants
There are 3 high-(1045, 1054, 1057) and 33 moderate-quality(829, 830, 835, 854, 873, 1046-1048, 1053, 1055, 1056, 1058-1079) RCTs or crossover trials incorporated into this analysis. There are 5 low-quality RCTs in Appendix 1.(831, 1080-1083)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: muscle relaxants, low back pain, and chronic low back pain radicular pain syndrome, carisoprodol cyclobenzaprine, diazepam, metaxalone methocarbamol, baclofen, chlorzoxazone, dantrolene, orphenadrine, tizanadine, clinical trial or randomized controlled trail or random, systematic reviews or reviews, population study or epidemiological study or prospective cohort to find 7,086 articles. Of those we reviewed 54 articles and included 34 articles (32 RCTs and 2 reviews).

SYSTEMIC GLUCOCORTICOSTEROIDS (AKA "Steroids")

Glucocorticosteroids are used to treat symptomatic herniated discs both through local injections (e.g., epidural glucocorticosteroid injections) and oral agents to attempt to reduce localized inflammation and swelling.(13, 1084-1110)

1. Recommendation: Systemic Glucocorticosteroids for Acute or Subacute Radicular Pain Syndromes
Systemic glucocorticosteroids are recommended for treatment of acute and subacute radicular pain syndromes. (56% panel agreement. 44% felt oral steroids should be Not Recommended.)
>   *Strength of Evidence* – **Recommended, Evidence (C)**
>   *Level of Confidence* – Moderate

Indications – Moderate to severe acute and subacute radicular pain syndromes where the goal is to improve function with the understanding there are no demonstrable impacts on the necessity for surgery. One study suggested that the patient should have an ODI >30.(1111) Recommend as part of an overall active care strategy that includes pregressive increases in activity designed to promote early activity, self-care, and self-efficacy.
Frequency/Dose – One 15-day course of oral prednisone (5 days at 60mg, then 5 days at 40mg, then 5 days at 20mg).(1111)
Indications for Discontinuation – Intolerable adverse effects, e.g., agitation, non-tolerance or other adverse effects.
Benefits – Modestly improved function compared with placebo.(1111)
Harms – Short term worsening of glucose control in diabetics is likely. Anxiety and insomnia are frequent. May exacerbate hypertension. Longer term and higher dose use has been particularly associated with adverse effects such as osteonecrosis, glaucoma, mood swings, infection, osteoporosis, and weight gain.

2. Recommendation: Systemic Glucocorticosteroids for Chronic Radicular Pain Syndromes
There is no recommendation for or against systemic glucocorticosteroids for treatment of chronic radicular pain syndromes.
>   *Strength of Evidence* – **No Recommendation, Insufficient Evidence (I)**

Level of Confidence – Moderate

3. Recommendation: Systemic Glucocorticosteroids for Acute, Subacute, or Chronic Low Back Pain
Systemic glucocorticosteroids are not recommended for treatment of acute, subacute, or chronic low back pain.

Strength of Evidence – **Moderately Not Recommended, Evidence (B)** – Acute LBP
Not Recommended, Insufficient Evidence (I) – Subacute or chronic LBP
Level of Confidence – High

Rationale for Recommendations
Glucocorticosteroids to treat radicular pain syndromes and LBP have been assessed in quality studies.(1111-1114) The single blinded trial for treatment of radicular pain that included long-term follow-up suggested long-lasting benefits compared with placebo suggesting apparent efficacy.(1111) Other trials had followed subjects inadequately or used less steroid, although still suggesting benfit. However, trials uniformly have shown no benefit for treatment of LBP. One moderate-quality trial found comparable (in)efficacy for treatment of LBP with intramuscular compared with intraarticular steroids (2408).

Systemic glucocorticosteroids are either minimally invasive or not invasive depending on the chosen administration route, have adverse effects, but are low cost. Glucocorticosteroids are not recommended for management of LBP, but are recommended for acute and subacute radicular pain syndromes where their efficacy has been documented.

Evidence for the Use of Systemic Glucocorticosteroids (aka "Steroids")
There are 3 high-(1111, 1112, 1115) and 3 moderate-quality(1113, 1114, 1116) RCTs incorporated into this analysis.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: acute low back pain, subacute low back pain, chronic low back pain, radicular pain syndrome, sciatica, spinal stenosis, Epidural Glucocorticosteroid Injection, Dexamethasone, Glucocorticosteroid injection, Methylprednisolone, Triamcinolone, Steroid injection, Corticosteroid injection, betamethasone, Peridural Injection, Extradural Injection, Epidural Injection, clinical trial, randomized controlled trial, random, systematic review, review, population study, epidemiological study, and prospective cohort as well as reviewed references to find 44,715 articles (24 articles from reference lists). Of the 44.691 articles, we reviewed 190 articles and included 105 articles (all RCTs).

THALIDOMIDE
Thalidomide is a sedative-hypnotic and multiple myeloma medication. Case reports have found it efficacious in treating CRPS (1117-1119); thus, thalidomide is under investigation as an agent with possible wider benefit for this condition. However, severe birth defects (phocomelia) have resulted when the drug has been taken during pregnancy.

Recommendation: Thalidomide for Chronic Low Back Pain
Thalidomide is not recommended for treatment of chronic low back pain.

Strength of Evidence – **Not Recommended, Insufficient Evidence (I)**
Level of Confidence – High

Rationale for Recommendation
There are no quality studies evaluating thalidomide for treatment of chronic pain syndromes. This medication has severe adverse effects and should never be used by patients who are pregnant or have the potential to become pregnant. Peripheral neuropathy (apparently dose dependent)(1120) is another potentially severe adverse effect and occurs in as many as 80% of patients. Risk of thrombosis has also been reported. Therefore, thalidomide cannot be recommended for the treatment of LBP.

Evidence for the Use of Thalidomide
There are no quality studies incorporated into this analysis.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following terms: thalidomide, and chronic low back pain to find 13,020 articles. Of the 13,020 articles we reviewed zero articles.

TUMOR NECROSIS FACTOR-ALPHA INHIBITORS

Tumor necrosis factor alpha is thought to have a role in resorption of herniated intervertebral discs and also in producing the pain associated with herniated discs. Adalimumab and infliximab are monoclonal antibodies against tumor necrosis factor alpha. Etanercept is a tumor necrosis factor receptor inhibitor. They have been used for a number of rheumatological conditions, as well as in uncontrolled trials of sciatica.(1121-1123)

1. *Recommendation: Tumor Necrosis Factor Alpha for Radicular Pain*
   Tumor necrosis factor-α inhibitors are moderately not recommended for treatment of radicular pain syndromes.
   > *Strength of Evidence* **–** Moderately Not Recommended, Evidence (B)
   > *Level of Confidence* – Moderate

2. *Recommendation: Tumor Necrosis Factor Alpha for Acute, Subacute, or Chronic Low Back Pain*
   Tumor necrosis factor-α inhibitors are not recommended for treatment of acute, subacute, or chronic low back pain.
   > *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
   > *Level of Confidence* – Moderate

Rationale for Recommendations
Most RCT data including over 1 year of follow-up failed to find beneficial effects of infliximab for lumbar radicular pain syndromes (1124-1126), although one study reported benefits (2409). Thus, there is no consistent quality evidence that tumor necrosis factor-α inhibitors have beneficial effects on the treatment of radicular pain syndromes. These agents are invasive and have significant adverse effects, including leucopenia, thrombocytopenia, pancytopenia, predisposition to serious infection, and a lupus-like autoantibody syndrome. Since potential adverse effects can be severe, proof of efficacy is essential before these inhibitors could be recommended. They are costly and also have not been assessed in acute, subacute, or chronic LBP syndromes.

Evidence for the Use of Tumor Necrosis Factor Alpha Inhibitors

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates and an updated search was conducted using PubMed for publications between 1/1/2013 and 11/15/2017. We used the following search terms: tumor necrosis factors, tumor, necrosis, factor-α, inhibitors, radicular, syndromes, sciatica, subacute, low, back, pain, chronic, and random* to find 22,806 articles. Of the 22,806 articles we considered for inclusion 61. Of the 61 articles considered for inclusion, 4 are randomized controlled trials and 57 systematic reviews.

COMPLEMENTARY OR ALTERNATIVE METHODS OR DIETARY SUPPLEMENTS, ETC.
Some interventions for LBP are classified as dietary supplements or as complementary or alternative treatments. A few of these interventions include homeopathic treatments, naturopathic treatments, vitamins, herbal remedies, spiritual healing, touch for healing, craniosacral therapy, aromatherapy, energy healing, and neural therapy.(1127-1136) Tuina-focused integrative Chinese medical therapies emphasize anatomy and physiology when used for the treatment of LBP.(1137) Most of these interventions (certain exceptions discussed below) do not have quality evidence of efficacy for low back pain. As there are many interventions shown to be efficacious for the treatment of acute, subacute, chronic, radicular and post-operative LBP, it is strongly recommended that patients be treated with therapies proven to be efficacious for these conditions, whether or not the intervention is considered complementary, alternative, or a dietary supplement, etc.

Recommendation: Complementary or Alternative Treatments or Dietary Supplements, etc., for Acute, Subacute, or Chronic Low Back Pain
Complementary or alternative treatments or dietary supplements, etc. (other than those specifically described below) are not recommended for treatment of acute, subacute, or chronic low back pain.

> *Strength of Evidence –* **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence –* Low

Rationale for Recommendation
Except where described elsewhere, quality studies regarding complementary or alternative interventions or dietary supplements have not been identified or do not exist. Available trials frequently have significant methodological weaknesses. These interventions are not proven efficacious for the treatment of acute, subacute, or chronic LBP or for radicular pain syndromes or other back-related problems. There are other interventions shown to be efficacious.

Evidence for the Use of Complementary or Alternative Treatments or Dietary Supplements
There are 7 moderate-quality RCTs incorporated into this analysis.(1138-1144) There is 1 low-quality RCT in Appendix 1.(1145)

We searched PubMed, EBSCO, Cochrane Review, and Google scholar without limits on publication dates. We used the following search terms: Complementary alternative medicine, homeopathic treatments, naturopathic treatments, spiritual healing, touch for healing, craniosacral therapy, aromatherapy, energy healing, and neural therapy, subacute low back pain, chronic low back pain, low back pain, clinical trial, randomized controlled trial, random, systematic review, population study, epidemiological study, and prospective cohort to find 4,436 articles. Of the 4,436 articles, we reviewed 13 articles and included 9 articles.

MEDICAL FOODS

Theramine, an amino acid formulation (AAF), has been used as a prescription medical food to theoretically reduce pain and inflammatory processes through dietary management.(1146) Theramine purportedly may increase the production of serotonin, nitric oxide, histamine, and gamma-aminobutyric acid by providing precursors to these neurotransmitters.(1146)

Recommendation: Medical Foods for Acute, Subacute, Chronic, Radicular and Post-operative Low Back Pain
There is no recommendation for or against use of medical foods, including theramine, for treatment of acute, subacute, chronic, radicular and post-operative low back pain.

> *Strength of Evidence –* **No Recommendation, Insufficient Evidence (I)**

Rationale for Recommendation
There are no placebo-controlled trials identified. There is one moderate-quality trial comparing theramine with low dose naproxen.(1146) This may have biases similar to a non-treatment or wait-listed control group. Theramine is not invasive, has low adverse effects but cost quickly becomes high. In the absence of trials demonstrating efficacy, there is no recommendation for or against theramine.

Evidence for the Use of Medical Foods
There is 1 moderate-quality RCT incorporated into this analysis.(1146)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar with no limits on publication dates. We used the following terms: medical food theramine, theramine, subacute low back pain, chronic low back pain and low back pain. This search found 8 articles and we included 1 article.

HERBAL AND OTHER PREPARATIONS
Herbal treatments have been utilized to treat LBP, including Camphora molmol, Salix alba, Melaleuca alternifolia, Angelica sinensis, Aloe vera, Thymus officinalis, Menthe piperita, Arnica montana, Curcuma longa, Tanacetum parthenium, Harpagophytum procumbens, and Zingiber officinale. Evidence of efficacy varies across these compounds. (Creams and ointments, including capsicum, are reviewed separately.)

1. *Recommendation: Herbal Treatments for Acute, Subacute, or Chronic Low Back Pain*
   There is no recommendation for or against the use of Harpagoside, Camphora molmol, Melaleuca alternifolia, Angelica sinensis, Aloe vera, Thymus officinalis, Menthe piperita, Arnica montana, Curcuma longa, Tanacetum parthenium, or Zingiber officinale,(1147) for treatment of acute, subacute, or chronic low back pain.

   > *Strength of Evidence* **– No Recommendation, Insufficient Evidence (I)**

2. *Recommendation: Willow Bark for Acute, Subacute, or Chronic Low Back Pain*
   Willow bark (salix) is not recommended for treatment of acute, subacute, or chronic low back pain.

   > *Strength of Evidence –* **Not Recommended, Insufficient Evidence (I)**
   > *Level of Confidence –* Low

Rationale for Recommendations
Treatments are diverse with limited comparability between treatment regimens. Herbal treatments/supplements for any condition are not well regulated in the U.S. and research regarding

therapeutic and biologically available dosage is limited or non-existent. There is a potential for a placebo effect to be misinterpreted as a sign of efficacy.

There is evidence suggesting that harpagoside is effective in the treatment of LBP.(1148, 1149) There is one trial comparing harpagoside with a low dose (12.5mg) of Vioxx (see below).(1149) As this was a low dose of Vioxx, it may be reasonable to infer that harpagoside is somewhat less efficacious than NSAIDs. Safety of this agent also needs to be addressed in larger trials over longer durations. However, in patients who do not tolerate a NSAID or who have contraindications, this may be a reasonable medication for treating chronic LBP. Providers should be cautioned that there are no quality long-term safety data. However, there is little, if any, control over the quality and dosing of these compounds in contrast with pharmaceuticals and thus, there is no recommendation.

There is evidence that salicin is effective in the treatment of LBP,(1150, 1151) as this is the plant from which salicylates were derived. There also is evidence that Salix (willow bark) inhibits platelet aggregation, though less strongly than aspirin or other salicylates.(1152) While willow bark appears mildly effective in short-term trials, when compared to a low dose of rofecoxib there is no difference, but this also suggests that willow bark is inferior to NSAIDs for the treatment of LBP. A rationale basis for using this agent is not apparent when, as it is directly related to salicylates, it may contain other compounds with potential adverse effects and is more expensive than most generic NSAIDs. **If salicylates are used as treatment, generic aspirin is preferable to Willow bark or salicin.**

Harpagoside and salicin are taken orally. Neither have long-term demonstrated efficacy and safety. Adverse effects appear low. They are not costly. Both appear likely to be substantially inferior to prescription dose NSAIDs.

There is no quality evidence to support the use of most of these agents including Camphora molmol, Melaleuca alternifolia, Angelica sinensis, Aloe vera, Thymus officinalis, Menthe piperita, Arnica montana, Curcuma longa, Tanacetum parthenium, and Zingiber officinale,(1147) for LBP or post-operative patients.

Evidence for the Use of Herbal Treatments
There are 2 high-(1148, 1149) and 4 moderate-quality(1150, 1151, 1153, 1154) RCTs incorporated into this analysis.

We searched PubMed, EBSCO, Cochrane Review, and Google scholar without limits on publication dates. We used the following search terms: herbal preparations, herbal remedies, herbal medicine, herbalism, Harpagoside, Camphora molmol, Melaleuca Alternifolia, Angelica Sinensis, Aloe Vera, Thymus Officinalis, Menthe Peperita, Arnica Montana, Curcuma Longa, Tancaetum Parthenium, Zingiber Officinale, Harpagophytum, Willow Bark Extract, chronic low back pain, low back pain, clinical trial, randomized controlled trial, random, systematic review, population study, epidemiological study, and prospective cohort to find 5,197 articles. Of the 5,197 articles, we reviewed 10 articles and included 8 articles (6 original articles, 2 reviews).

CAPSAICIN, "SPORTS CREAMS," AND OTHER CREAMS; OINTMENTS AND TOPICAL AGENTS
Capsaicin is applied to the skin as a cream or ointment and is thought to reduce pain by stimulating other nerve endings, thus it is thought to be potentially effective through distraction. Rado-Salil ointment is a proprietary formulation of 14 agents, the two most common of which are menthol (55.1%)

and methylsalicylate (26.5%). There are many other commercial products that similarly cause either a warm or cool feeling in the skin. All of these agents are thought to work through a counter-irritant mechanism (i.e., feeling the dermal sensation rather than the LBP). There is evidence that capsaicin compounds should not be used chronically due to reported adverse effects on neurons.(1155) Other topical medications include dimethyl sulfoxide (DMSO), and N-Acetylcysteine (NAC) in addition to those previously reviewed. DMSO, a free radical scavenger, has been used for years. CRPS is one of the few indications for its use (see Chronic Pain Guideline).

1. *Recommendation:* Capsaicin for Acute or Subacute Low Back Pain or Temporary Flares of Chronic Low Back Pain
   Capsaicin (capsicum) is moderately recommended for treatment of acute or subacute low back pain or temporary flare-ups of chronic low back pain. Long-term use is not recommended. Capsaicin appears superior to Spiroflor. Other creams and ointments may be useful, although there is no quality evidence to guide recommendations.

   Indications – For acute, subacute, or temporary flare-ups of chronic LBP. However, other treatments appear to likely have greater efficacy (e.g., NSAIDs, progressive exercise program, etc.). Yet, capsaicin may be a useful adjunct. These compounds may also be used in those patients who prefer topical treatments over oral treatments and other more efficacious treatments, but have only mild LBP.

   Indications for Discontinuation – Resolution of LBP, lack of efficacy, or development of adverse effects that necessitate discontinuation. Recommended not to be used more than 1 month due to concerns about adverse effects, aggregate costs, and acknowledgement that the patient should be transitioning to an active treatment program.
   Benefits –Modest reductions in pain through distraction.
   Harms – Local irritation and theoretical neuronal death with longer term use.(1156)
   > *Strength of Evidence* – **Moderately Recommended, Evidence (B)**
   > *Level of Confidence* – Moderate

2. *Recommendation: Spiroflor for Acute, Subacute, or Chronic Low Back Pain*
   Spiroflor is not recommended for treatment of acute, subacute, or chronic low back pain as it appears less efficacious then capsaicin and there are other treatments that are efficacious.
   > *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
   > *Level of Confidence* – Low

3. *Recommendation: Topical NSAIDs or Other Creams and Ointments for Acute, Subacute, or Chronic Low Back Pain*
   There is no recommendation for or against the use of topical NSAIDs or other creams and ointments for treatment of acute, subacute, or chronic low back pain.
   > *Strength of Evidence* – **No Recommendation, Insufficient Evidence (I)**
   > *Level of Confidence* – Low

4. *Recommendation: DMSO for Chronic Low Back Pain*
   DMSO is not recommended for treatment of chronic low back pain.
   > *Strength of Evidence* –**Not Recommended, Insufficient Evidence (I)**
   > *Level of Confidence* – Low

5. *Recommendation: N-Acetylcysteine for Chronic Low Back Pain*

N-Acetylcysteine is not recommended for treatment of chronic low back pain.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

6. *Recommendation: EMLA Cream for Chronic Low Back Pain*
EMLA cream is not recommended for treatment of chronic low back pain.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

7. *Recommendation: Wheatgrass Cream for Chronic Low Back Pain*
Wheatgrass cream is not recommended for treatment of chronic low back pain.
> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

Rationale for Recommendations
Capsicum compounds have evidence of efficacy in quality studies, although they do not appear particularly potent. There is evidence that capsicum is superior to Spiroflor. There are many other commercially available creams and ointments, but no quality studies for the purposes of treating LBP. These agents are topical, thus not invasive, and have low adverse effects. Over an extended period of time they are not inexpensive, but they are not expensive for short-term use. There are no studies of long-term chronic use, so there is no information about long-term efficacy or dermal or other toxicity. Capsaicin is moderately recommended for treatment of LBP. It may be reasonable to combine capsicum with NSAIDs for additional reductions in LBP through different mechanisms, although that has not been tested in a trial. For other topical agents, see the Chronic Pain Guideline.

Evidence for the Use of Capsaicin, "Sports Creams," or Other Creams and Ointments
There are 2 high-(1157, 1158) and 3 moderate-quality(1151, 1159, 1160) RCTs incorporated into this analysis.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following terms: topical NSAIDs, creams, ointments, NAC, DMSO, ELMA, cream, wheatgrass cream, capsaicin, capsicum, subacute, low back pain, and chronic low back pain to find 22,850 articles. Of the 22,850 articles we reviewed 5 articles and all were included.

VITAMINS
Vitamins have been used to treat essentially all disorders. There has been particular interest in anti-oxidants. However, all anti-oxidants are simultaneously pro-oxidants,(1161, 1162) thus evidence of potential harm from vitamins, particularly vitamin E, is accumulating.(1163-1165) There is poor evidence that vitamins or minerals have beneficial therapeutic effects in normal or over-nourished societies.

Recommendation: Vitamins for Treatment of Acute, Subacute, Chronic, or Post-operative Low Back Pain, or Radicular Pain
In the absence of documented deficiencies or other nutritional deficit states, the use of vitamins is not recommended for treatment of patients with acute, subacute, chronic, or post-operative low back pain or with radiculopathy.
> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

Rationale for Recommendation
There are few trials of vitamins. There is no consistent evidence of efficacy. Various types of vitamins have been suggested for musculoskeletal conditions such as chronic low back pain because of their anti-inflammatory and antinociceptive properties. These vitamins, minerals, and supplements include glucosamine, bromelain, variations of B vitamins, vitamin C, zinc and manganese.(1128) Studies have suggested a correlation between non-specific musculoskeletal pain and vitamin D deficiency, but no significant correlation has been demonstrated in patients with low back pain and vitamin D deficiency.(1166, 1167) This has been complicated by the difficulty of diagnosing vitamin D deficiency.(1168) Randomized controlled trials are needed for better understanding vitamin D repletion in patients with chronic low back pain.(1169)

Evidence for the Use of Vitamins
There is 1 moderate-quality RCT incorporated into this analysis.(1170) There is 1 low-quality RCT in Appendix 1.(1145) (In addition, there are two RCTs that appear to be high quality published in German that are reviewed in Appendix 1.(1171, 1172) However, these were not considered for the development of guidance as the ACOEM methodology requires publications in English.(9))

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: vitamins and low back pain to find 79,341 articles. Of the 79,341 articles, we reviewed 10 articles and included 10 articles.

Allied Health Professionals, Physical and Occupational Therapy, and Other Physical Methods (Devices, Therapies, Electrical Therapies, Acupuncture, and Neuroreflexotherapy)

This section discusses devices, physical methods, and other modalities that have been used to treat LBP. As many of the physical methods described in this section can be administered by other health professionals including physical and occupational therapists and chiropractors, referrals and components of physical and occupational therapy are addressed.

Studies of Referrals to Allied Health Professionals
There are many RCTs that have compared the results of LBP treatments between different health care providers in an attempt to provide evidence for efficacy of one array of treatments over another. However, there are numerous, major methodological weaknesses to this approach that limits the value of these studies including: 1) employment of multiple active, often diverse treatments, 2) lack of a systematic, controlled method to employ the treatments (e.g., not knowing what interventions were employed in what sequence under what circumstances), 3) inability to determine how any one patient was (typically) treated, and 4) lack of control for these potentially confounding variables. Perhaps the single greatest weakness with those studies is that in large part, due to the progress of science, the comparison groups are often no longer treated in the manner that most of these studies utilized (e.g., using bed rest for the general treatment arm). Thus, these studies are largely unusable for purposes of specific evidence-based decision making and guideline development. Throughout this Guideline, these studies are reviewed, but they are nearly always excluded from the decision-making process due to the

aforementioned insurmountable problems. However, guidance on the number of visits for these interventions with allied health professionals (e.g., physical therapists, occupational therapists, chiropractors) may be helpful for treatment of LBP, including guiding a conditioning program, as well as other modalities as indicated elsewhere.

- It should be expected that most patients with more severe acute and subacute LBP conditions receive 8 to 12 visits with allied health professionals over 6 to 8 weeks, as long as functional improvement and program progression are documented. Patients with mild symptoms may require either no therapy appointments or few appointments. Those with moderate problems may require 5 to 6 visits. (The number of recommended visits is the consensus of the Evidence-based Practice Spine Panel.)

- During an episode of therapy, the use of physical agents and manual procedures should be weaned and treatment frequency should decrease. This promotes the patient's active participation in the program and allows transition to an independent self-management program.

- Patients with chronic LBP who have not had prior treatment should follow similar guidance as those with acute LBP. Other chronic LBP patients may need more treatment. Factors influencing the number of visits needed include the content of prior treatment, patient response to prior treatment, their retention of information, and the exercises they were taught.

PHYSICAL AND OCCUPATIONAL THERAPY

The term "physical therapy" is used here in the generic sense to include physical medicine and therapeutic and rehabilitative evaluations and procedures. Physical therapists are major health care providers who render many of these services through multiple, specific interventions (e.g., exercise, ultrasound, manipulation, iontophoresis, etc.).(687, 694, 1173-1185) The majority, if not all, of these interventions are also employed by other health care practitioners. Most occupational therapists are trained to recognize both psychological and physical issues that may influence the treatment of back pain. Each of these specific interventions is discussed in individual topical sections within these Guidelines. However, there are a few RCTs of "physical therapy." The studies in this section include numerous interventions and lack structuring of treatments within the arms of these trials. Thus, there are no strong conclusions that may be drawn from this body of evidence with respect to the value of individual modalities and comparisons between generic treatment programs are weak. These studies of "physical therapy" are reviewed here for completeness. More recent physical therapy literature has explored treatment based on identifying subgroups. The three most commonly seen classification systems are McKenzie, Delitto, et al., and O'Sullivan. There is also research exploring the impact of fear-avoidance beliefs on low back pain, with treatment approaches based on the presence or absence of fear avoidant beliefs.

Recommendation: Physical Therapy, Occupational Therapy, or Other Professionals for Mild to Moderate Acute, Subacute, or Chronic Low Back Pain
A course of 4 to 6 appointments is typically recommended to initiate a directed therapeutic exercise program for mild to moderate acute, subacute, or chronic low back pain. In self-motivated patients or in rapidly resolving cases, one or two visits may suffice.
Indications – Mild to moderate LBP that is felt to be mostly manageable by self-care.
Frequency – Four to six visits to initiate and then reinforce an exercise program is typically helpful. In self-motivated patients and rapidly resolving cases, one or two visits may suffice. More appointments

may be indicated for cases where there is incomplete resolution, lack of a plateau and/or ongoing functional improvements after reaching six visits (see Exercise Section).

Benefits – Increased probability of engaging in an exercise program. Potential reinforcement with provider recommendations.

Harms – Medicalization, prolongation and increased risk of chronicity.

> *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

(See Exercise Section regarding recommendations and education for moderate to severe LBP which may require more prolonged services.)

Evidence for the Use of Physical and Occupational Therapy
There are 4 high-(1186-1189), 49 moderate-quality RCTs (one with 3 reports),(606, 618, 645, 664, 667, 670, 686, 691, 696, 698, 711, 716, 720, 1174, 1190-1225) and 4 secondary analyses(1226-1229) incorporated into this analysis. These studies are heterogeneous with numerous simultaneous interventions, thus sound conclusions cannot be drawn from them (see individual treatment modalities to ascertain the available evidence on specific treatment interventions). There are 2 low-quality RCTs (one targeting unrelated conditions)(1230, 1231) and 4 other studies(748, 1232-1234) in Appendix 1.

We searched PubMed, EBSCO, Google Scholar, and Cochrane Review with no limits on publication dates. The following search terms were used "(Physical OR occupational) AND therapy AND (subacute low back pain OR chronic low back pain)" to find 5498 articles. Of those 5498 articles, we reviewed 68 articles, included 68 articles (68 RCTs, and zero reviews).

Devices
Many devices have been used to treat LBP, including shoe insoles and lifts, taping, lumbar supports and braces, magnets, bedding/mattresses, and hyperbaric oxygen.

SHOE INSOLES AND SHOE LIFTS

1. *Recommendation: Shoe Insoles and Lifts for Treatment of Acute Low Back Pain*
   Shoe insoles and lifts are not recommended for treatment of acute low back pain as there other treatments that have been shown to be beneficial. Patients with a significant leg length discrepancy found in the context of treatment for acute LBP may be reasonable candidates for a shoe insole.

   > *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
   > *Level of Confidence* – Moderate

2. *Recommendation: Shoe Insoles and Lifts for Treatment of Subacute or Chronic Low Back Pain, Radicular Pain, or Other Back-related Conditions*
   Shoe insoles and lifts are not recommended for treatment of subacute or chronic low back pain or radicular pain syndromes or other back-related conditions other than in circumstances of leg length discrepancy over 2cm. In the absence of significant leg length discrepancy, shoe insoles and lifts are not recommended as there are other treatments shown to have demonstrable benefits and minor leg length discrepancies appear unlikely to result in meaningful adverse health effects.

   > *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
   > *Level of Confidence* – Moderate

3. *Recommendation: Shoe Insoles and Lifts for Significant Leg Length Discrepancy*
   Shoe lifts are recommended for treatment of chronic or recurrent low back pain among individuals with significant leg length discrepancy of more than 2cm.

   Indications – Leg length discrepancies that are confirmed on repeated measurements as over 2cm.

   Frequency/Duration – Daily use of shoe lifts.

   Indications for Discontinuation – Patient exhibits lift intolerance. There are substantial numbers of subjects (35%) who do not tolerate shoe insoles as the shoes become too tight.(1235)
   Benefits – Theoretical reduction in LBP.
   Harms – Discomfort associated with accommodation, especially short-term.

   > *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
   > *Level of Confidence* – Low

4. *Recommendation: Shoe Insoles and Lifts for Prevention of Low Back Pain*
   Shoe insoles and lifts are not recommended for prevention of low back pain.

   > *Strength of Evidence* – **Not Recommended, Evidence (C)**
   > *Level of Confidence* – Moderate

5. *Recommendation: Shoe Insoles for Patients with Prolonged Walking Requirements*
   There is no recommendation for or against the use of shoe insoles for patients with chronic low back pain who have prolonged walking requirements.

   > *Strength of Evidence* – **No Recommendation, Insufficient Evidence (I)**
   > *Level of Confidence* – Low

Rationale for Recommendations
Some individuals have lower extremities that are substantially different in length, referred to as "leg length discrepancies" which are generally defined as over 2 to 3cm. These discrepancies are theoretically linked to increased risk of LBP. However, robust prospective cohort studies to substantiate this purported risk factor have not been reported. In theory, shoe lifts may ameliorate this leg length discrepancy and thereby reduce LBP. A nonsystematic review noted that the "role of leg length discrepancy (LLD) both as a biomechanical impediment and a predisposing factor for associated musculoskeletal disorders has been a source of controversy for some time." Shoe insoles or orthotics are sometimes used for primary prevention purposes to theoretically reduce risk of LBP through the reduction in the force generated from heel strike.

There is one quality study reported comparing shoe insoles in patients with LBP which is likely mostly chronic. All of these studies, even those attempting blinding, suffer from probable unblinding of participants and placebo effects. The length of trials ranged from a few weeks to a few months. Shoe insoles are relatively low cost, not invasive, and have little potential for adverse effects. However, there is no recommendation for or against the use of shoe insoles for chronic LBP patients with prolonged walking requirements. For all other spinal pain patients, including those without prolonged walking requirements, there is no quality evidence of efficacy. Shoe insoles and lifts are not recommended for the primary prevention of low back pain as there is no quality evidence of their efficacy. There are other interventions with greater likelihood of efficacy in preventing spinal pain. Shoe insoles and inserts are moderate cost, particularly when considering frequency of replacements. They are not invasive, but

problems with discomfort are relatively common, and non-compliance rates of more than 50% have been reported.

Evidence for the Use of Shoe Insoles and Lifts
There are 3 moderate-quality RCTs or crossover trials incorporated into this analysis.(1235-1237) There are 3 low-quality RCTs in Appendix 1.(1238-1240)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: shoe insoles and lifts, subacute, chronic, radicular and sciatica to find 347 articles. Of the 347 articles, we reviewed 9 and included 4 articles.

KINESIOTAPING (including KT Tape and RockTape) AND TAPING
Taping and kinesiotaping (including KT tape and Rocktape) are used on the extremities and the spine particularly in sports settings.

Recommendation: Kinesiotaping and Taping for Treatment of Acute, Subacute, Chronic Low Back Pain, Radicular Pain, or Other Back-related Conditions
Kinesiotaping and taping are not recommended for treatment of acute, subacute, or chronic low back pain or radicular pain syndromes or other back-related conditions.

> *Strength of Evidence* – **Not Recommended, Evidence (C)**
> *Level of Confidence* – Moderate

Rationale for Recommendation
There are no consistent quality studies demonstrating kinesiotaping and taping are efficacious for the treatment of acute, subacute, or chronic LBP or radicular pain syndromes or other back-related problems. One moderate-quality study suggested it may be effective, however, three found it ineffective.(1241-1244) The theory is that taping supports the muscles, although most of the spine muscles are small and deep, thus the rationale for taping the back seems limited. Taping has occasionally been used as a technique to teach posture. However, there are concerns about the value of this technique as there also is some controversy regarding appropriate postures for work and lifting. These interventions are not invasive, but there are generally minor adverse effects among patients who do not tolerate tape or the adhesives. However, tape is expensive and there are other interventions shown to be efficacious.

Evidence for Use of Kinesiotaping and Taping
There are 4 moderate-quality RCTs incorporated into this analysis.(1241-1244) There are 2 low-quality RCT in Appendix 1.(1245, 1246)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar with no limits on publication dates. The following search terms were used "(kinesiotaping AND taping) AND (subacute low back pain chronic low back pain radicular pain syndromes (including 'sciatica') spinal stenosis, sacroiliitis spondylolisthesis)" to find 13,533 articles. Of those 13,533, we reviewed 5 articles, and included 5 articles (5 RCTs and zero reviews).

LUMBAR SUPPORTS

Lumbar supports range from soft wrap-around appliances to reinforced braces to rigid braces and have been used to treat various phases of lumbar pain(832, 1247-1251) and post-surgical rehabilitation. They have also been used for prevention of low back pain.(192, 1252-1255) The rigid devices have been used particularly in post-operative lumbar fusion with a goal to facilitate boney union.

1. *Recommendation: Lumbar Supports for Prevention of Low Back Pain*
   Lumbar supports are not recommended for prevention of low back pain.

   > *Strength of Evidence* – **Not Recommended, Evidence (C)**
   > *Level of Confidence* – Moderate

2. *Recommendation: Lumbar Supports for Treatment of Acute, Subacute and Chronic Low Back Pain*
   Lumbar supports are not recommended for treatment of low back pain.

   > *Strength of Evidence* **–** Not Recommended, Evidence (C)
   > *Level of Confidence* – Moderate

3. *Recommendation: Lumbar Supports after fusion surgery for Low Back Disorders*
   Rigid lumbar supports are recommended for post-operative fusion patients.

   Benefits – Facilitate fusion.
   Harms – Discomfort, dermal irritation.

   > *Strength of Evidence* **–** Recommended, Insufficient Evidence (I)
   > *Level of Confidence* – High

Rationale for Recommendations
The overall quality of the available evidence is relatively limited and there is no clear evidence of efficacy for the use of lumbar supports for short- or long-term treatment or prevention of low back pain. Lumbar supports also attempt to enforce reduced mobility in contrast to evidence that increasing activity levels reduces LBP (see Bed Rest and Aerobic Exercises). Thus, the theoretical construct for a beneficial use of lumbar supports for either treatment or prevention of LBP appears tenuous, although they may be useful for specific treatment of spondylolisthesis, documented instability, or post-operative treatment.

Soft braces have been used to prevent LBP and studied in workers in high risk industries (warehousing, airline baggage handling). Theoretical mechanisms for the prevention of LBP include provision of trunk support and prevention of pain-producing events, reminders of "proper lifting technique," and an increase in intra-abdominal pressure and a decrease in intradiscal pressure.(1256) However, limiting movement to avoid pain is contrary to the cognitive behavioral approaches to LBP shown to be helpful. Proper lifting technique is problematic and reviewed elsewhere, and there is no quality evidence that such devices reduce intradiscal pressure. Reported compliance rates are poor (about 40%)(135, 1257) and complaints include excessive heat, restrictive movements, discomfort with sitting, rubbing or pinching of skin, and feelings of bruised ribs.(135, 1257)

Lumbar supports are low to moderate cost. They are not invasive, but they have minor and widely prevalent adverse effects resulting in low compliance rates. There are other interventions with evidence of efficacy especially for treatment (NSAIDs, exercise, cognitive-behavioral, etc.), and also for prevention (exercise).

Evidence for the Use of Lumbar Supports

There are 10 moderate-quality RCTs incorporated into this analysis.(135, 207, 832, 1250, 1255, 1257-1261) There are 4 low-quality RCTs in Appendix 1.(1262-1265)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: lumbar supports, subacute low back pain and chronic low back pain to find 31,235 articles. Of the 31,235 articles we reviewed eleven articles and included all eleven articles.

MAGNETS
Proponents believe that magnetic fields have therapeutic value in the treatment of musculoskeletal disorders.

Recommendation: Magnets for Treatment of Acute, Subacute, or Chronic Low Back Pain
Magnets are moderately not recommended for treatment of acute, subacute, or chronic low back pain.

> *Strength of Evidence* – **Moderately Not Recommended, Evidence (B)**
> *Level of Confidence* – High

Rationale for Recommendation
Two moderate-quality RCTs suggest a lack of efficacy and none support efficacy.(1266, 1267) Magnets are not invasive, have no adverse effects, and are low cost. However, other treatments have proven efficacy.

Evidence for the Use of Magnets
There are 2 moderate-quality RCT/crossover trial incorporated into this analysis.(1266, 1267)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following terms: magnets, subacute low back pain, chronic low back pain, radicular pain syndromes (including 'sciatica'), Spinal stenosis, spinal fractures, sacroiliitis, and spondylolisthesis to find 437 articles. Of the 437 articles we reviewed 2 articles and included 2 articles.

HYPERBARIC OXYGEN
Hyperbaric oxygen (HBO) involves the administration of oxygen in a pressurized chamber to increase the oxygen delivery to the tissues of the body. It has been used to treat a number of conditions with problematic microvascular blood supply, including diabetic foot ulcers and decubitus ulcers. Oxygen may be titrated to higher concentrations up to 100%. Small individual patient chambers or a large walk-in multi-patient chamber may be used. There also are "topical" hyperbaric oxygen treatments that do not involve the use of chambers.

1. *Recommendation: Hyperbaric Oxygen for Treatment of Chronic Low Back Pain*
   Hyperbaric oxygen is not recommended for treatment of chronic low back pain.

   > *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
   > *Level of Confidence* – Moderate

2. *Recommendation: Topical Hyperbaric Oxygen for Treatment of Chronic Low Back Pain*
   Topical hyperbaric oxygen is not recommended for treatment of chronic low back pain.

   > *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
   > *Level of Confidence* – Moderate

Rationale for Recommendations
There are no quality trials identified. Hyperbaric oxygen is costly, and in the absence of evidence of efficacy, is not recommended (see Chronic Pain Guideline for other conditions).

Evidence for the Use of Hyperbaric Oxygen
There are no quality studies incorporated into this analysis.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without any limits on publication dates. We used the following search terms: Topica Hyperbaric Oxygen, Hyperbaric Oxygen, HBO and Chronic Low back pain to find 4, 600 articles. Of the 4, 600 articles, we reviewed 0 articles and included 0 articles.

IONTOPHORESIS
Iontophoresis is a drug delivery system utilizing electrical current to transdermally deliver either glucocorticosteroids or NSAIDs and that has apparent efficacy in the extremities where the dermis and adipose tissue overlying the target tissue is thin and penetration of the medicine to the target tissue is possible, which does not describe the spine.

Recommendation: Iontophoresis for Treatment of Low Back Pain
There is no recommendation for or against iontophoresis for treatment of acute, subacute, or chronic low back pain or radicular pain syndromes or other back-related conditions.

      Strength of Evidence – **No Recommendation, Insufficient Evidence (I)**

Rationale for Recommendation
Iontophoresis is not shown to be efficacious for the treatment of acute, subacute, or chronic LBP or radicular pain syndromes or other back-related problems. It is not invasive and is not low cost. There are other interventions shown to be efficacious.

Evidence for Use of Iontophoresis
There are no quality studies evaluating the use of iontophoresis for the treatment of LBP.

We searched PubMed, EBSCO, Google Scholar, Cochrane review with no limits on publication dates. We used following search terms chronic low back pain radicular pain syndromes (including 'sciatica') spinal stenosis, sacroiliitis, spondylolisthesis to find 54 articles. Of 54 articles, we reviewed zero articles and included zero articles.

MASSAGE

Massage is a commonly used treatment for LBP.(796, 799, 1268-1275) Massage is theorized to aid muscle and mental relaxation which could hypothetically result in increased pain tolerance through endorphin release.(1276-1278) Other theories are that massage may enhance local blood flow that could increase clearance of chemical pain mediators or stimulate large diameter nerve fibers that have an inhibitory input on T-cells in the spinal cord, resulting in decreased pain.(1276, 1279, 1280)

1. *Recommendation: Massage for Select Subacute or Chronic Low Back Pain*
   Massage is recommended for select use in subacute or chronic low back pain as an adjunct to more efficacious treatments consisting primarily of a graded aerobic and strengthening exercise program.

Indication – For time-limited use in subacute and chronic LBP patients without underlying serious pathology such as fracture, tumor, osteoporosis, or infection as an adjunct to a conditioning program that has both graded aerobic exercise and strengthening exercises. Massage is recommended to assist in increasing the patient's functional activity levels and comfort more rapidly although the primary treatment focus should remain on the conditioning program. In patients not involved in a conditioning program or who are non-compliant with graded increases in activity levels, this intervention is not recommended.

Frequency/Duration – Six to 10 sessions of 30 to 35 minutes each, 1 or 2 times a week for 4 to 10 weeks. Objective improvements should be shown approximately half way through the regimen to continue this treatment course.

Indications for Discontinuation – Resolution, intolerance, lack of benefit, or non-compliance with aerobic and strengthening exercises.
Benefits – Modest reduction in pain.
Harms – Short term discomfort during massage, and potentially longer term afterwards with more vigorous massage.

> *Strength of Evidence* – **Recommended, Evidence (C)**
> *Level of Confidence* – Low

2. Recommendation: Massage for Treatment of Acute Low Back Pain or Chronic Radicular Pain Syndromes

Massage is recommended for select use in acute low back pain or chronic radicular pain syndromes in which low back pain is a substantial symptom component.

Indications – Patients with acute LBP or chronic radicular pain syndromes. For acute LBP, patients should have already had NSAIDs/acetaminophen, aerobic exercise, directional exercises, cold/heat instituted with insufficient results as they typically resolve acute LBP. Massage is recommended as an adjunct to more efficacious treatments to assist in increasing functional activity levels more rapidly although it is recommended that the primary treatment focus remain on the conditioning program. In patients not involved in a conditioning program or who are non-compliant with graded increases in activity levels, this intervention is not recommended.

Frequency/Duration – Objective benefit (functional improvement along with symptom reduction and opioid reduction) should be demonstrated after a trial of 5 sessions in order for further treatment to continue, for up to 10 visits during which a transition to a conditioning program is accomplished.

Indications for Discontinuation – Resolution, intolerance, or lack of benefit.

Benefits – Modest reduction in pain
Harms – Short term discomfort during massage, and potentially longer term afterwards with more vigorous massage.

> *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

3. Recommendation: Mechanical Devices for Administering Massage
Mechanical devices for administering massage are not recommended.(1281, 1282)
> *Strength of Evidence* – **Not Recommended, Evidence (C)**
> *Level of Confidence* – Moderate

Rationale for Recommendations
Massage is a commonly used treatment for LBP. Relatively few higher quality trials of massage have been reported, varying massage methods have been used, methods and patient populations differed substantially between trials, and long-term followup is largely lacking in most trials(1283) resulting in heterogeneous results. Many trials have utilized massage as a control treatment for other interventions.(1250) Trials suggest modest benefits.

Two studies used mechanical massage devices – one was negative,(1281) and the other showed no differences with modest overall reductions in pain similar to two other interventions demonstrating that mechanical massage devices have not been shown to be beneficial.(1282)

The two highest quality studies involving manual massage techniques suggest benefits of massage compared to other modalities for treatment of subacute and chronic LBP.(1284, 1285) Higher quality studies utilized massage therapists to administer the treatments, suggesting that the experience of the massage provider and quality of the massage may be important factors.

Massage is not invasive, has low risk of adverse effects aside from short-term pain, (1284) and is moderately costly in aggregate. It is recommended for treatment of subacute and chronic LBP, but only as an adjunct to a conditioning program. It is also recommended for select use in acute LBP or radicular pain syndromes. Mechanical devices are not recommended.(1281, 1282)

Evidence for the Use of Massage
There are 14 moderate-quality RCTs incorporated into this analysis.(550, 640, 861, 1250, 1281, 1282, 1284-1291) There are 5 low-quality RCTs in Appendix 1.(1274, 1292-1295)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: massage, subacute low back pain, low back pain, radicular low back pain, massage, clinical trial, randomized controlled trial, random, systematic review, review, population study, epidemiological study, and prospective cohort to find 11,944 articles. Of those 11,944 articles, we reviewed 26 articles and included 25 articles (18 RCTs and 7 reviews). We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following terms: Mechanical devices for administering massage subacute low back pain, chronic low back pain, radicular pain syndromes, and sciatica to find 2,084 articles. Of the 2,084 articles, we reviewed zero articles and included zero articles.

REFLEXOLOGY
Reflexology is a treatment that focuses on massage of reflex points which are believed to be linked to physiological responses and healing of other tissues including those in the back.(1296)

1. *Recommendation: Reflexology for Treatment of Chronic Low Back Pain*
   Reflexology is not recommended for treatment of chronic low back pain.

   Strength of Evidence – **Not Recommended, Evidence (C)**
   Level of Confidence – Moderate

2. *Recommendation: Reflexology for Treatment of Acute, Subacute, Radicular, Post-operative Low Back Pain or Other Low Back Conditions*
Reflexology is not recommended for treatment of acute or subacute low back pain or other low back conditions.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Moderate

Rationale for Recommendations
Reflexology has not been shown to be clearly efficacious for the treatment of chronic LBP in either of two moderate-quality studies.(1297, 1298) There is no evidence of efficacy for the use of reflexology for other LBP conditions. Other treatments have been shown to be efficacious.

Evidence for the Use of Reflexology
There are 2 moderate-quality RCTs incorporated into this analysis.(1297, 1298) There is 1 low-quality RCT in Appendix 1.(1299)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar with limits on publication dates from 2011-2012. We used the following terms: reflexology, subacute low back pain, chronic low back pain, radicular pain syndromes (including 'sciatica'), Spinal stenosis, spinal fractures, and spondylolisthesis to find 116 articles. Of the 116 articles we reviewed 3 articles and included 3 articles.

CHIROPRACTIC CARE
There are RCTs of "chiropractic care" which are reviewed here for completeness. Because of the broad realm of chiropractic care, including different manipulation techniques,(1300) the lack of structuring of treatment arms within these particular trials of chiropractic care, inclusions of multiple co-interventions, and questions about the adequacy of control group treatments, no strong conclusions can be drawn from this particular body of evidence with respect to the value of individual modalities or even comparisons between generic programs. Sound conclusions cannot be drawn from these RCTs of multiple modalities. (See individual treatment modalities to ascertain the available evidence on specific treatment interventions.)

Evidence for the Use of Chiropractic Care
There are 11 moderate-quality RCTs incorporated into this analysis.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following terms: chiropractic care, chiropractor, and low back pain to find articles. Of the articles we reviewed, 9 articles and all were included.

MYOFASCIAL RELEASE
Myofascial release is a manual soft tissue technique to attempt to stretch and apply traction on target tissue(s). It is most commonly used in the periscapular area to treat non-specific muscle soreness.

Recommendation: Myofascial Release for Treatment of Low Back Pain
There is no recommendation for or against the use of myofascial release for treatment of acute, subacute, chronic, post-operative low back pain, radicular pain syndromes or other back-related conditions.

Rationale for Recommendation
There are no placebo or sham trials. There is one comparative trial and it does not show clear efficacy.(1301) Thus, myofascial release is not shown to be efficacious for LBP, although there are other techniques to be investigated. Myofascial release is not invasive and is not low cost and there is no recommendation for or against its use. However, there are other interventions shown to be efficacious.

Evidence for Use of Myofascial Release
There is 1 moderate-quality RCT incorporated into this analysis.(1301)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar with no limits on publication dates. The search terms used were "(sub-acute low back pain OR chronic low back pain OR radicular pain syndrome OR sciatica OR Spinal stenosis OR spinal fractures OR sacroiliitis OR spondylolisthesis) AND myofascial release" to find 1357 articles. Of those 1357 articles, we reviewed one and included (1 RCT and zero review).

TRACTION

Traction is the distraction of structures within the lumbar spine by application of tension along the axis of the spinal column that is most frequently used to treat radicular syndromes.(588, 1283, 1302-1310) Duration and magnitude of force is adjustable and sometimes varied. Types of traction include motorized, manual, bed rest, pulley-weight, gravitational, suspension, and inverted, with manual and motorized being most commonly used. Trials with subgroups of patients have appeared promising for a minority of patients, but full validation studies are yet to be reported.(570, 1302)

Recommendation: Traction for Treatment of Low Back Pain
Traction is not recommended for treatment of acute, subacute, or chronic low back pain or radicular pain syndromes.

Strength of Evidence – **Strongly Not Recommended, Evidence (A)** (Subacute, Chronic)
Moderately Not Recommended, Evidence (B) (Radicular)
Not Recommended, Insufficient Evidence (I) (Acute, Post-operative LBP)
Level of Confidence – Moderate

Rationale for Recommendation
There are quality studies that have evaluated the value of traction in treating LBP, although most of the literature has significant limitations. The higher quality studies appear to have successfully blinded participants in contrast with many other studies. Nearly all of the highest quality studies failed to show meaningful benefits from traction.(570, 1311-1316)

Traction has long been used to treat sciatica with a belief that this therapy produces negative intradiscal pressures that result in improved rates of disc resorption. However, this has not been borne out and more studies show a lack of efficacy (1307, 1311, 1317-1319) than show efficacy for those patients.(1316, 1320, 1321) Traction is non-invasive, does not have adverse effects, but is moderately costly. There are interventions that are effective that should be employed. Traction is not recommended for treatment of low back conditions or radicular pain syndromes.

Evidence for the Use of Traction

There is 1 high- (with 2 reports)(1311, 1313) and 19 moderate-quality(570, 699, 1059, 1258, 1282, 1306, 1314-1326) RCTs incorporated into this analysis. There are 4 low-quality RCTs in Appendix 1.(1307, 1327-1329)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: traction, subacute low back pain, chronic low back pain, and radicular pain syndromes (including sciatica) to find 6,348 articles. Of the 6,348 articles, we included 19 articles.

DECOMPRESSION AND DECOMPRESSIVE DEVICES

Decompression through traction is a treatment that utilizes a therapeutic table and traction mechanism. Its intent is to reduce intradiscal pressure, thus allowing for disc decompression. The theory is that decompression will externally decompress the nerve root and help relieve pain and other symptoms.

Recommendation: Decompression through Traction and Spinal Decompressive Devices for Treatment of Acute, Subacute, or Chronic Low Back Pain or Radicular Pain Syndromes
Decompression through traction and spinal decompressive devices is not recommended for treatment of acute, subacute, chronic, post-operative low back pain, or radicular pain syndromes.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Moderate

Rationale for Recommendation
There is no clear evidence for efficacy of this treatment.(1308, 1330) Decompression through traction and spinal decompressive devices are not recommended for the treatment of acute, subacute, chronic, or radicular pain syndromes. There is insufficient evidence to recommend this treatment which is moderately costly, though not invasive.

Evidence for the Use of Decompression through Traction and Decompressive Devices
We searched PubMed, EBSCO, Cochrane Review, and Google Scholar with no limits on publication dates and an updated search was conducted using PubMed for publication between 1/1/2013 and 11/15/2017 using the following terms: Decompression through traction, spinal decompressive devices, subacute low back pain, chronic low back pain, radicular pain syndromes, sciatica, and random*)" to find 1828 articles. Of the 1828 articles, we considered 23 for inclusion. Of the 23 considered for inclusion, 2 are randomized controlled trials and 1 is a systematic review.

MANIPULATION AND MOBILIZATION

Manipulation and mobilization are two types of manual therapy that include wide arrays of different techniques and schools of thought.(102, 1331-1335) Some consider these two interventions to be on a spectrum of velocity and applied force. In general, mobilization involves assisted, low-force, low-velocity movement. Manipulation involves high-force, high-velocity, and low-amplitude action with a focus on moving a target joint. As commonly used, "adjustment" is generally a synonym for manipulation.

From the standpoint of evidence-based practice guidelines development, there are numerous types of manipulation utilized in different studies. It seems unlikely that if there is an effect of manipulation, that it should be the same regardless of diagnosis, technique, or any other factors. This results in difficulties

with comparing methods, techniques, or results across the available literature. These differences appear to be largely unstated in the available systematic reviews, which have aggregated all studies.

1. *Recommendation: Manipulation or Mobilization of the Lumbar Spine for Treatment of Acute or Subacute Low Back Pain or Radicular Pain Syndromes without Neurological Deficit*
Manipulation or mobilization of the lumbar spine is recommended for select treatment of acute or subacute low back pain, or radicular pain syndromes without neurological deficit. Manipulation may also be considered for treatment of *severe*, acute LBP concurrently with directional preference exercises, aerobic exercise, and NSAIDs with the *goal to improve motion* and hopefully to decrease pain and enable more efficient exercise.

Indications – Acute, subacute LBP, and radicular syndromes without neurological deficits. Patients should generally have had NSAIDs and/or acetaminophen, directional and aerobic exercise instituted and have insufficient results over 1 to 2 weeks. Indications include unresolving acute or sub-acute LBP with: 1) patient preference especially with positive past experience for the same/similar problem; or 2) health conditions with increased risk of harms from NSAIDS/ acetaminophen; or 3) patient aversion to medication use or intolerance to aerobic exercise and directional exercises; and/or 4) persisting activity intolerance or unacceptable pain level after 7 to 10 days and a trial of NSAIDS, acetaminophen or aerobic exercise.

Frequency/Duration – Most patients with more severe LBP conditions may receive up to 12 visits over 6 to 8 weeks,(595, 712, 1336-1338) as long as functional improvement (and not minor improvements in pain ratings) and progression away from passive modalities to a more active HEP and self-directed activity program are documented when re-evaluated after 3 to 6 visits. There is no quality evidence that more than 12 visits are helpful for an episode of LBP. Compliance, including with conditioning exercises and efficacy should be demonstrated. Patients likely to benefit from manipulation exceeding these ranges may have complicating circumstances associated with slower recovery times or delayed treatment response, though nevertheless should show significant early therapeutic effects.

Indications for Discontinuation – Increased pain or development of a radicular pain problem is an indication for immediate discontinuation. Failure to progress in functional improvement after 3 to 6 visits should result in reassessment and either a change to an alternative manipulation program or discontinuation. For any episode of acute or subacute pain, or for a treatment trial for chronic back pain, treatment should be discontinued by the 12th manipulation session, except in those cases (noted above) where continued functional improvement is demonstrated.

Benefits – Potential for faster resolution of pain and improved function.
Harms – Worsening of LBP, especially immediately after manipulation.

> *Strength of Evidence* **– Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

2. *Recommendation: Regular or Routine Manipulation or Mobilization*
Regular or routine manipulation or mobilization is not recommended as there is no evidence of efficacy.
There is no evidence that prophylactic treatment is effective for primary prevention (before the first episode of pain) or for secondary prevention (after recovery from an episode of back pain), and prophylactic treatment is not recommended. There is also no evidence that manipulation on a regular or routine basis is beneficial.

Strength of Evidence – **Not Recommended, Insufficient Evidence (I)**
Level of Confidence – High

3. *Recommendation: Manipulation or Mobilization for Chronic Pain*
Manipulation or mobilization of the lumbar spine is recommended for short-term relief of chronic pain or as a component of an active treatment program focusing on active exercises for acute exacerbations.

Frequency/Duration – 1 to 3 times a week for 2 weeks;(1339-1341) total treatments dependent on response to therapy with most higher-quality studies suggesting a maximum of 6 appointments.(679, 861, 1193, 1342) Substantial functional progress (e.g., return to work or activities, increasing ability to tolerate exercise, reduced impairing medication use) should be documented at each follow-up visit. Treatment plan should be reassessed after each 2-week interval. Most guidelines suggest that if there is significant response in the above outcomes, it is worth considering another 2 weeks of treatment. If no response to 2 weeks of application of a particular manipulation treatment, it should be discontinued and 2 weeks of a different method of manipulation/mobilization or other treatment should be considered. If there is no response after 4 weeks and two 2-week trials of different manipulation/mobilization techniques, it is unlikely that further manipulation/mobilization will be helpful.

Indications for Discontinuation **–** Lack of demonstrated continued functional response after 6 manipulation/mobilization sessions (2 trials of 2 or more different methods), resolution of symptoms, or failure to participate in an active rehabilitation program.
Benefits – Potential for faster resolution of pain and improved function.
Harms – Worsening of LBP, especially immediately after manipulation.

Strength of Evidence – **Recommended, Evidence (C)**
Level of Confidence – Low

4. *Recommendation: Manipulation for Treatment of Radicular Pain Syndromes with Acute Neurological Deficits*
Manipulation is not recommended for treatment of radicular pain syndromes with progressive motor loss. Patients often have radicular pain in the lower extremity without clear evidence of neurological impingement and these patients do not have demonstrated contraindications for manipulation(1343, 1344) and may be considered in Recommendation #1 above. The available studies attempting to directly address this question provide somewhat contradictory evidence.(1343, 1345) There also are concerns about the use of manipulation in the presence of acute or progressive neurological deficits.

Strength of Evidence – **Not Recommended, Insufficient Evidence (I)**
Level of Confidence – Low

5. *Recommendation: Manipulation/Mobilization of Non-adjacent Areas for Low Back Pain*
Manipulation or mobilization of regions outside of/not adjacent to the lumbopelvic area (e.g., cervical spine, lower extremity) is not recommended for treatment of low back pain.

Strength of Evidence – **Not Recommended, Insufficient Evidence (I)**
Level of Confidence – High

Rationale for Recommendations
The highest quality sham-manipulation trial suggested no benefits of manipulation.(1346) There are many additional moderate quality studies evaluating manipulation, although there are problems with

quality of the available literature,(1347-1349) use of mixtures of manipulation with exercises and other treatments precluding conclusions on efficacy of spinal manipulation, and suboptimal statistical testing that have been noted.(1350, 1351) There are comparative trials with "usual care" (which often is not "usual" today and/or contain numerous uncontrolled co-interventions) but no quality studies demonstrating superiority of manipulation for LBP patients compared with the other treatment strategies (e.g., NSAIDs, progressive walking program, directional exercises, and heat) contained in this guideline. One comparative trial suggested adjunctive Manual-thrust manipulation was modestly superior to mechanical-assisted manipulation (MAM) at 4 weeks but not longer-term. Both also treated with ibuprofen, with no differences between MAM and largely unstructured "usual medical care."(1334)

The manipulation literature resulted in the publication of a clinical prediction rule (CPR) that appeared quite promising.(595, 1352) Yet, a subsequent attempt to validate this CPR failed.(649, 658) It is also somewhat concerning that of the five highest quality studies, three found no benefit,(812, 1336, 1338) one resulted in the CPR subsequently not validated(595) and only one was positive for comparing manipulation with non-thrust manipulation.(1337) However, most of the evidence continues to suggest manipulation is approximately as efficacious as common physiotherapy interventions such as stretching or strengthening exercises for treatment of acute and chronic LBP. These weaknesses have resulted in a decrease in the strength of evidence rating for manipulation for acute pain to "I" from "B."

Manipulation is not without risks. Reported but rare fatal outcomes have been associated with cervical *not* lumbar manipulation. Adverse effects reported include vertebrobasilar accidents (neck manipulation only) and disc herniation or progression to cauda equina syndrome. One study suggested lower risk among a manipulated group compared to non-manipulated patients but was not randomized and likely had considerable selection and spectrum biases.(1353) The mean age of vertebrobasilar accidents in the case reports is 38 and the risk has been reportedly due to cervical manipulation with a rotary component.(1354) Twenty-nine of the vertebrobasilar accidents resulted in death.

Manipulation is not invasive, is of moderate to high cost in aggregate, but does have rare adverse effects.(1355-1359) However, the adverse effects are primarily from cervical, not lumbar manipulation. If other interventions that have evidence of efficacy have failed, it may be acceptable to use manipulation as a secondary treatment option adjunct to a program of evidence-based functional restoration if tied to signs of objective functional recovery within 2 weeks that is faster than the progress expected with the rate of usual spontaneous recovery. For acute, severe LBP, it may also be reasonable to initially prescribe manipulation in addition to aerobic exercise, directional exercise and NSAID. Minimum and maximum dosage thresholds of manipulation are difficult to extract from these studies. In general, the studies assessed treatment effects early on and with a limited number of encounters. Studies generally suggest that a treatment effect from manipulation would be expected within the first 2 weeks and first few visits. A decision to continue manipulation should be based on establishing a positive early treatment response for functional outcomes (e.g., distance walking, work ability/limitations).

Nearly all studies excluded patients with symptoms consistent with sciatica.(1343) Leg pain was allowed, but the definition of "leg" vs. lower extremity pain was not specified. Essentially all have eliminated those with neurological deficits. Thus, there is lack of demonstrated efficacy on patients with sciatica and concerns exist about reports of increased symptoms of neurological compression after manipulation.

There are no quality studies for adjustments or manipulations of the neck/cervical spine or other areas outside of the lumbopelvic region. High-velocity rotary cervical spine manipulations have reportedly had severe consequences, though these are rare. Adjustments or manipulations are not invasive, are of moderate cost, but have rare severe complications. Therefore, adjustments or manipulations of the cervical spine to treat LBP or other lower back problems are not indicated.

Evidence for the Use of Manipulation and Mobilization
There are 1 high-(812) and 36 moderate-quality RCTs incorporated into this analysis (5 with multiple reports).(549, 595, 618, 639, 679, 691, 832, 852, 861, 1193, 1197, 1258, 1318, 1321, 1334-1338, 1342-1346, 1352, 1360-1377) There are 14 low-quality RCTs in Appendix 1.(624, 1378-1390)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following terms: manipulation, mobilization, subacute low back pain, chronic low back pain, and radicular pain syndromes to find 21,394 articles. Of the 21,394 articles we reviewed 39 articles and all were included.

MANIPULATION UNDER ANESTHESIA (MUA) AND MEDICATION-ASSISTED SPINAL MANIPULATION (MASM)
Manipulation under anesthesia (MUA) and medication-assisted spinal manipulation (MASM) involves the administration of anesthesia or medication followed by manipulation of the spine with the intended effect of relieving LBP.(1391-1396) Proponents believe this method of manipulation is superior to manipulation without anesthesia due to factors including the reduction in resistance to movement that occurs after the administration of the anesthetic. However, such reductions in resistance may increase the likelihood of injuries to the patient.(1397)

Recommendation: MUA and MASM for Treatment of Acute, Subacute, or Chronic Low Back Pain
MUA and MASM are not recommended for treatment of acute, subacute, or chronic low back pain.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Moderate

Rationale for Recommendation
MUA and MASM have been evaluated in chronic LBP patients in one RCT; however, that study used a complex mixture of interventions and changed multiple interventions between the two groups.(1398) Thus, there is no quality study reported comparing these with either a non-interventional control or other conservative treatment. There are also no quality studies that solely evaluate MUA or MASM. MUA/MASM is high cost, is invasive when combined with injections, and has the potential for significant adverse effects (e.g., herniations, fracture)(1399) although no reports of complications with the use of more modern osteopathic and chiropractic techniques as the result of anesthesia or subsequent to 1986 were found.(1400)

Evidence for the Use of MUA and MASM
There is 1 moderate-quality RCT incorporated into this analysis.(1398)

We searched PubMed, EBSCO, Google Scholar, and Cochrane Review with no limits on publication dates. The following search terms were used: "(manipulation under anesthesia OR medication assisted spinal

manipulation) AND (low back pain OR chronic low back pain)" to find 15,391 articles. Of those 15,391 articles, we reviewed 9 articles, included 7 articles (4 RCTs and 3 reviews).

HOT AND COLD THERAPIES
Cold and heat are believed to have therapeutic benefits to modify the disease processes (e.g., cold to reduce acute inflammation and swelling, and heat to speed healing through increased blood supply).(330, 1401-1403) However, some practitioners believe that these various modalities are all distractants that do not materially alter the clinical course. Others believe the distractants allow increased activity levels, thus even though there may be no direct action of these modalities and the disease processes, this theory supports using these modalities through indirect mechanism(s) of action.

Cryotherapies
Cold or cryotherapies involve applications of cold or cooling devices to the skin, such as towels moistened with cold water, ice wrapped in a blanket, ice massage, cold water and/or ice placed in a "water bottle," gel packs, cooling sprays, or single-use chemical packets that produce cooling on breaking one pouch inside the other to start a chemical reaction.(1404) There also are chemical sprays which produce cooling based on evaporation; however, the administration of these sprays is considerably more expensive. There is considerably less scientific literature focused on this set of therapeutics, and essentially no quality research on moist versus dry cryotherapy.(1405)

Cryotherapy purportedly delays or reduces inflammation.(1401) Application of cold will result in vasoconstriction, though a subsequent vasodilatory response to reassert homeostasis is also likely. Similar to heat therapies, most researchers believe that cryotherapies do not directly result in healing. Rather, the general beliefs are that these may distract the patient from other painful stimuli, thus allowing faster resumption of normal activities or increased tolerance of therapeutic exercises. Despite the lack of evidence for direct healing benefits because of the potential for increased function and earlier recovery, the use of cryotherapies for the patient's benefits may still be worthwhile, particularly as the cost for some of these methods for intervention is essentially nil.

1. *Recommendation: Cryotherapies for Treatment of Acute, Subacute, or Chronic Low Back Pain*
   Self-applications of low-tech cryotherapies are recommended for treatment of acute low back pain. Cryotherapies may be tried for subacute or chronic low back pain, though they may be less beneficial.

   Indications – Moderate to severe acute LBP patients with sufficient symptoms that an NSAID/acetaminophen and progressive graded activity are believed to be insufficient. May be tried as well for subacute or chronic pain, but suggested threshold for discontinuation is lower, particularly as active modalities are generally far preferable to passive modalities for rehabilitation of non-acute LBP.
   Indications for Discontinuation – Non-tolerance, including exacerbation of LBP.
   Benefits – Potential modest reduction in LBP. Self-efficacy, although relying on a passive modality.
   Harms – Cold injuries. Time may be devoted to passive modality instead of active exercises.
   > *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
   > *Level of Confidence* – Low

2. *Recommendation: Routine Use of Cryotherapies for Treatment of Low Back Pain*

Routine use of cryotherapies in health care provider offices or home use of a high-tech device is not recommended for treatment of low back pain. However, single use of low-tech cryotherapy (ice in a plastic bag) for severe exacerbations is reasonable.

Strength of Evidence – **Not Recommended, Insufficient Evidence (I)**
Level of Confidence – Moderate

Rationale for Recommendations
One trial with scant results suggests ice better than heat or alternating ice-heat for chronic LBP,(1404) thus, precluding strong conclusions. Self-applications of cryotherapies using towels or reusable devices are not invasive, are without complications, and do not have any appreciable costs. These are recommended as potential distractants or counter-irritants. Other forms of cryotherapy can be considerably more expensive, including chemicals or cryotherapeutic applications in clinical settings, and are not recommended.

Evidence for the Use of Cryotherapies
There is 1 moderate-quality RCT incorporated into this analysis.(1404) There is 1 low-quality RCT in Appendix 1.(1406)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following terms: cryotherapies, ice, cold, ice pack, cold pack, and low back pain to find 17,506 articles. Of the 17,506 articles we reviewed one article and included one article.

Heat Therapies
There are many forms of heat therapy for treatment of LBP. These include hot packs, moist hot packs, sauna, warm baths, infrared, diathermy, and ultrasound. The depth of penetration of heat is minimal for local convective means, but the other modalities have deeper penetration.(1407) A particular methodological problem with most of these studies is that, despite occasional attempts at and claims of successful blinding, it is essentially impossible to blind the patient from these interventions as they produce noticeable, perceptible tissue warming. Some of these heat-related modalities have been shown to reduce pain ratings more than placebo (see below), it is less clear whether there are meaningful long-term benefits.

Hot Packs, Heat Wraps, and Moist Heat
The application of warmth or heat is frequently divided into dry or moist heat. Moist heat involves the application of a wet towel or other device that brings the warmed water into direct contact with the skin. Dry heat does not involve direct application of water on the skin surface. Thus, a water bottle is still generally classified as dry heat. Hot or heat packs are common household items or commercial products that are heated and then applied to the skin. In the simplest form, a heated towel is used. Heat wraps include devices that produce heat at greater depth than typical convective heat.(1408, 1409) Some chemical products, frequently marked as glove warmers for cold ambient conditions, are also now available that produce warmth. Electrical blankets are another of the more commonly used sources of dry heat.(1410)

Moist heat most commonly involves heating wet towels, soaking a towel in warm water, or using commercial products that are soaked in a warm bath prior to application on the skin surface. Some

patients heat moist towels in a microwave oven; however, this is ill-advised as the potential for steam burns is considerable.

1. *Recommendation: Heat Therapy for Treatment of Acute, Subacute, or Chronic Low Back Pain*
Self-applications of heat therapy, including a heat wrap, are recommended for treatment of acute, subacute, or chronic low back pain. However, use in chronic LBP is suggested to be minimized to flare-ups with the primary emphasis in chronic LBP patients being placed on functional restoration elements including aerobic and strengthening exercises. Application of moist heat by a health care provider in conjunction with an exercise program may have some short-term value in the treatment of acute LBP for a single treatment primarily for demonstrative and educational purposes. However, education regarding home application should be part of the treatment.

   Indications – Acute, subacute, or chronic LBP.
   Frequency/Duration – Self-applications may be periodic or continuous and include different regimens – e.g., 15 to 20 minutes, 3 to 5 times a day. These applications should be home-based as there is no evidence for particular efficacy of provider-based heat treatments.
   Indications for Discontinuation – Intolerance, increased pain, or development of a burn or other adverse event.
   Benefits – Potential modest reduction in LBP. Self-efficacy, although relying on a passive modality.
   Harms – Heat injuries. Time may be devoted to passive modality instead of active exercises.

   > *Strength of Evidence* – **Recommended, Evidence (C)**
   > *Level of Confidence* – Low

2. *Recommendation: Application of Heat Therapy by a Health Care Provider for Chronic Low Back Pain*
Application of heat (such as infrared, moist heat, whirlpool) by a health care provider is not recommended for chronic low back pain as the patient can perform this application independently.

   > *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
   > *Level of Confidence* – Moderate

Rationale for Recommendations
Heat therapy in the form of a commercial heat wrap is studied in a few trials.(834, 1411-1414) Caution should be taken in interpreting these heat wrap studies as their design was suboptimal to determine true efficacy particularly compared with standard care. For example, a low dose of ibuprofen (1,200mg a day) was used as one of the control arms, yet detailed data on efficacy of that arm are not reported. Another study used only education as the control, thus appearing to the patient to be doing nothing and biasing in favor of the heat wrap.(1415) Still, there appears to be some evidence of efficacy. Non-proprietary self-applications of heat therapies are not invasive, have low adverse effects provided excessive heat is not used, and may have no associated costs. Thus, heat therapy is recommended for management of LBP.

Evidence for the Use of Hot Packs, Heat Wraps, and Moist Heat
There are 8 moderate-quality RCTs (one with 2 reports) incorporated into this analysis.(834, 1410-1417)
There are 6 low-quality RCTs in Appendix 1.(702, 1418-1422)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: self-applied heat therapy, heat wrap, hot packs, moist heat, heating

pad, subacute low back pain, acute low back pain, chronic low back pain low back pain, clinical trial, randomized controlled trial, random, systematic review, population study, epidemiological study, and prospective cohort to find 1,775 articles. Of the 1,775 articles, we reviewed 0 articles and included 0 articles. We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: heat application by a health care provider, heat therapy, heat, infrared, moist heat, whirlpool, heat pack, low back pain and chronic low back pain to find 33,710 articles. Of the 33,710 articles, we reviewed 18 articles and included 18 articles.

DIATHERMY

Diathermy is a type of heat treatment that has been used clinically to heat tissue and has been used to treat low back pain.(1423) There are two forms of diathermy – short wave and microwave. (High-dose diathermy is also used to coagulate tissue.) Proponents of diathermy utilize it to treat a wide range of conditions as they believe it penetrates deeper than hot packs or heating pads and stimulates healing.

Recommendation: Diathermy for Treatment of Low Back Pain
Diathermy is not recommended for treatment of any low back pain-related condition.

> *Strength of Evidence* – **Not Recommended, Evidence (C)**
> *Level of Confidence* – Moderate

Rationale for Recommendation
Trials suggest a lack of efficacy of diathermy.(1315, 1371) Multiple other trials have utilized diathermy as a no-effect/low-effect control group or as part of a control group.(1315, 1360, 1371) It also has not been shown to be more effective than placebo diathermy. Diathermy has lack of efficacy, is not invasive, has low adverse effects and is of moderate cost. Therefore, diathermy is not recommended for treatment of LBP. No trial has assessed diathermy in patients with sciatica alone. However, one moderate-quality trial evaluated diathermy and included a comparison with sham diathermy with substantial numbers of patients that could be classified as having sciatica.(1315) No quality evidence of benefit for the treatment of acute, subacute or chronic LBP patients with pain in a lower extremity with diathermy is available. Among acute, subacute, and chronic sciatica patients, diathermy is not recommended.

Evidence for the Use of Diathermy
There are 6 moderate-quality RCTs (one with 4 reports) incorporated into this analysis.(663, 665, 852, 1315, 1360, 1371, 1424-1426) Two studies were primarily designed to evaluate the efficacy of manipulative therapies and utilized diathermy as a control group. There are 5 low-quality RCT in Appendix 1.(1427-1431)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: Diathermy, heat therapy, Electrical induced heat, low back pain, subacute low back pain, chronic low back pain radicular pain syndromes (including 'sciatica'), Spinal stenosis, spinal fractures, sacroiliitis, and spondylolisthesis, to find 68,489 articles. Of the 68,489 articles, we reviewed 14 articles, and included 13 articles (12 RCTs and 1 Review).

INFRARED THERAPY
Infrared is a heat treatment created by various devices producing electromagnetic radiation in the infrared spectrum.

Recommendation: Infrared Therapy for Treatment of Acute, Subacute, Chronic, Post-operative or Radicular Low Back Pain
There is no recommendation for or against the use of infrared therapy in the home for treatment of acute, subacute, chronic, radicular or post-operative low back pain.

> *Strength of Evidence* – **No Recommendation, Insufficient Evidence (I)**

Rationale for Recommendations
Infrared is of moderate cost, not invasive, and has little potential for adverse effects. It is more expensive than other alternatives such as heat and has not been shown to be superior to less expensive forms of heat therapy. There is limited evidence on which to base a recommendation and available information conflicts. Therefore, there is no recommendation regarding the use of infrared therapy for treatment of low back pain.

Evidence for the Use of Infrared Therapy
There are 5 moderate-quality RCTs incorporated into this analysis.(686, 1318, 1416, 1432, 1433) There are 2 low-quality RCT in Appendix 1.(1286, 1422)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar with no limits on publication dates. We used the following terms: infrared, near-infrared spectroscopies, spectroscopies, near-infrared, NIR spectroscopy, NIR spectroscopies, spectroscopies, NIR, spectrometry near-infrared, near-infrared spectrometries, subacute low back pain, and chronic low back pain. Of the 1,443 articles, we reviewed 1 article and included 1 articles. We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following terms: provider-based infrared therapy, and low back pain to find 35 articles. Of the 35 articles we reviewed one article and included one article.

ULTRASOUND
Ultrasound has been used for treatment of low back pain.(1283, 1434-1437) Ultrasound treatment is achieved using a wand or probe to administer ultrasound waves which are generated by a piezoelectric effect of crystals within the head of the instrument and result in a deep heat, with purported increases in tissue relaxation, improved blood flow, and scar tissue breakdown. Continuous ultrasound at 1.5 to 2 W/cm^2 is capable of heating lumbar periarticular tissue. "The higher intensity ultrasound resulted in greater and faster temperature increase."(1438) Ultrasound waves can be continuous or pulsed; the latter can reduce the heating effect and is commonly used for acute injuries to minimize edema. The head of the ultrasound instrument should be kept in constant motion to minimize discomfort and prevent tissue damage.

Therapeutic ultrasound has more than 60 years of clinical history. It has been frequently used for the treatment of pain, soft-tissue lesions, and a host of musculoskeletal disorders, although it is used more for upper extremity musculoskeletal disorders than for spine-related disorders.(1439)

Recommendation: Ultrasound for Treatment of Low Back Pain
There is no recommendation for or against the use of ultrasound for treatment of low back pain. In situations where deeper heating is desirable, a limited trial of ultrasound is reasonable for treatment of acute low back pain, but only if performed as an adjunct with exercise.

> *Strength of Evidence* – **No Recommendation, Insufficient Evidence (I)**

Rationale for Recommendation
There is one small study,(1440) but no large-size quality studies of ultrasound for the treatment of LBP. Most studies used ultrasound as either part of a group of interventions, as a control or as a sham treatment that also limits the ability to develop guidance. Ultrasound is not invasive, has few adverse effects, but is moderately costly. Therefore, there is no recommendation for or against its use in treatment of LBP.

Evidence for the Use of Ultrasound
There are 1 high-(1441) and 19 moderate-quality RCT incorporated into this analysis.(590, 594, 597, 603, 665, 691, 698, 702, 715, 723, 852, 1059, 1289, 1336, 1440, 1442-1445) There is 1 low-quality RCT in Appendix 1.(1381)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms Ultrasound therapy, sub-acute low back pain, chronic low back pain to find 73,183 articles. Of the 73,183 articles, we reviewed 6 articles and included 6 articles (5 RCTs and 1 review).

LOW-LEVEL LASER THERAPY
Low-level laser treatment usually involves laser energy that does not induce significant heating. It is theorized that a mechanism of action is through photoactivation of the oxidative chain.(1446)

Recommendation: Low-level Laser Therapy for Treatment of Low Back Pain
Low-level laser therapy is not recommended for treatment of low back pain.

> *Strength of Evidence* – **Not Recommended, Evidence (C)**
> *Level of Confidence* – Moderate

Rationale for Recommendation
There are different lasers and different treatment regimens. There are multiple trials available. Among the highest quality studies with successful randomization, most indicate a lack of efficacy.(1447-1451) One study suggests this is ineffective for either acute or chronic LBP.(1447) One of the positive studies appears to have significant problems with baseline differences, which seem likely to be significantly responsible for at least some of the subsequent differences found.(1448) Low-level laser therapy is not invasive, not likely to have significant adverse effects, but some of these intensive treatment regimens would be quite costly. Longer term evaluation, utilization of objective measures, and standardization of the treatment regimens is required prior to consideration of a recommendation for utilization in treatments for chronic LBP. There are alternative effective treatments that promote patient independence and autonomy.

Evidence for the Use of Low-level Laser Therapy
There are 3 high-(1448, 1449, 1451) and 5 moderate-quality RCTs(853, 1432, 1447, 1448, 1450, 1452) incorporated into this analysis. There are 2 low-quality RCTs in Appendix 1.(1453, 1454)

We searched PubMed, EBSCO, Google Scholar, and Cochrane Review with no limits on publication dates. The following search terms were used "(low level laser therapy) AND (chronic low back pain OR back pain)" to find 71,156 articles. Of those 71,156 articles, we reviewed 8 articles and included 7 articles (all RCTs).

ACUPUNCTURE

Acupuncture originated in China and is based in part on the theory that many diseases are manifestations of an imbalance between yin and yang as reflected by disruption of normal vital energy flow (qi) in specific locations, referred to as meridians.(1455-1463) Needling along one of the 361 classical acupuncture points on these meridians is believed to restore balance. This stimulation is classically done with thin, solid, metallic needles which are then manipulated (or turned) manually or stimulated electrically (electroacupuncture). In addition to needling, acupuncture frequently involves moxibustion and cupping. Besides traditional Chinese acupuncture, there are many other types of acupuncture that have arisen, including accessing non-traditional acupuncture points.(1464) Acupuncture has been used for treatment of low back pain.(646, 1270, 1434, 1464-1467)

1. *Recommendation: Acupuncture for Treatment of Acute, or Subacute, Radicular and Post-operative Low Back Pain*
Acupuncture is not recommended for treatment of acute, subacute, radicular, or post-operative low back pain.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Moderate

2. *Recommendation: Acupuncture for Treatment of Chronic to Severe Low Back Pain*
Acupuncture is recommended for select use in the treatment of chronic moderate to severe low back pain as an adjunct to more efficacious treatments.

Indications – Chronic LBP patients should have had NSAIDs and/or acetaminophen, strengthening and aerobic exercise instituted and have insufficient results. Acupuncture may be considered as a treatment for chronic LBP as a limited course during which time there are clear objective and functional goals to be achieved. Consideration is for time-limited use in patients with chronic LBP without underlying serious pathology as an adjunct to a conditioning program that has both graded aerobic exercise and strengthening exercises. Acupuncture is only recommended to assist in increasing functional activity levels more rapidly and the primary attention should remain on the conditioning program. In those not involved in a conditioning program, or who are non-compliant with graded increases in activity levels, this intervention is not recommended.

Frequency/Duration – Evidence does not support specific Chinese meridian approaches, as needling the affected area appears sufficient. Patterns used in quality studies ranging from weekly for a month to 20 appointments over 6 months. However, the norm is generally no more than 8 to 12 sessions. An initial trial of 5 to 6 appointments is recommended in combination with a conditioning program of aerobic and strengthening exercises. Future appointments should be tied to improvements in objective measures and would justify an additional 6 sessions, for a total of 12 sessions.

Indications for Discontinuation – Resolution, intolerance, or non-compliance, including non-compliance with aerobic and strengthening exercises.

Harms – Rare needling of deep tissue, such as artery, lung, etc. and resultant complications. Use of acupuncture may theoretically increase reliance on passive modality(ies) for chronic pain.

Benefits – Modest reduction in pain.

> *Strength of Evidence* – **Recommended, Evidence (C)**

Level of Confidence – Low

Rationale for Recommendations
Quality studies evaluating efficacy of acupuncture for treating chronic LBP, are largely positive, although they somewhat conflict. There is no quality evidence on acute or subacute LBP, radicular pain syndromes, post-operative or other LBP-related conditions. The mechanism(s) of action is (are) unclear. The possibility that acupuncture is not superior to other treatments cannot be eliminated. Studies generally fail to control for attention bias, and also suggest that needling in locations other than traditional acupuncture points and/or sham acupuncture treatments may provide equal benefit(1465, 1468, 1469) which leads to questions regarding whether it is the needling rather than the acupuncture per se that was of benefit. There are a lack of systematized acupuncture approaches. There also is no quality evidence for many other forms of acupuncture outside of traditional Chinese or the sham acupuncture (e.g., Japanese, French, scalp, hand, foot, auricular, etc.).

Acupuncture performed by skilled professionals is minimally invasive, has minimal adverse effects, and is moderately costly although it could be high cost with ongoing treatments. In some of the studies that demonstrated efficacy for patients with chronic LBP, longer lasting benefits were found beyond the treatment period. Despite significant reservations regarding its true mechanism of action, a limited course of acupuncture may be recommended for treatment of chronic LBP as an adjunct to a conditioning program. It is not recommended for other back-pain related conditions as there is no evidence of its efficacy and particularly for acute pain, it would not be expected to materially alter the natural history.

Evidence for the Use of Acupuncture
There are 10 high-(1465, 1468-1477) (one with 2 reports) and 25 moderate-quality(745, 833, 861, 1079, 1284, 1432, 1447, 1452, 1478-1495) RCTs (one with 2 reports) incorporated into this analysis. Trials enrolling only the elderly were not included.(1077, 1496-1498) There are 5 low-quality RCTs(1130, 1499-1502) and 1 other study(1503) in Appendix 1.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following terms: acupuncture, chronic low back pain, subacute low back pain, radicular pain, and sciatica to find 54,349 articles. Of the 52,349 articles we reviewed 32 articles and included 32 articles.

NEUROREFLEXOTHERAPY

Neuroreflexotherapy is an alternative treatment that was developed in Spain and involves implantation of numerous epidermal staples in "trigger" points in the back as well as burins (small metallic punches) in "referred tender points in the ear"(1504) at depths up to 2mm.(1505, 1506) In contrast with acupuncture, the sites are chosen by dermatomal innervation. Implantation does not require anesthesia and staples remain in place for up to 90 days. Significant reductions in LBP have been reported at 1 year in uncontrolled studies.(1507)

1. *Recommendation: Neuroreflexotherapy for Treatment of Moderate to Severe Chronic Low Back Pain*
   Neuroreflexotherapy is recommended for treatment of moderate to severe chronic low back pain in patients who have failed management with NSAIDs, progressive aerobic exercise program or other exercises, or manipulation.
   Harms – Irritant or allergic reactions to the metals.

Benefits – Modest reductions in low back pain.

    Strength of Evidence – **Recommended, Evidence (C)**
    Level of Confidence – Moderate

2. *Recommendation: Neuroreflexotherapy for Treatment of Acute or Subacute Low Back Pain or Radicular Pain*
   There is no recommendation for or against the use of neuroreflexotherapy for treatment of acute or subacute low back pain or radicular pain syndromes.

       Strength of Evidence – **No Recommendation, Insufficient Evidence (I)**

Rationale for Recommendations
Neuroreflexotherapy may be modestly efficacious for the treatment of chronic LBP.(1504, 1508) It appears to have some analogy to treatment with non-traditional acupuncture and superficial needling. Reports are mostly foreign language and this treatment is currently largely unavailable in the U.S. There are reports of relatively few adverse effects. Thus, neuroreflexotherapy is minimally invasive, has some adverse effects, and is moderate cost. It needs to be replicated by other research groups in other settings. It has not been shown to be efficacious for the treatment of acute or subacute LBP or radicular pain syndromes. There are other treatments that have been shown to be efficacious.

Evidence for the Use of Neuroreflexotherapy
There is 1 high-(1504) and 1 moderate-quality(1508) RCT incorporated into this analysis.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar with no limits on publication dates. The following search terms were used: Neuroreflexotherapy AND (sub-acute low back pain OR Chronic low back pain)" to find 218 articles. Of those, we reviewed 3 articles and included 2 articles (2 RCT, zero reviews).

Electrical Therapies
There are multiple forms of electrical therapies used to treat musculoskeletal pain. These include interferential therapy, transcutaneous electrical stimulation (TENS), neuromuscular electrical stimulation (NMES), percutaneous electrical nerve stimulation (PENS), microcurrent electrical stimulation, H-wave® Device Stimulation, and high voltage galvanic therapy. The mechanism(s) of action, if any, are unclear.

INTERFERENTIAL THERAPY
Interferential therapy (IFT) is a form of electrical stimulation using amplitude modulation of two out-of-phase medium-frequency currents to produce a low-frequency current that has been used to treat low back pain.(1434, 1509) This procedure is similar to TENS and differs by having less impedance in the tissues and is reportedly more comfortable than traditional TENS treatment. IFT is commonly used in the U.K.

Recommendation: Interferential Therapy for Treatment of Acute, Subacute or Chronic Low Back Pain, Chronic Radicular Pain Syndromes or Other Back Disorders
There is no recommendation for or against the use of interferential therapy for treatment of acute, subacute or chronic low back pain, chronic radicular pain syndromes, or other back-related disorders.

    Strength of Evidence – **No Recommendation, Insufficient Evidence (I)**

Rationale for Recommendation

Evidence is conflicting regarding whether interferential therapy produces any benefits in comparison with no treatment among acute, subacute and chronic LBP patients. There also is no quality evidence that interferential therapy produces any incremental benefits when added to a treatment regimen. Interferential therapy is non-invasive, does not have significant adverse effects, but is moderately costly.

Evidence for the Use of Interferential Therapy
There are 1 high-(1510) and 7 moderate-quality RCTs incorporated into this analysis.(1212, 1282, 1511-1515)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar with limits on dates for 2011-2012. We used the following terms: interferential therapy, subacute low back pain, chronic low back pain, radicular pain syndromes (including 'sciatica'), spinal stenosis, spinal fractures, sacroiliitis, spondylolisthesis, clinical trial or randomized controlled trial, systematic reviews or reviews to find 106 articles. Of the 106 articles we reviewed 10 articles and included 8 RCTs (2 review articles).

TRANSCUTANEOUS ELECTRICAL NEUROSTIMULATION (TENS) AND NEUROMUSCULAR ELECTRICAL STIMULATION (NMES)
Transcutaneous electrical nerve stimulation (TENS) has been used to treat LBP.(588, 1434, 1516-1522) TENS is a modality to control pain through electrical stimulation delivered by pads placed on the surface of the skin for the treatment of many painful conditions including both non-inflammatory and inflammatory disorders.(1510, 1523-1526) Neuromuscular electrical stimulation is somewhat similar, but considered a stronger device that causes muscular contraction and thus purportedly re-educates muscles.(1527)

1. *Recommendation: TENS and NMES for Treatment of Acute or Subacute Low Back Pain or Acute Radicular Pain Syndromes*
   TENS and NMES are not recommended for treatment of acute or subacute low back pain or acute radicular pain syndromes.

   Strength of Evidence – **Not Recommended, Insufficient Evidence (I)**
   Level of Confidence – Moderate

2. *Recommendation: TENS for Treatment of Chronic Low Back Pain or Chronic Radicular Pain Syndrome*
   TENS is recommended for select use in treatment of chronic low back pain or chronic radicular pain syndrome as an adjunct for more efficacious treatments.

   Indications – Chronic LBP insufficiently managed with prior NSAIDs, aerobic exercise, and strengthening exercise with which compliance is documented. Many providers would also require failure with TCA and/or SNRI anti-depressants. TENS (single or dual channel) may be recommended as treatment for chronic LBP when clear objective and functional goals are being achieved which includes objective functional improvements such as return to work, increased exercise tolerance and reductions in medication use. TENS is used as adjunctive treatment in chronic pain conditions to support graded aerobic exercise and strengthening exercises. For patients who are not involved in a conditioning program or who are non-compliant with graded increases in activity levels, this intervention is not recommended. There is no quality evidence that more complex TENS units beyond the single or dual channel models are more efficacious, thus those models are not recommended.

TENS units should be trialed prior to purchase to demonstrate efficacy and increase function. Two or 3 visits with a therapist may be necessary to instruct the patient in the application and use of the unit and to determine the most effective electrode placement and current parameters. If the patient has a TENS unit, then electrical stimulation for pain management should not be performed as part of any ongoing rehabilitative program. Either a low-intensity prolonged (30 plus minutes) stimulation through an active electrode over the painful area or a higher intensity over the painful area for 15 to 30 minutes (commonly referred to as hyperstimulation analgesia) are the two most common treatment protocols.(1528) High-frequency stimulation is generally 80 to 200 Hz, whereas low-frequency is generally 4 to 8 Hz.

Indications for Discontinuation – Resolution, intolerance, or non-compliance including non-compliance with aerobic and strengthening exercises.
Benefits – Modest pain reduction. Potential improved exercise and exertion tolerances.
Harms – Minor skin irritation.

> *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

3. Recommendation: NMES for Treatment of Chronic Low Back Pain or Chronic Radicular Pain Syndrome
> **There is no recommendation for or against the use of NMES for chronic low back pain or chronic radicular pain syndrome as an adjunct for more efficacious treatments.**

> *Strength of Evidence* – **No Recommendation, Insufficient Evidence (I)**
> *Level of Confidence* – Low

Rationale for Recommendations
There are quality studies evaluating the utility of TENS, particularly for chronic LBP. There is insufficient evidence on NMES and thus no recommendations regarding this treatment. There was no quality study identified evaluating acute LBP, and one with a minority of patients having subacute LBP.(1529) There are studies evaluating TENS for sciatica patients. In reviewing these studies, there is not clear evidence of benefit. Of the high-quality studies for chronic LBP, 3(1510, 1530, 1531) suggest benefit and 2(1040, 1532) suggest no benefit. While the highest quality study(1531) did find benefit, not all of the higher quality trials did, thus the evidence conflicts. There is no study finding strong evidence of major benefits, thus any benefit appears likely to be modest.

TENS is not invasive, has no significant adverse effects, and is moderately costly. It has no clear benefits and is not recommended for treatment of acute, subacute, or chronic LBP or radicular pain syndromes. In rare cases where more efficacious strategies have been exhausted, it may be reasonable to prescribe TENS for select subacute LBP patients, but only as an adjunct to a conditioning program.

Evidence for the Use of Transcutaneous Electrical Nerve Stimulation (TENS) and Neuromuscular Electrical Stimulation (NMES)
There are 5 high-(1040, 1510, 1530-1532) and 25 moderate-quality RCTs or crossover trials(1281, 1330, 1480, 1484, 1487, 1488, 1496, 1529, 1533-1549) incorporated into this analysis. There are 7 low-quality RCTs in Appendix 1.(1500, 1550-1555)

We searched PubMed, EBSCO, Cochrane Review, Google Scholar without limits on publication dates. We used the following search terms: Transcutaneous Electrical Nerve Stimulation, TENS, Electrical Stimulation, subacute low back pain, chronic low back pain, radicular pain syndromes, sciatica, spinal

stenosis, spinal fractures, sacroiliitis, and spondylolisthesis to find 11,703 articles. Of the 11,703 articles, we reviewed 58 articles and included 40 articles (40 RCTs and 9 summaries).

PERCUTANEOUS ELECTRICAL NERVE STIMULATION (PENS)

Percutaneous electrical nerve stimulation (PENS) involves inserting needles to a depth of 1 to 4 centimeters around a nerve serving a painful area. The techniques described in the studies differ.

Recommendation: PENS for Treatment of Acute, Subacute, or Chronic Low Back Pain or Radicular Pain Syndromes
PENS is not recommended for treatment of acute, subacute, or chronic low back pain or radicular pain syndromes.

> *Strength of Evidence –* **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence –* Moderate

Rationale for Recommendation
PENS has been evaluated in small-scale, short-term studies, but there are no high-quality studies.(1529, 1556-1558) The two highest quality studies suggest no efficacy.(1529, 1559) Four of the RCTs were reported by one group (*JAMA* reported a significant potential financial conflict of interest for this group's study following publication of the article). All of the studies that showed improvement over placebo or sham treatment failed to show any improvement over baseline in the placebo treated group, which is unusual. Most studies of chronic LBP report a 2-week outcome for treatment with PENS, which generally is insufficient for chronic pain patients. The one study that evaluated duration of improvement after PENS treatment was stopped and found no effect 4 weeks after treatment ceased. No study documented a significant improvement in function. Hseih and Lee did not find the use of one-time PENS to be superior to a combination of diclofenac, mephenoxalone, and an antacid.(1529) There were no studies that compared PENS to heat therapies. Although Ghoname, et al., found PENS to be superior to exercise, the exercise consisted of simple spinal flexion and extension while seated, which would appear insufficient.(1556)

PENS has not been convincingly demonstrated to be superior to other less expensive and/or proven interventions. Most PENS studies have been conducted in chronic non-radicular back pain patients. In acute LBP, the natural history is to resolve, and PENS has not been shown to accelerate that natural healing process. Short-term pain relief can be achieved more easily with analgesics. PENS is minimally invasive and no significant adverse effects have been reported (although most articles failed to include a section on complications). However, it is high cost.

Evidence for the Use of PENS

A comprehensive literature search was conducted using PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates using the following terms: percutaneous electrical nerve field stimulat*, percutaneious electrical nerve stimulat*, PENS, PNRS, NSS2 Bridge, NSS1 NeuroStim; Back, low back pain, Random* to find 42,805 articles. Of the 42,805 articles, we reviewed 123 articles and included 19 articles (18 randomized controlled trials and 1 systematic review).

MICROCURRENT ELECTRICAL STIMULATION

Microcurrent electrical stimulation is a type of electrotherapy. Proponents believe that it will relieve pain and contribute to healing while using lower currents than are used in TENS or interferential and galvanic stimulation. If effective, this modality does not work through distraction, as the current is too low to be perceived.

Recommendation: Microcurrent Electrical Stimulation for Treatment of Acute, Subacute, or Chronic Low Back Pain or Radicular Pain Syndrome
Microcurrent electrical stimulation is not recommended for treatment of acute, subacute, or chronic low back pain or for radicular pain syndrome.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

Rationale for Recommendation
One small study has suggested a lack of efficacy.(1560) Microcurrent electrical stimulation is not recommended as other modalities have been shown to be effective in the treatment of acute, subacute, and chronic LBP. Microcurrent electrical stimulation is not invasive, has little potential for adverse effects, and is moderately costly.

Evidence for the Use of Microcurrent Electrical Stimulation
There is 1 moderate-quality study incorporated into this analysis.(1560)

We searched PubMed, EBSCO, Google Scholar, and Cochrane Review with no limits on publication dates. The following search terms were used:"Micro current electrical stimulation, sub-acute low back pain, chronic low back pain, radicular pain syndromes including sciatica" to find 869 articles. Of those 869 articles, we reviewed one article and included one article.

H-WAVE® DEVICE STIMULATION
Proponents believe these electrical currents stimulate healing.

Recommendation: H-Wave® Device Stimulation for Treatment of Low Back Pain and Radicular Pain Syndromes
There is no recommendation for or against H-Wave® Device stimulation for treatment of acute, subacute, or chronic low back pain or radicular pain syndromes.

> *Strength of Evidence* – **No Recommendation, Insufficient Evidence (I)**

Rationale for Recommendation
Other modalities have been shown to be effective in the treatment of acute, subacute and chronic LBP and radicular pain syndromes. H-Wave® Device stimulation is more costly than other self-administered electrical stimulation modalities. It is not invasive and has low adverse effects, but is moderate cost and becomes high cost after 6 weeks.

Evidence for the Use of H-Wave® Device Stimulation
There are no quality studies evaluating H-Wave® Device stimulation for the treatment of acute, subacute, or chronic LBP or radicular pain syndromes.
We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following terms: H-Wave® Device stimulation, subacute low back pain, chronic low back

pain, and radicular pain syndromes (including 'sciatica') to find 154 articles. Of the 154 articles we reviewed zero articles and included zero articles.

HIGH-VOLTAGE GALVANIC THERAPY
High-voltage galvanic is an electrical therapy.

Recommendation: High-voltage Galvanic Therapy for Treatment of Low Back Pain
There is no recommendation for or against high-voltage galvanic therapy for treatment of acute, subacute, or chronic low back pain or for radicular pain syndromes or other back-related conditions.

> *Strength of Evidence –* **No Recommendation, Insufficient Evidence (I)**

Rationale for Recommendation
High-voltage galvanic is not shown to be efficacious for the treatment of acute, subacute, or chronic LBP or radicular pain syndromes or other back-related problems. It is not invasive, but is not low cost. There are other interventions shown to be efficacious.

Evidence for the Use of High-voltage Galvanic
There are no quality studies evaluating the use of high-voltage galvanic for the treatment of LBP.

We search PubMed, EBSCO, Cochrane Review, and Google Scholar with no limits on publication dates. The following search terms were used "High-voltage galvanic) AND (sub-acute low back pain OR radicular pain syndromes OR spinal stenosis OR spinal fractures OR sacroiliitis)" to find 27 articles. Of those 27 articles, we reviewed zero articles and included zero articles.

INVERSION THERAPY
Inversion has been used for treatment of patients with herniated discs(1324, 1561) and low back pain.(1562)

Recommendation: Inversion Therapy for Treatment of Radicular Pain or Low Back Pain
There is no recommendation for or against the use of inversion therapy for treatment of either radicular pain or low back pain.

> *Strength of Evidence –* **No Recommendation, Insufficient Evidence (I)**
> *Level of Confidence –* Low

Rationale for Recommendation
The overall quality of the literature base for inversion therapy is poor. Two trials have attempted to address treatment in patients with radiculopathy, with one suggesting lower surgical rates in the inversion therapy group,(1561) yet many outcome data may be confounded. Most results for treatment of LBP were also negative in another study.(1562) Trial inclusion criteria (age, body mass index) would restrict most patients from this treatment.(1561) Inversion therapy is not invasive, has moderate adverse effects especially in older individuals but the evidence base is too weak to support an evidence-based recommendation for or against treatment. There are many other effective treatments.

Evidence for the Use of Inversion Therapy
There is 1 moderate-quality RCT incorporated into this analysis.(1561) There are 2 low-quality RCTs in Appendix 1.(1324, 1562)

We searched PubMed, CINAHL, Cochrane Library and Google Scholar without date limits using the following terms; Inversion table, inversion tables, inversion therapy, inversion therapy table, inversion therapies, inversion traction therapy, inversion traction, subacute low back pain, chronic low back pain, low back pain, controlled clinical trial, controlled trials, randomized controlled trial, randomized controlled trials, random allocation, random, randomized, randomization, randomly; systematic, systematic review, retrospective, and prospective studies. We found and reviewed 10 articles in PubMed, 3 in CINAHL, 7 in Cochrane Library, and 2,100 in Google Scholar. We considered for inclusion 1 from PubMed, 0 from CINAHL, 1 from Cochrane Library, 2 from Google Scholar and 0 from other sources. Of the 4 articles considered for inclusion, 3 randomized trials and 1 systematic studies met the inclusion criteria.

Injection Therapies………………………………………………………………

There are several types of injections included in this section. These include epidural injections (caudal, interlaminar and transforaminal), intradiscal injections, chemonucleolysis, tender or "trigger point" injections, facet joint injections, sacroiliac joint injections, intrathecal drugs, ligamentous injections (prolotherapy), and botulinum injections.

LUMBAR EPIDURAL INJECTIONS

Epidural glucocorticosteroid injections deliver the steroid close to the herniated disc or area of spinal stenosis.(1084, 1088-1090, 1092, 1093, 1102, 1104-1106, 1563-1581) The three approaches most commonly used are caudal, interlaminar, and transforaminal.(1582-1585) The technical performance including precise placement of these injections is reportedly related to the efficacy.(1586) Interlaminar epidural injections are the least technical and place the steroid immediately adjacent to the dural sac in the posterior spinal column. Fluoroscopic guidance improves the placement accuracy of injection, as blind targeting has been shown to be 77% accurate.(1587) Injections have also been performed after epiduroscopy.(1588) Transforaminal injections most closely target the herniated disc and neurological impingement with the least volume of agent,(1582, 1589) but are technically more difficult and fluoroscopic or CT guidance is usually used.(1590) Transforaminal injections also necessitate better diagnostic precision to ensure proximity to the affected level.(1585) A technique has also been described using electrical stimulation to assist with nerve root identification.(1591) As these injections are most frequently performed as a combination of a glucocorticoid with an anesthetic, they are considered both diagnostic and therapeutic.(1592)

1. *Recommendation: Epidural Glucocorticosteroid Injections for Treatment of Acute or Subacute Radicular Pain*
   An epidural glucocorticosteroid injection is recommended as an option for treatment of acute or subacute radicular pain syndromes. Its purpose is to provide a few weeks of partial pain relief while awaiting spontaneous improvement and remaining as active as practical. An epidural steroid injection may cause short-term improvement(1575, 1593-1597) which may assist in successfully accruing sufficient time to ascertain if non-operative care will succeed. An "option" means there should be no requirement that a patient receive and fail treatment with epidural glucocorticosteroids, especially repeated injections, prior to discectomy.

   Indications –A radicular pain syndrome consistent with herniation and neurological impingement of one nerve root (e.g.,, L5 or S1). Should also have physical signs such as decreased dermatomal sensation, decreased reflex in the expected distribution (e.g., S1), myotomal motor deficit (i.e., in the same nerve distribution), and/or straight leg raise. Symptoms should have lasted at least 3 weeks while having been treated with

NSAID(s) unless contraindicated or not tolerated, and without evidence of trending towards spontaneous resolution.

Frequency/Duration – Each injection's results should be evaluated with objective improvement before scheduling an additional injection, such as improved functional ability or reduction in opioids requirements. Medications most often used in the RCTs were triamcinolone and methylprednisolone combined with an anesthetic (most often bupivacaine). There are no head-to-head comparisons of different medications to ascertain the optimum medication(s) and/or dose(s).

Indications for Discontinuation – A second epidural steroid injection is not recommended if following the first injection there has been sufficient resolution of the symptoms, particularly leg symptoms, or a decrease in symptoms to a tolerable level. If there has been no response to a first epidural injection, there would be no recommendation for a second injection. In patients who respond with a pharmacologically appropriate 3 to 6 weeks of temporary, partial relief of leg pain, but who then have a worsening of leg pain and function, and who are not (yet) interested in surgical discectomy, a repeat epidural steroid injection is an option. Generally, there are not benefits beyond 3 injections for a given episode of radicular pain. Patients requesting a fourth injection should be counseled for discectomy or considered to have chronic radicular symptoms for which epidural steroids are not recommended.

Benefits – Short to intermediate term reduction in pain. Theoretical, though likely infrequent avoidance of surgery if sufficient pain reduction occurs.

Harms – Rare complications of paralysis, infections.

> *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Moderate

2. *Recommendation: Epidural Glucocorticosteroid Injections for Treatment of Acute Flare-ups of Spinal Stenosis*
   Epidural glucocorticosteroid injections are moderately not recommended for treatment of spinal stenosis.(1598) (Friedly 14)

   > *Strength of Evidence* – **Moderately Not Recommended, Evidence (B)**
   > *Level of Confidence* – Moderate

3. *Recommendation: Epidural Glucocorticosteroid Injections for Treatment of Acute, Subacute, or Chronic Low Back Pain without Radicular Symptoms*
   Epidural glucocorticosteroid injections are not recommended for treatment of acute, subacute, or chronic low back pain in the absence of significant radicular symptoms. They are also not recommended as first- or second-line treatment in individuals with LBP symptoms that predominate over leg pain. They are not recommended as treatment for any chronic problem.

   > *Strength of Evidence* – **Not Recommended, Evidence (C)**
   > *Level of Confidence* – High

Rationale for Recommendations
The natural history of sciatica and disc herniations is natural resolution for a majority of patients.(1599) Glucocorticosteroid injections have been evaluated in moderate to high-quality studies. Most of the 7 high-quality studies that included acute to subacute pain patients with followups over 3 to 6 weeks demonstrated short-term reductions in short-term leg and back pain ratings for those with herniated intervertebral discs. Data also suggest that benefits disappear by approximately 6 weeks with no long-

term benefits. Most of the evidence suggests no change in function or the need for surgery. Importantly, there is good evidence across numerous studies that the natural history of symptoms from a herniated disc trend towards resolution over time. Thus, the purpose of these injections for acute radicular pain syndromes is perhaps best stated as "buying time" through a period of natural recovery that decreases the patient's pain while herniated disc shrinkage or resorption occurs.

The American Academy of Neurology's guideline has recommended against routine use of these injections.(1600) Systematic reviews have arrived at contradictory conclusions. Those with the highest standards for evidence have generally not found glucocorticosteroid injections to be a cost effective treatment. Most of the RCTs have studied blind interlaminar epidural injection. Fluoroscopic guidance may improve results; however, that theory has not been well tested. Evidence of efficacy appears relatively consistent in the higher quality studies, however, as all suggest short term benefits and no long term benefits, the assessment of the value of that time with incremental benefit appears critical and there is no clear method to assign a value.

Complications are infrequent, but in rare cases may be serious(1086, 1101, 1103, 1107, 1566, 1601-1606) including infection (meningitis, epidural abscess, etc.) and hemorrhage related to penetration of an anatomical variant artery. A resulting epidural hematoma may compress the nerve or spinal cord(1582) and generally requires emergency surgery. Suppression of the pituitary-adrenal axis does occur.(1607) Uncontrolled data suggest psychological factors may be associated with treatment failure,(1608) but that is not a universal finding. There are radiation exposure concerns for fluoroscope operators and patients that should be addressed(1092) and longer term potential risks of osteoporotic fractures.(1094)

Since the relief from epidural steroid injections is brief, and since by definition chronic non-specific back pain and chronic radicular pain with or without prior back surgery are chronic problems, epidural steroid injections are not recommended as a transient treatment for these long-term problems. There also is no quality evidence that accomplishing these injections earlier in the course of the syndrome results in any improvement in the condition. On the contrary, there is some evidence inferred suggesting it may make no difference.

One high-quality trial found no or minimal short-term benefit of epidural glucocorticosteroid injection for treatment of spinal stenosis.(1598) Two moderate-quality RCTs similarly suggested only minor short-term symptom reduction of spinal stenosis.(1609, 1610) No long-term benefits were reported in another trial (2410). Therefore, epidural glucocorticosteroid injections are not recommended for treatment of spinal stenosis.

Technique may be important as well as the anatomical approach chosen.(1586) However, there is insufficient evidence presently to recommend one technique over the other for an initial approach (caudal vs. interlaminar vs. transforaminal), other than to note that there is evidence that endoscopy for steroid injection has not been shown to be beneficial.(1611) Although it is suspected that fluoroscopic or CT guidance for these injections is helpful, there is not sufficient evidence for guidance on that topic. Predictive factors of unresponsive patients include greater number of medications used for pain, greater number of past treatments for pain, walking less, and coughing, household chores, sitting, unemployment due to pain,(1574, 1612) as well as potential sex differences.(1613)

Most studies assessed only one injection, although three studies used a series of *up to* 3 injections over 6 weeks,(1593, 1594, 1597) and there is no quality study that performed 3 injections without an assessment after each injection to determine whether an additional injection was appropriate and recommended. Thus, there is no quality evidence to either support or require a series of 3 injections. There is no evidence that there is a limit of 3 in a year or lifetime, although if there is no clear benefit, then repeated injections are not recommended.

Current practice in the U.S. is generally to obtain an MRI or CT prior to an epidural injection. Yet, at least four of the trials solely relied on the clinical examination to address the level targeted with subsequent epidural glucocorticosteroid injection, and thus there is some evidence that imaging may not be necessary.(1110, 1593, 1594, 1614) Additional studies may be needed to determine whether imaging is required or not, as if unnecessary, it can be eliminated and markedly reduce costs.

Epidural glucocorticoid injections are invasive, have some adverse effects,(1594) and are costly. The number needed to treat (NNT) to achieve partial pain relief at 3 weeks was 11.4, but there was no benefit from weeks 6 to 52.(1594) These injections are an option in acute radiculopathy, but as a second-line treatment after prior treatment with NSAIDs, possibly a short course of an oral corticosteroid and a suggested waiting period of at least 3 weeks.

Evidence for the Use of Lumbar Epidural Injections

We searched PubMed, EBSCO, Google Scholar, and Cochrane Review with no limits on publication dates and then an updated search was conducted using PubMed for publications between 1/1/2013 and 11/15/2017. We used the following search terms: acute low back pain, subacute low back pain, chronic low back pain, radicular pain syndrome, sciatica, spinal stenosis, Epidural Glucocorticosteroid Injection, Dexamethasone, Glucocorticosteroid injection, Methylprednisolone, Triamcinolone, Steroid injection, Corticosteroid injection, betamethasone, Peridural Injection, Extradural Injection, Epidural Injection, clinical trial, randomized controlled trial, random, systematic review, review, population study, epidemiological study, and prospective cohort as well as reviewed references to find 44,715 articles. Of the 44.691 articles, we reviewed 190 articles and included 59 articles (59 randomized controlled trials and 0 systematic reviews).

INTRADISCAL STEROIDS
Injections of glucocorticoids into the intervertebral disc, often performed under fluoroscopy or other imaging modalities, are classified as "intradiscal steroids."(1615-1617) The theory is that these injections help to reduce the degree to which the disc is both herniated and/or producing an inflammatory response. Proponents believe that these injections are better directed at the target tissue. The weakness in the theory is that the target tissue may be that which is impinged by the herniated nucleus pulposus material.

1. *Recommendation: Intradiscal Steroid Injections for Treatment of Acute Low Back Pain*
   Intradiscal steroid injections are not recommended for treatment of acute low back pain.
       Strength of Evidence – **Not Recommended, Insufficient Evidence (I)**
       Level of Confidence – Moderate

2. *Recommendation: Intradiscal Steroid Injections for Treatment of Subacute or Chronic Low Back Pain*
   Intradiscal steroid injections are not recommended for treatment of subacute or chronic low back pain.

Strength of Evidence – **Not Recommended, Evidence (C)**
Level of Confidence – Moderate

Rationale for Recommendations
For radicular pain and herniated discs, one study is available but it did not include a placebo group, thus there is no evidence regarding efficacy for intradiscal injection.(1618) For chronic LBP, two moderate-quality trials suggest lack of efficacy(1619, 1620) and one suggests efficacy.(1621) Thus, the data somewhat conflict and there is also no pattern of consistent results in the highest quality trial. There is no clear evidence that these injections improve on the natural history of acute LBP. Compared to epidural injections or compared to no treatment, benefits have not been demonstrated. These injections are invasive, have adverse effects and are moderate to high cost.

Evidence for the Use of Intradiscal Steroids
There are 5 moderate-quality(1618-1622) RCTs incorporated into this analysis.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: Intradiscal steroid injections, Epidural steroid injections, sub-acute, chronic, low, back and pain to find 2,675 articles. Of the articles, 2,675 we reviewed eight articles and included seven articles.

CLONIDINE
Clonidine is an α-agonist most typically used as an anti-hypertensive. As $α_2$ adrenoceptor agonists may affect nociceptive processing,(1623) clonidine has been used to treat CRPS (see Chronic Pain Guideline). Adverse effects include hypotension, dry mouth, drowsiness, and dizziness. Clonidine in combination with monoamine oxidase inhibitors or beta blockers has a complex effect on neuronal catecholamines and may precipitate a hypertensive crisis on discontinuance.

1. *Recommendation: Epidural Clonidine for Treatment of Radicular Pain*
    Epidural clonidine is not recommended for treatment of radicular pain.

    Strength of Evidence – **Not Recommended, Evidence (C)**
    Level of Confidence – Moderate

2. *Recommendation: Epidural Clonidine for Treatment of Chronic Low Back Pain*
    There is no recommendation for or against the use of epidural clonidine for treatment of chronic low back pain.

    Strength of Evidence – **No Recommendation, Insufficient Evidence (I)**
    Level of Confidence – Moderate

3. *Recommendation: Intramuscular Clonidine for Treatment of Pyriformis Syndrome*
    There is no recommendation for or against the use of intramuscular clonidine for treatment of pyriformis syndrome.
    Strength of Evidence – **No Recommendation, Insufficient Evidence (I)**
    Level of Confidence – Low

4. *Recommendation: Intramuscular Clonidine for Treatment of Other Low Back Conditions*
    There is no recommendation for or against the use of intramuscular clonidine for treatment of other low back conditions.

Strength of Evidence – **No Recommendation, Insufficient Evidence (I)**
Level of Confidence – Low

Rationale for Recommendations
There is evidence epidural clonidine is inferior to epidural steroid injection for radicular pain.(1624) It is also invasive, has adverse effects and thus, epidural clonidine is not recommended for treatment of radicular pain. A trial of intramuscular clonidine plus bupivacaine superior to bupivacaine plus saline for pyriformis syndrome.(1625) However, prior to recommendation intramuscular injections for pyriformis syndrome need to be independently replicated.(1625)

Evidence for the Use of Clonidine
There are 1 high-(1625) and 1 moderate-quality(1624) RCTs evaluating the use of clonidine for chronic low back pain. There is 1 other study in Appendix 1.(1626)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: clonidine, acute low back pain, subacute low back pain, radicular pain syndrome, sciatica, spinal stenosis, and sacroiliitis to find 1,493 articles. Of the 1,493 articles, we reviewed 4 articles and included four articles.

CHEMONUCLEOLYSIS (CHYMOPAPAIN AND COLLAGENASE)
Chymopapain is an enzyme that has long been used to treat herniated discs.(1627-1629) While collagenase has been utilized more recently,(1630) both enzymes are injected into the disc. **Chymopapain is no longer available in the U.S. due to reimbursement problems.** Caution is warranted in those increasingly limited numbers of countries that allow this procedure.(1631)

TENDER AND TRIGGER POINT INJECTIONS
Trigger points are a physical examination finding that is interpreted as abnormal. This finding involves an examiner's opinion that the degree of tenderness particularly on palpating a muscle is abnormally great.(1632) Although controversial, perhaps the most widely accepted criteria for tenderness are the American College of Rheumatology's former criteria for fibromyalgia, and involve an acknowledgement that there is "pain" on 4kg of palpation pressure at a given tender point to diagnose that condition,(1633) but for purposes of tender or trigger points those locations are not necessary. Ideally, examiners seek a palpable "knot" or nodule of muscle tissue and palpating this nodule both reproduces the patient's symptoms and produces a distal radiation of symptoms, such as tingling in the upper extremity denoting a trigger point. However, most patients merely have tender points without radiation of symptoms. In common usage, the terms "trigger" and "tender" are used interchangeably. Studies have attempted to address both findings, although research studies' descriptions of methods have not been particularly clear on distinguishing one condition from another.

Tender and trigger points are primarily diagnosed in the periscapular area, although some are found in the lumbosacral area. These points are integrally involved in "myofascial pain syndrome" and "fibromyalgia." Most practitioners believe these are two distinct entities, while others believe that these are related conditions on a continuum of the same basic disorder.(1632) Robust basic epidemiological studies are lacking. It appears that many people are tender to palpation thus what differentiates normal from abnormal individuals is unclear. There are multiple weaknesses in these theories, including a lack of identification of how common these findings are in normal people, the lack of purely objective

findings, subjectivity involved on the part of the examiner, and weaknesses in the pathophysiological theories.

These injections into muscle "knots" typically consist of an anesthetic with or without glucocorticoid.(1632, 1634) The goals of injection are generally thought to involve anesthesia, anti-inflammatory medication, and allowing deep-tissue massage of the area to work out the muscle knot.

1. *Recommendation: Trigger and/or Tender Point Injections for Treatment of Acute Low Back Pain*
Trigger and/or tender point injections are not recommended for treatment of acute low back pain.

>   *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
>   *Level of Confidence* – Moderate

2. *Recommendation: Trigger and/or Tender Point Injections for Treatment of Subacute or Chronic Low Back Pain*
Trigger and/or tender point injections may be recommended as a reasonable second or tertiary option for treatment of subacute or chronic low back pain that is not resolving. These injections are recommended to consist either solely of a topical anesthetic (e.g., bupivacaine) or dry needling without an injection. Repeated injections should be linked to subjective *and* objective improvements. The use of therapeutic injections without participation in an active therapy program or in the context of maintaining employment is not recommended. An alternative option to these injections is acupuncture.

Indications – Subacute or chronic LBP that is not resolving with more conservative means (e.g., NSAID, progressive aerobic exercises, other exercises).

Frequency/Duration – Allow at least 3 to 4 weeks between injections. If results are not satisfactory after first set of injections, a second set is reasonable. If there are not subjective *and* objective improvements at that point, further injections are not recommended.

Indications for Discontinuation – Resolution, intolerance, or completing 2 sets of injections without materially affecting the condition.
Benefits – Modest reduction in pain and potential to speed resolution.
Harms – Hematoma, medicalization of otherwise benign conditions.

>   *Strength of Evidence* – **Recommended, Evidence (C)**
>   *Level of Confidence* –Low

3. *Recommendation: Trigger Point Injections Using Glucocorticosteroids*
Glucocorticosteroids are not recommended for use in trigger point injections.(1635)

>   *Strength of Evidence* – **Not Recommended, Evidence (C)**
>   *Level of Confidence* –Moderate

Rationale for Recommendations
The literature on this subject is relatively heterogeneous. The main subject of these studies may be arbitrarily categorized into LBP,(1478) trigger points,(1636) or tender points.(1637, 1638) Nevertheless, there are quality studies for subacute and chronic LBP patients. There are no quality studies evaluating this treatment in acute LBP, and the one study that might have included acute LBP patients can be reasonably concluded to suggest that this treatment is not recommended in that population.(1637) These injections are invasive, have rare adverse effects,(1478) and are moderately costly depending on the number administered. There are no studies evaluating these injections on a longer term basis,

though there are studies suggesting benefits lasting up to 14 days.(1478) There is no evidence that a steroid is required for efficacy of these injections, particularly those that are tender point injections (see also Shoulder Disorders guideline). As glucocorticosteroids also have adverse effects, their use in these injections is not recommended.

Evidence for the Use of Tender and Trigger Point Injections
There is 1 high-(1637) and 5(1478, 1636, 1638-1640) moderate-quality RCTs or crossover trials incorporated into this analysis.

We searched PubMed, EBSCO, Google Scholar, and Cochrane Review with no limits on publication dates. The following search terms used were "(Trigger OR tender point injections) AND (chronic low back pain)" to find 43,945 articles. Of those articles, we reviewed 8 articles, included 13 articles (6 RCTs and 7 reviews).

DIAGNOSTIC FACET JOINT INJECTIONS (INTRAARTICULAR AND NERVE BLOCKS)
Facet (zygapophysial) joints are prone to degenerative joint disease, particularly osteoarthrosis, and are thought to be pain-generating sources.(609, 622, 635, 703, 721, 1107, 1641-1649) Facet joint pain prevalence estimates vary from 5 to 90%.(622) Because of the overlapping innervation of the facet joints themselves (each is served by two medial branch nerves – a given medial branch nerve innervates the caudal portion of the facet joint at its level, and the rostral portion of the next lower facet joint) there has been considerable debate regarding whether these injections are truly diagnostic of underlying pathology. Moreover, careful skin mapping shows that the area of skin served by the cervical and lumbar medial branch nerves is more cephalad (in the neck) and more lateral and caudad (in the low back) than the location of the joint itself. Thus, it is often difficult to correlate degenerative joint disease changes seen on imaging studies with the actual nerve involved.

Two types of diagnostic facet injections are performed. The intra-articular injection is performed by injecting a local anesthetic under fluoroscopic or other imaging guidance directly into the facet joint. The second is a medial nerve branch block which is performed by injecting anesthetic along the nerves supplying the facet joints.(1650) (Datta 13) Either can be used to diagnose facet syndrome, but a medial branch block has been used when rhizotomy procedures have been considered.(1643, 1647, 1651) A positive block is considered to occur when there is complete, or nearly complete, relief of the pain the patient has been experiencing for the length of time expected for the anesthetic used.(333, 1652, 1653) The positions of the needle should be verified by fluoroscopy and documented with permanent images. The intra-articular blocks are sometimes combined with a glucocorticosteroid injection and thus, they are potentially a combined diagnostic and therapeutic intervention.(1654) Nerve root blocks are performed prior to attempts at radiofrequency lesioning.(1655)

Another indication for diagnostic intra-articular injections is lumbar segmental rigidity where the block can be both diagnostic and therapeutic.(60) In cases of chronic LBP, loss of mobility at one or more levels, particularly in the L3-S1 segments, is not uncommon. Injections for this indication may be combined with exercise to restore mobility and facilitate the rehabilitation process.

1. *Recommendation: Diagnostic Facet Joint Injection for Chronic Low Back Pain*
   Diagnostic facet joint injections are not recommended for evaluation of patients with chronic low back pain, including that which is significantly exacerbated by extension and rotation or associated with lumbar rigidity.

Strength of Evidence – **Not Recommended, Evidence (C)**
Level of Confidence – Low

2. *Recommendation: Diagnostic Facet Joint Injections for Acute or Subacute Low Back Pain or Radicular Pain Syndromes*
Diagnostic facet joint injections are not recommended for acute or subacute low back pain or radicular pain syndromes.

Strength of Evidence – **Not Recommended, Insufficient Evidence (I)**
Level of Confidence – Low

3. *Recommendation: Diagnostic Medial Branch Blocks for Acute or Subacute Low Back Pain or Radicular Pain Syndromes*
Diagnostic medial branch blocks are not recommended for acute or subacute low back pain or radicular pain syndromes.(1656)

Strength of Evidence – **Not Recommended, Evidence (C)**
Level of Confidence – Low

Rationale for Recommendations
Most studies now suggest a lack of utility of diagnostic facet joint injections.(1657-1659) Few studies suggest diagnostic utility of facet joint injections.(1660) Some have suggested a small minority of patients fulfill diagnostic criteria.(60)

One study of radicular pain patients found injection of an anesthetic was diagnostically non-specific.(1661) One study of medial branch blocks reported equal value of those blocks compared with peri-capsular blocks raising some question as to the efficacy vs. inefficacy of either.(1656)

The results of a trial comparing intra-articular injection vs. periarticular injection vs. saline injection also raises concerns about the validity of this construct,(1657) although the resulting improvements in all three groups could be argued to be worth the intervention in select significantly affected patients with chronic LBP thought to be facet mediated. Still, the results demonstrated that relief was not long lasting. Efficacy of facet joint injections is not well established in quality studies' original data. It has been reported that the peri-procedure administration of sedatives may confound the results of facet joint pain.(1662) This may contribute to suboptimal results for these injections. In patients with chronic LBP who have failed initial therapy, a negative diagnostic injection suggests that subsequent therapy directed at facet joint would not be useful. Improved, but still suboptimum range of motion (measured inclinometrically) may be an indication for therapeutic intra-articular injections in cases of lumbar segmental rigidity. Diagnostic medial branch blocks are primarily used to infer a need for rhizotomy.

Diagnostic facet injections are not recommended for acute or subacute LBP or radicular pain syndromes. These injections are invasive. Although they have relatively few adverse effects, the aggregate costs are high.

Evidence for the Use of Diagnostic Facet Joint Injections
We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates and then an updated search was conducted using PubMed for publications between 1/1/2013 and 11/15/2017. We used the following search terms: diagnostic facet joint injections, back, nerve blocks, intraarticular blocks, intraarticular injections, intra-articular injections, medial nerve branch block, subacute low back pain, radicular pain syndrome, sciatica, and random* to find 3,098 articles. Of the

3,098 articles, we reviewed 20 articles and 10 articles were included (6 randomized controlled trials and 4 systematic reviews).

THERAPEUTIC FACET JOINT INJECTIONS
Therapeutic facet joint injections involve injections of a combination of a local anesthetic with glucocorticosteroids for purposes of relieving pain from the facet to facilitate an active therapy program or to maintain employment.(1641, 1645, 1659, 1663) These are usually performed as combined diagnostic and therapeutic injections, rather than first performing an anesthetic injection followed by a second injection that includes glucocorticosteroid.(1643, 1651, 1654, 1655, 1664) They also may be accomplished either as an intra-articular or as a pericapsular injection, using a number of techniques.(1656, 1657, 1665)

1. *Recommendation: Therapeutic Facet Joint Injections for Treatment of Chronic Low Back Pain*
Therapeutic facet joint injections are not recommended (62% Panel agreement; 19% agreed with Recommended and 19% agreed with No Recommendation.) Indications are nevertheless provided for the potential to seek approval from a workers' compensation carrier for highly select patients with chronic LBP.

Indications:	Chronic LBP thought to be isolated to one or at most 2 facet joints. Generally with increased pain with extension and axial rotation. Failed to gain sufficient relief with non-invasive treatment options including at least multiple NSAID(s), aerobic exercise, and strengthening exercise. A trial of manipulation to assess functional gain is also generally warranted before consideration of therapeutic facet joint injection(s).
Benefits:	Potential to improve pain and possibly function.
Harms:	Medicalization, higher opioids use, infection.
Frequency/Dose/Duration:	Usually combination of anesthetic and glucocorticosteroid. Steroids used in trials included: Methylprednisolone acetate 20mg (2411, 2412), 40mg (2413), 80mg (2414), betamethasone, triamcinolone hexacetonide 20mg (2408), dexamethasone sodium phosphate 3.3mg (2415). If there is 80% relief and objective improvement in function, yet symptoms recur, a second injection may be reasonable. Repeated and recurrent injections are not recommended.
Indications for Discontinuation:	Resolution of pain, complications necessitating discontinuation of therapy or device removal, or loss of therapeutic effect.

Strength of Evidence – **Not Recommended, Insufficient Evidence (I)**
Level of Confidence – Low

2. *Recommendation: Therapeutic Facet Joint Injections for Treatment of Acute, Subacute, or Radicular Non-specific Axial Pain*
Therapeutic facet joint injections are not recommended for treatment of acute, subacute, or radicular non-specific axial pain.

*Strength of Evidence – **Not Recommended, Insufficient Evidence (I)***
Level of Confidence – Moderate

3. *Recommendation: Therapeutic Facet Joint Injections for Treatment of Chronic Non-specific Axial Pain*
   Therapeutic facet joint injections are moderately not recommended for treatment of chronic non-specific axial pain.

   *Strength of Evidence – **Moderately Not Recommended, Evidence (B)***
   Level of Confidence – Moderate

4. *Recommendation: Therapeutic Facet Joint Injections for Patients with a Prior Injection*
   Repeat use of intra-articular therapeutic facet joint injections are moderately not recommended for patients who have failed to achieve lasting functional improvements with a prior injection.

   *Strength of Evidence – **Moderately Not Recommended, Evidence (B)***
   Level of Confidence – Moderate

Rationale for Recommendations
Degenerative facet joints become ubiquitous with age.(53-55) High- and moderate-quality studies suggest lack of efficacy of therapeutic facet joint injections for treatment of chronic LBP,(1649, 1657, 1666-1668) although one study suggested modest efficacy.(1669) One comparative trial found comparable (in)efficacy with radiofrequency injections which also appear ineffective (see below) (2416, 2417). Another moderate quality trial found comparable (in)efficacy with intramuscular compared with facet joint injections with steroids for treatment of LBP (2408).

Therapeutic facet joint injections are typically performed to address a joint that is felt to be symptomatic on a diagnostic facet joint block. They also have been performed to address a purported cause of segmental rigidity.(60, 61) This involves injection of a local anesthetic and a glucocorticosteroid. Facet injections are not advocated for acute or subacute LBP or radicular pain syndromes.

Both the American Pain Society and NICE guidelines recommend against these injections. (1670, 1671) These injections are invasive, have relatively low adverse effects, and are costly. Most of the quality studies available on this topic do not support these injections. If they are performed highly selectively, there should be evidence of enduring reductions of pain plus objective functional benefits along with a lack of needing to repeat the treatment other than rarely.

Evidence for the Use of Therapeutic Facet Joint Injections
We searched PubMed, EBSCO, Cochrane Library and Google Scholar without limits on publication dates then an updated search was done in PubMed for publication between 1/1/2013 and 11/15/2017. We used the following search terms: inject*, therapeutic facet joint injections, subacute low back pain, chronic low back pain, radicular pain, sciatica, back, and random* to find 4,560 articles. Of the 4,560 articles, we reviewed 448 articles and included 448 articles (19 randomized controlled trials and 429 systematic reviews).

FACET JOINT HYALURONIC ACID INJECTIONS
Facet joint injections with hyaluronic acid are being attempted for treatment of facet degenerative joint disease. These injections are analogous to similar injections in the knee and other arthritic joints.

Recommendation: Facet Joint Injections with Hyaluronic Acid for Treatment of Facet Degenerative Joint Disease

Facet joint injections with hyaluronic acid are not recommended for treatment of facet degenerative joint disease.

> *Strength of Evidence –* **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence –* Low

Rationale for Recommendation

There are no placebo- or sham-controlled trials. Weekly injections of hyaluronic acid have been studied in one moderate-quality study and appear to be largely ineffective compared to facet steroid injections that appear no more effective than placebo.(1672) As studied, this intervention is invasive, requiring a series of 18 injections performed at 3 levels, likely has some side effects, and is high cost. While the comparative pain and disability score reductions could be interpreted as somewhat promising, additional studies are needed prior to recommending this fairly invasive intervention and would need to show superiority of these injections.

Evidence for use of Facet Joint Hyaluronic Acid Injections

There is 1 moderate-quality RCT incorporated into this analysis.(1672)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: Facet, joint, hyaluronic, acid, injections, subacute, radicular, syndromes, sciatica, Spinal, stenosis, chronic, low, back, and pain to find 24,887 articles. Of the 24,887 articles, we reviewed one articles and included one articles.

SACROILIAC JOINT INJECTIONS

The sacroiliac joints (SIJs) are believed to cause a minority of chronic LBP cases, with estimates ranging from 10 to 26.6%. The most commonly performed interventions are sacroiliac joint injections either with or without fluoroscopic or other imaging guidance.(1645, 1673) The injection targets the tenderest area and generally consists of a glucocorticosteroid combined with a local anesthetic agent. The combination of agents is frequently designed to attempt to be both diagnostic and therapeutic. However, the diagnostic precision of these injections is likely limited by factors that include the inability to inject the joint directly without fluoroscopic or other imaging, as well as the infiltration and diffusion of medication into surrounding tissues that could be potential pain generators.(1674) The use of fluoroscopically guided, CT guided, or unguided SI joint corticosteroid injections have been suggested by some to be effective for low back pain and spondyloarthropathy.(1675-1677) Other resources have found the evidence to be limited or poor.(1678, 1679)

1. *Recommendation: Sacroiliac Joint Corticosteroid Injections for Treatment of Sacroiliitis*
   Sacroiliac joint corticosteroid injections are recommended as a treatment option for patients with a specific known cause of sacroiliitis, i.e., proven rheumatologic inflammatory arthritis involving the sacroiliac joints.
   Indications – Symptoms of sacroiliitis of at least 1 to 2 months duration with prior treatment that has included NSAIDs.

   Frequency/Duration – Each injection should be evaluated before additional injections are scheduled, rather than scheduling a series of injections.

Indications for Discontinuation – Resolution of the symptoms of sacroiliitis or decrease in symptoms to a tolerable level.
Benefits – Short to intermediate term reduction in pain.
Harms – Rare complications of paralysis, infections; medicalization.

> *Strength of Evidence* – **Recommended, Evidence (C)**
> *Level of Confidence* – Low

2. *Recommendation: Sacroiliac Joint Injections for Treatment of Acute Low Back Pain*
Sacroiliac joint injections are not recommended for treatment of acute low back pain including low back pain thought to be sacroiliac joint related; subacute or chronic non-specific low back pain, including pain attributed to the sacroiliac joints, but without evidence of inflammatory sacroiliitis (rheumatologic disease); or any radicular pain syndrome.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

Rationale for Recommendations
Some patients appear to have SIJ pain that is not due to spondyloarthropathies. In one quality study, a short-term response to glucocorticoid injection into the soft tissue above the joint was demonstrated.(1680) In limb joints, injection outside a joint has not been demonstrated to improve pain coming from a joint, so the mechanism for this finding is puzzling. The other two quality studies were both of populations of spondyloarthropathy patients, thus applicability to working populations is unclear. Whether fluoroscopic guidance is needed is unclear and controversial.(1681) Without fluoroscopic guidance, the joint itself is usually not injected as this is a difficult joint on which to perform arthrocentesis without imaging guidance. It is not clear if actual joint injection results in appreciably lower success rates as an injection in the local proximity may be just as effective. Injection in the local proximity should perhaps be classified as a tender point injection, and not as a sacroiliac joint injection. There is no surgical procedure of proven efficacy to help patients tentatively identified as having "sacroiliac joint pain" by diagnostic injection. There are no quality studies showing a long-term improvement in pain or function in those receiving sacroiliac joint injections for chronic non-specific LBP.

For patients with proven rheumatologic inflammatory disease of the sacroiliac joints (e.g., ankylosing spondylitis), SIJ injection has evidence of efficacy and the same sort of disease in extremity joints is commonly managed successfully with corticosteroid injection therapy. Sacroiliac joint diagnostic injections with topical anesthetic are not recommended. If an injection is felt to be necessary, then it is recommended that it be combined with a glucocorticosteroid injection and it should be performed with imaging guidance to insure the arthritic joint is successfully injected.

SIJ injections are minimally invasive, have low adverse effects, and are moderate cost if performed with fluoroscopy. They are recommended for treatment of proven inflammatory arthritis of the sacroiliac joints.

Evidence for the Use of Sacroiliac Joint Injections
We searched PubMed, EBSCO, Cochrane Review, and Google Scholar with no limits on publication dates then an updated search was done in PubMed for publication between 1/1/2013 and 11/15/2017., We used the following search terms: sacroiliac joint corticosteroid injections, sacroiliitis, subacute low back pain, chronic low back pain, and low back pain to find 373 articles. Of the 675 articles, we reviewed 21

articles and included 21 articles (15 randomized controlled trials, 2 systematic reviews, and 4 Case-Series).

INTRATHECAL DRUGS
The use of intrathecal drug delivery systems (aka, "pain pumps") for acute pain is common and frequently effective utilizing morphine, fentanyl and other agents for perioperative and post-operative pain control. Those uses are reviewed in other chapters (e.g., see Hip and Groin Disorders guideline).(1682-1685) Occasionally, treatment of severe pain has been attempted using opioids administered parenterally by these devices.(1682-1689)

Recommendation: Intrathecal Drug Delivery Systems for Chronic Non-malignant Pain Conditions
Intrathecal drug delivery systems are not recommended for treatment of chronic nonmalignant pain conditions.

Harms – Device complications, fatalities.
Benefits – Less debility, reduced accidents risks, risks of dependency or addiction.
> *Strength of Evidence* **– Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – High

Rationale for Recommendation
Intrathecal drug delivery systems have not been evaluated in quality studies to determine whether treatment with these systems is superior to oral medication(s) or other treatment options for chronic nonmalignant pain patients. A placebo-controlled trial for gabapentin was negative (2418). Administrations via pain pumps for chronic non-malignant and malignant pain are limited, but there are studies evaluating parenteral opioids for pain in chronic cervicothoracic patients that while suggesting short-term relief of pain, do not demonstrate long-term benefits. A quality cost-benefit analysis in an RCT is not available (2419). The medications used were potent and not intended for chronic use.(1688, 1690) Deaths have been associated with intrathecal opioid use, including one-day post-implantation.(1686) Granulomas appear to frequently develop;(1691) the expected "permanency" of neurologic abnormalities associated with their formation has not been established.(1692)

Ziconotide has been used in intrathecal delivery systems, but with only several days duration; thus, there was insufficient time to ascertain efficacy commensurate with the invasiveness of this delivery system.(1693) It is not known whether there is a reduced incidence of intrathecal granuloma formation with this drug since its use has not been widely applied over the long term. Ziconotide has a narrow therapeutic margin and has been associated with severe neuropsychiatric adverse effects. Since it does not share pharmacologic actions with narcotics, there is no known method to determine prospectively whether a patient will respond favorably to this drug.(1694)

Intrathecal opioid delivery systems are invasive and costly, with possible significant adverse effects including elevated mortality (2420) and potential long-term sequelae from both implantation/ retention of the devices, including granuloma formation, and those associated with the concurrent use of intrathecal opioids.(1695) Thus, with a lack of documented efficacy, invasiveness, serious adverse effects and marked costs, these devices are not recommended. For new patients, there are few barriers for implementing this guideline. For existing patients, this guideline should not be interpreted as requiring device removal.

Evidence for the Use of Intrathecal Drugs
We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates and then an updated search was conducted using PubMed for publications between 1/1/2013 and 11/15/2017. We used the following search terms: Intrathecal Pain Pumps, Intrathecal, drug, delivery, system, chronic, low, back, pain, and random* to find 67,313 articles. Of the 67,313 articles, we reviewed 14 articles and 14 articles were included (12 randomized controlled trials and 2 systematic reviews).

PROLOTHERAPY INJECTIONS

Prolotherapy injections attempt to address a theoretical cause or mechanism for chronic LBP.(102, 1696-1701) This purported therapy involves repeated injections of irritating, osmotic, and chemotactic agents (e.g., dextrose, glucose, glycerin, zinc sulphate, phenol, guaiacol, tannic acid, pumice flour, sodium morrhuate), combined with an injectable anesthetic agent to reduce pain, into back structures, especially ligaments, with the theoretical construct that they will strengthen these tissues.(1702, 1703) Prolotherapy injections alone have been mostly found to not be more effective than control injections for patients with chronic LBP.(1697, 1704, 1705)

Recommendation: Prolotherapy Injections for Treatment of Acute, Subacute, or Chronic Low Back Pain or Radicular Pain Syndromes
Prolotherapy injections are strongly not recommended for treatment of acute, subacute, or chronic low back pain or any radicular pain syndrome.

> *Strength of Evidence* – **Strongly Not Recommended, Evidence (A)**
> *Level of Confidence* – High

Rationale for Recommendation
Although there is considerable heterogeneity in the available literature, the highest quality studies showed no benefit of prolotherapy injections.(685, 1696, 1706-1708)

Prolotherapy injections are invasive and have a stated purpose of causing irritation. There are reports of deaths from accidental intrathecal injections,(1696) post-procedure "lumbar puncture headaches,"(1708, 1709) and increased LBP (88%).(685) The intravenous injections (e.g., diazepam, midazolam) given to tolerate the procedure and large volumes of lidocaine used may increase the risks from these procedures. These injections are costly. As the highest quality studies fail to show benefits, these injections are not recommended for the treatment of LBP.

Table 10. Outcomes from Prolotherapy Injections vs. Saline Injections and Exercise vs. Normal Activity among 110 Chronic LBP Patients

	VAS Baseline (0-100)	VAS at 1 year Follow up	Roland-Morris Disability Score at Baseline (0-23)	Roland-Morris Disability Score at 1 year Follow up
Injection glucose and lignocaine	51.9	18.6	13.7	5.5
Injection of saline	55.0	18.4	14.3	4.5
Exercise	54.6	20.5	13.0	4.8
Normal activity	52.3	16.5	15.0	5.1
	VAS baseline	VAS at 2-year follow-up	Roland-Morris disability score at baseline	Roland-Morris disability score at 2-year follow-up
Injection glucose and lignocaine	51.9	18.4	13.7	4.9
Injection of saline	55.0	16.4	14.3	4.2
Exercise	54.6	18.0	13.0	3.9

Normal activity	52.3	16.6	15.0	5.2

Adapted from Yelland MJ, Glasziou PP, Bogduk N, Schluter PJ, McKernon M. *Spine*. 2003.

Evidence for the Use of Prolotherapy Injections
There is 2 high-(1706, 1707) and 5 moderate-quality(1321, 1398, 1696, 1708, 1710) RCTs incorporated into this analysis.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following terms: prolotherapy injections, proliferation therapy, regenerative injection therapy, subacute low back pain, chronic low back pain, radicular pain, and sciatica to find 465 articles. Of the 465 articles, we reviewed 16 articles, and included 12 (6 RCTs and 6 systematic reviews).

BOTULINUM INJECTIONS
Botulinum injections have been used to produce muscle paresis and have anti-nociceptive properties.(1711) Adherents beliefs include that this "rest through weakness" is useful as a treatment for a number of musculoskeletal disorders including LBP.(1712, 1713) It has been used for upper back and myofascial pain,(685, 1714, 1715) LBP,(1713, 1716-1718) and piriformis syndrome.(1645, 1712, 1719-1724)

Recommendation: Botulinum Injections for Treatment of Chronic Low Back Pain
There is no recommendation for or against the use of botulinum injections for treatment of acute, subacute, or chronic low back pain or radicular pain syndromes or other low back-related problems.
> *Strength of Evidence* – **No Recommendation, Insufficient Evidence (I)**
> *Level of Confidence* – Low

Rationale for Recommendation
Two high-quality studies directly conflict, with one suggesting benefits(1725) while the other suggesting no benefits.(1720) One moderate-quality trial suggested benefits.(1722) Thus, the quality data conflict and there are no sizable quality studies with long-term follow-up. It is concerning that these injections induce weakness, yet many of the most successful interventions identified in systematic reviews in other sections of this guideline build strength and/or endurance. Botulinum injections are invasive, have adverse effects that include fatalities,(1725) and are costly and with conflicting data have no recommendation.

Evidence for the Use of Botulinum Injections
There are 2 high-(1720, 1725) and 2 moderate-quality(1722, 1726) RCTs incorporated into this analysis. There are 2 low-quality RCTs in Appendix 1.(1721, 1727)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms: botulinum injections, botulinum toxin A, subacute low back pain, chronic low back pain, spinal stenosis, spinal fractures, sacroiliitis or spondylolisthesis to find 1,898 articles. Of the 1,898 articles, we reviewed 5 articles and included all 5 articles (4 RCTs, 1 prospective study).

RADIOFREQUENCY NEUROTOMY, NEUROTOMY, AND FACET RHIZOTOMY
Facet joints (aka zygapophysial joints) have been thought to be the source of pain for some patients with chronic LBP.(1728-1733) Patients who experience pain relief from the injection of anesthetic along the nerve roots innervating the joints ("diagnostic blocks") have been considered candidates for various

neurotomy procedures.(1734) Surgical neurotomy involves the transecting or cutting of the nerves supplying the facet joints. Less invasive procedures involving electrodes to create nerve lesions (denervation) have largely replaced this surgical procedure.(1730)

Radiofrequency neurotomy involves the use of a radiofrequency electrode to create a heat lesion to coagulate the nerve supplying the joint. If the theory is correct and the patient is correctly diagnosed, the procedure will result in complete relief of LBP. If there are other sources of pain that have other nerves for conduction of pain impulses or the radiofrequency (RF) lesion does not encompass the nerve due to either anatomic variants or technical errors, the procedure is thought to be less successful or not at all successful.(1642, 1735)

1. *Recommendation: Radiofrequency Neurotomy, Neurotomy, or Facet Rhizotomy for Treatment of Chronic Low Back Pain*
   Radiofrequency neurotomy, neurotomy, or facet rhizotomy are not recommended for treatment of patients with chronic LBP, including patients who are confirmed with diagnostic blocks but who do not have radiculopathy and who have failed conservative treatment. (64% panel agreement; 36% of panel agreed with limited indications as indicated below.)

   Indications – Patients with chronic LBP without radiculopathy who failed conservative treatments and who have had a confirmed diagnosis by medial branch blocks.(1736)

   Frequency/Duration – One procedure might be tried as an option after failure of non-invasive treatments including NSAIDs and a quality exercise program or as a means to help with participation in an active rehabilitation program. There is no recommendation for repeated procedures. It is reasonable to attempt a second lesion after 26 weeks in patients who had greater than 80% improvement in pain from first procedure for the first 8 weeks with a late return of pain.(1737) There is no recommendation for a third or for additional procedures. There is logically a limit as to how many times it is possible to permanently destroy the same nerve.

   Indications for Discontinuation – Resolution of symptoms. If there is no response to the first procedure, there is no evidence that a second lesion will be beneficial.

   Benefits – Possible pain reduction
   Harms – Medicalization, procedural complications. Successful denervation of joints should increase risk of Charcot joints.
   > *Strength of Evidence* – **Not Recommended, Evidence (C)**
   > *Level of Confidence* –Low

2. *Recommendation: Radiofrequency Neurotomy, Neurotomy, or Facet Rhizotomy for Treatment of Other Lumbar Spinal Conditions*
   Radiofrequency neurotomy, neurotomy, or facet rhizotomy are not recommended for treatment of all other lumbar spinal conditions.
   > *Strength of Evidence* – **Not Recommended, Evidence (C)**
   > *Level of Confidence* – Low

Rationale for Recommendations
High-quality studies supporting surgical neurotomy using sham were not found. The highest quality, sham-controlled studies are largely negative.(1738, 1739) Another moderate quality study of RF added to steroid injection also found nearly all measures (e.g., ODI, NRS, MQS) were negative between groups (2421). The largest sized trial found neurotomy ineffective compared with an exercise program for

treatment of LBP, or SI joint pain or intervertebral disc pain (2422). The next lower quality study is more favorable, but used unconventional statistical testing with 90% confidence intervals, rendering it unusable(1740) and the next study suffered an apparent randomization failure.(1741) Two comparative trials found comparable (in)efficacy with intraarticular glucocorticoid injections which also appear ineffective, which suggests the procedure may have no significant benefit (see above) (2416, 2417).The lowest quality study had worrisome results in the placebo.(1742) There is a poor correlation between pain relief from a block and relief from radiofrequency neurotomy (2423). Available systematic reviews also discuss additional significant methodological concerns.(59) These concerns further limit the robustness of conclusions. As results are permanent, there should be good evidence of long-term benefit prior to recommending this procedure. Permanently denervated joints in the appendicular skeleton are called Charcot joints, and over long-term follow-up they do not do well; there are no long-term results reported for those potential adverse effects. All studies suggested the need for further research.

The theoretical basis of cutting or ablating nerve fibers seems sound as procedures that eliminate the pathway to conduct sensations of pain should be effective for the treatment of chronic pain syndromes. However, the history of cutting or otherwise ablating nerves to treat numerous pain conditions throughout the body is suboptimal, with a not infrequent increased risk for developing additional chronic pain problems(1743) that were only widely recognized after long-term follow-up studies were reported. There have been many attempts at this type of procedure over several decades. However, perhaps due to pain fiber regeneration, alternate pathways for conduction, phantom pain, ongoing neurological stimulation, and/or conduction from the transected or ablated nerve fibers, no procedure to date has been shown to be effective for the treatment of pain that involves cutting or ablating nerve fibers. An interesting finding in two of these studies is the possibility that patients with higher degree of successful blocks, (e.g., >80%) as opposed to the 50% threshold that is more widely employed, have better outcomes.(1740, 1742) However, as this has not been proven, it cannot be adopted as guidance at this time.

It is noteworthy how few patients thought to be candidates for the procedure actually have successful blocks (43.5%[679] to 54.3%(1739)). This suggests that the number of patients who could be successfully treated with this therapy, especially if the supposition in the prior paragraph proves true and the procedure is proven effective, would likely be quite small.

Radiofrequency lesioning is invasive, has adverse effects, and is costly. With the highest quality studies mostly suggesting a lack of efficacy, the overall evidence base does not support this treatment. Additional quality research is needed in this area as outlined above, as it is currently an experimental procedure for purposes of treating acute, subacute, and chronic LBP, and radicular pain syndromes and/or "discogenic" LBP. There are no quality studies identified to support surgical neurotomy or rhizotomy and thus they are not recommended.

Evidence for the Use of Radiofrequency Neurotomy, Neurotomy, and Facet Rhizotomy
We searched PubMed, EBSCO, Cochrane Review, and Google Scholar with no limits on publication dates then an updated search was done in PubMed for publication between 1/1/2013 and 11/15/2017. We used the following terms: radiofrequency neurotomy, neurotomy, facet rhizotomy, subacute low back pain, chronic low back pain, low back pain, back, random*. Of the 389 articles, we reviewed 58 articles and included 58 articles (31 are randomized controlled trials and 29 systematic reviews).

DORSAL ROOT GANGLIA RADIOFREQUENCY LESIONING
Radiofrequency lesioning of the dorsal root ganglia has been attempted for treatment of chronic sciatica and some other pain syndromes.(1728, 1732, 1744)

Recommendation: Radiofrequency Lesioning for Treatment of Chronic Sciatica
Radiofrequency lesioning of the dorsal root ganglia is moderately not recommended for treatment of chronic sciatica.

>*Strength of Evidence* – **Moderately Not Recommended, Evidence (B)**
>*Level of Confidence* – Moderate

Rationale for Recommendation
Radiofrequency lesioning is invasive, has adverse effects, and is costly. It has been shown to not be efficacious in a high-quality study.(1745)

Evidence for the Use of Dorsal Root Ganglia Radiofrequency Lesioning
There is 1 high-quality RCT incorporated into this analysis.(1745)

We searched PubMed, EBSCO, Cochrane review and Google Scholar without any limits on publication dates. We used the following search terms "Radiofrequency lessoning of the dorsal root ganglia for chronic sciatica, radicular pain syndromes (including 'sciatica')" to find 8414 articles. Of those, we reviewed 5 articles and included 3 (1 RCT and 2 reviews).

INTRADISCAL ELECTROTHERMAL THERAPY (IDET)
Intradiscal electrothermal therapy (IDET) involves the heating of an intradiscal probe through electrical current. The goal is to coagulate tissue and theoretically result in improvement in pain thought to be derived from the disc or surrounding structures.(1746-1748) As this is a relatively new intervention, techniques have not been standardized.

Recommendation: Intradiscal Electrothermal Therapy (IDET) for Treatment of Low Back Pain
IDET is not recommended for treatment of acute, subacute, or chronic low back pain or any other back-related disorder.

>*Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
>*Level of Confidence* – Low

Rationale for Recommendation
There are two high-quality RCTs(1749, 1750) that conflict regarding whether IDET has any value in treating chronic LBP. It is unclear whether heterogeneity of patients' clinical findings may in part explain these differences. Another problem is the reliance on discography as the primary diagnostic requirement for IDET, as it has low diagnostic value (see Discography). IDET has not been clearly shown to be beneficial. It is costly and invasive although it may have a relatively low complication rate.(1751) Thus, there is not adequate evidence to recommend this procedure.

Evidence for the Use of IDET
There are 2 high-quality RCTs incorporated into this analysis.(1749, 1750)

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following terms: IDET, intradiscal electrothermal therapy, and low back pain to find 1174 articles. Of the 1174 articles we reviewed two articles and included two articles.

PERCUTANEOUS INTRADISCAL RADIOFREQUENCY THERMOCOAGULATION (PIRFT)
Percutaneous intradiscal radiofrequency thermocoagulation involves the same principle as that of IDET. However, the heating of an intradiscal probe is through radiofrequency instead of electrical current. The theoretical mechanisms of efficacy are essentially the same as for IDET.(1752-1754)

Recommendation: Percutaneous Intradiscal Radiofrequency Thermocoagulation for Treatment of Acute, Subacute, or Chronic Low Back Pain
Percutaneous intradiscal radiofrequency thermocoagulation is moderately not recommended for treatment of acute, subacute, or chronic low back pain particularly including discogenic low back pain.
> *Strength of Evidence* – **Moderately Not Recommended, Evidence (B)**
> *Level of Confidence* – Moderate

Rationale for Recommendation
There is no evidence of efficacy in two quality studies, including one high quality study.(1752, 1755) A third moderate-quality trial is not a purely sham-controlled trial and has problems with interpretation. Thus, the procedure is not recommended.

Evidence for the Use of Percutaneous Intradiscal Radiofrequency Thermocoagulation
There is 1 high-(1752) and 2 moderate-quality(1754, 1755) RCTs incorporated into this analysis.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates. We used the following search terms "(Percutaneous intradiscal radiofrequency thermocoagulation) AND (subacute OR chronic OR low OR back OR pain)" to find 611 articles. Of the articles, we reviewed 5 articles and included 5 articles (3 RCTs and 2 reviews).

Surgical Considerations. . . . .
This guideline will address only the non-emergent surgical treatment of the most common acute, subacute, and chronic back problems. The indications for emergent surgery for red flag conditions including spinal cord compression, cauda equina syndrome, unstable fractures, epidural abscess, or hematoma, etc., will not be discussed, as treatment of these conditions is outside the scope of these guidelines, as are other indications for surgery (e.g., neoplasms). This guideline does discuss recognition of red flag conditions that require expedited referral to a surgeon qualified to deal with spine emergencies (see Red Flags).

Within the first 3 months after onset of acute low back symptoms, surgery is considered only for serious spinal pathology or nerve root compression not responsive to an adequate trial of conservative therapy. Disc herniation may impinge on a nerve root typically causing mostly lower extremity and sometimes lumbosacral symptoms accompanied by nerve root dysfunction. However, the presence of a herniated disc on an imaging study does not necessarily imply nerve root dysfunction. Studies of asymptomatic adults commonly demonstrate intervertebral disc herniations that apparently do not cause symptoms.

Some studies show spontaneous disc resorption without surgery. Many patients with strong clinical findings of nerve root compression due to disc herniation and/or spinal stenosis recover activity

tolerance within 1 month. There is no quality evidence that delaying surgery for this period worsens outcomes in the absence of progressive nerve root compromise.(1756) With or without surgery, more than 70% of patients with apparent surgical indications eventually recover to their pre-morbid activity level, including those with severe initial presenting signs of neurological compromise.(1757, 1758) Spine surgery for patients with clear indications appears to speed short- to mid-term recovery. However, surgery results in pain improvements in fewer than 40% of patients with questionable physiologic findings, which is the rate of response of pain to placebo surgery.(1201, 1759) Surgery generally increases the risk for future spine procedures with higher complication rates especially associated with more invasive procedures such as fusion.(1760-1763) Yet, reoperation rates are reportedy lower after fusion compared with decompressive surgery for spinal spondylolisthesis.(1762) In older patients and repeat procedures, the rate of complications is higher.(1764, 1765) Patients with comorbid conditions such as cardiac or respiratory disease, diabetes, or mental illness, may be poor candidates for surgery. Comorbidity should be weighed and discussed carefully with the patient.

If surgery is a consideration, counseling regarding likely outcomes, risks, and benefits and especially expectations is important. Patients with acute LBP alone (in the absence of objective findings of radiculopathy), without findings of serious spinal pathology (such as tumor, fracture, infection, hematoma), rarely benefit from surgery, although a second opinion from a spine surgeon to the effect that surgery is not recommended and is unlikely to be helpful may be reassuring to the patient.

Before surgery, physicians may consider referral for psychological screening to improve surgical outcomes, possibly including standard tests such as the second edition of the Minnesota Multiphasic Personality Inventory (MMPI-2).(1766) In addition, physicians may look for non-organic signs (e.g., Waddell) during the physical exam as these have been shown to correlate with poorer surgical outcome.

LUMBOSACRAL NERVE ROOT DECOMPRESSION

Nerve root decompression is performed for symptomatic nerve root compression by disc herniation and/or spinal stenosis. Direct methods of nerve root decompression include standard open discectomy, laminotomy, foraminotomy, facetectomy, and laminectomy. The only indirect method of nerve root decompression shown to be potentially effective is chemonucleolysis with chymopapain.

Endoscopic removal of a herniated disc fragment, while performed percutaneously, is a similar operation to standard open discectomy and is considered below. Standard open discectomy can be done with or without the use of an operating microscope or loop magnification and with or without endoscopic "tubes" to minimize the size of the skin incision and muscle dissection.

DISCECTOMY, MICRODISCECTOMY, SEQUESTRECTOMY, ENDOSCOPIC DECOMPRESSION

There are multiple surgical techniques that have been used to surgically relieve pressure on lumbosacral nerve roots causing radicular pain syndromes.(1767-1771) These include open discectomy (with or without microscope),(1772-1777) automated percutaneous discectomy,(1778-1780) epidural percutaneous discectomy,(1781) sequestrectomy, and endoscopic procedures.(1782-1786) More recent techniques include percutaneous laser disc decompression,(1787) automated percutaneous discectomies (also known as nucleoplasty),(1788, 1789) disc coblation, and endoscopic approaches.(1790) The same surgical approaches are also sometimes used to address less common spinal pathology (e.g., facet joint arthropathy with consequent nerve root impingement). This section reviews the indications for discectomy for a herniated lumbar disc.

1. *Recommendation: Lumbar Discectomy for Radiculopathy*
   Lumbar discectomy is moderately recommended to speed recovery in patients with radiculopathy due to ongoing nerve root compression who continue to have significant pain and functional limitation after 4 to 6 weeks of time and appropriate conservative therapy. For patients who are candidates for discectomy (other than for cauda equina syndrome and the rare progressive major neurologic deficit), there is evidence that there is no need to rush surgical decisions as there is no difference in long-term functional recovery whether the surgery is performed early or delayed. Open discectomy, microdiscectomy, and endoscopic discectomy are all potentially appropriate ways to perform discectomy. The decision as to which of these procedures to choose should be left to the surgeon and the patient until quality evidence becomes available to provide evidence-based guidance. Other procedures such as laser discectomy and/or PERC involve indirect procedures with limited access to the disc contents.

   Indications – All of the following should be present: 1) radicular pain syndrome with current dermatomal pain and/or numbness, or myotomal muscle weakness all consistent with a herniated disc; 2) imaging findings by MRI, or CT with or without myelography that confirm persisting nerve root compression at the level and on the side predicted by the history and clinical examination; and 3) continued significant pain and functional limitation after 4 to 6 weeks of time and appropriate non-operative therapy that usually includes NSAID(s). Progressive neurological deficits are considered a separate indication.

   Benefits – Earlier pain relief
   Harms – Operative complications that very rarely include severe adverse effects or fatality comparable with other moderate surgical procedures.
   > *Strength of Evidence* – **Moderately Recommended, Evidence (B)**
   > *Level of Confidence* – High

2. *Recommendation: Discectomy for Treatment of Acute, Subacute, or Chronic Low Back Pain without Radiculopathy*
   Discectomy is moderately not recommended for treatment of acute, subacute, or chronic low back pain without radiculopathy.
   > *Strength of Evidence* – **Moderately Not Recommended, Evidence (B)**
   > *Level of Confidence* – High

3. *Recommendation: Discectomy for Back or Radicular Pain Syndrome*
   Percutaneous discectomy (nucleoplasty), laser discectomy, and disc coblation therapy are not recommended for treatment for any back or radicular pain syndrome.
   > *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
   > *Level of Confidence* – Low

Rationale for Recommendations
There are no sham-controlled surgical trials. All moderate-quality comparative trials demonstrate short- to intermediate-benefits, but not long-term benefits from nerve root decompression surgery compared with conservative treatment for patients with radicular symptoms from disc herniation unresponsive to 4 to 6 or more weeks of prior non-operative treatment.(1756, 1791-1793) However, as up to 75% of patients with radicular symptoms from herniated discs may become minimally symptomatic or asymptomatic without surgery,(1756, 1791-1794) sufficient time should pass prior to consideration of surgery. Also, there is no need to rush patients into surgery as there is consistent evidence of a lack of differences in long-term functional recovery.(1756, 1791-1793)

Quality literature is insufficient on the comparative values of open discectomy, microdiscectomy, or endoscopic discectomy. There are no quality trails of endoscopic decompression identified or percutaneous lumbar laser disc decompression.(1795) Also, there is no quality evidence that automated percutaneous discectomy, laser discectomy, or coblation therapy is an effective treatment for any back or radicular pain problem. There are trials on techniques to minimize postoperative epidural fibrosis, but surgical technique is beyond the scope of this guideline.(1796)

Discectomy is invasive, costly and has adverse effects. However, there is consistent, moderate-quality evidence that lumbar discectomy is an effective operation to speed recovery in patients with radiculopathy due to ongoing nerve root compression who have not improved significantly after 4 to 6 weeks of time and appropriate conservative therapy and it is thus recommended.

Evidence for the Use of Discectomy

We searched PubMed, EBSCO, Cochrane Review, and Google scholar without limits on publication dates and then an updated search was done in PubMed for publication between 1/1/2013 and 11/15/2017. We used the following search terms: percutaneous discectomy, nucleoplasty, laser discectomy, disc coblation therapy, discectomy, microdiscectomy, sequestrectomy, chemonucleolysis, endoscopic, decompression, subacute, low back pain, chronic low back pain, radicular pain, radiculopathy, sciatica, clinical trial, randomized controlled trial, random, systematic review, population study, epidemiological study, and prospective cohort to find 5,829 articles. Of the 5,829 articles, we reviewed 39 articles and 39 articles were included (28 randomized controlled trials and 11 systematic reviews).

ADHESIOLYSIS
Epidural adhesiolysis attempts to use hypertonic saline and glucocorticoids with a catheter and/or endoscopy to address adhesions that particularly develop after surgery and are proposed by some to be related to post-operative pain and failed back surgery syndrome.(1797, 1798) Epidural adhesiolysis is also known as percutaneouslysis of epidural adhesions, epidural neurolysis, epidural decompressive neuroplasty, and Racz neurolysis.(1799-1803)

Recommendation: Adhesiolysis for Treatment of Low Back Pain
Adhesiolysis is not recommended for treatment of acute, subacute, or chronic low back pain, arachnoiditis, or spinal stenosis or radicular pain syndromes.

> *Strength of Evidence* **– Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

Rationale for Recommendation
There are no sham-controlled trials. All studies comparing different adhesiolysis techniques were conducted by the same research group. The only other trial was an unblinded comparison of adhesiolysis with physiotherapy.(1804) Independent replication of the suggested modest benefits is needed before a recommendation may be made.

Adhesiolysis has been reported to show encouraging results in relatively small case studies and other uncontrolled or poorly controlled studies.(1797) No large scale, controlled clinical trials involving adhesiolysis have been reported.

Adhesiolysis is a relatively new procedure, is invasive, and has complications including serious ones such as dural puncture, spinal cord compression, infection, catheter shearing, hematoma, cardiac

dysrhythmias, myelopathy, paralysis, and blindness.(511, 515, 1802, 1805-1807) It is also costly. Large scale, high-quality, multi-center studies with long-term follow-up are needed prior to consideration of this intervention for recommendation.

Evidence for the Use of Adhesiolysis
There is 1 high-(1808) and 4 moderate-quality(515, 1804, 1809, 1810) RCTs incorporated into this analysis. There is 1 low-quality RCT in Appendix 1.(1806)

One of the studies (which suggested that approximately half of the relief was gone at 12 months)(1801) has been labeled by its authors with an incorrect study design which raises concerns about selection bias, spectrum bias, and a potential uncontrolled confounder due to enrolling subjects into multiple studies.

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar with no limits on publication dates. We used the following terms: adhesiolysis, subacute low back pain, chronic low back pain, radicular pain syndromes, and spinal stenosis. Of the 675 articles, we reviewed 10 articles and included 6 articles.

DECOMPRESSIVE SURGERY FOR SPINAL STENOSIS (LAMINOTOMY/FACETECTOMY, LAMINECTOMY)

Spinal stenosis means insufficient room for neural elements in the spinal canal and/or neural foramina. It can be congenital (e.g., short pedicles, narrow canal diameter) or acquired (degenerative enlargement of facets and ligaments and in addition the formation of osteophytes), or both. Stenosis can be in the central canal, in the lateral recess, or in the neural foramen. These degenerative changes are referred to as lumbar spondylosis. The typical symptom of lumbar spinal stenosis is neurogenic claudication, or leg pain that develops during walking and that is promptly relieved by rest. Standing may exacerbate the pain. Acquired lumbar spondylosis is a natural aging phenomenon with a strong genetic component that can become symptomatic.

Decompressive surgery for spinal stenosis involves techniques that remove bone from one or more structures to expand a narrowed spinal canal/neural foramen that impinges on neural structures.(16, 1811-1821) **Laminotomy** is removal of a portion of the lamina, usually to permit access to the central spinal canal to gain access to another structure such as a herniated disc or a neural foramen.**Laminectomy** refers to the complete removal of the lamina. It was traditionally performed as part of a discectomy, but is not performed any longer for that sole indication.(1822, 1823) **Hemilaminectomy** refers to removal of the left half or the right half of the lamina. **Facetectomy** is removal of part of or at times all of a facet joint. **Posterior decompression** is a term usually used to include any of the above surgeries for spinal stenosis. **Fusion** is sometimes recommended at the same time as a spinal stenosis decompression.(1824) The fusion section of these guidelines should be consulted for the indications for spine fusion performed simultaneously with decompression.

Recommendation: Decompression Surgery for Treatment of Spinal Stenosis
Decompression surgery is moderately recommended as an effective treatment for patients with symptomatic spinal stenosis (neurogenic claudication) that is intractable to conservative management. Caution is warranted among elderly with multiple comorbidities.(1825)
Indications – All of the following should be present: 1) radicular-type pain involving usually multiple dermatomes with pain and/or numbness, or myotomal muscle weakness all consistent with the nerve root levels affected; 2) imaging findings by MRI, or CT with or without myelography that confirm spinal stenosis and corroborate the dermatomal and myotomal findings predicted by the history and clinical examination; and 3) continued significant

pain and functional limitation after at least 4 to 6 weeks of time and appropriate non-operative therapy that usually includes flexion exercises plus aerobic exercise (walking or cycling),(593) and NSAIDs. Progressive neurological deficits are considered a separate indication.

Benefits – Relief of spinal stenosis-related symptoms.

Harms – Rare, but serious complications include infection, paralysis and death.

> *Strength of Evidence* – **Moderately Recommended, Evidence (B)**
> *Level of Confidence* – Moderate

Rationale for Recommendation

The highest of the moderate-quality trials reported comparable results from physical therapy (PT) consisting of flexion exercises plus aerobic exercises versus decompressive surgery over 2 years,(593) although it is noteworthy that 57% of the PT group crossed over to surgery. One trial found no significant differences between a decompressive device and epidural steroid injection.(1826) One moderate-quality trial comparing decompressive surgery with non-operative management and found superiority of decompression surgery for patients with symptomatic spinal stenosis (neurogenic claudication) that is intractable despite conservative management.(1827, 1828) The few other trials compare various operative procedures. These procedures are commonly performed in settings of either central canal stenosis, lateral recess, or neuroforaminal stenosis. Decompressive surgery is invasive, has significant adverse effects and is costly, but if there is insufficient improvement with non-operative management and/or progressive neurological deficits, it is recommended.

There is no quality evidence of benefit to adding lumbar fusion to decompression.(1829) Fusion has no role in the surgical treatment of spinal stenosis, rather the role of fusion is to treat instability if proven to be present (see Fusion).

Evidence for the Use of Decompressive Surgery

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates and then an updated search was done in PubMed for publication between 1/1/2013 and 11/15/2017. We used the following search terms: decompression surgery, decompression, back, microdiscectomy, lumbar laminectomy, open decompression, microdecompression, spinal stenosis, herniated disc and spondylolisthesis to find 8,102 articles. Of the 8,102 articles we reviewed 90 articles and 49 articles were included (30 randomized controlled trials and 19 systematic reviews).

SPINAL FUSION

Lumbar fusion involves the surgical fusion of one or more vertebral segments by inserting bone grafts (with or without instrumentation) so that the previously mobile involved segments heal together to form a single bone mass. A spinal motion segment consists of two adjacent vertebra, the connecting ligaments, two facet joints, and the interposed disc. The proposed goal of lumbar fusion is similar to that in fusing other joints in the body – that instability and pain will be significantly improved, if not resolved.(558, 1830-1863)

The U.S. has the highest rate of lumbar fusion surgery in the world (twice that of Norway, 5-fold that of England). There has been a 55% increase in spine surgery rates in the 1980s, a 6-fold variation in spine

surgery rates among U.S. cities, and 10-fold variation in spine fusion rates(1864) without evidence of beneficial outcomes.

There are some diagnoses for which fusion is either non-controversial or less controversial. These include unstable vertebral fractures or where surgery is being done for tumor, infection (osteomyelitis and/or discitis), or other disease processes that have led to spinal motion segment instability. Treatment of these conditions is outside the scope of these guidelines.

1. *Recommendation: Lumbar Fusion for Treatment of Chronic Non-specific Low Back Pain*
   Lumbar fusion is moderately not recommended as a treatment for chronic non-specific low back pain.(1865-1870)

   > *Strength of Evidence –* **Moderately Not Recommended, Evidence (B)**
   > *Level of Confidence –*Moderate

2. *Recommendation: Lumbar Fusion for Treatment of Isthmic Spondylolisthesis*
   Lumbar fusion is recommended as an effective treatment for isthmic spondylolisthesis.(1871)
   Indications – LBP with documented instability. Either i) ≥5mm of translation of the superior vertebral body on the inferior body from the full extension film to the full flexion films, and/or ii) a total angular movement during flexion and extension at the unstable level that is at least 20 degrees greater than the motion present at an adjacent disc. Lumbar fusion is also indicated for grades 3, 4, and 5 spondylolisthesis; 2) a decompressive laminectomy at an area of degenerative instability as in the case of a coexisting spondylolisthesis or scoliosis when a discectomy is performed at the same level; 3) a decompressive laminectomy performed at an area of degenerative instability, as in the case of a coexisting spondylolisthesis or scoliosis where there is gross movement on flexion-extension radiographs; and 4) a decompressive laminectomy at an area of degenerative instability as in the case of a coexisting spondylolisthesis or scoliosis where an adequate decompression requires the removal of greater than 50% of both facets or the complete removal of a unilateral facet complex.(1872)
   Benefits – Reduction in back pain and neurological compromise if present.
   Harms – Operative complications, rare severe outcomes (e.g., paralysis, fatalities), increased further re-operative risk, cost, increased risk of disability.

   > *Strength of Evidence –* **Recommended, Evidence (C)**
   > *Level of Confidence –*Moderate

3. *Recommendation: Lumbar Fusion for Treatment of Degenerative Spondylolisthesis*
   Lumbar fusion is recommended as an effective treatment for degenerative spondylolisthesis.
   Indications – LBP with documented instability. Either i) ≥5mm of translation of the superior vertebral body on the inferior body from the full extension film to the full flexion films, and/or ii) a total angular movement during flexion and extension at the unstable level that is at least 20 degrees greater than the motion present at an adjacent disc. Lumbar fusion is also indicated for grades 3, 4, and 5 spondylolisthesis; 2) a decompressive laminectomy at an area of degenerative instability as in the case of a coexisting spondylolisthesis or scoliosis when a discectomy is performed at the same level; 3) a decompressive laminectomy performed at an area of degenerative instability, as in the case of a coexisting spondylolisthesis or scoliosis where there is gross movement on flexion-extension radiographs; and 4) a decompressive laminectomy at an area of degenerative instability as in the case of a coexisting spondylolisthesis or scoliosis where an adequate decompression requires

the removal of greater than 50% of both facets or the complete removal of a unilateral facet complex.(1872)
Benefits – Reduction in back pain and neurological compromise if present.
Harms – Operative complications, rare severe outcomes (e.g., paralysis, fatalities), increased further re-operative risk, cost, increased risk of disability.

> *Strength of Evidence* – **Recommended, Evidence (C)**
> *Level of Confidence* – Moderate

4. *Recommendation: Lumbar Fusion for Treatment of Radiculopathy from Disc Herniation or Chronic Low Back Pain*
Lumbar fusion is not recommended to treat radiculopathy from disc herniation or for most patients with chronic low back pain after lumbar discectomy. Exceptions are rare but include large foraminal herniations with need to remove the facet joint to access the disc.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Moderate

5. *Recommendation: Spinal Fusion with Third Discectomy*
Spinal fusion is recommended as an option at the time of discectomy if a patient is having the third lumbar discectomy on the same disc.
Indications – Meeting indications for a third discectomy on the same disc.
Benefits – Theoretical reduced risk of 4th surgery on the same disc.
Harms – Longer recovery, greater rate of complications, higher costs.

> *Strength of Evidence* – **Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

6. *Recommendation: Spinal Fusion for Treatment of Spinal Stenosis without Concomitant Instability or Deformity*
Lumbar fusion is not recommended for treatment of spinal stenosis unless concomitant instability or deformity has been proven.(1827, 1828)

> *Strength of Evidence* – **Not Recommended, Evidence (C)**
> *Level of Confidence* – Moderate

Rationale for Recommendations: General Issues Regarding Fusion
There are many quality studies on fusion, although most are somewhat handicapped as they have heterogenous populations of patients and insufficient sample sizes with which to assess differences between diagnostic entities. There are no RCTs on patients with what are widely considered as unequivocal indications for lumbar fusion surgery such as unstable fracture, spinal infections, or tumors. There are many trials showing equivalent outcomes in non-operatively managed, neurologically-intact patients with thoracolumbar burst fractures compared with various surgeries.(1873-1876) Treatment of these conditions is outside the scope of this guideline. This guideline also does not address human bone morphogenetic protein-2(1873-1893) or osteoconductive bone graft extenders.(1874, 1894-1900)

There are no RCTs using lumbar fusion for either acute or subacute non-specific LBP. Lumbar fusion has been proposed as treatment for spondylolisthesis,(1901) disc herniation, spinal stenosis, and chronic non-specific LBP (also referred to as degeneration of the disc, discogenic LBP, micro instability, black disc disease, and lumbar spondylosis).

There are numerous methodological issues affecting the quality of the literature on this subject and these methodological issues impair the ability to draw robust evidence-based conclusions. These difficulties have been widely noted(34, 1847, 1853, 1858, 1902-1906) and these quality problems in the underlying original research are underscored by the sharply differing conclusions in the systematic reviews. Many of these conflicts likely originate from the problem that case series tend to show benefits while subsequent RCTs may or may not support the original impressions from the uncontrolled or less well designed studies.

Chronic LBP patients can be extremely difficult to manage, particularly when the pain is severe, narcotics and other drug issues are present, adherence to exercise regimens is weak, psychosocial stressors are present, and coping skills are poor (2425). Patients without indications often come to view these surgical procedures as potential cures. Lumbar fusion is the most invasive of the commonly performed lumbar surgeries. It is high cost and has significant risks of complications. However, for a select few chronic LBP patients with specific indications, it may be recommended.

Rationale for Recommendations: Fusion Complication Rates
Compared with matched non-surgical controls, patients on worker's compensation reportedly have worse outcomes with over 5.5-fold greater permanent disability status, greater opioid use, greater than 3.6-fold days of work lost and 26% of surgical patients underwent a second surgery.(1854) Risks of increased opioids use among those with prior use and 13% without pre-operative use becoming chronic users after fusion surgery suggest risks are considerable (2426). Following lumbar fusion, reoperation rates within 2 years have been estimated to range from 5.4 to 22% in the recent well-designed RCTs.(1907, 1908) A 1990s population-based study found the reoperation rate following lumbar fusion was 17 to 21% when assessed at 11-year follow up.(1909) There appears to be increased risk of reoperation if the initial diagnosis is herniated disc, degeneration of the disc, or spinal stenosis. Patients subjected to more invasive procedures have increased blood loss, longer operative times, and/or poorer outcomes in all higher quality studies where such data have been reported.(1907, 1910-1916) Overall, reported complication rates range from 1.4 to 40% (excluding scoliosis).(1902, 1907, 1913, 1917)

Rationale for Recommendations: Instability Issues
There is controversy in the medical literature about the definition of proven spinal instability. The Evidence-based Practice Spine Panel recognizes the controversy(1918) and recommends the following definition be used with flexion-extension bending films done standing with a 72 inch tube to film distance: These films should be taken digitally, and a CD with the films and the software to permit viewing and computer measurement of the translation distance should be retained and kept available for review. The first criterion is ≥5mm of translation of the superior vertebral body on the inferior body from the full extension film to the full flexion films. The other criterion is having a total angular movement during flexion and extension at the unstable level that is at least 20 degrees greater than the motion present at an adjacent disc.

Rationale for Recommendations: Fusion for Chronic Non-Specific Low Back Pain
The terms degeneration of the disc, degenerative disc disease, "discogenic back pain," "black disc disease," "micro instability," and "lumbar spondylosis" are used interchangeably to describe the same group of patients with chronic LBP in whom the pain generating structure is not defined. Discography has been used to attempt to define the lower back disc structures as the pain source, but has been largely unsuccessful in so doing (see Discography above). Chronic back pain thought to arise from

degeneration of the disc is complex and can be difficult to treat. Current surgical treatment modalities are controversial. Since there is no reliable method to identify the source of a patient's pain, surgery for pain would presumably be unlikely to be helpful. Nevertheless, this theory has been attempted to be tested.

There are 3 moderate-quality comparative trials of fusion vs. rehabilitation programs for treatment of chronic LBP and two of them suggest fusion is inferior to rehabilitation.(1865-1870, 1907, 1912, 1913, 1919, 1920) The third study reported surgical fusion improved upon standard conservative care,(1912, 1919) however, the wait-listed control group's treatment consisted of "more of the same" that previously failed,(1921) while anticipating surgery and thus likely biasing the design. In addition, Fritzell's patients were highly selected (each surgeon did on average 2 fusions for chronic back pain each year). They had a much lower incidence of depressive symptoms than is seen in typical chronic LBP populations. Benefits from fusion were on average small (on average 30% improvement), and about 1 in 6 patients became pain free. The study was not blinded and improvement in outcomes from fusion over non-operative treatment decreased over time.(1922) These studies demonstrate that if there is a benefit from fusion, it is not much.(1865-1867) A meta-analysis of RCTs found that at an average 11 years after surgery/randomization, there is no demonstrable benefit for fusion surgery among these patients and there was more adjacent segment disease among those undergoing fusion surgery although it was not clinical significant (2393-2398).

In a pooled study, the surgical group incurred reoperations (23%), worse disability (53% vs. 32% disability pensions) and greater fear avoidant beliefs.(1923) There are no published RCTs of lumbar fusion in a US workers' compensation population. There are four retrospective cohort studies in worker's compensation systems, and these show the results of fusion are significantly worse than in a non-workers' compensation population.(480, 1854, 1924, 1925) Thus, there is not quality evidence to support fusion for chronic non-specific LBP in any population, and evidence of considerably worse outcomes in workers.

Rationale for Recommendation: Fusion for Isthmic Spondylolisthesis
For isthmic spondylolisthesis, there is one moderate-quality trial comparing fusion with non-operative care that reported benefits of surgery.(1871) Thus, fusion is recommended for this indication. The literature available pertains to lumbar fusion for treatment of Grade 1 and Grade 2 spondylolisthesis. There is no quality evidence on Grade 3, Grade 4, and Grade 5 spondylolisthesis, but these are rare conditions, and when nerve roots are compromised, fusion is widely viewed as indicated.

Rationale for Recommendation: Lumbar Fusion for Treatment of Degenerative Spondylolisthesis
There is one moderate quality trial comparing fusion with non-operative care for degenerative spondylolisthesis. This trial reported negative results, however the trial reported approximately 40% crossovers and so it may have inadvertently negated the value of the trial as there were no differences in the "intention to treat" analysis, but better outcomes for fusion in the "as treated" analysis.(1917) One comparative trial of spinal fusion with spinal fusion plus decompressive surgery for treatment of adult spondylolisthesis found no additive benefits of the decompressive surgery.(122) Another trial of unilateral compared with bilateral fusion found no significant differences.(1926) Thus, the highest quality evidence suggests there may be a beneficial effect of fusion surgery for treatment of isthmic spondylolisthesis and it is also believed to be true for degenerative spondylolisthesis and thus it is recommended. The literature available pertains to lumbar fusion for treatment of Grade 1 and Grade 2

spondylolisthesis. There is no quality evidence on Grade 3, Grade 4, and Grade 5 spondylolisthesis, but these are rare conditions, and when nerve roots are compromised, fusion is widely viewed as indicated.

Rationale for Recommendation: Lumbar Fusion for Treatment of Radiculopathy from Disc Herniation or Chronic Low Back Pain
There are no quality trials in these patients. Without other indications for more extensive surgery, far less invasive surgical options (e.g., non-operative management, discectomy etc.) than fusion are available and are recommended for treatment. Thus, fusion for these patients is not recommended.

Rationale for Recommendation: Spinal Fusion with Third Discectomy
There are no quality trials on these patients. If there is a second herniation of the same disc, repeat discectomy results in comparable outcomes and is recommended.(1927-1930) However, among those having undergone two prior discectomies, it is believed to be a reasonable option to attempt fusion to avoid the theoretical need for a 4th discectomy.

Rationale for Recommendation: Spinal Fusion for Treatment of Spinal Stenosis without Concomitant Instability or Deformity
Decompressive surgery (reviewed above), is a less extensive surgical approach that resolves these issues. Additionally, one moderate-quality trial reported no advantage of fusion over decompression for foraminal stenosis.(1931) In the absence of proven instability or deformity, fusion is not recommended.

Rationale for Recommendations: Other
There are many other comparative trials with different approaches and techniques. One pattern present is quality evidence of higher rates of fusion from use of an electromagnetic device compared with sham in all three high- and moderate-quality trials.(1932-1934)

Evidence for the Use of Spinal Fusion
We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without limits on publication dates and then an updated search was conducted using PubMed for publications between 1/1/2013 and 11/15/2017. We used the following search terms: fusion, spinal fusion, spondylodesis, spondylosyndesis, back, chronic low back pain, and random* to find 47,070 articles. Of the 47,070 articles we reviewed 270 articles and included 270 articles (109 randomized controlled trials and 161 systematic reviews).

DISC REPLACEMENT
Artificial disc replacement was devised as an alternative to fusion for the patient with chronic non-specific LBP thought to be disc-related(1859, 1935-1938) as well as for focal lumbar stenosis.(1939) Its theoretical advantage is that it preserves motion in the involved vertebral segment thus purportedly decreasing the chances of degenerative changes developing at the adjacent motion segments. The term "adjacent segment disease" is used to describe patients with degenerative changes (that are presumed to be painful) at the spinal level above or below a spinal motion segment that has been treated, for example, by spinal fusion.(1940)

1. *Recommendation: Disc Replacement for Subacute or Chronic Lumbar Radiculopathy or Myelopathy*

There is no recommendation for artificial disc replacement as a treatment for subacute or chronic radiculopathy or myelopathy.[xv]

> *Strength of Evidence –* **No Recommendation, Evidence (I)**
> *Level of Confidence –* Low

Recommendation: Disc Replacement for Treatment of Chronic Non-specific Low Back Pain or Other Spinal Pain Syndrome
Artificial disc replacement is not recommended as a treatment for chronic non-specific low back pain or any other spinal pain syndrome.
> *Strength of Evidence –* **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence –* Low

Rationale for Recommendation
There is one moderate-quality trial comparing disc replacement with only ~2 weeks of a rehabilitation program, showing some evidence of superiority over 2 years based on Oswestry Disability Index scores, however, the study reported actually worse adjacent segment disease and facet degeneration in the surgical arm(1941-1943) and no significant advantage in range of motion.(1944) The rehabilitation was so short that it may likely be susceptible to both undertreatment and attention biases. A few comparative RCTs suggest potential superiority of disc replacement to fusion over short to intermediate terms.(1908, 1945-1950) Results from trials are not generalizable to those with multi-level degeneration of the disc. One trial has now been reported to 5 years of follow up, suggesting superiority over fusion(1949), but no longer-term quality studies have been reported.

Available RCTs compare disc replacement to fusion (1945, 1949, 1951) and as noted in the fusion section of this Guideline, fusion has not been shown to improve the outcomes over modern non-operative care. The follow-up in the published RCTs is now up to 5 years. Some may consider this too short to be considered standard treatment. There is evidence that higher volume surgical centers have shorter hospital stays and lower complication rates.(1952) Complication rates are not inconsiderable and surgical candidates should be fully apprised of these reported complications which include 2.8 adverse events per patient, 5% device failures, 5% neurological deteriorations at 24 months compared with baseline, and 33.3% failure to have at least a 25% decrease in the ODI at 24 months compared with baseline. Additional research including demonstrated long-term safety and efficacy would be needed prior to a recommendation in support.

Evidence for the Use of Disc Replacement
We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without using any limitation on publication dates and then an updated search was done in PubMed for publication between 1/1/2013 and 11/15/2017. We used the following search terms: disc replacement, back, spinal fractures, randomized clinical trial or randomized controlled trial or random, systematic review or reviews, population study or epidemiological study or prospective cohort to find 3666 articles. Of the 3666 articles we reviewed 64 articles and included 31 articles (16 randomized controlled trials and 15 systematic reviews).

[xv] The spinal cord terminates in adults at approximately L1/L2. The term myelopathy refers to cord compression and thus generally does not happen in the lumbar spine.

VERTEBROPLASTY

Vertebroplasty, first reported in 1987,(1953) involves using image guidance to inject polymethylmethacrylate within the vertebral body, in order to stabilize vertebral fractures caused by osteoporosis,(1954-1960) vertebral osteonecrosis, or malignancies of the spinal column.(1961-1969) This procedure is most common among elderly osteoporotic patients who have delayed healing of compression fractures of the vertebral body(ies),(1970) but it is sometimes performed on younger patients with acute vertebral fractures due to osteoporosis. A work-related minor trauma may be the event that caused the osteoporotic pathologic fracture.

1. *Recommendation: Vertebroplasty for Treatment of Low Back or Thoracic Pain Due to Vertebral Compression Fractures*
   Vertebroplasty is strongly not recommended as a routine treatment for patients with low back or thoracic pain due to vertebral compression fractures.(1971, 1972)

   > *Strength of Evidence* – **Strongly Not Recommended, Evidence (A) [Subacute, Chronic]**
   > *Level of Confidence* – High
   > *Strength of Evidence* – **Not Recommended, Evidence (C) [Acute]**
   > *Level of Confidence* – Moderate

2. *Recommendation: Vertebroplasty for Treatment of Select Patients with Low Back or Thoracic Pain Due to Vertebral Compression Fractures*
   There is no recommendation for or against the use of vertebroplasty for treatment of highly select patients with low back or thoracic pain due to unusual vertebral compression fractures.

   Indications – Patients who are not included in the two available high-quality trials. These include patients who have had fractures despite bisphosphonate therapy, pathologic fractures due to neoplasms in the vertebral body, or multiple simultaneous compression fractures (three or more). Candidates for vertebroplasty should have these types of unusual vertebral body compression fractures, should generally have severe pain, passage of at least 2 months, and failure of other treatment options including medical management.

   > *Strength of Evidence* – **No Recommendation, Insufficient Evidence (I)**
   > *Level of Confidence* – Low

Rationale for Recommendations
There are multiple (2009, 2430) high-quality, sham-controlled RCTs that evaluated the efficacy of vertebroplasty and failed to find significant improvements in the patients who underwent vertebroplasty compared with a sham procedure. (1971, 1972) These results are in contrast with two moderate-quality RCTs(1973, 1974) and other low-quality studies that had reported pain relief and other functional improvements that had appeared promising.(1966, 1975-1983) There is one other quality trial which reported pain relief and increased mobility; however, that trial is of lower quality, was short term (2 weeks), and had a substantially lower sample size than both of the 2009 studies, and appears biased against pain treatment.(1984) In addition, substantial complications occur with this procedure including deaths (1966, 1972, 1985, 1986) and subsequent fractures (2399, 2400). The results of the two high-quality RCTs indicate that vertebroplasty is strongly not recommended for nearly all patients with vertebral compression fractures. It remains unclear whether there are highly selected unusual patients – such as severely affected patients, patients with 3 or more simultaneous compression fractures, or patients with pathologic fractures due to neoplasms(1987)– who were outside the scope of these two quality trials, who might still derive benefit from this procedure. This procedure is invasive, has

complications,(1988, 1989) and is costly. Therefore, vertebroplasty is not recommended other than for highly select patients who have failed other interventions (including quality medical management) and for whom there are no other options available, whose significant pain is not resolving, and especially those for whom bisphosphonate therapy has failed.

Evidence for the Use of Vertebroplasty

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without using any limitation on publication dates and then an updated search was done in PubMed for publication between 1/1/2013 and 11/15/2017. We used the following search terms: vertebroplasty,back, spinal fractures, randomized clinical trial or randomized controlled trial or random, systematic review or reviews, population study or epidemiological study or prospective cohort to find 5,167 articles. Of the 5,167 articles we reviewed 57 articles and included 30 articles (21 randomized controlled trials and 10 systematic reviews).

KYPHOPLASTY

Kyphoplasty, first introduced in 1998, has been used similarly to vertebroplasty to restore vertebral body height and improve sagittal alignment of the spine.(1964, 1985, 1990-2000) Kyphoplasty involves injection of polymethylmethacrylate within a cavity in the vertebral body that has been created by percutaneously insertion of a balloon through the involved pedicle(s).(2001) It has been suggested that kyphoplasty may be appropriate as a prophylactic procedure.(2002)

Recommendation: Kyphoplasty for Treatment of Low Back or Thoracic Pain Due to Vertebral Compression Fractures

There is no recommendation for or against the use of kyphoplasty for the treatment of low back or thoracic pain due to vertebral compression fractures.

Indications – Vertebral body compression fractures among patients with severe pain; patients who have had fractures despite bisphosphonate therapy may also be candidates. May be helpful in cases of multiple myeloma and multiple compressions for pain control.

> *Strength of Evidence* – **No Recommendation, Insufficient Evidence (I)**
> *Level of Confidence* –Low

Rationale for Recommendation

There are no quality studies comparing kyphoplasty with a sham procedure. There is one moderate-quality study comparing kyphoplasty with an unstructured, unblinded, non-interventional control that included cancer patients.(2003) This study also differentially utilized passive treatments between the two groups, such as bed rest and braces that may have confounded the results. The other moderate-quality study compared two types of cement and found the calcium phosphate cement to be inferior for burst fractures.(2001) There are comparative clinical trials and other low-quality studies suggesting benefit.(1994, 2004, 2005) These have been compiled into meta-analyses with a conclusion of efficacy (as well as efficacy of vertebroplasty).(2006-2008) Yet, as kyphoplasty is similar to vertebroplasty, and two high-quality, sham-controlled trials for vertebroplasty are now reported documenting a lack of benefit,(1971, 1972) and despite the Wardlaw study which included patients with neoplasia, it appears reasonable to assume the same lack of benefit will eventually be shown for kyphoplasty for treatment of non-cancer patients. It remains unclear whether there are highly selected, unusual patients such as those severely affected, patients with 3 or more simultaneous compression fractures, or patients with pathologic fractures due to neoplasms,(1987) who may derive benefit from this procedure. Kyphoplasty

has also been found to be associated with subsequent, adjacent vertebral compression fractures.(2009, 2010{Frankel, 2007 #8912, 2011-2013), 2399-2402) Kyphoplasty is invasive, has complications, and is costly. There is no recommendation for or against kyphoplasty other than for highly selected patients who have failed other interventions (including quality medical management), and in whom there are no other options available, whose significant pain is not resolving, and especially those for whom bisphosphonate therapy has failed.

Evidence for the Use of Kyphoplasty

We searched PubMed, EBSCO, Cochrane Review, and Google Scholar without using any limitation on publication dates and then an updated search was done in PubMed for publication between 1/1/2013 and 11/15/2017. We used the following search terms: Kyphoplasty, Back, Spinal fractures, Randomized Controlled Trial, Random, Randomized, Systematic Review, Reviews, Population study, Epidemiological study, and Prospective cohort to find 5,213 articles. Of the 5,213 articles, we reviewed 39 articles and included 21 articles (17 randomized controlled trials and 4 systematic reviews).

SACROILIAC FUSION SURGERY
Sacroiliac joint-related surgical procedures are increasingly performed (2431-2438).

Recommendation: Sacroiliac Surgery for Treatment of Low Back Pain Disorders
Sacroiliac joint fusion surgery and other sacroiliac joint surgical procedures are not recommended for treatment of low back pain disorder.

> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

Rationale for Recommendation
There are two trials with several reports comparing SI joint fusion surgery with non-operative management (2431-2432, 2439, 2440). Both trials excluded patients with worker's compensation (2439). Patients included in the larger US-based study had either SI joint disruption or degenerative SI joints (2431), but only had degenerative disease in the European study (2440). Neither of the two trials included a functional restoration program with progressive aerobic and strengthening exercises combined with CBT or sham-control (1973, 1974, 2030). Yet, in treatment of LBP, the analogous procedure of lumbar fusion has been shown to be ineffective compared with a quality rehabilitation program (see Lumbar Fusion section). There also are SI joint fusion case series (2433). Thus, there are no quality trials comparing SI joint fusion with a quality rehabilitative program.

The two moderate-quality RCTs suggest improved pain and function, but the comparison groups' treatments are ill-defined exercise and neither routinely incorporated CBT (2431, 2440). Prior studies of SI joint fusion reported relatively poor results (one study found that 18% of patients operated on were "satisfied;" 65% required additional surgery) (2014) but used different techniques than the more recent studies. Other surgical series have reported better results with unpublished results as high as 90% good or excellent.(2015-2017) Sacroiliac joint surgery is invasive, has adverse effects (10% of those ambulatory pre-operatively in one recent series using the recent appliances were not fully ambulatory 6mo. post-operatively (2433), is costly, but without quality trials addressing either sham- or quality functional restoration-control, there is no recommendation. SI fusion is a reasonable option for

treatment of severe pelvic fractures with or without instability.(67) There may be limited uses for post-traumatic, unstable SI joints that requires further definition in quality studies.

Evidence for the Use of Sacroiliac Surgery

We searched PubMed, EBSCO, Cochrane review, Google scholar without limits on publication dates. We used following search terms: sacroiliac joint fusion surgery, sacroiliac surgery, chronic low back pain, radicular pain, sciatica, and sacroiliitis to find 17026 articles. Of 17026 articles, we reviewed 12 articles and included 9 articles (9 randomized controlled trials and 0 systematic reviews).

IMPLANTABLE SPINAL CORD STIMULATORS

Spinal cord stimulators (SCSs) deliver electrical impulses to the spinal cord area through electrodes that are implanted by laminotomy or percutaneously.(2018-2021) Proponents believe that this device is successful via the gate-control theory in which stimulating nerve fibers closes other paths of pain conduction;(2022) however, this mechanism is poorly understood.(2023) (This review includes only evidence concerning indications for treatment of LBP with or without lower extremity pain. The use of SCSs for the treatment of complex regional pain syndrome is discussed in the Chronic Pain Guideline.)

Recommendation: Spinal Cord Stimulators for Treatment of Acute, Subacute, or Chronic Low Back Pain or Radicular Pain Syndromes or Failed Back Surgery Syndrome
Spinal cord stimulators are not recommended for treatment of acute, subacute, chronic low back pain, radicular pain syndromes or failed back surgery syndrome. Indications are provided for highly select circumstances when a worker has primarily radicular extremity pain, all other indicated treatments have failed, the patient has inadequate function, and the provider wishes to seek approval from a worker's compensation carrier for consideration of possible coverage despite the lack of quality evidence of efficacy in these patients.

> *Indications:* See Table 11.
> *Benefits:* Potential to improve pain and possibly function.
> *Harms:* Medicalization, paralysis, higher opioids use, fatalities. One-third of patients reportedly have adverse effects (2024).
> *Frequency/Dose/Duration:* N/A
> *Indications for Discontinuation:* Resolution of pain, complications necessitating discontinuation of therapy or device removal, or loss of therapeutic effect.
> *Strength of Evidence* – **Not Recommended, Insufficient Evidence (I)**
> *Level of Confidence* – Low

Rationale for Recommendation
There are few quality studies evaluating SCS for the treatment of LBP, none of which compared SCS with a non-surgical treatment such as a quality multi-disciplinary rehabilitation program or a sham procedure.(2025, 2026) Problems with study design have been noted for many years (2207, 2446), but to date have not been addressed in quality studies.

Reports with worker's compensation patients include a controlled, 2-year cohort study of workers' compensation patients in Washington State which found a low success rate, lack of long-term benefits, and increased opioid use among those receiving stimulators. (2027) Cost effectiveness was also not shown

in Washington State (2447), resulting in a decision to not cover the procedure for worker's compensation patients (2207).[xvi] Others have opined worker's compensation results in worse outcomes (2204, 2402).

One moderate-quality study showed reduced pain ratings by 6 and 12 months after implantation, but improvements diminished over time.(2025) One study of SCSs for complex regional pain syndrome also found diminished differences over time – SCS recommendations for the treatment of complex regional pain syndrome Type I are addressed in the Chronic Pain Guideline.(2028) A recent RCT found better efficacy with high-frequency stimulation than with traditional SCS, but had no sham- or functional restoration-controlled arm, similar to the weaknesses of prior studies (2448).

A non-RCT of 40 patients with chronic LBP with intractable leg pain attempted to determine whether operating when the patient was awake and able to provide feedback would improve outcomes.(2029) Leg scores pre-operatively at 6 months were 7.38, 4.18, 5.55, and 6.27. Total pain scores were 69.11, 54.79, 58.64, and 63.01. There appears to be a lack of lasting benefit.

Spinal cord stimulators are costly (2442),[xvii] invasive, have reported serious complications (including surgical procedures for loose leads, repairs, and surgical removal of the devices), and have a significant revision rate.(2030, 2031) Without quality evidence of enduring efficacy compared with either sham-control or a quality functional restoration program, SCS is not recommended. Potential indications are provided in Table 11 in the event that there is a patient with predominant radicular pain, unamenable to surgery, with inadequate function after complying with functional restoration program components for at least 6 months who wishes to seek potential approval from a worker's compensation insurer.

Evidence for the Use of Implantable Spinal Cord Stimulators

We searched PubMed, EBSCO, Google Scholar, and Cochrane Review with no limits on publication dates and then an updated search was conducted using PubMed for publications between 1/1/2013 and 11/15/2017. We used the following search terms: Spinal cord stimulator, spinal cord stimulation,) sub-acute low back pain, chronic low back pain, radicular pain syndromes, sciatica, back, and random to find 9106 articles. Of the 9106 articles, we reviewed 31 articles and included 31 articles (9 randomized controlled trials and 22 systematic reviews).

Table 11. Selection Criteria for Implantable Spinal Cord Stimulator in a Chronic Radiculopathy Patient*

1. Clear diagnosis of chronic radiculopathy including supportive evidence on electrodiagnostic study. Leg pain should predominate over axial back pain (2449)

[xvii] A cost-effectiveness analysis from Canada has been used to support cost-effectiveness of SCS. The cost analyses for conservative care included annual, 3-day hospitalizations for breakthrough pain ($9,405 total), 24 annual visits with a family physician, and physician therapy charges over 5 years (estimated at $8,680). Five-year costs were estimated at $28,123 SCS versus $38,029 for conservative care. Hospitalization for breakthrough pain ($9,405) is highly unusual in the U.S., and without that expense (without consideration of the other unusual numbers of visits), the fiscal advantage of SCS completely disappeared. As the study contains unusual assumptions and elimination of hospitalization causes the purported fiscal advantage of the SCS to disappear, the conclusions of this study do not appear applicable to typical U.S. patients. A second cost-effectiveness estimate in the United Kingdom reported approximately 4.8-fold higher costs in those receiving SCS (2442). Neither study had surgical costs reasonably close to US costs.

2. Poor or inadequate response to surgical treatment such as discectomy.
3. Poor or inadequate response to functional restoration program with treatment generally for at least 6 months.** Program shoud have been in an experienced interdisciplinary clinic with proven good outcomes that included core, emphasized elements of progressive aerobic exercise, strengthening, and cognitive behavioral therapy, and for which the patient demonstrated good compliance.
4. Remedial surgery inadvisable or not feasible.
5. Major psychiatric disorders have been treated with expected responses. Somatization disorder not amenable to treatment disqualifies the patient for use of invasive procedures, as the risk of the procedure is higher than the expected success rate. The candidate should have a successful independent, psychological evaluation and a structured interview performed by a psychologist specialized in chronic pain management including appropriate psychometric testing (see Chronic Pain guideline, Appendix 1). The psychological evaluation should be performed by a practitioner who is not employed by the requesting or treating physicians.***
6. Willingness to stop inappropriate drug use before implantation.
7. No indication that secondary gain is directly influencing pain or disability complaints.
8. Ability to give informed consent for the procedure.
9. Successful results of at least 50% pain reduction from a trial of a temporary external stimulator of approximately 2-3 days and reduction of use of opioid medication or other medication with significant adverse effects or functional improvement such as return to work that may be evaluated by an occupational or physical therapist prior to and before discontinuation of the trial.

*Adapted from Kumar K, Hunter G, Demeria D. Spinal cord stimulation in treatment of chronic benign pain: challenges in treatment planning and present status, a 22-year experience. *Neurosurgery*. 2006;58(3):481-96[; Lee AW, Pilitsis JG. Spinal cord stimulation: indications and outcomes. *Neurosurg Focus*. 2006;21(6):E3[38]; Segal R, Stacey BR, Rudy TE, et al. Spinal cord stimulation revisited. *Neurol Res*. 1998;20(5):391-6.(873)
**Some authors advocate earlier intervention,(37, 859); however, quality evidence is lacking.
***Presence of depression is common in patients with chronic pain, requires evaluation and may require treatment. Depression that is particularly severe may require treatment prior to assessing appropriateness of SCS, however, the presence of depression does not preclude SCS.

Rehabilitation for Delayed Recovery

If an individual fails to recover within the appropriate biological healing timeframe, the acute care paradigms of specific diagnosis and treatment change to biopsychosocial approaches that address pain, function, work, and psychological distress that impede progress. Such programs focus on restoration of work-related function. These programs include work conditioning, work hardening, functional rehabilitation, behavioral interventions, chronic pain programs, and other interdisciplinary approaches. They may also include education about risk/rewards of declined surgical procedures.(548)

Initiation of these programs should be considered in the subacute stage if disability is not adequately explained by physical findings (see Chronic Pain Guideline). Chronicity by itself is a major predictor of poor outcome.(2032) The longer it takes to resolve the disability (delayed recovery), the higher the cost, the less likely patients are to return to work at all, the greater the risk for costly medical care, and the greater the likelihood for costs to be shifted from the workers' compensation system to other payment systems (e.g., long-term disability, Social Security Disability Insurance). The increased costs of rehabilitation programs may be justified by cost benefit analysis of program outcomes. Consistent with the above, earlier intervention should be considered.

See the recommendations in the Chronic Pain Guideline for the following:
- Work Conditioning, Work Hardening, Early Intervention Programs, and Back Schools for Chronic Pain
- Interdisciplinary Pain Rehabilitation Programs, Multidisciplinary Rehabilitation Programs, Chronic Pain Management Programs, and Functional Restoration Programs
- Participatory Ergonomics Programs for Patients with Chronic Pain

- [Psychological Evaluation for Chronic Pain Patients](#)
- [Cognitive Behavioral Therapy for Patients with Chronic Pain](#)
- [Fear Avoidance Belief Training](#)
- [Biofeedback](#)

References

1.   Melhorn J, Talmage J, Ackerman III W, Hyman M. *AMA Guides® to the Evaluation of Disease and Injury Causation, second edition*. Chicago, IL: American Medical Association; 2014.
2.   Center for the Evaluative Clinical Sciences. Spine surgery. A Report by the Dartmouth Atlas of Health Care. CMS-FDA Collaborative. 2006.
3.   Centers for Disease Control and Prevention. Vital signs: overdoses of prescription opioid pain relievers---United States, 1999--2008. *MMWR Morbidity and mortality weekly report*. 2011;60:1487-1492.
4.   Centers for Disease Control and Prevention (CDC). Vital signs: risk of overdose from methadone used for pain relief-United States, 1999-2010. *MMWR Morbidity and mortality weekly report*. 2012;61::493-497.
5.   Institute of Medicine. Standards for Developing Trustworthy Clinical Practice Guidelines. Available at: http://nationalacademies.org/hmd/Activities/Quality/ClinicPracGuide.aspx. 2011.
6.   The AGREE Research Trust. Appraisal of Guidelines for Research & Evaluation II (AGREE II) Instrument. 2009.
7.   American College of Occupational and Environmental Medicine. Methodology for the Update of the Occupational Medicine Practice Guidelines. Available at: https://insights.ovid.com/crossref?an=00043764-200803000-00006. 2008.
8.   American College of Occupational and Environmental Medicine. Summary: Methodology for Updates to the ACOEM Practice Guidelines. Available at: https://acoem.org/acoem/media/PracticeResources/Methodology-2017-Update.pdf. 2017.
9.   Harris JS, Sinnott PL, Holland JP, et al. Methodology to update the practice recommendations in the American College of Occupational and Environmental Medicine's Occupational Medicine Practice Guidelines, second edition. *Journal of occupational and environmental medicine / American College of Occupational and Environmental Medicine*. 2008;50:282-295.
10.   Griffith LE, Hogg-Johnson S, Cole DC, et al. Low-back pain definitions in occupational studies were categorized for a meta-analysis using Delphi consensus methods. *Journal of clinical epidemiology*. 2007;60:625-633.
11.   Heliovaara M, Sievers K, Impivaara O, et al. Descriptive epidemiology and public health aspects of low back pain. *Annals of medicine*. 1989;21:327-333.
12.   Livshits G, Popham M, Malkin I, et al. Lumbar disc degeneration and genetic factors are the main risk factors for low back pain in women: the UK Twin Spine Study. *Annals of the rheumatic diseases*. 2011;70:1740-1745.
13.   Balague F, Mannion AF, Pellise F, Cedraschi C. Non-specific low back pain. *Lancet*. 2012;379:482-491.
14.   Hoy D, Brooks P, Blyth F, Buchbinder R. The Epidemiology of low back pain. *Best practice & research*. 2010;24:769-781.
15.   U.S. Department of Labor, Bureau of Labor Statistics. Nonfatal Occupational Injuries and Illnesses Requiring Days Away from Work, 2012. 2013.
16.   Atlas SJ, Chang Y, Keller RB, Singer DE, Wu YA, Deyo RA. The impact of disability compensation on long-term treatment outcomes of patients with sciatica due to a lumbar disc herniation. *Spine*. 2006;31:3061-3069.
17.   Eccleston S, Petrova P, Zhao X. *The Anatomy of Workers' Compensation Medical Costs and Utilization, 6th Ed (10 volume)*. Cambridge, MA: Workers Compensation Research Institute; 2007.
18.   Webster BS, Snook SH. The cost of 1989 workers' compensation low back pain claims. *Spine*. 1994;19:1111-1115; discussion 1116.
19.   Silverstein B, Viikari-Juntura E, Kalat J. Use of a prevention index to identify industries at high risk for work-related musculoskeletal disorders of the neck, back, and upper extremity in Washington state, 1990-1998. *American journal of industrial medicine*. 2002;41:149-169.
20.   Bekelman JE, Li Y, Gross CP. Scope and impact of financial conflicts of interest in biomedical research: a systematic review. *Jama*. 2003;289:454-465.
21.   Shah RV, Albert TJ, Bruegel-Sanchez V, Vaccaro AR, Hilibrand AS, Grauer JN. Industry support and correlation to study outcome for papers published in Spine. *Spine*. 2005;30:1099-1104; discussion 1105.
22.   Steinbrook R. Peer review and federal regulations. *N Engl J Med*. 2004;350:103-104.
23.   Al-Saeed O, Al-Jarallah K, Raeess M, Sheikh M, Ismail M, Athyal R. Magnetic resonance imaging of the lumbar spine in young arabs with low back pain. *Asian spine journal*. 2012;6:249-256.

24.   Kalichman L, Guermazi A, Li L, Hunter DJ. Association between age, sex, BMI and CT-evaluated spinal degeneration features. *Journal of back and musculoskeletal rehabilitation*. 2009;22:189-195.

25.   Klaassen Z, Tubbs RS, Apaydin N, Hage R, Jordan R, Loukas M. Vertebral spinal osteophytes. *Anatomical science international*. 2011;86:1-9.

26.   Brinjikji W, Luetmer PH, Comstock B, et al. Systematic literature review of imaging features of spinal degeneration in asymptomatic populations. *AJNR American journal of neuroradiology*. 2015;36:811-816.

27.   Mannion AF, Muntener M, Taimela S, Dvorak J. Comparison of three active therapies for chronic low back pain: results of a randomized clinical trial with one-year follow-up. *Rheumatology (Oxford, England)*. 2001;40:772-778.

28.   Kankaanpaa M, Taimela S, Airaksinen O, Hanninen O. The efficacy of active rehabilitation in chronic low back pain. Effect on pain intensity, self-experienced disability, and lumbar fatigability. *Spine*. 1999;24:1034-1042.

29.   Airaksinen O, Brox JI, Cedraschi C, et al. Chapter 4. European guidelines for the management of chronic nonspecific low back pain. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2006;15 Suppl 2:S192-300.

30.   Cohen I, Rainville J. Aggressive exercise as treatment for chronic low back pain. *Sports medicine (Auckland, NZ*. 2002;32:75-82.

31.   Danielsen JM, Johnsen R, Kibsgaard SK, Hellevik E. Early aggressive exercise for postoperative rehabilitation after discectomy. *Spine*. 2000;25:1015-1020.

32.   Fardon DF, Williams AL, Dohring EJ, Murtagh FR, Gabriel Rothman SL, Sze GK. Lumbar disc nomenclature: version 2.0: recommendations of the combined task forces of the North American Spine Society, the American Society of Spine Radiology, and the American Society of Neuroradiology. *Spine*. 2014;39:E1448-1465.

33.   Kamper SJ, Maher CG, Hancock MJ, Koes BW, Croft PR, Hay E. Treatment-based subgroups of low back pain: a guide to appraisal of research studies and a summary of current evidence. *Best practice & research Clinical rheumatology*. 2010;24:181-191.

34.   Gibson JN, Waddell G. Surgery for degenerative lumbar spondylosis. *The Cochrane database of systematic reviews*. 2005:CD001352.

35.   Gross DP, Battie MC, Asante A. Development and validation of a short-form functional capacity evaluation for use in claimants with low back disorders. *J Occup Rehabil*. 2006;16:53-62.

36.   Hahne AJ, Ford JJ. Functional restoration for a chronic lumbar disk extrusion with associated radiculopathy. *Physical therapy*. 2006;86:1668-1680.

37.   Poiraudeau S, Rannou F, Revel M. Functional restoration programs for low back pain: a systematic review. *Ann Readapt Med Phys*. 2007;50:425-429.

38.   Boyle G. Review of the McGill Pain Questionnaire. In: Plake B, Impara J, eds. *The 14th Mental Measurements Yearbook*. Lincoln: Buros Institute; 2001.

39.   Boyle GJ, Boerresen BH, Jang DM. Factor Analyses Of The Mcgill Pain Questionnaire (Mpq) In Acute And Chronic Pain Patients. *Psychological reports*. 2015;116:797-820.

40.   Byrne M, Troy A, Bradley LA, et al. Cross-validation of the factor structure of the McGill Pain Questionnaire. *Pain*. 1982;13:193-201.

41.   Melzack R. The McGill Pain Questionnaire: major properties and scoring methods. *Pain*. 1975;1:277-299.

42.   Fairbank JC, Couper J, Davies JB, O'Brien JP. The Oswestry low back pain disability questionnaire. *Physiotherapy*. 1980;66:271-273.

43.   Roland M, Fairbank J. The Roland-Morris Disability Questionnaire and the Oswestry Disability Questionnaire. *Spine*. 2000;25:3115-3124.

44.   Bruns D. Clinical and Forensic Standards for the Psychological Assessment of Patients with Chronic Pain. *Psychol Inj and Law*. 2014;7:297-316.

45.   Grotle M, Brox JI, Vollestad NK. Functional status and disability questionnaires: what do they assess? A systematic review of back-specific outcome questionnaires. *Spine (Phila Pa 1976)*. 2005;30:130-140.

46.   Frymoyer JW. Back pain and sciatica. *N Engl J Med*. 1988;318:291-300.

47.   Ropper AH, Zafonte RD. Sciatica. *N Engl J Med*. 2015;372:1240-1248.

48.   Warren JC, Dearborn HAS. Cases of Sciatica. *The New England Journal of Medicine, Surgery and Collateral Branches of Science*. 1821;10:111-117.

49.   Colorado Division of Workers' Compensation. *Rule 17, Exhibit 9: Chronic Pain Disorder Medical Treatment Guidelines*: Colorado Department of Labor and Employment: Division of Worker Compensation 2012.

50.   Bruns D, Disorbio JM. Assessment of biopsychosocial risk factors for medical treatment: a collaborative approach. *Journal of clinical psychology in medical settings*. 2009;16:127-147.

51.   Fardon DF, Milette PC. Nomenclature and classification of lumbar disc pathology. Recommendations of the Combined task Forces of the North American Spine Society, American Society of Spine Radiology, and American Society of Neuroradiology. *Spine (Phila Pa 1976)*. 2001;26:E93-E113.

52.   Hou L, Hsu A, Veeravagu A, Boakye M. Spinal gout in a rental transplant patient: a case report and literature review. *Surgical neurology*. 2007;67:65-73.

53.   Kalichman L, Li L, Kim DH, et al. Facet joint osteoarthritis and low back pain in the community-based population. *Spine*. 2008;33:2560-2565.

54.   Eubanks JD, Lee MJ, Cassinelli E, Ahn NU. Prevalence of lumbar facet arthrosis and its relationship to age, sex, and race: an anatomic study of cadaveric specimens. *Spine*. 2007;32:2058-2062.

55.   Schwarzer AC, Wang SC, Bogduk N, McNaught PJ, Laurent R. Prevalence and clinical features of lumbar zygapophysial joint pain: a study in an Australian population with chronic low back pain. *Ann Rheum Dis*. 1995;54:100-106.

56.   Cheung KM, Karppinen J, Chan D, et al. Prevalence and pattern of lumbar magnetic resonance imaging changes in a population study of one thousand forty-three individuals. *Spine*. 2009;34:934-940.

57.   de Schepper E, Damen J, van Meurs J, Ginai A, Popham M, Hofman A, et al. The association between lumbar disc degeneration and low back pain: the influence of age, gender, and individual radiographic features. *Spine*. 2010;35:531-536.

58.   Endean A, Palmer KT, Coggon D. Potential of magnetic resonance imaging findings to refine case definition for mechanical low back pain in epidemiological studies: a systematic review. *Spine*. 2011;36:160-169.

59.   Hooten WM, Martin DP, Huntoon MA. Radiofrequency neurotomy for low back pain: evidence-based procedural guidelines. *Pain Med*. 2005;6:129-138.

60.   Mayer TG, Gatchel RJ, Keeley J, McGeary D, Dersh J, Anagnostis C. A randomized clinical trial of treatment for lumbar segmental rigidity. *Spine (Phila Pa 1976)*. 2004;29:2199-2205; discussion 2206.

61.   Mayer TG, Robinson R, Pegues P, Kohles S, Gatchel RJ. Lumbar segmental rigidity: can its identification with facet injections and stretching exercises be useful? *Archives of physical medicine and rehabilitation*. 2000;81:1143-1150.

62.   Slipman CW, Sterenfeld EB, Chou LH, Herzog R, Vresilovic E. The predictive value of provocative sacroiliac joint stress maneuvers in the diagnosis of sacroiliac joint syndrome. *Archives of physical medicine and rehabilitation*. 1998;79:288-292.

63.   van der Wurff P, Buijs EJ, Groen GJ. Intensity mapping of pain referral areas in sacroiliac joint pain patients. *Journal of manipulative and physiological therapeutics*. 2006;29:190-195.

64.   van der Wurff P, Buijs EJ, Groen GJ. A multitest regimen of pain provocation tests as an aid to reduce unnecessary minimally invasive sacroiliac joint procedures. *Arch Phys Med Rehabil*. 2006;87:10-14.

65.   Merskey H, Bogduk N, Eds. *Classification of Chronic Pain: Descriptions of Chronic Pain Syndromes and Definitions of Pain Terms*. Seattle, Wash: IASP Press; 1994.

66.   Van der Wurff P MW, Hagmeijer RH. Clinical tests of the sacroilliac joint. *Manual therapy*. 2000;5.

67.   Foley BS, Buschbacher RM. Sacroiliac joint pain: anatomy, biomechanics, diagnosis, and treatment. *American journal of physical medicine & rehabilitation / Association of Academic Physiatrists*. 2006;85:997-1006.

68.   Maigne JY, Planchon CA. Sacroiliac joint pain after lumbar fusion. A study with anesthetic blocks. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2005;14:654-658.

69.   Katz V, Schofferman J, Reynolds J. The sacroiliac joint: a potential cause of pain after lumbar fusion to the sacrum. *Journal of spinal disorders & techniques*. 2003;16:96-99.

70.   Battistone MJ, Manaster BJ, Reda DJ, Clegg DO. Radiographic diagnosis of sacroiliitis--are sacroiliac views really better? *The Journal of rheumatology*. 1998;25:2395-2401.

71.   Hansen HC, Helm S, 2nd. Sacroiliac joint pain and dysfunction. *Pain Physician*. 2003;6:179-189.

72.   Clare HA, Adams R, Maher CG. Reliability of McKenzie classification of patients with cervical or lumbar pain. *Journal of manipulative and physiological therapeutics*. 2005;28:122-127.

73.   Kilpikoski S, Airaksinen O, Kankaanpaa M, Leminen P, Videman T, Alen M. Interexaminer reliability of low back pain assessment using the McKenzie method. *Spine*. 2002;27:E207-214.

74.   Razmjou H, Kramer JF, Yamada R. Intertester reliability of the McKenzie evaluation in assessing patients with mechanical low-back pain. *The Journal of orthopaedic and sports physical therapy*. 2000;30:368-383; discussion 384-369.

75.   Riddle DL, Rothstein JM. Intertester reliability of McKenzie's classifications of the syndrome types present in patients with low back pain. *Spine*. 1993;18:1333-1344.

76.   Donelson R, Silva G, Murphy K. Centralization phenomenon. Its usefulness in evaluating and treating referred pain. *Spine*. 1990;15:211-213.

77.   Karas R, McIntosh G, Hall H, Wilson L, Melles T. The relationship between nonorganic signs and centralization of symptoms in the prediction of return to work for patients with low back pain. *Physical therapy*. 1997;77:354-360; discussion 361-359.

78.   Sufka A, Hauger B, Trenary M, et al. Centralization of low back pain and perceived functional outcome. *The Journal of orthopaedic and sports physical therapy*. 1998;27:205-212.

79.   Werneke M, Hart DL, Cook D. A descriptive study of the centralization phenomenon. A prospective analysis. *Spine*. 1999;24:676-683.

80.   Donelson R, Aprill C, Medcalf R, Grant W. A prospective study of centralization of lumbar and referred pain. A predictor of symptomatic discs and anular competence. *Spine*. 1997;22:1115-1122.

81.   Kopp JR, Alexander AH, Turocy RH, Levrini MG, Lichtman DM. The use of lumbar extension in the evaluation and treatment of patients with acute herniated nucleus pulposus. A preliminary report. *Clinical orthopaedics and related research*. 1986:211-218.

82.   Laslett M, Oberg B, Aprill CN, McDonald B. Centralization as a predictor of provocation discography results in chronic low back pain, and the influence of disability and distress on diagnostic power. *The spine journal : official journal of the North American Spine Society*. 2005;5:370-380.

83.   Long AL. The centralization phenomenon. Its usefulness as a predictor or outcome in conservative treatment of chronic low back pain (a pilot study). *Spine*. 1995;20:2513-2520; discussion 2521.

84.   Donelson R, Long A, Spratt K, Fung T. Influence of directional preference on two clinical dichotomies: acute versus chronic pain and axial low back pain versus sciatica. *PM & R : the journal of injury, function, and rehabilitation*. 2012;4:667-681.

85.   Long A, Donelson R, Fung T. Does it matter which exercise? A randomized control trial of exercise for low back pain. *Spine*. 2004;29:2593-2602.

86.   Skytte L, May S, Petersen P. Centralization: its prognostic value in patients with referred symptoms and sciatica. *Spine*. 2005;30:E293-299.

87.   Takasaki H, May S. Mechanical diagnosis and therapy has similar effects on pain and disability as 'wait and see' and other approaches in people with neck pain: a systematic review. *Journal of physiotherapy*. 2014;60:78-84.

88.   Melzack R. From the gate to the neuromatrix. *Pain*. 1999;Suppl 6:S121-126.

89.   Melzack R. Pain and the neuromatrix in the brain. *Journal of dental education*. 2001;65:1378-1382.

90.   Melzack R. Evolution of the neuromatrix theory of pain. The Prithvi Raj Lecture: presented at the third World Congress of World Institute of Pain, Barcelona 2004. *Pain Pract*. 2005;5:85-94.

91.   Melzack R, Katz J. Pain the 21st century: the neuromatrix and beyond. In: Young G, Kane A, Nicholson K, eds. *Psychological Knowledge in Court: PTSD, Pain, and TBI*. New York: Springer; 2006.

92.   Apkarian AV, Baliki MN, Geha PY. Towards a theory of chronic pain. *Progress in neurobiology*. 2009;87:81-97.

93.   Apkarian AV, Sosa Y, Sonty S, et al. Chronic back pain is associated with decreased prefrontal and thalamic gray matter density. *The Journal of neuroscience : the official journal of the Society for Neuroscience*. 2004;24:10410-10415.

94.   Barad MJ, Ueno T, Younger J, Chatterjee N, Mackey S. Complex regional pain syndrome is associated with structural abnormalities in pain-related regions of the human brain. *J Pain*. 2014;15:197-203.

95.   Geha PY, Baliki MN, Harden RN, Bauer WR, Parrish TB, Apkarian AV. The brain in chronic CRPS pain: abnormal gray-white matter interactions in emotional and autonomic regions. *Neuron*. 2008;60:570-581.

96.   Hashmi JA, Baliki MN, Huang L, et al. Shape shifting pain: chronification of back pain shifts brain representation from nociceptive to emotional circuits. *Brain : a journal of neurology*. 2013;136:2751-2768.

97.   Mansour AR, Farmer MA, Baliki MN, Apkarian AV. Chronic pain: the role of learning and brain plasticity. *Restorative neurology and neuroscience*. 2014;32:129-139.

98.   Mutso AA, Petre B, Huang L, et al. Reorganization of hippocampal functional connectivity with transition to chronic back pain. *Journal of neurophysiology*. 2014;111:1065-1076.

99.   Schuh-Hofer S, Wodarski R, Pfau DB, et al. One night of total sleep deprivation promotes a state of generalized hyperalgesia: a surrogate pain model to study the relationship of insomnia and pain. *Pain*. 2013;154:1613-1621.

100.   Staud R, Robinson ME, Price DD. Temporal summation of second pain and its maintenance are useful for characterizing widespread central sensitization of fibromyalgia patients. *J Pain*. 2007;8:893-901.

101.   van der Windt D, Simons E, Riphagen I, et al. Physical examination for lumbar radiculopathy due to disc herniation in patients with low-back pain. *The Cochrane database of systematic reviews*. 2010;17:CD007431.

102.   Dagenais S, Tricco AC, Haldeman S. Synthesis of recommendations for the assessment and management of low back pain from recent clinical practice guidelines. *The spine journal : official journal of the North American Spine Society*. 2010;10:514-529.

103.   Krawiec CJ, Denegar CR, Hertel J, Salvaterra GF, Buckley WE. Static innominate asymmetry and leg length discrepancy in asymptomatic collegiate athletes. *Manual therapy*. 2003;8:207-213.

104.   Waddell G, McCulloch JA, Kummel E, Venner RM. Nonorganic physical signs in low-back pain. *Spine*. 1980;5:117-125.

105.   Gaines WG, Jr., Hegmann KT. Effectiveness of Waddell's nonorganic signs in predicting a delayed return to regular work in patients experiencing acute occupational low back pain. *Spine*. 1999;24:396-400; discussion 401.

106.   McIntosh G, Frank J, Hogg-Johnson S, Bombardier C, Hall H. Prognostic factors for time receiving workers' compensation benefits in a cohort of patients with low back pain. *Spine*. 2000;25:147-157.

107.   Waddell G, Morris E, Di Paola M, Bircher M, Finlayson D. A concept of illness tested as an improved basis for surgical decisions in low-back disorders. *Spine*. 1986;11:712-719.

108.   Nicholas M, Linton S, Watson P, Main C, "Decade of the Flags" Working Group. Early identification and management of psychological risk factors ("yellow flags") in patients with low back pain: a reappraisal. *Physical therapy*. 2011;91:737-753.

109.   Keefe FJ, Block AR, Williams RB, Jr., Surwit RS. Behavioral treatment of chronic low back pain: clinical outcome and individual differences in pain relief. *Pain*. 1981;11:221-231.

110.   Waddell G, Richardson J. Observation of overt pain behaviour by physicians during routine clinical examination of patients with low back pain. *Journal of psychosomatic research*. 1992;36:77-87.

111.   Fishbain DA, Cutler RB, Rosomoff HL, Rosomoff RS. Are opioid-dependent/tolerant patients impaired in driving-related skills? A structured evidence-based review. *J Pain Symptom Manage*. 2003;25:559-577.

112.   Fishbain DA, Cutler RB, Rosomoff HL, Rosomoff RS. Is there a relationship between nonorganic physical findings (Waddell signs) and secondary gain/malingering? *The Clinical journal of pain*. 2004;20:399-408.

113.   Main CJ, Waddell G. Behavioral responses to examination. A reappraisal of the interpretation of "nonorganic signs". *Spine*. 1998;23:2367-2371.

114.   Chou R, Shekelle P. Will this patient develop persistent disabling low back pain? *JAMA : the journal of the American Medical Association*. 2010;303:1295-1302.

115.   Goertz CM, Pohlman KA, Vining RD, Brantingham JW, Long CR. Patient-centered outcomes of high-velocity, low-amplitude spinal manipulation for low back pain: a systematic review. *Journal of electromyography and kinesiology : official journal of the International Society of Electrophysiological Kinesiology*. 2012;22:670-691.

116.   Leonardi M, Bickenbach J, Ustun TB, Kostanjsek N, Chatterji S. The definition of disability: what is in a name? *Lancet*. 2006;368:1219-1221.

117.   Tate DG, Pledger C. An integrative conceptual framework of disability. New directions for research. *The American psychologist*. 2003;58:289-295.

118.   van Abbema R, Lakke SE, Reneman MF, et al. Factors associated with functional capacity test results in patients with non-specific chronic low back pain: a systematic review. *J Occup Rehabil*. 2011;21:455-473.

119.   Verkerk K, Luijsterburg PA, Miedema HS, Pool-Goudzwaard A, Koes BW. Prognostic factors for recovery in chronic nonspecific low back pain: a systematic review. *Phys Ther*. 2012;92:1093-1108.

120.   Carragee E, Alamin T, Cheng I, Franklin T, Hurwitz E. Does minor trauma cause serious low back illness? *Spine*. 2006;31:2942-2949.

121.   Carragee E, Alamin T, Cheng I, Franklin T, van den Haak E, Hurwitz E. Are first-time episodes of serious LBP associated with new MRI findings? *The spine journal : official journal of the North American Spine Society*. 2006;6:624-635.

122.   Carragee EJ. Single-level posterolateral arthrodesis, with or without posterior decompression, for the treatment of isthmic spondylolisthesis in adults. A prospective, randomized study. *The Journal of bone and joint surgery*. 1997;79:1175-1180.

123.   Eriksen W. Do people who were passive smokers during childhood have increased risk of long-term work disability? A 15-month prospective study of nurses' aides. *Eur J Public Health*. 2004;14:296-300.

124.   Garg A, Boda S, Hegmann KT, et al. The NIOSH lifting equation and low-back pain, Part 1: Association with low-back pain in the backworks prospective cohort study. *Human factors*. 2014;56:6-28.

125.   Garg A, Kapellusch JM, Hegmann KT, et al. The NIOSH lifting equation and low-back pain, Part 2: Association with seeking care in the backworks prospective cohort study. *Human factors*. 2014;56:44-57.

126.   Hestbaek L, Leboeuf-Yde C, Kyvik KO. Are lifestyle-factors in adolescence predictors for adult low back pain? A cross-sectional and prospective study of young twins. *BMC musculoskeletal disorders*. 2006;7:27.

127.   Hoogendoorn WE, van Poppel MN, Bongers PM, Koes BW, Bouter LM. Systematic review of psychosocial factors at work and private life as risk factors for back pain. *Spine*. 2000;25:2114-2125.

128.   Kapellusch JM, Garg A, Boda S, et al. Association between lifting and use of medication for low back pain: results from the Backworks Prospective Cohort Study. *Journal of occupational and environmental medicine / American College of Occupational and Environmental Medicine*. 2014;56:867-877.

129.   Linton SJ. A review of psychological risk factors in back and neck pain. *Spine*. 2000;25:1148-1156.

130.   Papageorgiou AC, Croft PR, Thomas E, Ferry S, Jayson MI, Silman AJ. Influence of previous pain experience on the episode incidence of low back pain: results from the South Manchester Back Pain Study. *Pain*. 1996;66:181-185.

131. Smedley J, Egger P, Cooper C, Coggon D. Prospective cohort study of predictors of incident low back pain in nurses. *BMJ*. 1997;314:1225-1228.

132. Tubach F, Leclerc A, Landre MF, Pietri-Taleb F. Risk factors for sick leave due to low back pain: a prospective study. *Journal of occupational and environmental medicine / American College of Occupational and Environmental Medicine*. 2002;44:451-458.

133. Van Nieuwenhuyse A, Crombez G, Burdorf A, et al. Physical characteristics of the back are not predictive of low back pain in healthy workers: A prospective study. *BMC musculoskeletal disorders*. 2009;10:doi:10.1186/1471-2474-1110-1182.

134. Van Nieuwenhuyse A, Somville PR, Crombez G, et al. The role of physical workload and pain related fear in the development of low back pain in young workers: evidence from the BelCoBack Study; results after one year of follow up. *Occup Environ Med*. 2006;63:45-52.

135. van Poppel MN, Koes BW, van der Ploeg T, Smid T, Bouter LM. Lumbar supports and education for the prevention of low back pain in industry: a randomized controlled trial. *Jama*. 1998;279:1789-1794.

136. Heuch I, Heuch I, Hagen K, Zwart JA. Body mass index as a risk factor for developing chronic low back pain: a follow-up in the Nord-Trondelag Health Study. *Spine*. 2013;38:133-139.

137. Karahan A, Kav S, Abbasoglu A, Dogan N. Low back pain: prevalence and associated risk factors among hospital staff. *Journal of advanced nursing*. 2009;65:516-524.

138. Knox JB, Orchowski JR, Owens B. Racial differences in the incidence of acute low back pain in United States military service members. *Spine*. 2012;37:1688-1692.

139. Ozguler A, Leclerc A, Landre MF, Pietri-Taleb F, Niedhammer I. Individual and occupational determinants of low back pain according to various definitions of low back pain. *Journal of epidemiology and community health*. 2000;54:215-220.

140. Frymoyer JW, Newberg A, Pope MH, Wilder DG, Clements J, MacPherson B. Spine radiographs in patients with low-back pain. An epidemiological study in men. *The Journal of bone and joint surgery American volume*. 1984;66:1048-1055.

141. Wickstrom G. Effect of work on degenerative back disease. A review. *Scandinavian journal of work, environment & health*. 1978;4 Suppl 1:1-12.

142. Miranda H, Viikari-Juntura E, Punnett L, Riihimaki H. Occupational loading, health behavior and sleep disturbance as predictors of low-back pain. *Scandinavian journal of work, environment & health*. 2008;34:411-419.

143. Shiri R, Karppinen J, Leino-Arjas P, Solovieva S, Viikari-Juntura E. The association between smoking and low back pain: a meta-analysis. *The American journal of medicine*. 2010;123:87 e87-35.

144. Leino-Arjas P, Solovieva S, Kirjonen J, Reunanen A, Riihimaki H. Cardiovascular risk factors and low-back pain in a long-term follow-up of industrial employees. *Scandinavian journal of work, environment & health*. 2006;32:12-19.

145. Cust G, Pearson JC, Mair A. The prevalence of low back pain in nurses. *International nursing review*. 1972;19:169-179.

146. Pedersen O, Petersen R, Schack Staffeldt E. Back pain and isometric back muscle strength of workers in a Danish factory. *Scandinavian journal of rehabilitation medicine*. 1975;7:125-128.

147. Watson KD, Papageorgiou AC, Jones GT, et al. Low back pain in schoolchildren: the role of mechanical and psychosocial factors. *Archives of disease in childhood*. 2003;88:12-17.

148. Aro S, Leino P. Overweight and musculoskeletal morbidity: a ten-year follow-up. *International journal of obesity*. 1985;9:267-275.

149. Barton JE, Haight RO, Marsland DW, Temple TE, Jr. Low back pain in the primary care setting. *The Journal of family practice*. 1976;3:363-366.

150. Bostman OM. Body mass index and height in patients requiring surgery for lumbar intervertebral disc herniation. *Spine*. 1993;18:851-854.

151. Bovenzi M, Zadini A. Self-reported low back symptoms in urban bus drivers exposed to whole-body vibration. *Spine*. 1992;17:1048-1059.

152. Gyntelberg F. One year incidence of low back pain among male residents of Copenhagen aged 40-59. *Danish medical bulletin*. 1974;21:30-36.

153. Heliovaara M, Knekt P, Aromaa A. Incidence and risk factors of herniated lumbar intervertebral disc or sciatica leading to hospitalization. *Journal of chronic diseases*. 1987;40:251-258.

154. Karvonen MJ, Viitasalo JT, Komi PV, Nummi J, Jarvinen T. Back and leg complaints in relation to muscle strength in young men. *Scand J Rehabil Med*. 1980;12:53-59.

155. Leboeuf-Yde C. Body weight and low back pain. A systematic literature review of 56 journal articles reporting on 65 epidemiologic studies. *Spine*. 2000;25:226-237.

156. Raanaas R, Anderson D. A questionnaire survey of Norwegian taxi drivers' musculoskeletal health, and work-related risk factors. *Int J Industr Ergonom*. 2008;38:280-290.

157.   Videman T, Sarna S, Battie MC, et al. The long-term effects of physical loading and exercise lifestyles on back-related symptoms, disability, and spinal pathology among men. *Spine*. 1995;20:699-709.

158.   Wright D, Barrow S, Fisher AD, Horsley SD, Jayson MI. Influence of physical, psychological and behavioural factors on consultations for back pain. *British journal of rheumatology*. 1995;34:156-161.

159.   Croft PR, Rigby AS. Socioeconomic influences on back problems in the community in Britain. *Journal of epidemiology and community health*. 1994;48:166-170.

160.   Hershkovich O, Friedlander A, Gordon B, et al. Associations of body mass index and body height with low back pain in 829,791 adolescents. *American journal of epidemiology*. 2013;178:603-609.

161.   Shiri R, Karppinen J, Leino-Arjas P, Solovieva S, Viikari-Juntura E. The association between obesity and low back pain: a meta-analysis. *American journal of epidemiology*. 2010;171:135-154.

162.   Leino-Arjas P, Kaila-Kangas L, Solovieva S, Riihimaki H, Kirjonen J, Reunanen A. Serum lipids and low back pain: an association? A follow-up study of a working population sample. *Spine*. 2006;31:1032-1037.

163.   Battie MC, Videman T, Levalahti E, Gill K, Kaprio J. Heritability of low back pain and the role of disc degeneration. *Pain*. 2007;131:272-280.

164.   Kalichman L, Hunter DJ. The genetics of intervertebral disc degeneration. Familial predisposition and heritability estimation. *Joint, bone, spine : revue du rhumatisme*. 2008;75:383-387.

165.   Elders LA, Burdorf A. Prevalence, incidence, and recurrence of low back pain in scaffolders during a 3-year follow-up study. *Spine*. 2004;29:E101-106.

166.   Hoogendoorn WE, Bongers PM, de Vet HC, et al. Psychosocial work characteristics and psychological strain in relation to low-back pain. *Scandinavian journal of work, environment & health*. 2001;27:258-267.

167.   Verbunt JA, Smeets RJ, Wittink HM. Cause or effect? Deconditioning and chronic low back pain. *Pain*. 2010;149:428-430.

168.   van Oostrom SH, Monique Verschuren WM, de Vet HC, Picavet HS. Ten year course of low back pain in an adult population-based cohort--the Doetinchem cohort study. *Eur J Pain*. 2011;15:993-998.

169.   Heneweer H, Vanhees L, Picavet HS. Physical activity and low back pain: a U-shaped relation? *Pain*. 2009;143:21-25.

170.   Thiese MS, Hegmann KT, Garg A, Porucznik C, Behrens T. The predictive relationship of physical activity on the incidence of low back pain in an occupational cohort. *Journal of occupational and environmental medicine / American College of Occupational and Environmental Medicine*. 2011;53:364-371.

171.   Bergenudd H, Nilsson B, Uden A, Willner S. Bone mineral content, gender, body posture, and build in relation to back pain in middle age. *Spine*. 1989;14:577-579.

172.   Biering-Sorensen F. Physical measurements as risk indicators for low-back trouble over a one-year period. *Spine (Phila Pa 1976)*. 1984;9:106-119.

173.   Bigos SJ, Spengler DM, Martin NA, et al. Back injuries in industry: a retrospective study. II. Injury factors. *Spine*. 1986;11:246-251.

174.   Chaffin DB, Park KS. A longitudinal study of low-back pain as associated with occupational weight lifting factors. *American Industrial Hygiene Association journal*. 1973;34:513-525.

175.   Dehlin O, Hedenrud B, Horal J. Back symptoms in nursing aides in a geriatric hospital. An interview study with special reference to the incidence of low-back symptoms. *Scand J Rehabil Med*. 1976;8:47-53.

176.   Harkness EF, Macfarlane GJ, Nahit ES, Silman AJ, McBeth J. Risk factors for new-onset low back pain amongst cohorts of newly employed workers. *Rheumatology (Oxford)*. 2003;42:959-968.

177.   Wai EK, Roffey DM, Bishop P, Kwon BK, Dagenais S. Causal assessment of occupational lifting and low back pain: results of a systematic review. *The spine journal : official journal of the North American Spine Society*. 2010;10:554-566.

178.   Waters TR, Putz-Anderson V, Garg A, Fine LJ. Revised NIOSH equation for the design and evaluation of manual lifting tasks. *Ergonomics*. 1993;36:749-776.

179.   Wai EK, Roffey DM, Bishop P, Kwon BK, Dagenais S. Causal assessment of occupational bending or twisting and low back pain: results of a systematic review. *The spine journal : official journal of the North American Spine Society*. 2010;10:76-88.

180.   Roffey DM, Wai EK, Bishop P, Kwon BK, Dagenais S. Causal assessment of occupational standing or walking and low back pain: results of a systematic review. *The spine journal : official journal of the North American Spine Society*. 2010;10:262-272.

181.   Roffey DM, Wai EK, Bishop P, Kwon BK, Dagenais S. Causal assessment of occupational pushing or pulling and low back pain: results of a systematic review. *The spine journal : official journal of the North American Spine Society*. 2010;10:544-553.

182.   Bible JE, Choemprayong S, O'Neill KR, Devin CJ, Spengler DM. Whole-body vibration: is there a causal relationship to specific imaging findings of the spine? *Spine*. 2012;37:E1348-1355.

183.   Roffey DM, Wai EK, Bishop P, Kwon BK, Dagenais S. Causal assessment of occupational sitting and low back pain: results of a systematic review. *The spine journal : official journal of the North American Spine Society*. 2010;10:252-261.

184.   Waters T, Genaidy A, Barriera Viruet H, Makola M. The impact of operating heavy equipment vehicles on lower back disorders. *Ergonomics*. 2008;51:602-636.

185.   Garg A, Hegmann KT, Moore JS, et al. Study protocol title: a prospective cohort study of low back pain. *BMC musculoskeletal disorders*. 2013;14:84.

186.   Engst C, Chhokar R, Miller A, Tate RB, Yassi A. Effectiveness of overhead lifting devices in reducing the risk of injury to care staff in extended care facilities. *Ergonomics*. 2005;48:187-199.

187.   Garg A, Owen B. Reducing back stress to nursing personnel: an ergonomic intervention in a nursing home. *Ergonomics*. 1992;35:1353-1375.

188.   Garg A, Owen BD, Carlson B. An ergonomic evaluation of nursing assistants' job in a nursing home. *Ergonomics*. 1992;35:979-995.

189.   Hegmann K, Garg A. Chapter 15. Ergonomic issues in medical centers. In: McCunney R, ed. *Medical Center Occupational Health and Safety*. Philadelphia: Lippincott Williams & Wilkins; 1999.

190.   Owen BD, Garg A. Reducing risk for back pain in nursing personnel. *AAOHN J*. 1991;39:24-33.

191.   Owen BD, Garg A. Reducing back stress through an ergonomic approach: weighing a patient. *International journal of nursing studies*. 1994;31:511-519.

192.   Bigos SJ, Holland J, Holland C, Webster JS, Battie M, Malmgren JA. High-quality controlled trials on preventing episodes of back problems: systematic literature review in working-age adults. *The spine journal : official journal of the North American Spine Society*. 2009;9:147-168.

193.   Martimo KP, Verbeek J, Karppinen J, et al. Manual material handling advice and assistive devices for preventing and treating back pain in workers. *Cochrane Database Syst Rev*. 2007:CD005958.

194.   Hadler N. *Occupational Muscoluskeletal Disorders*. Baltimore, MD: Lippincott, Williams & Wilkins; 2005.

195.   Waddell G. The Back Pain Revolution. Edinburgh, Scotland: Churchill Livingstone; 2004.

196.   Bigos SJ, Battie MC, Spengler DM, et al. A longitudinal, prospective study of industrial back injury reporting. *Clinical orthopaedics and related research*. 1992:21-34.

197.   Williams RA, Pruitt SD, Doctor JN, et al. The contribution of job satisfaction to the transition from acute to chronic low back pain. *Archives of physical medicine and rehabilitation*. 1998;79:366-374.

198.   Currie SR, Wang J. More data on major depression as an antecedent risk factor for first onset of chronic back pain. *Psychological medicine*. 2005;35:1275-1282.

199.   Coudeyre E, Rannou F, Tubach F, et al. General practitioners' fear-avoidance beliefs influence their management of patients with low back pain. *Pain*. 2006;124:330-337.

200.   Linton SJ, Vlaeyen J, Ostelo R. The back pain beliefs of health care providers: are we fear-avoidant? *J Occup Rehabil*. 2002;12:223-232.

201.   Stadnik TW, Lee RR, Coen HL, Neirynck EC, Buisseret TS, Osteaux MJ. Annular tears and disk herniation: prevalence and contrast enhancement on MR images in the absence of low back pain or sciatica. *Radiology*. 1998;206:49-55.

202.   Eyring EJ. The biochemistry and physiology of the intervertebral disk. *Clinical orthopaedics and related research*. 1969;67:16-28.

203.   Battie MC, Videman T, Kaprio J, et al. The Twin Spine Study: contributions to a changing view of disc degeneration. *The spine journal : official journal of the North American Spine Society*. 2009;9:47-59.

204.   Karppinen J, Solovieva S, Luoma K, Raininko R, Leino-Arjas P, Riihimaki H. Modic changes and interleukin 1 gene locus polymorphisms in occupational cohort of middle-aged men. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2009;18:1963-1970.

205.   Matsui H, Kanamori M, Ishihara H, Yudoh K, Naruse Y, Tsuji H. Familial predisposition for lumbar degenerative disc disease. A case-control study. *Spine*. 1998;23:1029-1034.

206.   Sokoloff L. *The Biology of Degenerative Joint Disease*. Chicago, Ill: University of Chicago Press; 1969.

207.   Calmels P, Queneau P, Hamonet C, et al. Effectiveness of a lumbar belt in subacute low back pain: an open, multicentric, and randomized clinical study. *Spine (Phila Pa 1976)*. 2009;34:215-220.

208.   Dai F, Belfer I, Schwartz CE, et al. Association of catechol-O-methyltransferase genetic variants with outcome in patients undergoing surgical treatment for lumbar degenerative disc disease. *The spine journal : official journal of the North American Spine Society*. 2010;10:949-957.

209.   Caplan PS, Freedman LM, Connelly TP. Degenerative joint disease of the lumbar spine in coal miners--a clinical and x-ray study. *Arthritis and rheumatism*. 1966;9:693-702.

210.   Lawrence J, Aitken-Swan J. Rheumatism in miners: Part 1. Rheumatic complaints. *Br J Ind Med*. 1952;9:1-12.

211.   Schmorl G, Junghanns H. *The Human Spine in Health and Disease*. New York, NY: Grune and Stratton; 1971.

212.   Lawrence J, de Graaff R, Laine A. Degenerative joint disease in random samples and occupational groups. In: Jeffrey M, Ball J, eds. *The Epidemiology of Chronic Rheumatism*; 1963:98-119.

213.   Battie MC, Videman T. Lumbar disc degeneration: epidemiology and genetics. *The Journal of bone and joint surgery*. 2006;88 Suppl 2:3-9.

214.   Patel A, WR; S, Daubs M, Brodke D, Cannon-Albright L. Evidence for an inherited predisposition to lumbar disc disease. *The Journal of bone and joint surgery American volume*. 2011;93::225-229.

215.   Abdel-Moty E, Fishbain DA, Khalil TM, et al. Functional capacity and residual functional capacity and their utility in measuring work capacity. *The Clinical journal of pain*. 1993;9:168-173.

216.   Chan G, Tan V, Koh D. Ageing and fitness to work. *Occupational medicine*. 2000;50:483-491.

217.   Gouttebarge V, Wind H, Kuijer PP, Frings-Dresen MH. Reliability and validity of Functional Capacity Evaluation methods: a systematic review with reference to Blankenship system, Ergos work simulator, Ergo-Kit and Isernhagen work system. *International archives of occupational and environmental health*. 2004;77:527-537.

218.   Gross DP, Battie MC. Functional capacity evaluation performance does not predict sustained return to work in claimants with chronic back pain. *J Occup Rehabil*. 2005;15:285-294.

219.   Ilmarinen J, Tuomi K, Eskelinen L, Nygard CH, Huuhtanen P, Klockars M. Background and objectives of the Finnish research project on aging workers in municipal occupations. *Scandinavian journal of work, environment & health*. 1991;17 Suppl 1:7-11.

220.   Jones T, Kumar S. Functional capacity evaluation of manual materials handlers: a review. *Disability and rehabilitation*. 2003;25:179-191.

221.   Kaplan GM, Wurtele SK, Gillis D. Maximal effort during functional capacity evaluations: an examination of psychological factors. *Arch Phys Med Rehabil*. 1996;77:161-164.

222.   King PM, Tuckwell N, Barrett TE. A critical review of functional capacity evaluations. *Physical therapy*. 1998;78:852-866.

223.   Kuijer PP, Gouttebarge V, Brouwer S, Reneman MF, Frings-Dresen MH. Are performance-based measures predictive of work participation in patients with musculoskeletal disorders? A systematic review. *International archives of occupational and environmental health*. 2012;85:109-123.

224.   Mahmud N, Schonstein E, Schaafsma F, et al. Functional capacity evaluations for the prevention of occupational re-injuries in injured workers. *The Cochrane database of systematic reviews*. 2010:CD007290.

225.   Marfeo EE, Haley SM, Jette AM, et al. Conceptual foundation for measures of physical function and behavioral health function for Social Security work disability evaluation. *Archives of physical medicine and rehabilitation*. 2013;94:1645-1652 e1642.

226.   Mayer TG, Gatchel RJ, Mayer H, Kishino ND, Keeley J, Mooney V. A prospective two-year study of functional restoration in industrial low back injury. An objective assessment procedure. *JAMA*. 1987;258:1763-1767.

227.   McFadden S, MacDonald A, Fogarty A, Le S, Merritt BK. Vocational assessment: a review of the literature from an occupation-based perspective. *Scandinavian journal of occupational therapy*. 2010;17:43-48.

228.   Oesch P, Meyer K, Bachmann S, Hagen KB, Vollestad NK. Comparison of two methods for interpreting lifting performance during functional capacity evaluation. *Physical therapy*. 2012;92:1130-1140.

229.   Reneman MF, Kool J, Oesch P, Geertzen JH, Battie MC, Gross DP. Material handling performance of patients with chronic low back pain during functional capacity evaluation: a comparison between three countries. *Disability and rehabilitation*. 2006;28:1143-1149.

230.   Spanjer J, Groothoff JW, Brouwer S. Instruments used to assess functional limitations in workers applying for disability benefit: a systematic review. *Disability and rehabilitation*. 2011;33:2143-2150.

231.   Tramposh AK. The functional capacity evaluation: measuring maximal work abilities. *Occup Med*. 1992;7:113-124.

232.   Tuomi K, Ilmarinen J, Martikainen R, Aalto L, Klockars M. Aging, work, life-style and work ability among Finnish municipal workers in 1981-1992. *Scandinavian journal of work, environment & health*. 1997;23 Suppl 1:58-65.

233.   Wind H, Gouttebarge V, Kuijer PP, Sluiter JK, Frings-Dresen MH. Effect of Functional Capacity Evaluation information on the judgment of physicians about physical work ability in the context of disability claims. *International archives of occupational and environmental health*. 2009;82:1087-1096.

234.   Gibson L, Strong J. Safety issues in functional capacity evaluation: findings from a trial of a new approach for evaluating clients with chronic back pain. *Journal of occupational rehabilitation*. 2005;15:237-251.

235.   Gibson L, Strong J, Wallace A. Functional capacity evaluation as a performance measure: evidence for a new approach for clients with chronic back pain. *The Clinical journal of pain*. 2005;21:207-215.

236.   Spanjer J, Krol B, Brouwer S, Popping R, Groothoff JW, van der Klink JJ. Reliability and validity of the Disability Assessment Structured Interview (DASI): a tool for assessing functional limitations in claimants. *Journal of occupational rehabilitation*. 2010;20:33-40.

237.   Brouwer S, Dijkstra PU, Stewart RE, Goeken LN, Groothoff JW, Geertzen JH. Comparing self-report, clinical examination and functional testing in the assessment of work-related limitations in patients with chronic low back pain. *Disability and rehabilitation*. 2005;27:999-1005.

238.   Gross DP, Battie MC. Construct validity of a kinesiophysical functional capacity evaluation administered within a worker's compensation environment. *Journal of occupational rehabilitation*. 2003;13:287-295.

239.   Reneman MF, Jaegers SM, Westmaas M, Goeken LN. The reliability of determining effort level of lifting and carrying in a functional capacity evaluation. *Work (Reading, Mass*. 2002;18:23-27.

240.   Reneman MF, Schiphorts Preuper HR, Kleen M, Geertzen JH, Dijkstra PU. Are pain intensity and pain related fear related to functional capacity evaluation performances of patients with chronic low back pain? *J Occup Rehabil*. 2007;17:247-258.

241.   Schiphorst Preuper HR, Reneman MF, Boonstra AM, et al. Relationship between psychological factors and performance-based and self-reported disability in chronic low back pain. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2008;17:1448-1456.

242.   Smeets RJ, van Geel AC, Kester AD, Knottnerus JA. Physical capacity tasks in chronic low back pain: what is the contributing role of cardiovascular capacity, pain and psychological factors? *Disability and rehabilitation*. 2007;29:577-586.

243.   Eriksen J, Sjogren P, Bruera E, Ekholm O, Rasmussen NK. Critical issues on opioids in chronic non-cancer pain: an epidemiological study. *Pain*. 2006;125:172-179.

244.   Pransky GS, Dempsey PG. Practical aspects of functional capacity evaluations. *J Occup Rehabil*. 2004;14:217-229.

245.   Gross DP, Battie MC. The prognostic value of functional capacity evaluation in patients with chronic low back pain: part 2: sustained recovery. *Spine*. 2004;29:920-924.

246.   Gross DP, Battie MC, Cassidy JD. The prognostic value of functional capacity evaluation in patients with chronic low back pain: part 1: timely return to work. *Spine*. 2004;29:914-919.

247.   Hall H, McIntosh G, Melles T, Holowachuk B, Wai E. Effect of discharge recommendations on outcome. *Spine*. 1994;19:2033-2037.

248.   Chou R, Fu R, Carrino JA, Deyo RA. Imaging strategies for low-back pain: systematic review and meta-analysis. *Lancet*. 2009;373:463-472.

249.   Rubinstein SM, van Tulder M. A best-evidence review of diagnostic procedures for neck and low-back pain. *Best practice & research*. 2008;22:471-482.

250.   Bigos S, Bowyer O, Braen G, et al. Acute low back problems in adults. Clinical practice guidline No. 14. Rockville, MD: AHCPR publication No. 95-0642. Agency for Health Care Policy and Research, Public Health Services, U.S. Department of Health and Human Services.; 1994.

251.   Andersson GB, Cocchiarella L. *Guides to the Evaluation of Permanent Impairment. 5th edition*. Chicago, Ill: American Medical Association; 2001.

252.   Deyo RA, Mirza SK, Heagerty PJ, Turner JA, Martin BI. A prospective cohort study of surgical treatment for back pain with degenerated discs; study protocol. *BMC musculoskeletal disorders*. 2005;6:24.

253.   Djais N, Kalim H. The role of lumbar spine radiography in the outcomes of patients with simple acute low back pain. *APLAR J Rheum*. 2005;8:45-50.

254.   Kendrick D, Fielding K, Bentley E, Kerslake R, Miller P, Pringle M. Radiography of the lumbar spine in primary care patients with low back pain: randomised controlled trial. *Bmj*. 2001;322:400-405.

255.   Kerry S, Hilton S, Dundas D, Rink E, Oakeshott P. Radiography for low back pain: a randomised controlled trial and observational study in primary care. *Br J Gen Pract*. 2002;52:469-474.

256.   Chou R, Deyo RA, Jarvik JG. Appropriate use of lumbar imaging for evaluation of low back pain. *Radiologic clinics of North America*. 2012;50:569-585.

257.   Brenner DJ, Hall EJ. Computed tomography--an increasing source of radiation exposure. *The New England journal of medicine*. 2007;357:2277-2284.

258.   Fazel R, Krumholz HM, Wang Y, et al. Exposure to low-dose ionizing radiation from medical imaging procedures. *The New England journal of medicine*. 2009;361:849-857.

259.   Jarvik JG, Hollingworth W, Martin B, et al. Rapid magnetic resonance imaging vs radiographs for patients with low back pain: a randomized controlled trial. *Jama*. 2003;289:2810-2818.

260.   Deyo RA, Diehl AK, Rosenthal M. Reducing roentgenography use. Can patient expectations be altered? *Archives of internal medicine*. 1987;147:141-145.

261.   Carrino JA, Lurie JD, Tosteson AN, et al. Lumbar spine: reliability of MR imaging findings. *Radiology*. 2009;250:161-170.

262.   Hu ZJ, He J, Zhao FD, Fang XQ, Zhou LN, Fan SW. An assessment of the intra- and inter-reliability of the lumbar paraspinal muscle parameters using CT scan and magnetic resonance imaging. *Spine*. 2011;36:E868-874.

263.   Lei D, Rege A, Koti M, Smith FW, Wardlaw D. Painful disc lesion: can modern biplanar magnetic resonance imaging replace discography? *Journal of spinal disorders & techniques*. 2008;21:430-435.

264.   Lurie JD, Moses RA, Tosteson AN, et al. Magnetic resonance imaging predictors of surgical outcome in patients with lumbar intervertebral disc herniation. *Spine*. 2013;38:1216-1225.

265.   O'Neill C, Kurgansky M, Kaiser J, Lau W. Accuracy of MRI for diagnosis of discogenic pain. *Pain physician*. 2008;11:311-326.

266.   Sheehan NJ. Magnetic resonance imaging for low back pain: indications and limitations. *Annals of the rheumatic diseases*. 2010;69:7-11.

267.   Suri P, Boyko EJ, Goldberg J, Forsberg CW, Jarvik JG. Longitudinal associations between incident lumbar spine MRI findings and chronic low back pain or radicular symptoms: retrospective analysis of data from the longitudinal assessment of imaging and disability of the back (LAIDBACK). *BMC musculoskeletal disorders*. 2014;15:152.

268.   Suri P, Hunter DJ, Katz JN, Li L, Rainville J. Bias in the physical examination of patients with lumbar radiculopathy. *BMC musculoskeletal disorders*. 2010;11:275.

269.   Wassenaar M, van Rijn RM, van Tulder MW, et al. Magnetic resonance imaging for diagnosing lumbar spinal pathology in adult patients with low back pain or sciatica: a diagnostic systematic review. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2012;21:220-227.

270.   Chou D, Samartzis D, Bellabarba C, et al. Degenerative magnetic resonance imaging changes in patients with chronic low back pain: a systematic review. *Spine*. 2011;36:S43-53.

271.   Hanly JG, Mitchell MJ, Barnes DC, MacMillan L. Early recognition of sacroiliitis by magnetic resonance imaging and single photon emission computed tomography. *The Journal of rheumatology*. 1994;21:2088-2095.

272.   Boden SD, McCowin PR, Davis DO, Dina TS, Mark AS, Wiesel S. Abnormal magnetic-resonance scans of the cervical spine in asymptomatic subjects. A prospective investigation. *The Journal of bone and joint surgery*. 1990;72:1178-1184.

273.   Boden SD, Sumner DR. Biologic factors affecting spinal fusion and bone regeneration. *Spine (Phila Pa 1976)*. 1995;20:102S-112S.

274.   Chung CB, Vande Berg BC, Tavernier T, et al. End plate marrow changes in the asymptomatic lumbosacral spine: frequency, distribution and correlation with age and degenerative changes. *Skeletal radiology*. 2004;33:399-404.

275.   Haig AJ, Tong HC, Yamakawa KS, et al. Predictors of pain and function in persons with spinal stenosis, low back pain, and no back pain. *Spine (Phila Pa 1976)*. 2006;31:2950-2957.

276.   Haig AJ, Tong HC, Yamakawa KS, et al. Spinal stenosis, back pain, or no symptoms at all? A masked study comparing radiologic and electrodiagnostic diagnoses to the clinical impression. *Arch Phys Med Rehabil*. 2006;87:897-903.

277.   Healy JF, Healy BB, Wong WH, Olson EM. Cervical and lumbar MRI in asymptomatic older male lifelong athletes: frequency of degenerative findings. *Journal of computer assisted tomography*. 1996;20:107-112.

278.   Jarvik JJ, Hollingworth W, Heagerty P, Haynor DR, Deyo RA. The Longitudinal Assessment of Imaging and Disability of the Back (LAIDBack) Study: baseline data. *Spine*. 2001;26:1158-1166.

279.   Jensen MC, Brant-Zawadzki MN, Obuchowski N, Modic MT, Malkasian D, Ross JS. Magnetic resonance imaging of the lumbar spine in people without back pain. *N Engl J Med*. 1994;331:69-73.

280.   Kjaer P, Leboeuf-Yde C, Sorensen JS, Bendix T. An epidemiologic study of MRI and low back pain in 13-year-old children. *Spine (Phila Pa 1976)*. 2005;30:798-806.

281.   Mikhael MA, Ciric IS, Kudrna JC, Hindo WA. Recognition of lumbar disc disease with magnetic resonance imaging. *Computerized radiology : official journal of the Computerized Tomography Society*. 1985;9:213-222.

282.   Parkkola R, Rytokoski U, Kormano M. Magnetic resonance imaging of the discs and trunk muscles in patients with chronic low back pain and healthy control subjects. *Spine*. 1993;18:830-836.

283.   Salminen JJ, Erkintalo MO, Pentti J, Oksanen A, Kormano MJ. Recurrent low back pain and early disc degeneration in the young. *Spine*. 1999;24:1316-1321.

284.   Savage RA, Whitehouse GH, Roberts N. The relationship between the magnetic resonance imaging appearance of the lumbar spine and low back pain, age and occupation in males. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 1997;6:106-114.

285.   Schelhas K, et al. Cervical discogenic pain. Prospective correlation of magnetic resonance imaging and discography in asymptomatic subjects and pain sufferers. 1996;21:300-312.

286.   Tong HC, Haig AJ, Yamakawa KS, Miner JA. Specificity of needle electromyography for lumbar radiculopathy and plexopathy in 55- to 79-year-old asymptomatic subjects. *American journal of physical medicine & rehabilitation / Association of Academic Physiatrists*. 2006;85:908-912; quiz 913-905, 934.

287.   Visuri T, Ulaska J, Eskelin M, Pulkkinen P. Narrowing of lumbar spinal canal predicts chronic low back pain more accurately than intervertebral disc degeneration: a magnetic resonance imaging study in young Finnish male conscripts. *Military medicine*. 2005;170:926-930.

288.   Weinreb JC, Wolbarsht LB, Cohen JM, Brown CE, Maravilla KR. Prevalence of lumbosacral intervertebral disk abnormalities on MR images in pregnant and asymptomatic nonpregnant women. *Radiology*. 1989;170:125-128.

289.   Weishaupt D, Zanetti M, Hodler J, Boos N. MR imaging of the lumbar spine: prevalence of intervertebral disk extrusion and sequestration, nerve root compression, end plate abnormalities, and osteoarthritis of the facet joints in asymptomatic volunteers. *Radiology*. 1998;209:661-666.

290.   Boos N, Rieder R, Schade V, Spratt KF, Semmer N, Aebi M. 1995 Volvo Award in clinical sciences. The diagnostic accuracy of magnetic resonance imaging, work perception, and psychosocial factors in identifying symptomatic disc herniations. *Spine*. 1995;20:2613-2625.

291.   Carragee EJ, Chen Y, Tanner CM, Truong T, Lau E, Brito JL. Provocative discography in patients after limited lumbar discectomy: A controlled, randomized study of pain response in symptomatic and asymptomatic subjects. *Spine (Phila Pa 1976)*. 2000;25:3065-3071.

292.   Kleinstuck F, Dvorak J, Mannion AF. Are "structural abnormalities" on magnetic resonance imaging a contraindication to the successful conservative treatment of chronic nonspecific low back pain? *Spine*. 2006;31:2250-2257.

293.   Waris E, Eskelin M, Hermunen H, Kiviluoto O, Paajanen H. Disc degeneration in low back pain: a 17-year follow-up study using magnetic resonance imaging. *Spine*. 2007;32:681-684.

294.   Modic MT, Obuchowski NA, Ross JS, et al. Acute low back pain and radiculopathy: MR imaging findings and their prognostic role and effect on outcome. *Radiology*. 2005;237:597-604.

295.   Alexander LA, Hancock E, Agouris I, Smith FW, MacSween A. The response of the nucleus pulposus of the lumbar intervertebral discs to functionally loaded positions. *Spine*. 2007;32:1508-1512.

296.   Karadimas EJ, Siddiqui M, Smith FW, Wardlaw D. Positional MRI changes in supine versus sitting postures in patients with degenerative lumbar spine. *Journal of spinal disorders & techniques*. 2006;19:495-500.

297.   Ferreiro Perez A, Garcia Isidro M, Ayerbe E, Castedo J, Jinkins JR. Evaluation of intervertebral disc herniation and hypermobile intersegmental instability in symptomatic adult patients undergoing recumbent and upright MRI of the cervical or lumbosacral spines. *Eur J Radiology*. 2007;62:444-448.

298.   Hirasawa Y, Bashir WA, Smith FW, Magnusson ML, Pope MH, Takahashi K. Postural changes of the dural sac in the lumbar spines of asymptomatic individuals using positional stand-up magnetic resonance imaging. *Spine*. 2007;32:E136-140.

299.   Gilbert JW, Wheeler GR, Lingreen RA, Johnson RR. Open stand-up MRI: a new instrument for positional neuroimaging. *Journal of spinal disorders & techniques*. 2006;19:151-154.

300.   Wildermuth S, Zanetti M, Duewell S, et al. Lumbar spine: quantitative and qualitative assessment of positional (upright flexion and extension) MR imaging and myelography. *Radiology*. 1998;207:391-398.

301.   Weishaupt D, Quick HH, Nanz D, Schmidt M, Cassina PC, Debatin JF. Ligating clips for three-dimensional MR angiography at 1.5 T: in vitro evaluation. *Radiology*. 2000;214:902-907.

302.   Aota Y, Niwa T, Yoshikawa K, Fujiwara A, Asada T, Saito T. Magnetic resonance imaging and magnetic resonance myelography in the presurgical diagnosis of lumbar foraminal stenosis. *Spine*. 2007;32:896-903.

303.   Bischoff RJ, Rodriguez RP, Gupta K, Righi A, Dalton JE, Whitecloud TS. A comparison of computed tomography-myelography, magnetic resonance imaging, and myelography in the diagnosis of herniated nucleus pulposus and spinal stenosis. *J Spinal Disord*. 1993;6:289-295.

304.   Chawalparit O, Churojana A, Chiewvit P, Thanapipatsir S, Vamvanij V, Charnchaowanish P. The limited protocol MRI in diagnosis of lumbar disc herniation. *Journal of the Medical Association of Thailand = Chotmaihet thangphaet*. 2006;89:182-189.

305.   Pui MH, Husen YA. Value of magnetic resonance myelography in the diagnosis of disc herniation and spinal stenosis. *Australas Radiol*. 2000;44:281-284.

306.   Karppinen J, Malmivaara A, Tervonen O, et al. Severity of symptoms and signs in relation to magnetic resonance imaging findings among sciatic patients. *Spine*. 2001;26:E149-154.

307.   Schenk P, Laubli T, Hodler J, Klipstein A. Magnetic resonance imaging of the lumbar spine: findings in female subjects from administrative and nursing professions. *Spine*. 2006;31:2701-2706.

308.   Beattie PF, Meyers SP, Stratford P, Millard RW, Hollenberg GM. Associations between patient report of symptoms and anatomic impairment visible on lumbar magnetic resonance imaging. *Spine*. 2000;25:819-828.

309.   Boos N, Semmer N, Elfering A, et al. Natural history of individuals with asymptomatic disc abnormalities in magnetic resonance imaging: predictors of low back pain-related medical consultation and work incapacity. *Spine*. 2000;25:1484-1492.

310.   Borenstein DG. Epidemiology, etiology, diagnostic evaluation, and treatment of low back pain. *Current opinion in rheumatology*. 2001;13:128-134.

311.   Carragee EJ, Alamin TF, Miller JL, Carragee JM. Discographic, MRI and psychosocial determinants of low back pain disability and remission: a prospective study in subjects with benign persistent back pain. *The spine journal : official journal of the North American Spine Society*. 2005;5:24-35.

312.   Elfering A, Semmer N, Birkhofer D, Zanetti M, Hodler J, Boos N. Risk factors for lumbar disc degeneration: a 5-year prospective MRI study in asymptomatic individuals. *Spine*. 2002;27:125-134.

313.   Jarvik JG, Hollingworth W, Heagerty PJ, Haynor DR, Boyko EJ, Deyo RA. Three-year incidence of low back pain in an initially asymptomatic cohort: clinical and imaging risk factors. *Spine*. 2005;30:1541-1548; discussion 1549.

314.   Jarvik JG, Maravilla KR, Haynor DR, Levitz M, Deyo RA. Rapid MR imaging versus plain radiography in patients with low back pain: initial results of a randomized study. *Radiology*. 1997;204:447-454.

315.   Jia LS, Shi ZR. MRI and myelography in the diagnosis of lumbar canal stenosis and disc herniation. A comparative study. *Chinese medical journal*. 1991;104:303-306.

316.   Siddiqui AH, Rafique MZ, Ahmad MN, Usman MU. Role of magnetic resonance imaging in lumbar spondylosis. *Journal of the College of Physicians and Surgeons--Pakistan : JCPSP*. 2005;15:396-399.

317.   Videman T, Battie MC, Gibbons LE, Maravilla K, Manninen H, Kaprio J. Associations between back pain history and lumbar MRI findings. *Spine*. 2003;28:582-588.

318.   Ash LM, Modic MT, Obuchowski NA, Ross JS, Brant-Zawadzki MN, Grooff PN. Effects of diagnostic information, per se, on patient outcomes in acute radiculopathy and low back pain. *AJNR American journal of neuroradiology*. 2008;29:1098-1103.

319.   Barz T, Melloh M, Staub LP, et al. Nerve root sedimentation sign: evaluation of a new radiological sign in lumbar spinal stenosis. *Spine*. 2010;35:892-897.

320.   Lee JH, Lee SH. Physical examination, magnetic resonance image, and electrodiagnostic study in patients with lumbosacral disc herniation or spinal stenosis. *Journal of rehabilitation medicine : official journal of the UEMS European Board of Physical and Rehabilitation Medicine*. 2012;44:845-850.

321.   Li AL, Yen D. Effect of increased MRI and CT scan utilization on clinical decision-making in patients referred to a surgical clinic for back pain. *Canadian journal of surgery Journal canadien de chirurgie*. 2011;54:128-132.

322.   Lurie JD, Tosteson AN, Tosteson TD, et al. Reliability of magnetic resonance imaging readings for lumbar disc herniation in the Spine Patient Outcomes Research Trial (SPORT). *Spine*. 2008;33:991-998.

323.   Modic MT, Masaryk T, Boumphrey F, Goormastic M, Bell G. Lumbar herniated disk disease and canal stenosis: prospective evaluation by surface coil MR, CT, and myelography. *AJR American journal of roentgenology*. 1986;147:757-765.

324.   Yan L, Li J, Zhao W, Cui Z, Wang H, Sr., Xin L. The study of epidurography and multispiral CT scanning examinations in the diagnosis of lumbar nerve root canal stenosis. *Orthopedics*. 2010;33:732.

325.   Mayerhoefer ME, Stelzeneder D, Bachbauer W, et al. Quantitative analysis of lumbar intervertebral disc abnormalities at 3.0 Tesla: value of T(2) texture features and geometric parameters. *NMR in biomedicine*. 2012;25:866-872.

326.   Chang HS, Zidan I, Fujisawa N, Matsui T. Microsurgical posterolateral transmuscular approach for lumbar foraminal stenosis. *Journal of spinal disorders & techniques*. 2011;24:302-307.

327.   Hsieh MS, Tsai MD. Diagnosis of herniated intervertebral disc assisted by 3-dimensional, multiaxial, magnetic resonance imaging. *Journal of the Formosan Medical Association = Taiwan yi zhi*. 1999;98:347-355.

328.   Williams A, Murtagh F, Rothman S, Sze G. Lumbar disc nomenclature: version 2.0. Am Soc Neuroradiology; 2014.

329.   van Rijn RM, Wassenaar M, Verhagen AP, et al. Computed tomography for the diagnosis of lumbar spinal pathology in adult patients with low back pain or sciatica: a diagnostic systematic review. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2012;21:228-239.

330.   French SD, Green S, Buchbinder R, Barnes H. Interventions for improving the appropriate use of imaging in people with musculoskeletal conditions. *Cochrane Database Syst Rev*. 2010:CD006094.

331.   Boden SD. The use of radiographic imaging studies in the evaluation of patients who have degenerative disorders of the lumbar spine. *The Journal of bone and joint surgery American volume*. 1996;78:114-124.

332.   Ren XS, Selim AJ, Fincke G, et al. Assessment of functional status, low back disability, and use of diagnostic imaging in patients with low back pain and radiating leg pain. *Journal of clinical epidemiology*. 1999;52:1063-1071.

333.   Saal JS. General principles of diagnostic testing as related to painful lumbar spine disorders: a critical appraisal of current diagnostic techniques. *Spine (Phila Pa 1976)*. 2002;27:2538-2545; discussion 2546.

334.	Iversen T, Solberg TK, Romner B, et al. Accuracy of physical examination for chronic lumbar radiculopathy. *BMC musculoskeletal disorders*. 2013;14:206.

335.	Nakao S, Yoshida M, Yamada H, Hashizume H. A new 3-dimensional computed tomography imaging method to diagnose extraforaminal stenosis at the lumbosacral junction. *Journal of spinal disorders & techniques*. 2010;23:e47-52.

336.	Slebus FG, Braakman R, Schipper J, van Dongen KJ, Westendorp-de Seriere M. Non-corresponding radiological and surgical diagnoses in patients operated for sciatica. *Acta neurochirurgica*. 1988;94:137-143.

337.	Willen J, Danielson B. The diagnostic effect from axial loading of the lumbar spine during computed tomography and magnetic resonance imaging in patients with degenerative disorders. *Spine*. 2001;26:2607-2614.

338.	Beauvais C, Wybier M, Chazerain P, et al. Prognostic value of early computed tomography in radiculopathy due to lumbar intervertebral disk herniation. A prospective study. *Joint, bone, spine : revue du rhumatisme*. 2003;70:134-139.

339.	Carrera GF, Williams AL, Haughton VM. Computed tomography in sciatica. *Radiology*. 1980;137:433-437.

340.	Gilbert FJ, Grant AM, Gillan MG, et al. Low back pain: influence of early MR imaging or CT on treatment and outcome--multicenter randomized trial. *Radiology*. 2004;231:343-351.

341.	Kalichman L, Kim DH, Li L, Guermazi A, Hunter DJ. Computed tomography-evaluated features of spinal degeneration: prevalence, intercorrelation, and association with self-reported low back pain. *The spine journal : official journal of the North American Spine Society*. 2010;10:200-208.

342.	Carmody RF, Yang PJ, Seeley GW, Seeger JF, Unger EC, Johnson JE. Spinal cord compression due to metastatic disease: diagnosis with MR imaging versus myelography. *Radiology*. 1989;173:225-229.

343.	Kardaun JW, Schipper J, Braakman R. CT, myelography, and phlebography in the detection of lumbar disk herniation: an analysis of the literature. *Am J Neuroradiol*. 1989;10:1111-1122.

344.	Kent DL, Haynor DR, Larson EB, Deyo RA. Diagnosis of lumbar spinal stenosis in adults: a metaanalysis of the accuracy of CT, MR, and myelography. *AJR American journal of roentgenology*. 1992;158:1135-1144.

345.	Loblaw DA, Perry J, Chambers A, Laperriere NJ. Systematic review of the diagnosis and management of malignant extradural spinal cord compression: the Cancer Care Ontario Practice Guidelines Initiative's Neuro-Oncology Disease Site Group. *J Clin Oncol*. 2005;23:2028-2037.

346.	Thornbury JR, Fryback DG, Turski PA, et al. Disk-caused nerve compression in patients with acute low-back pain: diagnosis with MR, CT myelography, and plain CT. *Radiology*. 1993;186:731-738.

347.	Ben-Galim P, Reitman CA. The distended facet sign: an indicator of position-dependent spinal stenosis and degenerative spondylolisthesis. *The spine journal : official journal of the North American Spine Society*. 2007;7:245-248.

348.	Botwin KP, Skene G, Tourres-Ramos FM, Gruber RD, Bouchlas CG, Shah CP. Role of weight-bearing flexion and extension myelography in evaluating the intervertebral disc. *American journal of physical medicine & rehabilitation / Association of Academic Physiatrists*. 2001;80:289-295.

349.	Wilkinson AG, Sellar RJ. The influence of needle size and other factors on the incidence of adverse effects caused by myelography. *Clinical radiology*. 1991;44:338-341.

350.	Pedersen ON. Use of a 22-gauge Whitacre needle to reduce the incidence of side effects after lumbar myelography: a prospective randomised study comparing Whitacre and Quincke spinal needles. *European radiology*. 1996;6:184-187.

351.	Bakhsh A. Role of conventional lumbar myelography in the management of sciatica: An experience from Pakistan. *Asian journal of neurosurgery*. 2012;7:25-28.

352.	Engelhorn T, Rennert J, Richter G, Struffert T, Ganslandt O, Doerfler A. Myelography using flat panel volumetric computed tomography: a comparative study in patients with lumbar spinal stenosis. *Spine*. 2007;32:E523-527.

353.	Kebede T, Bedane A, Admassie D, Zenebe G. Patterns of lumbar myelographic findings in patients with LBP a 5 years retrospective study at Yehuleshet Higher Clinic, Addis Ababa, Ethiopia. *Ethiop Med J*. 2010;48:229-236.

354.	Baker RR, Holmes ER, 3rd, Alderson PO, Khouri NF, Wagner HN, Jr. An evaluation of bone scans as screening procedures for occult metastases in primary breast cancer. *Annals of surgery*. 1977;186:363-368.

355.	McNeil BJ. Value of bone scanning in neoplastic disease. *Seminars in nuclear medicine*. 1984;14:277-286.

356.	Finkelstein JA, Chapman JR, Mirza S. Occult vertebral fractures in ankylosing spondylitis. *Spinal cord*. 1999;37:444-447.

357.	Pillai A, Jain M. Management of clinical fractures of the scaphoid: results of an audit and literature review. *European journal of emergency medicine : official journal of the European Society for Emergency Medicine*. 2005;12:47-51.

358.	Chakravarty D, Sloan J, Brenchley J. Risk reduction through skeletal scintigraphy as a screening tool in suspected scaphoid fracture: a literature review. *Emergency medicine journal : EMJ*. 2002;19:507-509.

359.	Deutsch AL, Coel MN, Mink JH. Imaging of stress injuries to bone. Radiography, scintigraphy, and MR imaging. *Clinics in sports medicine*. 1997;16:275-290.

360.	Battafarano DF, West SG, Rak KM, Fortenbery EJ, Chantelois AE. Comparison of bone scan, computed tomography, and magnetic resonance imaging in the diagnosis of active sacroiliitis. *Semin Arthritis Rheum*. 1993;23:161-176.

361.   Zafeirakis A, Kasimos D, Sioka C, Aravanis I, Zoumboulidis A. Evaluation of a quantitative diagnostic sacroiliac bone scan index in cases of chronic low back pain in young male adults. *Hellenic journal of nuclear medicine*. 2005;8:19-26.

362.   Brenner AI, Koshy J, Morey J, Lin C, DiPoce J. The bone scan. *Seminars in nuclear medicine*. 2012;42:11-26.

363.   Delbeke D, Schoder H, Martin WH, Wahl RL. Hybrid imaging (SPECT/CT and PET/CT): improving therapeutic decisions. *Seminars in nuclear medicine*. 2009;39:308-340.

364.   Kosuda S, Kaji T, Yokoyama H, et al. Does bone SPECT actually have lower sensitivity for detecting vertebral metastasis than MRI? *J Nucl Med*. 1996;37:975-978.

365.   Leone A, Cianfoni A, Cerase A, Magarelli N, Bonomo L. Lumbar spondylolysis: a review. *Skeletal radiology*. 2011;40:683-700.

366.   Maus T. Imaging the back pain patient. *Physical medicine and rehabilitation clinics of North America*. 2010;21:725-766.

367.   O'Neill C, Owens DK. Role of single photon emission computed tomography in the diagnosis of chronic low back pain. *The spine journal : official journal of the North American Spine Society*. 2010;10:70-72.

368.   Rinkus K, Knaub M. Clinical and diagnostic evaluation of low back pain. *Seminars Spine Surg*. 2008;20:93-101.

369.   Takemitsu M, El Rassi G, Woratanarat P, Shah SA. Low back pain in pediatric athletes with unilateral tracer uptake at the pars interarticularis on single photon emission computed tomography. *Spine*. 2006;31:909-914.

370.   Hanly JG, Barnes DC, Mitchell MJ, MacMillan L, Docherty P. Single photon emission computed tomography in the diagnosis of inflammatory spondyloarthropathies. *The Journal of rheumatology*. 1993;20:2062-2068.

371.   Ryan RJ, Gibson T, Fogelman I. The identification of spinal pathology in chronic low back pain using single photon emission computed tomography. *Nuclear medicine communications*. 1992;13:497-502.

372.   Bodner RJ, Heyman S, Drummond DS, Gregg JR. The use of single photon emission computed tomography (SPECT) in the diagnosis of low-back pain in young patients. *Spine (Phila Pa 1976)*. 1988;13:1155-1160.

373.   Gunzburg R, Servais F, Verhas M. Tomoscintigraphy of the lumbar spine: prospects and clinical application. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 1994;3:308-311.

374.   Harisankar CN, Mittal BR, Bhattacharya A, Singh P, Sen R. Utility of single photon emission computed tomography/computed tomography imaging in evaluation of chronic low back pain. *Indian journal of nuclear medicine : IJNM : the official journal of the Society of Nuclear Medicine, India*. 2012;27:156-163.

375.   Pneumaticos SG, Chatziioannou SN, Hipp JA, Moore WH, Esses SI. Low back pain: prediction of short-term outcome of facet joint injection with bone scintigraphy. *Radiology*. 2006;238:693-698.

376.   Heydari A, Nargol AV, Jones AP, Humphrey AR, Greenough CG. EMG analysis of lumbar paraspinal muscles as a predictor of the risk of low-back pain. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2010;19:1145-1152.

377.   Tong HC. Specificity of needle electromyography for lumbar radiculopathy in 55- to 79-yr-old subjects with low back pain and sciatica without stenosis. *American journal of physical medicine & rehabilitation / Association of Academic Physiatrists*. 2011;90:233-238; quiz 239-242.

378.   Sandoval AE. Electrodiagnostics for low back pain. *Physical medicine and rehabilitation clinics of North America*. 2010;21:767-776.

379.   Kang PB, Preston DC, Raynor EM. Involvement of superficial peroneal sensory nerve in common peroneal neuropathy. *Muscle & nerve*. 2005;31:725-729.

380.   Ahern DK, Follick MJ, Council JR, Laser-Wolston N, Litchman H. Comparison of lumbar paravertebral EMG patterns in chronic low back pain patients and non-patient controls. *Pain*. 1988;34:153-160.

381.   Cholewicki J, Greene HS, Polzhofer GK, Galloway MT, Shah RA, Radebold A. Neuromuscular function in athletes following recovery from a recent acute low back injury. *The Journal of orthopaedic and sports physical therapy*. 2002;32:568-575.

382.   Demoulin C, Crielaard JM, Vanderthommen M. Spinal muscle evaluation in healthy individuals and low-back-pain patients: a literature review. *Joint, bone, spine : revue du rhumatisme*. 2007;74:9-13.

383.   Finneran MT, Mazanec D, Marsolais ME, Marsolais EB, Pease WS. Large-array surface electromyography in low back pain: a pilot study. *Spine*. 2003;28:1447-1454.

384.   Humphrey AR, Nargol AV, Jones AP, Ratcliffe AA, Greenough CG. The value of electromyography of the lumbar paraspinal muscles in discriminating between chronic-low-back-pain sufferers and normal subjects. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2005;14:175-184.

385.   Leach RA, Owens EF, Jr., Giesen JM. Correlates of myoelectric asymmetry detected in low back pain patients using hand-held post-style surface electromyography. *Journal of manipulative and physiological therapeutics*. 1993;16:140-149.

386.   Lehman G. Kinesiological research: the use of surface electromyography for assessing the effects of spinal manipulation. *Journal of electromyography and kinesiology : official journal of the International Society of Electrophysiological Kinesiology*. 2012;22:692-696.

387.   Meyer JJ. The validity of thoracolumbar paraspinal scanning EMG as a diagnostic test: an examination of the current literature. *Journal of manipulative and physiological therapeutics*. 1994;17:539-551.

388.   Mohseni-Bandpei M, MJ W, B R. Application of surface electromyography in the assessment of low back pain: a review article. *Phys Ther Rev J*. 2000;5:93-105.

389.   Reger SI, Shah A, Adams TC, et al. Classification of large array surface myoelectric potentials from subjects with and without low back pain. *Journal of electromyography and kinesiology : official journal of the International Society of Electrophysiological Kinesiology*. 2006;16:392-401.

390.   Ritvanen T, Zaproudina N, Nissen M, Leinonen V, Hanninen O. Dynamic surface electromyographic responses in chronic low back pain treated by traditional bone setting and conventional physical therapy. *Journal of manipulative and physiological therapeutics*. 2007;30:31-37.

391.   Roy SH, Bonato P, Knaflitz M. EMG assessment of back muscle function during cyclical lifting. *Journal of electromyography and kinesiology : official journal of the International Society of Electrophysiological Kinesiology*. 1998;8:233-245.

392.   Roy SH, De Luca CJ, Emley M, et al. Classification of back muscle impairment based on the surface electromyographic signal. *Journal of rehabilitation research and development*. 1997;34:405-414.

393.   Roy SH, Oddsson LI. Classification of paraspinal muscle impairments by surface electromyography. *Physical therapy*. 1998;78:838-851.

394.   Rzanny R, Grassme R, Reichenbach JR, Scholle HC, Kaiser WA. Investigations of back muscle fatigue by simultaneous 31P MRS and surface EMG measurements. *Biomedizinische Technik Biomedical engineering*. 2006;51:305-313.

395.   Sihvonen T, Partanen J, Hanninen O, Soimakallio S. Electric behavior of low back muscles during lumbar pelvic rhythm in low back pain patients and healthy controls. *Arch Phys Med Rehabil*. 1991;72:1080-1087.

396.   Mannion AF, Taimela S, Muntener M, Dvorak J. Active therapy for chronic low back pain part 1. Effects on back muscle activation, fatigability, and strength. *Spine*. 2001;26:897-908.

397.   Chisari C, Simonella C, Rossi B. A surface EMG analysis of sarcolemma excitability alteration and myofibre degeneration in Steinert disease. *Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology*. 2001;112:1925-1930.

398.   Drost G, Blok JH, Stegeman DF, van Dijk JP, van Engelen BG, Zwarts MJ. Propagation disturbance of motor unit action potentials during transient paresis in generalized myotonia: a high-density surface EMG study. *Brain : a journal of neurology*. 2001;124:352-360.

399.   Drost G, Stegeman DF, Schillings ML, et al. Motor unit characteristics in healthy subjects and those with postpoliomyelitis syndrome: a high-density surface EMG study. *Muscle & nerve*. 2004;30:269-276.

400.   Haig AJ, Gelblum JB, Rechtien JJ, Gitter AJ. Technology assessment: the use of surface EMG in the diagnosis and treatment of nerve and muscle disorders. *Muscle & nerve*. 1996;19:392-395.

401.   Huppertz HJ, Disselhorst-Klug C, Silny J, Rau G, Heimann G. Diagnostic yield of noninvasive high spatial resolution electromyography in neuromuscular diseases. *Muscle & nerve*. 1997;20:1360-1370.

402.   Lagueny A, Marthan R, Schuermans P, Le Collen P, Ferrer X, Julien J. Single fiber EMG and spectral analysis of surface EMG in myotonia congenita with or without transient weakness. *Muscle & nerve*. 1994;17:248-250.

403.   Lindeman E, Spaans F, Reulen JP, Leffers P, Drukker J. Surface EMG of proximal leg muscles in neuromuscular patients and in healthy controls. Relations to force and fatigue. *Journal of electromyography and kinesiology : official journal of the International Society of Electrophysiological Kinesiology*. 1999;9:299-307.

404.   Meekins GD, So Y, Quan D. American Association of Neuromuscular & Electrodiagnostic Medicine evidenced-based review: use of surface electromyography in the diagnosis and study of neuromuscular disorders. *Muscle Nerve*. 2008;38:1219-1224.

405.   Meyer M, Hilfiker P, Gygi A. Surface EMG for diagnosis of neuromuscular diseases. In: Desmedt J, ed. *Computer-aided Electromyography and Expert Systems*. Amsterdam: Elsevier; 1989:181-188.

406.   Nirkko AC, Rosler KM, Hess CW. Sensitivity and specificity of needle electromyography: a prospective study comparing automated interference pattern analysis with single motor unit potential analysis. *Electroencephalogr Clin Neurophysiol*. 1995;97:1-10.

407.   Ramaekers VT, Disselhorst-Klug C, Schneider J, et al. Clinical application of a noninvasive multi-electrode array EMG for the recording of single motor unit activity. *Neuropediatrics*. 1993;24:134-138.

408.   Sunnerhagen KS, Carlsson U, Sandberg A, Stalberg E, Hedberg M, Grimby G. Electrophysiologic evaluation of muscle fatigue development and recovery in late polio. *Arch Phys Med Rehabil*. 2000;81:770-776.

409.   van der Hoeven JH, Links TP, Zwarts MJ, van Weerden TW. Muscle fiber conduction velocity in the diagnosis of familial hypokalemic periodic paralysis--invasive versus surface determination. *Muscle & nerve*. 1994;17:898-905.
410.   Wenzel S, Herrendorf G, Scheel A, Kurth C, Steinhoff BJ, Reimers CD. Surface EMG and myosonography in the detection of fasciculations: a comparative study. *J Neuroimaging*. 1998;8:148-154.
411.   Wimalaratna HS, Tooley MA, Churchill E, Preece AW, Morgan HM. Quantitative surface EMG in the diagnosis of neuromuscular disorders. *Electromyography and clinical neurophysiology*. 2002;42:167-174.
412.   Xu Y, Fan D, Zhang J, Zheng J, Zhang S, Kang D. Trigemino-cervical response in patients with amyotrophic lateral sclerosis. *Electromyography and clinical neurophysiology*. 2005;45:71-74.
413.   Zange J, Grehl T, Disselhorst-Klug C, et al. Breakdown of adenine nucleotide pool in fatiguing skeletal muscle in McArdle's disease: a noninvasive 31P-MRS and EMG study. *Muscle & nerve*. 2003;27:728-736.
414.   Thompson JM, Erickson RP, Offord KP. EMG muscle scanning: stability of hand-held surface electrodes. *Biofeedback and self-regulation*. 1989;14:55-62.
415.   Pullman SL, Goodin DS, Marquinez AI, Tabbal S, Rubin M. Clinical utility of surface EMG: report of the therapeutics and technology assessment subcommittee of the American Academy of Neurology. *Neurology*. 2000;55:171-177.
416.   McGill S, Juker D, Kropf P. Appropriately placed surface EMG electrodes reflect deep muscle activity (psoas, quadratus lumborum, abdominal wall) in the lumbar spine. *Journal of biomechanics*. 1996;29:1503-1507.
417.   Haig AJ, Gelblum JB, Rechtien JJ, Gitter AJ. Guidelines in electrodiagnostic medicine. Technology review: the use of surface EMG in the diagnosis and treatment of nerve and muscle disorders. *Muscle Nerve Suppl*. 1999;8::S239-242.
418.   Butler HL, Hubley-Kozey CL, Kozey JW. Changes in electromyographic activity of trunk muscles within the sub-acute phase for individuals deemed recovered from a low back injury. *Journal of electromyography and kinesiology : official journal of the International Society of Electrophysiological Kinesiology*. 2013;23:369-377.
419.   McNeill T, Huncke B, Pesch RN. Chemonucleolysis: evaluation of effectiveness by electromyography. *Arch Phys Med Rehabil*. 1977;58:303-306.
420.   Ramprasad M, Shenoy DS, Singh SJ, Sankara N, Joseley SR. The magnitude of pre-programmed reaction dysfunction in back pain patients: experimental pilot electromyography study. *Journal of back and musculoskeletal rehabilitation*. 2010;23:77-86.
421.   Lariviere C, Gagnon D, Gravel D, Bertrand Arsenault A. The assessment of back muscle capacity using intermittent static contractions. Part I - Validity and reliability of electromyographic indices of fatigue. *Journal of electromyography and kinesiology : official journal of the International Society of Electrophysiological Kinesiology*. 2008;18:1006-1019.
422.   Cram JR, Garber A. The relationship between narrow and wide bandwidth filter settings during an EMG scanning procedure. *Biofeedback and self-regulation*. 1986;11:105-114.
423.   Drost G, Van Dijk JP, Stegeman DF, Van Engelen BG, Zwarts MJ. Maintaining constant voluntary force in generalized myotonia despite muscle membrane disturbances: insights from a high-density surface EMG study. *J Clin Neurophysiol*. 2004;21:114-123.
424.   Ershad N, Kahrizi S, Abadi MF, Zadeh SF. Evaluation of trunk muscle activity in chronic low back pain patients and healthy individuals during holding loads. *Journal of back and musculoskeletal rehabilitation*. 2009;22:165-172.
425.   Hanada EY, Johnson M, Hubley-Kozey C. A comparison of trunk muscle activation amplitudes during gait in older adults with and without chronic low back pain. *PM & R : the journal of injury, function, and rehabilitation*. 2011;3:920-928.
426.   Nishizono H, Saito Y, Miyashita M. The estimation of conduction velocity in human skeletal muscle in situ with surface electrodes. *Electroencephalogr Clin Neurophysiol*. 1979;46:659-664.
427.   Schneider J, Rau G, Silny J. A noninvasive EMG technique for investigating the excitation propagation in single motor units. *Electromyography and clinical neurophysiology*. 1989;29:273-280.
428.   de Graaf I, Prak A, Bierma-Zeinstra S, Thomas S, Peul W, Koes B. Diagnosis of lumbar spinal stenosis: a systematic review of the accuracy of diagnostic tests. *Spine*. 2006;31:1168-1176.
429.   Furness G, Reilly MP, Kuchi S. An evaluation of ultrasound imaging for identification of lumbar intervertebral level. *Anaesthesia*. 2002;57:277-280.
430.   Klauser A, Halpern EJ, Frauscher F, et al. Inflammatory low back pain: high negative predictive value of contrast-enhanced color Doppler ultrasound in the detection of inflamed sacroiliac joints. *Arthritis and rheumatism*. 2005;53:440-444.
431.   Rhodes DW, Bishop PA. A review of diagnostic ultrasound of the spine and soft tissue. *Journal of manipulative and physiological therapeutics*. 1997;20:267-273.
432.   Rubaltelli L, De Gerone E, Caterino G. Echographic evaluation of tubercular abscesses in lumbar spondylitis. *Journal of ultrasound in medicine : official journal of the American Institute of Ultrasound in Medicine*. 1990;9:67-70.
433.   Anderson DJ, Adcock DF, Chovil AC, Farrell JJ. Ultrasound lumbar canal measurement in hospital employees with back pain. *British journal of industrial medicine*. 1988;45:552-555.

434.   Lee SW, Chan CK, Lam TS, et al. Relationship between low back pain and lumbar multifidus size at different postures. *Spine*. 2006;31:2258-2262.

435.   Teyhen DS, Miltenberger CE, Deiters HM, et al. The use of ultrasound imaging of the abdominal drawing-in maneuver in subjects with low back pain. *The Journal of orthopaedic and sports physical therapy*. 2005;35:346-355.

436.   Pulkovski N, Mannion AF, Caporaso F, et al. Ultrasound assessment of transversus abdominis muscle contraction ratio during abdominal hollowing: a useful tool to distinguish between patients with chronic low back pain and healthy controls? *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2012;21 Suppl 6:S750-759.

437.   Oliveira L, Maher C, Latimer J, Hodges P, Shirley D. An investigation of the reproducibility of ultrasound measures of abdominal muscle activation in patients with chronic non-specific low back pain. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2009;18:1059-1065.

438.   Newman RI, Seres JL, Miller EB. Liquid crystal thermography in the evaluation of chronic back pain: a comparative study. *Pain*. 1984;20:293-305.

439.   Rubal BJ, Traycoff RB, Ewing KL. Liquid crystal thermography. A new tool for evaluating low back pain. *Phys Ther*. 1982;62:1593-1596.

440.   Leclaire R, Esdaile JM, Jequier JC, Hanley JA, Rossignol M, Bourdouxhe M. Diagnostic accuracy of technologies used in low back pain assessment. Thermography, triaxial dynamometry, spinoscopy, and clinical examination. *Spine*. 1996;21:1325-1330; discussion 1331.

441.   Sherman RA, Barja RH, Bruno GM. Thermographic correlates of chronic pain: analysis of 125 patients incorporating evaluations by a blind panel. *Arch Phys Med Rehabil*. 1987;68:273-279.

442.   Takahashi Y, Takahashi K, Moriya H. Thermal deficit in lumbar radiculopathy. Correlations with pain and neurologic signs and its value for assessing symptomatic severity. *Spine*. 1994;19:2443-2449; discussion 2449-2450.

443.   Uematsu S, Jankel WR, Edwin DH, et al. Quantification of thermal asymmetry. Part 2: Application in low-back pain and sciatica. *J Neurosurg*. 1988;69:556-561.

444.   Ash CJ, Shealy CN, Young PA, Van Beaumont W. Thermography and the sensory dermatome. *Skeletal Radiol*. 1986;15:40-46.

445.   So YT, Aminoff MJ, Olney RK. The role of thermography in the evaluation of lumbosacral radiculopathy. *Neurology*. 1989;39:1154-1158.

446.   Harper CM, Jr., Low PA, Fealey RD, Chelimsky TC, Proper CJ, Gillen DA. Utility of thermography in the diagnosis of lumbosacral radiculopathy. *Neurology*. 1991;41:1010-1014.

447.   Swerdlow B, Dieter JN. An evaluation of the sensitivity and specificity of medical thermography for the documentation of myofascial trigger points. *Pain*. 1992;48:205-213.

448.   Gillstrom P. Thermography in low back pain and sciatica. *Archives of orthopaedic and traumatic surgery*. 1985;104:31-36.

449.   Wong KW, Luk KD, Leong JC, Wong SF, Wong KK. Continuous dynamic spinal motion analysis. *Spine*. 2006;31:414-419.

450.   Okawa A, Shinomiya K, Komori H, Muneta T, Arai Y, Nakai O. Dynamic motion study of the whole lumbar spine by videofluoroscopy. *Spine*. 1998;23:1743-1749.

451.   Ahmadi A, Maroufi N, Behtash H, Zekavat H, Parnianpour M. Kinematic analysis of dynamic lumbar motion in patients with lumbar segmental instability using digital videofluoroscopy. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2009;18:1677-1685.

452.   Antti-Poika I, Soini J, Tallroth K, Yrjönen T, Konttinen Y. Clinical relevance of discography combined with CT scanning. A study of 100 patients. *J Bone Joint Surg Br*. 1990;72:480-485.

453.   Cloward RB. Cervical diskography. A contribution to the etiology and mechanism of neck, shoulder and arm pain. *Annals of surgery*. 1959;150::1052-1064.

454.   Grubb SA, Kelly CK. Cervical discography: clinical implications from 12 years of experience. *Spine* 2000;25:1382-1389.

455.   Ortiz AO, Johnson B. Discography. *Tech Vasc Interv Radiol*. 2002;5:207-216.

456.   Singh V. The role of cervical discography in interventional pain management. *Pain Physician*. 2004;7:249-255.

457.   Wieser ES, Wang JC. Surgery for neck pain. *Neurosurgery*. 2007;60:S51-56.

458.   Adams MA, Dolan P, Hutton WC. The stages of disc degeneration as revealed by discograms. *The Journal of bone and joint surgery*. 1986;68:36-41.

459.   Holt EP, Jr. Fallacy of cervical discography. Report of 50 cases in normal subjects. *JAMA*. 1964;188::799-801.

460.   Ohnmeiss DD, Guyer RD, Mason SL. The relation between cervical discographic pain responses and radiographic images. *Clin J Pain*. 2000;16:1-5.
461.   Simmons EH, Bhalla SK. Anterior cervical discectomy and fusion. A clinical and biomechanical study with eight-year follow-up. *The Journal of bone and joint surgery*. 1969;51:225-237.
462.   Alamin TF, Kim MJ, Agarwal V. Provocative lumbar discography versus functional anesthetic discography: a comparison of the results of two different diagnostic techniques in 52 patients with chronic low back pain. *The spine journal : official journal of the North American Spine Society*. 2011;11:756-765.
463.   Ohtori S, Koshi T, Yamashita M, et al. Surgical versus nonsurgical treatment of selected patients with discogenic low back pain: a small-sized randomized trial. *Spine*. 2011;36:347-354.
464.   Putzier M, Streitparth F, Hartwig T, Perka CF, Hoff EK, Strube P. Can discoblock replace discography for identifying painful degenerated discs? *European journal of radiology*. 2013;82:1463-1470.
465.   Fortin JD. Precision diagnostic disc injections. *Pain Physician*. 2000;3:271-288.
466.   Modic MT, Ross JS, Masaryk TJ. Imaging of degenerative disease of the cervical spine. *Clinical orthopaedics and related research*. 1989:109-120.
467.   Simmons EH, Segil CM. An evaluation of discography in the localization of symptomatic levels in discogenic disease of the spine. *Clinical orthopaedics and related research*. 1975:57-69.
468.   Slipman CW, Plastaras C, Patel R, et al. Provocative cervical discography symptom mapping. *The spine journal : official journal of the North American Spine Society*. 2005;5:381-388.
469.   Sandhu HS, Sanchez-Caso LP, Parvataneni HK, Cammisa FP, Jr., Girardi FP, Ghelman B. Association between findings of provocative discography and vertebral endplate signal changes as seen on MRI. *Journal of spinal disorders*. 2000;13:438-443.
470.   Connor PM, Darden BV, 2nd. Cervical discography complications and clinical efficacy. *Spine* 1993;18:2035-2038.
471.   Holt EP, Jr. Further reflections on cervical discography. *JAMA*. 1975;231:613-614.
472.   Klafta LA, Jr., Collis JS, Jr. An analysis of cervical discography with surgical verification. *J Neurosurg*. 1969;30:38-41.
473.   Shinomiya K, Nakao K, Shindoh S, Mochida K, Furuya K. Evaluation of cervical diskography in pain origin and provocation. *Journal of spinal disorders*. 1993;6:422-426.
474.   Roth DA. Cervical analgesic discography. A new test for the definitive diagnosis of the painful-disk syndrome. *Jama*. 1976;235:1713-1714.
475.   Zeidman SM, Thompson K, Ducker TB. Complications of cervical discography: analysis of 4400 diagnostic disc injections. *Neurosurgery*. 1995;37:414-417.
476.   Cohen SP, Larkin TM, Barna SA, Palmer WE, Hecht AC, Stojanovic MP. Lumbar discography: a comprehensive review of outcome studies, diagnostic accuracy, and principles. *Reg Anesth Pain Med*. 2005;30:163-183.
477.   Whitecloud TS, 3rd, Seago RA. Cervical discogenic syndrome. Results of operative intervention in patients with positive discography. *Spine* 1987;12:313-316.
478.   Carragee EJ, Barcohana B, Alamin T, van den Haak E. Prospective controlled study of the development of lower back pain in previously asymptomatic subjects undergoing experimental discography. *Spine (Phila Pa 1976)*. 2004;29:1112-1117.
479.   Carragee EJ, Lincoln T, Parmar VS, Alamin T. A gold standard evaluation of the "discogenic pain" diagnosis as determined by provocative discography. *Spine (Phila Pa 1976)*. 2006;31:2115-2123.
480.   Maghout Juratli S, Franklin GM, Mirza SK, Wickizer TM, Fulton-Kehoe D. Lumbar fusion outcomes in Washington State workers' compensation. *Spine*. 2006;31:2715-2723.
481.   Carragee EJ, Alamin TF, Carragee JM. Low-pressure positive Discography in subjects asymptomatic of significant low back pain illness. *Spine*. 2006;31:505-509.
482.   Kapoor SG, Huff J, Cohen SP. Systematic review of the incidence of discitis after cervical discography. *The spine journal : official journal of the North American Spine Society*. 2010;10:739-745.
483.   Sharma SK, Jones JO, Zeballos PP, Irwin SA, Martin TW. The prevention of discitis during discography. *The spine journal : official journal of the North American Spine Society*. 2009;9:936-943.
484.   Guyer RD, Collier R, Stith WJ, et al. Discitis after discography. *Spine* 1988;13:1352-1354.
485.   Parfenchuck TA, Janssen ME. A correlation of cervical magnetic resonance imaging and discography/computed tomographic discograms. *Spine* 1994;19:2819-2825.
486.   Pneumaticos SG, Reitman CA, Lindsey RW. Diskography in the evaluation of low back pain. *The Journal of the American Academy of Orthopaedic Surgeons*. 2006;14:46-55.
487.   Carragee EJ, Hurwitz EL, Cheng I, et al. Treatment of neck pain: injections and surgical interventions: results of the Bone and Joint Decade 2000-2010 Task Force on Neck Pain and Its Associated Disorders. *Spine*. 2008;33:S153-169.
488.   Hartog A. Interventional treatment for low back pain: general risks. *Physical medicine and rehabilitation clinics of North America*. 2010;21:819-823.

489.   Gill K, Jackson R. CT-Discography. In: Frymoyer J, ed. *The Adult Spine: Principles and Practice*. New York: Raven Press, Ltd.; 1991:443-456.

490.   Jackson RP, Becker GJ, Jacobs RR, Montesano PX, Cooper BR, McManus GE. The neuroradiographic diagnosis of lumbar herniated nucleus pulposus: I. A comparison of computed tomography (CT), myelography, CT-myelography, discography, and CT-discography. *Spine*. 1989;14:1356-1361.

491.   Jackson RP, Cain JE, Jr., Jacobs RR, Cooper BR, McManus GE. The neuroradiographic diagnosis of lumbar herniated nucleus pulposus: II. A comparison of computed tomography (CT), myelography, CT-myelography, and magnetic resonance imaging. *Spine*. 1989;14:1362-1367.

492.   Derby R, Howard MW, Grant JM, Lettice JJ, Van Peteghem PK, Ryan DP. The ability of pressure-controlled discography to predict surgical and nonsurgical outcomes. *Spine (Phila Pa 1976)*. 1999;24:364-371; discussion 371-362.

493.   Carragee EJ, Alamin TF, Miller J, Grafe M. Provocative discography in volunteer subjects with mild persistent low back pain. *The spine journal : official journal of the North American Spine Society*. 2002;2:25-34.

494.   Birney TJ, White JJ, Jr., Berens D, Kuhn G. Comparison of MRI and discography in the diagnosis of lumbar degenerative disc disease. *Journal of spinal disorders*. 1992;5:417-423.

495.   Collins CD, Stack JP, O'Connell DJ, et al. The role of discography in lumbar disc disease: a comparative study of magnetic resonance imaging and discography. *Clinical radiology*. 1990;42:252-257.

496.   Gibson MJ, Buckley J, Mawhinney R, Mulholland RC, Worthington BS. Magnetic resonance imaging and discography in the diagnosis of disc degeneration. A comparative study of 50 discs. *The Journal of bone and joint surgery*. 1986;68:369-373.

497.   Ito M, Incorvaia KM, Yu SF, Fredrickson BE, Yuan HA, Rosenbaum AE. Predictive signs of discogenic lumbar pain on magnetic resonance imaging with discography correlation. *Spine*. 1998;23:1252-1258; discussion 1259-1260.

498.   Linson MA, Crowe CH. Comparison of magnetic resonance imaging and lumbar discography in the diagnosis of disc degeneration. *Clinical orthopaedics and related research*. 1990:160-163.

499.   Madan S, Gundanna M, Harley JM, Boeree NR, Sampson M. Does provocative discography screening of discogenic back pain improve surgical outcome? *Journal of spinal disorders & techniques*. 2002;15:245-251.

500.   Osti OL, Fraser RD. MRI and discography of annular tears and intervertebral disc degeneration. A prospective clinical comparison. *The Journal of bone and joint surgery*. 1992;74:431-435.

501.   Schneiderman G, Flannigan B, Kingston S, Thomas J, Dillin WH, Watkins RG. Magnetic resonance imaging in the diagnosis of disc degeneration: correlation with discography. *Spine*. 1987;12:276-281.

502.   Carragee EJ, Chen Y, Tanner CM, Hayward C, Rossi M, Hagle C. Can discography cause long-term back symptoms in previously asymptomatic subjects? *Spine (Phila Pa 1976)*. 2000;25:1803-1808.

503.   Manchikanti L, Singh V, Pampati V, et al. Provocative discography in low back pain patients with or without somatization disorder: a randomized prospective evaluation. *Pain Physician*. 2001;4:227-239.

504.   Walsh TR, Weinstein JN, Spratt KF, Lehmann TR, Aprill C, Sayre H. Lumbar discography in normal subjects. A controlled, prospective study. *The Journal of bone and joint surgery American volume*. 1990;72:1081-1088.

505.   Derby R, Kim BJ, Chen Y, Seo KS, Lee SH. The relation between annular disruption on computed tomography scan and pressure-controlled diskography. *Arch Phys Med Rehabil*. 2005;86:1534-1538.

506.   Derby R, Kim BJ, Lee SH, Chen Y, Seo KS, Aprill C. Comparison of discographic findings in asymptomatic subject discs and the negative discs of chronic LBP patients: can discography distinguish asymptomatic discs among morphologically abnormal discs? *The spine journal : official journal of the North American Spine Society*. 2005;5:389-394.

507.   Derby R, Lee SH, Kim BJ, Chen Y, Aprill C, Bogduk N. Pressure-controlled lumbar discography in volunteers without low back symptoms. *Pain Med*. 2005;6:213-221; discussion 222-214.

508.   Nordin M, Carragee EJ, Hogg-Johnson S, et al. Assessment of neck pain and its associated disorders: results of the Bone and Joint Decade 2000-2010 Task Force on Neck Pain and Its Associated Disorders. *Journal of manipulative and physiological therapeutics*. 2009;32:S117-140.

509.   Kluner C, Kivelitz D, Rogalla P, Putzier M, Hamm B, Enzweiler C. Percutaneous discography: comparison of low-dose CT, fluoroscopy and MRI in the diagnosis of lumbar disc disruption. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2006;15:620-626.

510.   Kallewaard JW, Vanelderen P, Richardson J, Van Zundert J, Heavner J, Groen GJ. Epiduroscopy for patients with lumbosacral radicular pain. *Pain practice : the official journal of World Institute of Pain*. 2014;14:365-377.

511.   Manchikanti L, Singh V. Epidural lysis of adhesions and myeloscopy. *Current pain and headache reports*. 2002;6:427-435.

512.   Raffaeli W, Righetti D, Andruccioli J, Sarti D. Periduroscopy: general review of clinical features and development of operative models. *Acta neurochirurgica Supplement*. 2011;108:55-65.

513.   Bosscher HA, Heavner JE. Diagnosis of the vertebral level from which low back or leg pain originates. A comparison of clinical evaluation, MRI and epiduroscopy. *Pain practice : the official journal of World Institute of Pain*. 2012;12:506-512.

514.   Richardson J, McGurgan P, Cheema S, Prasad R, Gupta S. Spinal endoscopy in chronic low back pain with radiculopathy. A prospective case series. *Anaesthesia*. 2001;56:454-460.

515.   Manchikanti L, Rivera JJ, Pampati V, et al. Spinal endoscopic adhesiolysis in the management of chronic low back pain: a preliminary report of a randomized, double-blind trial. *Pain Physician*. 2003;6:259-267.

516.   Waddell G, Burton AK. Occupational health guidelines for the management of low back pain at work: evidence review. *Occup Med (Lond)*. 2001;51:124-135.

517.   Kapoor S, Shaw WS, Pransky G, Patterson W. Initial patient and clinician expectations of return to work after acute onset of work-related low back pain. *Journal of occupational and environmental medicine / American College of Occupational and Environmental Medicine*. 2006;48:1173-1180.

518.   Strand LI, Ljunggren AE, Haldorsen EM, Espehaug B. The impact of physical function and pain on work status at 1-year follow-up in patients with back pain. *Spine*. 2001;26:800-808.

519.   Talmage JB, Melhorn JM, eds. *A Physcian's Guide to Return to Work*. Chicago, Ill: AMA Press; 2007.

520.   Scheel IB, Hagen KB, Herrin J, Carling C, Oxman AD. Blind faith? The effects of promoting active sick leave for back pain patients: a cluster-randomized controlled trial. *Spine*. 2002;27:2734-2740.

521.   Coomes EN. A comparison between epidural anaesthesia and bed rest in sciatica. *British medical journal*. 1961;1:20-24.

522.   Dahm KT, Brurberg KG, Jamtvedt G, Hagen KB. Advice to rest in bed versus advice to stay active for acute low-back pain and sciatica. *The Cochrane database of systematic reviews*. 2010:CD007612.

523.   Deyo RA, Diehl AK, Rosenthal M. How many days of bed rest for acute low back pain? A randomized clinical trial. *N Engl J Med*. 1986;315:1064-1070.

524.   Evans C, Gilbert J, Taylor W, A H. A randomized controlled trial of flexion exercises, education, and bed rest for patients with acute low back pain. *Physiotherapy Canada* 1987;39:96-101.

525.   Gilbert JR, Taylor DW, Hildebrand A, Evans C. Clinical trial of common treatments for low back pain in family practice. *BMJ (Clinical research ed*. 1985;291:791-794.

526.   Hagen EM, Eriksen HR, Ursin H. Does early intervention with a light mobilization program reduce long-term sick leave for low back pain? *Spine*. 2000;25:1973-1976.

527.   Hofstee DJ, Gijtenbeek JM, Hoogland PH, et al. Westeinde sciatica trial: randomized controlled study of bed rest and physiotherapy for acute sciatica. *J Neurosurg*. 2002;96:45-49.

528.   Jensen RK, Leboeuf-Yde C, Wedderkopp N, Sorensen JS, Manniche C. Rest versus exercise as treatment for patients with low back pain and Modic changes. A randomized controlled clinical trial. *BMC Med*. 2012;10:22.

529.   Malmivaara A, Hakkinen U, Aro T, et al. The treatment of acute low back pain--bed rest, exercises, or ordinary activity? *N Engl J Med*. 1995;332:351-355.

530.   Molde Hagen E, Grasdal A, Eriksen HR. Does early intervention with a light mobilization program reduce long-term sick leave for low back pain: a 3-year follow-up study. *Spine*. 2003;28:2309.

531.   Rozenberg S, Delval C, Rezvani Y, et al. Bed rest or normal activity for patients with acute low back pain: a randomized controlled trial. *Spine*. 2002;27:1487-1493.

532.   Szpalski M, Hayez JP. How many days of bed rest for acute low back pain? Objective assessment of trunk function. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 1992;1:29-31.

533.   Vroomen PC, de Krom MC, Wilmink JT, Kester AD, Knottnerus JA. Lack of effectiveness of bed rest for sciatica. *N Engl J Med*. 1999;340:418-423.

534.   Waddell G, Feder G, Lewis M. Systematic reviews of bed rest and advice to stay active for acute low back pain. *Br J Gen Pract*. 1997;47:647-652.

535.   Wiesel SW, Cuckler JM, Deluca F, Jones F, Zeide MS, Rothman RH. Acute low-back pain. An objective analysis of conservative therapy. *Spine*. 1980;5:324-330.

536.   Wilkinson MJ. Does 48 hours' bed rest influence the outcome of acute low back pain? *Br J Gen Pract*. 1995;45:481-484.

537.   Delitto A, Cibulka MT, Erhard RE, Bowling RW, Tenhula JA. Evidence for use of an extension-mobilization category in acute low back syndrome: a prescriptive validation pilot study. *Phys Ther*. 1993;73:216-222; discussion 223-218.

538.   Williams MM, Hawley JA, McKenzie RA, van Wijmen PM. A comparison of the effects of two sitting postures on back and referred pain. *Spine*. 1991;16:1185-1191.

539.   Marin R, Cyhan T, Miklos W. Sleep disturbance in patients with chronic low back pain. *American journal of physical medicine & rehabilitation / Association of Academic Physiatrists*. 2006;85:430-435.

540.   Levy H, Hutton WC. Mattresses and sleep for patients with low back pain: a survey of orthopaedic surgeons. *J South Orthop Assoc*. 1996;5:185-187.

541.   Hagino C, Erfanian P. Before-after study to determine the effectiveness of an adjustable wood frame-foam and wool mattress bed-system (The Natura Mattress System) in reducing chronic back pain in adults. *J Can Chiropr Assoc*. 1997;41:16-26.

542.   Kovacs FM, Abraira V, Pena A, et al. Effect of firmness of mattress on chronic non-specific low-back pain: randomised, double-blind, controlled, multicentre trial. *Lancet*. 2003;362:1599-1604.

543.   Bergholdt K, Fabricius RN, Bendix T. Better backs by better beds? *Spine*. 2008;33:703-708.

544.   Garfin SR, Pye SA. Bed design and its effect on chronic low back pain--a limited controlled trial. *Pain*. 1981;10:87-91.

545.   Monsein M, Corbin TP, Culliton PD, Merz D, Schuck EA. Short-term outcomes of chronic back pain patients on an airbed vs innerspring mattresses. *MedGenMed*. 2000;2:E36.

546.   Beinart NA, Goodchild CE, Weinman JA, Ayis S, Godfrey EL. Individual and intervention-related factors associated with adherence to home exercise in chronic low back pain: a systematic review. *The spine journal : official journal of the North American Spine Society*. 2013;13:1940-1950.

547.   Bell JA, Burnett A. Exercise for the primary, secondary and tertiary prevention of low back pain in the workplace: a systematic review. *Journal of occupational rehabilitation*. 2009;19:8-24.

548.   Brede E, Mayer TG, Shea M, Garcia C, Gatchel RJ. Facilitating unequivocal and durable decisions in workers' compensation patients eligible for elective orthopedic surgery. *The journal of pain : official journal of the American Pain Society*. 2014;15:49-58.

549.   Brennan GP, Fritz JM, Hunter SJ, Thackeray A, Delitto A, Erhard RE. Identifying subgroups of patients with acute/subacute "nonspecific" low back pain: results of a randomized clinical trial. *Spine*. 2006;31:623-631.

550.   Browder DA, Childs JD, Cleland JA, Fritz JM. Effectiveness of an extension-oriented treatment approach in a subgroup of subjects with low back pain: a randomized clinical trial. *Physical therapy*. 2007;87:1608-1618; discussion 1577-1609.

551.   Colle F, Rannou F, Revel M, Fermanian J, Poiraudeau S. Impact of quality scales on levels of evidence inferred from a systematic review of exercise therapy and low back pain. *Archives of physical medicine and rehabilitation*. 2002;83:1745-1752.

552.   Delitto A, George SZ, Van Dillen LR, et al. Low back pain. *The Journal of orthopaedic and sports physical therapy*. 2012;42:A1-57.

553.   Hauggaard A, Persson A. Specific spinal stabilisation exercises in patients with low back pain: a systematic review. *Phys Ther Rev*. 2007;12:233-248.

554.   Hayden JA, van Tulder MW, Malmivaara A, Koes BW. Exercise therapy for treatment of non-specific low back pain. *Cochrane Database Syst Rev*. 2005:CD000335.

555.   Hendrick P, Milosavljevic S, Hale L, et al. The relationship between physical activity and low back pain outcomes: a systematic review of observational studies. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2011;20:464-474.

556.   Hendrick P, Te Wake AM, Tikkisetty AS, Wulff L, Yap C, Milosavljevic S. The effectiveness of walking as an intervention for low back pain: a systematic review. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2010;19:1613-1620.

557.   Mayer J, Mooney V, Dagenais S. Evidence-informed management of chronic low back pain with lumbar extensor strengthening exercises. *The spine journal : official journal of the North American Spine Society*. 2008;8:96-113.

558.   Mayer TG, Gatchel RJ, Brede E, Theodore BR. Lumbar surgery in work-related chronic low back pain: can a continuum of care enhance outcomes? *The spine journal : official journal of the North American Spine Society*. 2014;14:263-273.

559.   Mayer TG, Neblett R, Brede E, Gatchel RJ. The quantified lumbar flexion-relaxation phenomenon is a useful measurement of improvement in a functional restoration program. *Spine*. 2009;34:2458-2465.

560.   Escolar-Reina P, Medina-Mirapeix F, Gascon-Canovas JJ, et al. How do care-provider and home exercise program characteristics affect patient adherence in chronic neck and back pain: a qualitative study. *BMC health services research*. 2010;10:60.

561.   Fersum KV, Dankaerts W, O'Sullivan PB, et al. Integration of subclassification strategies in randomised controlled clinical trials evaluating manual therapy treatment and exercise therapy for non-specific chronic low back pain: a systematic review. *British journal of sports medicine*. 2010;44:1054-1062.

562.   Hayden JA, Cartwright JL, Riley RD, Vantulder MW, Chronic Low Back Pain IPDM-AG. Exercise therapy for chronic low back pain: protocol for an individual participant data meta-analysis. *Systematic reviews*. 2012;1:64.

563.	Jenkins EM, Borenstein DG. Exercise for the low back pain patient. *Bailliere's clinical rheumatology*. 1994;8:191-197.

564.	Kent P, Mjosund HL, Petersen DH. Does targeting manual therapy and/or exercise improve patient outcomes in nonspecific low back pain? A systematic review. *BMC Med*. 2010;8:22.

565.	Kilpikoski S, Alen M, Paatelma M, Simonen R, Heinonen A, Videman T. Outcome comparison among working adults with centralizing low back pain: secondary analysis of a randomized controlled trial with 1-year follow-up. *Adv Physiother*. 2009;11:210-217.

566.	Krein SL, Metreger T, Kadri R, et al. Veterans walk to beat back pain: study rationale, design and protocol of a randomized trial of a pedometer-based internet mediated intervention for patients with chronic low back pain. *BMC musculoskeletal disorders*. 2010;11:205.

567.	Laird RA, Kent P, Keating JL. Modifying patterns of movement in people with low back pain -does it help? A systematic review. *BMC musculoskeletal disorders*. 2012;13:169.

568.	Liddle SD, Gracey JH, Baxter GD. Advice for the management of low back pain: a systematic review of randomised controlled trials. *Manual therapy*. 2007;12:310-327.

569.	Lim EC, Poh RL, Low AY, Wong WP. Effects of Pilates-based exercises on pain and disability in individuals with persistent nonspecific low back pain: a systematic review with meta-analysis. *The Journal of orthopaedic and sports physical therapy*. 2011;41:70-80.

570.	Fritz JM, Cleland JA, Brennan GP. Does adherence to the guideline recommendation for active treatments improve the quality of care for patients with acute low back pain delivered by physical therapists? *Med Care*. 2007;45:973-980.

571.	Hettinga D, Jackson A, Moffett J, May S, Mercer C, Woby S. A systematic review and synthesis of higher quality evidence of the effectiveness of exercise interventions for non-specific low back pain of at least 6 weeks' duration. *Phys Ther Rev*. 2007;12:221-232.

572.	Hilde G, Hagen KB, Jamtvedt G, Winnem M. Advice to stay active as a single treatment for low back pain and sciatica. *The Cochrane database of systematic reviews*. 2002:CD003632.

573.	Macedo LG, Smeets RJ, Maher CG, Latimer J, McAuley JH. Graded activity and graded exposure for persistent nonspecific low back pain: a systematic review. *Physical therapy*. 2010;90:860-879.

574.	McDonough SM, Tully MA, O'Connor SR, et al. The back 2 activity trial: education and advice versus education and advice plus a structured walking programme for chronic low back pain. *BMC musculoskeletal disorders*. 2010;11:163.

575.	Medina-Mirapeix F, Escolar-Reina P, Gascon-Canovas JJ, Montilla-Herrador J, Collins SM. Personal characteristics influencing patients' adherence to home exercise during chronic pain: a qualitative study. *Journal of rehabilitation medicine*. 2009;41:347-352.

576.	Medina-Mirapeix F, Escolar-Reina P, Gascon-Canovas JJ, Montilla-Herrador J, Jimeno-Serrano FJ, Collins SM. Predictive factors of adherence to frequency and duration components in home exercise programs for neck and low back pain: an observational study. *BMC Musculoskelet Disord*. 2009;10:155.

577.	McFeely J, Gracey J. Postoperative exercise programmes for lumbar spine decompression surgery: a systematic review of the evidence. *Phys Ther Rev*. 2006;11:248-262.

578.	Oesch P, Kool J, Hagen KB, Bachmann S. Effectiveness of exercise on work disability in patients with non-acute non-specific low back pain: Systematic review and meta-analysis of randomised controlled trials. *Journal of rehabilitation medicine : official journal of the UEMS European Board of Physical and Rehabilitation Medicine*. 2010;42:193-205.

579.	Posadzki P, Ernst E. Yoga for low back pain: a systematic review of randomized clinical trials. *Clinical rheumatology*. 2011;30:1257-1262.

580.	Schaafsma F, Schonstein E, Ojajarvi A, Verbeek J. Physical conditioning programs for improving work outcomes among workers with back pain. *Scandinavian journal of work, environment & health*. 2011;37:1-5.

581.	Slade SC, Keating JL. Trunk-strengthening exercises for chronic low back pain: a systematic review. *Journal of manipulative and physiological therapeutics*. 2006;29:163-173.

582.	Smeets RJ, Vlaeyen JW, Hidding A, et al. Active rehabilitation for chronic low back pain: cognitive-behavioral, physical, or both? First direct post-treatment results from a randomized controlled trial [ISRCTN22714229]. *BMC musculoskeletal disorders*. 2006;7:5.

583.	Swinkels A, Cochrane K, Burt A, Johnson L, Lunn T, Sian Rees A. Exercise interventions for non-specific low back pain: an overview of systematic reviews. *Phys Ther Rev*. 2009;14:247-259.

584.	Teasell RW, Harth M. Functional restoration. Returning patients with chronic low back pain to work--revolution or fad? *Spine (Phila Pa 1976)*. 1996;21:844-847.

585.	Theodore BR, Mayer TG, Gatchel RJ. Cost-Effectiveness of Early Versus Delayed Functional Restoration for Chronic Disabling Occupational Musculoskeletal Disorders. *Journal of occupational rehabilitation*. 2014.

586.   Torstensen TA, Ljunggren AE, Meen HD, Odland E, Mowinckel P, af Geijerstam S. Efficiency and costs of medical exercise therapy, conventional physiotherapy, and self-exercise in patients with chronic low back pain - A pragmatic, randomized, single-blinded, controlled trial with 1-year follow-up. *Spine*. 1998;23:2616-2624.

587.   Stanton TR, Fritz JM, Hancock MJ, et al. Evaluation of a treatment-based classification algorithm for low back pain: a cross-sectional study. *Physical therapy*. 2011;91:496-509.

588.   van Middelkoop M, Rubinstein SM, Kuijpers T, et al. A systematic review on the effectiveness of physical and rehabilitation interventions for chronic non-specific low back pain. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2011;20:19-39.

589.   van Tulder M, Malmivaara A, Esmail R, Koes B. Exercise therapy for low back pain: a systematic review within the framework of the cochrane collaboration back review group. *Spine*. 2000;25:2784-2796.

590.   Chatzitheodorou D, Kabitsis C, Malliou P, Mougios V. A pilot study of the effects of high-intensity aerobic exercise versus passive interventions on pain, disability, psychological strain, and serum cortisol concentrations in people with chronic low back pain. *Phys Ther*. 2007;87:304-312.

591.   Choi B, Verbeek J, Tam W, Jiang J. Exercises for prevention of recurrences of low-back pain. *Cochrane Database Syst Rev* 2010;1::CD006555.

592.   Pescatello LE. *ACSM's Guidelines for Exercise Testing and Prescription. 9th ed*. Baltimore, MD: Lippincott Williams & Wilikins; 2014.

593.   Delitto A, Piva SR, Moore CG, et al. Surgery versus nonsurgical treatment of lumbar spinal stenosis: a randomized trial. *Annals of internal medicine*. 2015;162:465-473.

594.   Chan CW, Mok NW, Yeung EW. Aerobic exercise training in addition to conventional physiotherapy for chronic low back pain: a randomized controlled trial. *Arch Phys Med Rehabil*. 2011;92:1681-1685.

595.   Childs JD, Fritz JM, Flynn TW, et al. A clinical prediction rule to identify patients with low back pain most likely to benefit from spinal manipulation: a validation study. *Ann Intern Med*. 2004;141:920-928.

596.   Cuesta-Vargas AI, Adams N, Salazar JA, Belles A, Hazanas S, Arroyo-Morales M. Deep water running and general practice in primary care for non-specific low back pain versus general practice alone: randomized controlled trial. *Clinical rheumatology*. 2012;31:1073-1078.

597.   Doğan S, Tur B, Kurtaiş Y, Atay M. Comparison of three different approaches in the treatment of chronic low back pain. *Clinical rheumatology*. 2008;27:873-881.

598.   Evjenth O, Hamberg J. *Muscle Stretching In Manual Therapy: A Clinical Manual*. Alfta, Sweden: Alfta Rehab 1984.

599.   Fritz JM, Delitto A, Erhard RE. Comparison of classification-based physical therapy with therapy based on clinical practice guidelines for patients with acute low back pain: a randomized clinical trial. *Spine*. 2003;28:1363-1371; discussion 1372.

600.   Goldby LJ, Moore AP, Doust J, Trew ME. A randomized controlled trial investigating the efficiency of musculoskeletal physiotherapy on chronic low back disorder. *Spine*. 2006;31:1083-1093.

601.   Jousset N, Fanello S, Bontoux L, et al. Effects of functional restoration versus 3 hours per week physical therapy: a randomized controlled study. *Spine*. 2004;29:487-493; discussion 494.

602.   Kell RT, Asmundson GJ. A comparison of two forms of periodized exercise rehabilitation programs in the management of chronic nonspecific low-back pain. *Journal of strength and conditioning research / National Strength & Conditioning Association*. 2009;23:513-523.

603.   Murtezani A, Hundozi H, Orovcanec N, Sllamniku S, Osmani T. A comparison of high intensity aerobic exercise and passive modalities for the treatment of workers with chronic low back pain: a randomized, controlled trial. *European journal of physical and rehabilitation medicine*. 2011;47:359-366.

604.   Sculco AD, Paup DC, Fernhall B, Sculco MJ. Effects of aerobic exercise on low back pain patients in treatment. *The spine journal : official journal of the North American Spine Society*. 2001;1:95-101.

605.   Shnayderman I, Katz-Leurer M. An aerobic walking programme versus muscle strengthening programme for chronic low back pain: a randomized controlled trial. *Clinical rehabilitation*. 2013;27:207-214.

606.   Smeets R. Chronic low back pain: Physical training, graded activity with problem solving training, or both? The one-year post-treatment results of a randomized controlled trial. *Pain*. 2008;134:263-276.

607.   Tritilanunt T, Wajanavisit W. The efficacy of an aerobic exercise and health education program for treatment of chronic low back pain. *Journal of the Medical Association of Thailand = Chotmaihet thangphaet*. 2001;84 Suppl 2:S528-533.

608.   Weiner DK, Perera S, Rudy TE, Glick RM, Shenoy S, Delitto A. Efficacy of percutaneous electrical nerve stimulation and therapeutic exercise for older adults with chronic low back pain: a randomized controlled trial. *Pain*. 2008;140:344-357.

609.   Storheim K, Brox JI, Holm I, Koller AK, Bo K. Intensive group training versus cognitive intervention in sub-acute low back pain: short-term results of a single-blind randomized controlled trial. *Journal of rehabilitation medicine*. 2003;35:132-140.
610.   Schenk R, Jozefczyk C, Kopf A. A randomized trial comparing interventions in patients with lumbar posterior derangement. *The Journal of manual & manipulative therapy*. 2003;11:95-102.
611.   Stankovic A, Lazovic M, Kocic M, et al. Lumbar stabilization exercises in addition to strengthening and stretching exercises reduce pain and increase function in patients with chronic low back pain. *Türk Fiz Tıp Rehab Derg*. 2012;58:178-184.
612.   McKenzie R, May S. *The Lumbar Spine: Mechanical Diagnosis and Therapy (2nd Ed)*. Waikanae, New Zealand: Spinal Publications; 2003.
613.   Filiz M, Cakmak A, Ozcan E. The effectiveness of exercise programmes after lumbar disc surgery: a randomized controlled study. *Clin Rehabil*. 2005;19:4-11.
614.   Hides JA, Jull GA, Richardson CA. Long-term effects of specific stabilizing exercises for first-episode low back pain. *Spine*. 2001;26:E243-248.
615.   Soukup MG, Glomsrod B, Lonn JH, Bo K, Larsen S. The effect of a Mensendieck exercise program as secondary prophylaxis for recurrent low back pain. A randomized, controlled trial with 12-month follow-up. *Spine*. 1999;24:1585-1591; discussion 1592.
616.   Soukup MG, Lonn J, Glomsrod B, Bo K, Larsen S. Exercises and education as secondary prevention for recurrent low back pain. *Physiother Res Int*. 2001;6:27-39.
617.   Wittink H, Michel TH, Kulich R, et al. Aerobic fitness testing in patients with chronic low back pain: which test is best? *Spine*. 2000;25:1704-1710.
618.   Hurwitz EL, Morgenstern H, Chiao C. Effects of recreational physical activity and back exercises on low back pain and psychological distress: findings from the UCLA Low Back Pain Study. *American journal of public health*. 2005;95:1817-1824.
619.   Machado LA, Azevedo DC, Capanema MB, Neto TN, Cerceau DM. Client-centered therapy vs exercise therapy for chronic low back pain: a pilot randomized controlled trial in Brazil. *Pain Med*. 2007;8:251-258.
620.   Mannion AF, Muntener M, Taimela S, Dvorak J. A randomized clinical trial of three active therapies for chronic low back pain. *Spine*. 1999;24:2435-2448.
621.   Turner JA, Clancy S, McQuade KJ, Cardenas DD. Effectiveness of behavioral therapy for chronic low back pain: a component analysis. *J Consult Clin Psychol*. 1990;58:573-579.
622.   Kuukkanen T, Malkia E. Effects of a three-month therapeutic exercise programme on flexibility in subjects with low back pain. *Physiother Res Int*. 2000;5:46-61.
623.   Aina A, May S, Clare H. The centralization phenomenon of spinal symptoms--a systematic review. *Manual therapy*. 2004;9:134-143.
624.   Petersen T, Larsen K, Nordsteen J, Olsen S, Fournier G, Jacobsen S. The McKenzie method compared with manipulation when used adjunctive to information and advice in low back pain patients presenting with centralization or peripheralization: a randomized controlled trial. *Spine*. 2011;36:1999-2010.
625.   Werneke MW, Hart DL, Cutrone G, et al. Association between directional preference and centralization in patients with low back pain. *The Journal of orthopaedic and sports physical therapy*. 2011;41:22-31.
626.   Werneke MW, Hart DL, Resnik L, Stratford PW, Reyes A. Centralization: prevalence and effect on treatment outcomes using a standardized operational definition and measurement method. *The Journal of orthopaedic and sports physical therapy*. 2008;38:116-125.
627.   Long A, May S, Fung T. The comparative prognostic value of directional preference and centralization: a useful tool for front-line clinicians? *The Journal of manual & manipulative therapy*. 2008;16:248-254.
628.   Purepong N, Jitvimonrat A, Boonyong S, Thaveeratitham P, Pensri P. Effect of flexibility exercise on lumbar angle: a study among non-specific low back pain patients. *J Bodyw Mov Ther*. 2012;16:236-243.
629.   Schnebel BE, Simmons JW, Chowning J, Davidson R. A digitizing technique for the study of movement of intradiscal dye in response to flexion and extension of the lumbar spine. *Spine*. 1988;13:309-312.
630.   Seroussi RE, Krag MH, Muller DL, Pope MH. Internal deformations of intact and denucleated human lumbar discs subjected to compression, flexion, and extension loads. *Journal of orthopaedic research : official publication of the Orthopaedic Research Society*. 1989;7:122-131.
631.   Scannell JP, McGill SM. Disc prolapse: evidence of reversal with repeated extension. *Spine*. 2009;34:344-350.
632.   Faas A, van Eijk JT, Chavannes AW, Gubbels JW. A randomized trial of exercise therapy in patients with acute low back pain. Efficacy on sickness absence. *Spine*. 1995;20:941-947.
633.   Maitland G. The slump test: examination and treatment. *The Australian journal of physiotherapy*. 1985;31:215-219.
634.   Khalil TM, Asfour SS, Martinez LM, Waly SM, Rosomoff RS, Rosomoff HL. Stretching in the rehabilitation of low-back pain patients. *Spine*. 1992;17:311-317.

635.   Anema JR, Steenstra IA, Bongers PM, et al. Multidisciplinary rehabilitation for subacute low back pain: graded activity or workplace intervention or both? A randomized controlled trial. *Spine*. 2007;32:291.

636.   Johannsen F, Remvig L, Kryger P, et al. Exercises for chronic low back pain: a clinical trial. *The Journal of orthopaedic and sports physical therapy*. 1995;22:52-59.

637.   Lindstrom I, Ohlund C, Eek C, et al. The effect of graded activity on patients with subacute low back pain: a randomized prospective clinical study with an operant-conditioning behavioral approach. *Phys Ther*. 1992;72:279-290; discussion 291-273.

638.   Lindstrom I, Ohlund C, Eek C, Wallin L, Peterson LE, Nachemson A. Mobility, strength, and fitness after a graded activity program for patients with subacute low back pain. A randomized prospective clinical study with a behavioral therapy approach. *Spine*. 1992;17:641-652.

639.   Ljunggren AE, Weber H, Kogstad O, Thom E, Kirkesola G. Effect of exercise on sick leave due to low back pain. A randomized, comparative, long-term study. *Spine*. 1997;22:1610-1616; discussion 1617.

640.   Manniche C, Hesselsoe G, Bentzen L, Christensen I, Lundberg E. Clinical trial of intensive muscle training for chronic low back pain. *Lancet*. 1988;2:1473-1476.

641.   Rittweger J, Just K, Kautzsch K, Reeg P, Felsenberg D. Treatment of chronic lower back pain with lumbar extension and whole-body vibration exercise: a randomized controlled trial. *Spine*. 2002;27:1829-1834.

642.   Yilmaz F, Yilmaz A, Merdol F, Parlar D, Sahin F, Kuran B. Efficacy of dynamic lumbar stabilization exercise in lumbar microdiscectomy. *Journal of rehabilitation medicine*. 2003;35:163-167.

643.   Bentsen H, Lindgarde F, Manthorpe R. The effect of dynamic strength back exercise and/or a home training program in 57-year-old women with chronic low back pain. Results of a prospective randomized study with a 3-year follow-up period. *Spine*. 1997;22:1494-1500.

644.   Garg A, Thiese M, Hegmann K. Individual factors associated with prevalence of low back pain. *Presented at the 6th International Scientific Conference on Prevention of Work-Realted Musculoskeletal Disorders (PREMUS) Boston, MA*. 2007.

645.   O'Sullivan PB, Phyty GD, Twomey LT, Allison GT. Evaluation of specific stabilizing exercise in the treatment of chronic low back pain with radiologic diagnosis of spondylolysis or spondylolisthesis. *Spine*. 1997;22:2959-2967.

646.   van Tulder MW, Koes BW, Bouter LM. Conservative treatment of acute and chronic nonspecific low back pain. A systematic review of randomized controlled trials of the most common interventions. *Spine*. 1997;22:2128-2156.

647.   Sherman KJ, Cherkin DC, Wellman RD, et al. A randomized trial comparing yoga, stretching, and a self-care book for chronic low back pain. *Archives of internal medicine*. 2011;171:2019-2026.

648.   Staal JB, Hlobil H, Twisk JW, Smid T, Koke AJ, van Mechelen W. Graded activity for low back pain in occupational health care: a randomized, controlled trial. *Ann Intern Med*. 2004;140:77-84.

649.   Hallegraeff JM, de Greef M, Winters JC, Lucas C. Manipulative therapy and clinical prediction criteria in treatment of acute nonspecific low back pain. *Percept Mot Skills*. 2009;108:196-208.

650.   Pope RP, Herbert RD, Kirwan JD, Graham BJ. A randomized trial of preexercise stretching for prevention of lower-limb injury. *Medicine and science in sports and exercise*. 2000;32:271-277.

651.   Stankovic R, Johnell O. Conservative treatment of acute low-back pain. A prospective randomized trial: McKenzie method of treatment versus patient education in "mini back school". *Spine*. 1990;15:120-123.

652.   Stankovic R, Johnell O. Conservative treatment of acute low back pain. A 5-year follow-up study of two methods of treatment. *Spine*. 1995;20:469-472.

653.   Grunnesjo MI, Bogefeldt JP, Svardsudd KF, Blomberg SI. A randomized controlled clinical trial of stay-active care versus manual therapy in addition to stay-active care: functional variables and pain. *Journal of manipulative and physiological therapeutics*. 2004;27:431-441.

654.   Hartvigsen J, Morso L, Bendix T, Manniche C. Supervised and non-supervised Nordic walking in the treatment of chronic low back pain: a single blind randomized clinical trial. *BMC musculoskeletal disorders*. 2010;11:30.

655.   Kim D, Brown J. Efficacy and safety of lumbar epidural dexamethasone versus methylprednisolone in the treatment of lumbar radiculopathy: a comparison of soluble versus particulate steroids. *Clin J Pain*. 2011;27:518-522.

656.   Moffett JK, Torgerson D, Bell-Syer S, et al. Randomised controlled trial of exercise for low back pain: clinical outcomes, costs, and preferences. *Bmj*. 1999;319:279-283.

657.   Steenstra IA, Anema JR, van Tulder MW, Bongers PM, de Vet HC, van Mechelen W. Economic evaluation of a multi-stage return to work program for workers on sick-leave due to low back pain. *J Occup Rehabil*. 2006;16:557-578.

658.   Hancock MJ, Maher CG, Latimer J, Herbert RD, McAuley JH. Independent evaluation of a clinical prediction rule for spinal manipulative therapy: a randomised controlled trial. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2008;17:936-943.

659.	Hlobil H, Uegaki K, Staal JB, de Bruyne MC, Smid T, van Mechelen W. Substantial sick-leave costs savings due to a graded activity intervention for workers with non-specific sub-acute low back pain. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2007;16:919-924.

660.	Lindstrom I, C O, Nachemson A. Physical performance, pain, pain behavior and subjective disability in patients with subacute low back pain. *Scandinavian journal of rehabilitation medicine*. 1995;27:153-160.

661.	Moffett JK, Jackson DA, Gardiner ED, et al. Randomized trial of two physiotherapy interventions for primary care neck and back pain patients: 'McKenzie' vs brief physiotherapy pain management. *Rheumatology (Oxford, England)*. 2006;45:1514-1521.

662.	Aure OF, Nilsen JH, Vasseljen O. Manual therapy and exercise therapy in patients with chronic low back pain: a randomized, controlled trial with 1-year follow-up. *Spine*. 2003;28:525-531; discussion 531-522.

663.	Bi X, Zhao J, Zhao L, et al. Pelvic floor muscle exercise for chronic low back pain. *The Journal of international medical research*. 2013;41:146-152.

664.	Carr JL, Klaber Moffett JA, Howarth E, et al. A randomized trial comparing a group exercise programme for back pain patients with individual physiotherapy in a severely deprived area. *Disability and rehabilitation*. 2005;27:929-937.

665.	Costa LO, Maher CG, Latimer J, et al. Motor control exercise for chronic low back pain: a randomized placebo-controlled trial. *Phys Ther*. 2009;89:1275-1286.

666.	del Pozo-Cruz B, Adsuar JC, Parraca J, Del Pozo-Cruz J, Moreno A, Gusi N. A web-based intervention to improve and prevent low back pain among office workers: a randomized controlled trial. *The Journal of orthopaedic and sports physical therapy*. 2012;42:831-841.

667.	del Pozo-Cruz B, Parraca JA, del Pozo-Cruz J, Adsuar JC, Hill J, Gusi N. An occupational, internet-based intervention to prevent chronicity in subacute lower back pain: a randomised controlled trial. *Journal of rehabilitation medicine*. 2012;44:581-587.

668.	Dufour N, Thamsborg G, Oefeldt A, Lundsgaard C, Stender S. Treatment of chronic low back pain: a randomized, clinical trial comparing group-based multidisciplinary biopsychosocial rehabilitation and intensive individual therapist-assisted back muscle strengthening exercises. *Spine (Phila Pa 1976)*. 2010;35:469-476.

669.	Ferreira ML, Ferreira PH, Latimer J, et al. Comparison of general exercise, motor control exercise and spinal manipulative therapy for chronic low back pain: A randomized trial. *Pain*. 2007;131:31-37.

670.	Franca FR, Burke TN, Caffaro RR, Ramos LA, Marques AP. Effects of muscular stretching and segmental stabilization on functional disability and pain in patients with chronic low back pain: a randomized, controlled trial. *Journal of manipulative and physiological therapeutics*. 2012;35:279-285.

671.	Frost H, Klaber Moffett JA, Moser JS, Fairbank JC. Randomised controlled trial for evaluation of fitness programme for patients with chronic low back pain. *Bmj*. 1995;310:151-154.

672.	Garcia AN, Costa Lda C, da Silva TM, et al. Effectiveness of back school versus McKenzie exercises in patients with chronic nonspecific low back pain: a randomized controlled trial. *Physical therapy*. 2013;93:729-747.

673.	Hakkinen A, Ylinen J, Kautiainen H, Tarvainen U, Kiviranta I. Effects of home strength training and stretching versus stretching alone after lumbar disk surgery: a randomized study with a 1-year follow-up. *Archives of physical medicine and rehabilitation*. 2005;86:865-870.

674.	Harts CC, Helmhout PH, de Bie RA, Staal JB. A high-intensity lumbar extensor strengthening program is little better than a low-intensity program or a waiting list control group for chronic low back pain: a randomised clinical trial. *The Australian journal of physiotherapy*. 2008;54:23-31.

675.	Helmhout PH, Harts CC, Staal JB, Candel MJ, de Bie RA. Comparison of a high-intensity and a low-intensity lumbar extensor training program as minimal intervention treatment in low back pain: a randomized trial. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2004;13:537-547.

676.	Johnson RE, Jones GT, Wiles NJ, et al. Active exercise, education, and cognitive behavioral therapy for persistent disabling low back pain: a randomized controlled trial. *Spine*. 2007;32:1578-1585.

677.	Koumantakis GA, Watson PJ, Oldham JA. Supplementation of general endurance exercise with stabilisation training versus general exercise only. Physiological and functional outcomes of a randomised controlled trial of patients with recurrent low back pain. *Clin Biomech (Bristol, Avon)*. 2005;20:474-482.

678.	Lewis C, Souvlis T, Sterling M. Strain-Counterstrain therapy combined with exercise is not more effective than exercise alone on pain and disability in people with acute low back pain: a randomised trial. *Journal of physiotherapy*. 2011;57:91-98.

679.	Lewis JS, Hewitt JS, Billington L, Cole S, Byng J, Karayiannis S. A randomized clinical trial comparing two physiotherapy interventions for chronic low back pain. *Spine*. 2005;30:711-721.

680.   Macedo L. Effect of Motor Control Exercises Versus Graded Activity in Patients With Chronic Nonspecific Low Back Pain: A Randomized Controlled Trial. *Physical Therapy Journal*. 2012;92:363-377.

681.   Manniche C, Asmussen K, Lauritsen B, et al. Intensive dynamic back exercises with or without hyperextension in chronic back pain after surgery for lumbar disc protrusion. A clinical trial. *Spine*. 1993;18:560-567.

682.   Nagrale AV, Patil SP, Gandhi RA, Learman K. Effect of slump stretching versus lumbar mobilization with exercise in subjects with non-radicular low back pain: a randomized clinical trial. *The Journal of manual & manipulative therapy*. 2012;20:35-42.

683.   Shirado O, Doi T, Akai M, et al. Multicenter randomized controlled trial to evaluate the effect of home-based exercise on patients with chronic low back pain: the Japan low back pain exercise therapy study. *Spine (Phila Pa 1976)*. 2010;35:E811-819.

684.   Wajswelner H, Metcalf B, Bennell K. Clinical pilates versus general exercise for chronic low back pain: randomized trial. *Medicine and science in sports and exercise*. 2012;44:1197-1205.

685.   Yelland MJ, Glasziou PP, Bogduk N, Schluter PJ, McKernon M. Prolotherapy injections, saline injections, and exercises for chronic low-back pain: a randomized trial. *Spine*. 2004;29:9-16; discussion 16.

686.   Diab AA, Moustafa IM. Lumbar lordosis rehabilitation for pain and lumbar segmental motion in chronic mechanical low back pain: a randomized trial. *Journal of manipulative and physiological therapeutics*. 2012;35:246-253.

687.   Diaz Arribas MJ, Ramos Sanchez M, Pardo Hervas P, et al. Effectiveness of the physical therapy Godelive Denys-Struyf method for nonspecific low back pain: primary care randomized control trial. *Spine*. 2009;34:1529-1538.

688.   Ewert T, Limm H, Wessels T, et al. The comparative effectiveness of a multimodal program versus exercise alone for the secondary prevention of chronic low back pain and disability. *PM & R : the journal of injury, function, and rehabilitation*. 2009;1:798-808.

689.   Gatti R, Faccendini S, Tettamanti A, Barbero M, Balestri A, Calori G. Efficacy of trunk balance exercises for individuals with chronic low back pain: a randomized clinical trial. *The Journal of orthopaedic and sports physical therapy*. 2011;41:542-552.

690.   Hansen FR, Bendix T, Skov P, et al. Intensive, dynamic back-muscle exercises, conventional physiotherapy, or placebo-control treatment of low-back pain. A randomized, observer-blind trial. *Spine*. 1993;18:98-108.

691.   Hurwitz EL, Morgenstern H, Harber P, et al. A randomized trial of medical care with and without physical therapy and chiropractic care with and without physical modalities for patients with low back pain: 6-month follow-up outcomes from the UCLA low back pain study. *Spine*. 2002;27:2193-2204.

692.   Kluge J, Hall D, Louw Q, Theron G, Grove D. Specific exercises to treat pregnancy-related low back pain in a South African population. *Int J Gynaecol Obstet*. 2011;113:187-191.

693.   Kuukkanen T, Malkia E, Kautiainen H, Pohjolainen T. Effectiveness of a home exercise programme in low back pain: a randomized five-year follow-up study. *Physiotherapy research international : the journal for researchers and clinicians in physical therapy*. 2007;12:213-224.

694.   Lewis M, Morley S, van der Windt DA, et al. Measuring practitioner/therapist effects in randomised trials of low back pain and neck pain interventions in primary care settings. *Eur J Pain*. 2010;14:1033-1039.

695.   Limke JC, Rainville J, Pena E, Childs L. Randomized trial comparing the effects of one set vs two sets of resistance exercises for outpatients with chronic low back pain and leg pain. *European journal of physical and rehabilitation medicine*. 2008;44:399-405.

696.   Niemisto L, Lahtinen-Suopanki T, Rissanen P, Lindgren KA, Sarna S, Hurri H. A randomized trial of combined manipulation, stabilizing exercises, and physician consultation compared to physician consultation alone for chronic low back pain. *Spine*. 2003;28:2185-2191.

697.   Petersen T, Kryger P, Ekdahl C, Olsen S, Jacobsen S. The effect of McKenzie therapy as compared with that of intensive strengthening training for the treatment of patients with subacute or chronic low back pain: A randomized controlled trial. *Spine*. 2002;27:1702-1709.

698.   Balthazard P, de Goumoens P, Rivier G, Demeulenaere P, Ballabeni P, Deriaz O. Manual therapy followed by specific active exercises versus a placebo followed by specific active exercises on the improvement of functional disability in patients with chronic non specific low back pain: a randomized controlled trial. *BMC Musculoskelet Disord*. 2012;13:162.

699.   Diab A. The efficacy of lumbar extension traction for sagittal alignment in mechanical low back pain: A randomized trial. *J Back Musculoskeletal Rehabil*. 2013;26:213-220.

700.   Elnaggar IM, Nordin M, Sheikhzadeh A, Parnianpour M, Kahanovitz N. Effects of spinal flexion and extension exercises on low-back pain and spinal mobility in chronic mechanical low-back pain patients. *Spine*. 1991;16:967-972.

701.   Friedrich M, Gittler G, Arendasy M, Friedrich KM. Long-term effect of a combined exercise and motivational program on the level of disability of patients with chronic low back pain. *Spine*. 2005;30:995-1000.

702.   Kumar S, Sharma VP, Negi MP. Efficacy of dynamic muscular stabilization techniques (DMST) over conventional techniques in rehabilitation of chronic low back pain. *Journal of strength and conditioning research / National Strength & Conditioning Association*. 2009;23:2651-2659.

703.   Risch SV, Norvell NK, Pollock ML, et al. Lumbar strengthening in chronic low back pain patients. Physiologic and psychological benefits. *Spine*. 1993;18:232-238.

704.   Rydeard R, Leger A, Smith D. Pilates-based therapeutic exercise: effect on subjects with nonspecific chronic low back pain and functional disability: a randomized controlled trial. *The Journal of orthopaedic and sports physical therapy*. 2006;36:472-484.

705.   Snook SH, Webster BS, McGorry RW, Fogleman MT, McCann KB. The reduction of chronic nonspecific low back pain through the control of early morning lumbar flexion. A randomized controlled trial. *Spine (Phila Pa 1976)*. 1998;23:2601-2607.

706.   Tavafian SS, Jamshidi AR, Mohammad K. Treatment of chronic low back pain: a randomized clinical trial comparing multidisciplinary group-based rehabilitation program and oral drug treatment with oral drug treatment alone. *Clin J Pain*. 2011;27:811-818.

707.   Winters MV, Blake CG, Trost JS, et al. Passive versus active stretching of hip flexor muscles in subjects with limited hip extension: a randomized clinical trial. *Phys Ther*. 2004;84:800-807.

708.   Wright A, Lloyd-Davies A, Williams S, Ellis R, Strike P. Individual active treatment combined with group exercise for acute and subacute low back pain. *Spine*. 2005;30:1235-1241.

709.   Bendix AF, Bendix T, Vaegter K, Lund C, Frolund L, Holm L. Multidisciplinary intensive treatment for chronic low back pain: a randomized, prospective study. *Cleveland Clinic journal of medicine*. 1996;63:62-69.

710.   Bronfort G, Goldsmith CH, Nelson CF, Boline PD, Anderson AV. Trunk exercise combined with spinal manipulative or NSAID therapy for chronic low back pain: a randomized, observer-blinded clinical trial. *Journal of manipulative and physiological therapeutics*. 1996;19:570-582.

711.   Cambron JA, Gudavalli MR, Hedeker D, et al. One-year follow-up of a randomized clinical trial comparing flexion distraction with an exercise program for chronic low-back pain. *J Altern Complement Med*. 2006;12:659-668.

712.   Cherkin DC, Deyo RA, Battie M, Street J, Barlow W. A comparison of physical therapy, chiropractic manipulation, and provision of an educational booklet for the treatment of patients with low back pain. *N Engl J Med*. 1998;339:1021-1029.

713.   Engbert K, Weber M. The effects of therapeutic climbing in patients with chronic low back pain: a randomized controlled study. *Spine*. 2011;36:842-849.

714.   Faas A, Chavannes AW, van Eijk JT, Gubbels JW. A randomized, placebo-controlled trial of exercise therapy in patients with acute low back pain. *Spine*. 1993;18:1388-1395.

715.   Goren A, Yildiz N, Topuz O, Findikoglu G, Ardic F. Efficacy of exercise and ultrasound in patients with lumbar spinal stenosis: a prospective randomized controlled trial. *Clinical rehabilitation*. 2010;24:623-631.

716.   Hemmila HM, Keinanen-Kiukaanniemi SM, Levoska S, Puska P. Long-term effectiveness of bone-setting, light exercise therapy, and physiotherapy for prolonged back pain: a randomized controlled trial. *Journal of manipulative and physiological therapeutics*. 2002;25:99-104.

717.   Henchoz Y, de Goumoens P, Norberg M, Paillex R, So AK. Role of physical exercise in low back pain rehabilitation: a randomized controlled trial of a three-month exercise program in patients who have completed multidisciplinary rehabilitation. *Spine (Phila Pa 1976)*. 2010;35:1192-1199.

718.   Henchoz Y, de Goumoens P, So AK, Paillex R. Functional multidisciplinary rehabilitation versus outpatient physiotherapy for non specific low back pain: randomized controlled trial. *Swiss medical weekly*. 2010;140:w13133.

719.   Henchoz Y, Pinget C, Wasserfallen JB, et al. Cost-utility analysis of a three-month exercise programme vs usual care following multidisciplinary rehabilitation for chronic low back pain. *Journal of rehabilitation medicine*. 2010;42:846-852.

720.   Marshall P, Murphy B. Self-report measures best explain changes in disability compared with physical measures after exercise rehabilitation for chronic low back pain. *Spine (Phila Pa 1976)*. 2008;33:326-338.

721.   Pengel LH, Refshauge KM, Maher CG, Nicholas MK, Herbert RD, McNair P. Physiotherapist-directed exercise, advice, or both for subacute low back pain: a randomized trial. *Ann Intern Med*. 2007;146:787-796.

722.   Smeets RJ, Maher CG, Nicholas MK, Refshauge KM, Herbert RD. Do psychological characteristics predict response to exercise and advice for subacute low back pain? *Arthritis and rheumatism*. 2009;61:1202-1209.

723.   Unsgaard-Tondel M, Fladmark AM, Salvesen O, Vasseljen O. Motor control exercises, sling exercises, and general exercises for patients with chronic low back pain: a randomized controlled trial with 1-year follow-up. *Physical therapy*. 2010;90:1426-1440.

724.   Vad VB, Bhat AL, Tarabichi Y. The role of the Back Rx exercise program in diskogenic low back pain: a prospective randomized trial. *Archives of physical medicine and rehabilitation*. 2007;88:577-582.

725.   Vasseljen O, Unsgaard-Tondel M, Westad C, Mork PJ. Effect of core stability exercises on feed-forward activation of deep abdominal muscles in chronic low back pain: a randomized controlled trial. *Spine*. 2012;37:1101-1108.

726.   Gundewall B, Liljeqvist M, Hansson T. Primary prevention of back symptoms and absence from work. A prospective randomized study among hospital employees. *Spine*. 1993;18:587-594.

727.   Salah Frih B, Fendri Y, Jellad A, Boudoukhane S, Rejeb N. Efficacy and treatment compliance of a home-based rehabilitation programme for chronic low back pain: a randomized, controlled study. *Annals of physical and rehabilitation medicine*. 2009;52:485-496.

728.   AlBahel F, Ramadan Hafez A, Rahim Zakaria A, Al-Ahaideb A, Buragadda S, Rao Melam G. Kinesio taping for the treatment of mechanical low back pain. *World Applied Sciences J*. 2013;22:78-84.

729.   Alexandre NM, de Moraes MA, Correa Filho HR, Jorge SA. Evaluation of a program to reduce back pain in nursing personnel. *Revista de saude publica*. 2001;35:356-361.

730.   Bendix AF, Bendix T, Ostenfeld S, Bush E, Andersen. Active treatment programs for patients with chronic low back pain: a prospective, randomized, observer-blinded study. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 1995;4:148-152.

731.   Davies JE, Gibson T, Tester L. The value of exercises in the treatment of low back pain. *Rheumatology and rehabilitation*. 1979;18:243-247.

732.   Dettori JR, Bullock SH, Sutlive TG, Franklin RJ, Patience T. The effects of spinal flexion and extension exercises and their associated postures in patients with acute low back pain. *Spine*. 1995;20:2303-2312.

733.   Dolan P, Greenfield K, Nelson RJ, Nelson IW. Can exercise therapy improve the outcome of microdiscectomy? *Spine*. 2000;25:1523-1532.

734.   Donelson R, Grant W, Kamps C, Medcalf R. Pain response to sagittal end-range spinal motion. A prospective, randomized, multicentered trial. *Spine*. 1991;16:S206-212.

735.   Franca FR, Burke TN, Hanada ES, Marques AP. Segmental stabilization and muscular strengthening in chronic low back pain: a comparative study. *Clinics (Sao Paulo)*. 2010;65:1013-1017.

736.   Geisser ME, Wiggert EA, Haig AJ, Colwell MO. A randomized, controlled trial of manual therapy and specific adjuvant exercise for chronic low back pain. *Clin J Pain*. 2005;21:463-470.

737.   Gillan MG, Ross JC, McLean IP, Porter RW. The natural history of trunk list, its associated disability and the influence of McKenzie management. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 1998;7:480-483.

738.   Helewa A, Goldsmith CH, Lee P, Smythe HA, Forwell L. Does strengthening the abdominal muscles prevent low back pain--a randomized controlled trial. *The Journal of rheumatology*. 1999;26:1808-1815.

739.   Kellett KM, Kellett DA, Nordholm LA. Effects of an exercise program on sick leave due to back pain. *Phys Ther*. 1991;71:283-291; discussion 291-283.

740.   Sherman KJ, Wellman RD, Cook AJ, Cherkin DC, Ceballos RM. Mediators of yoga and stretching for chronic low back pain. *Evidence-based complementary and alternative medicine : eCAM*. 2013;2013:130818.

741.   Snook SH, Webster BS, McGorry RW. The reduction of chronic, nonspecific low back pain through the control of early morning lumbar flexion: 3-year follow-up. *Journal of occupational rehabilitation*. 2002;12:13-19.

742.   Spratt KF, Weinstein JN, Lehmann TR, Woody J, Sayre H. Efficacy of flexion and extension treatments incorporating braces for low-back pain patients with retrodisplacement, spondylolisthesis, or normal sagittal translation. *Spine*. 1993;18:1839-1849.

743.   Stafne SN, Salvesen KA, Romundstad PR, Stuge B, Morkved S. Does regular exercise during pregnancy influence lumbopelvic pain? A randomized controlled trial. *Acta obstetricia et gynecologica Scandinavica*. 2012;91:552-559.

744.   Timm KE. A randomized-control study of active and passive treatments for chronic low back pain following L5 laminectomy. *The Journal of orthopaedic and sports physical therapy*. 1994;20:276-286.

745.   Tsui ML, Cheing GL. The effectiveness of electroacupuncture versus electrical heat acupuncture in the management of chronic low-back pain. *J Altern Complement Med*. 2004;10:803-809.

746.   Danneels LA, Vanderstraeten GG, Cambier DC, et al. Effects of three different training modalities on the cross sectional area of the lumbar multifidus muscle in patients with chronic low back pain. *British journal of sports medicine*. 2001;35:186-191.

747.   Descarreaux M, Normand MC, Laurencelle L, Dugas C. Evaluation of a specific home exercise program for low back pain. *Journal of manipulative and physiological therapeutics*. 2002;25:497-503.

748.   Rackwitz B, Limm H, Wessels T, Ewert T, Stucki G. Practicability of segmental stabilizing exercises in the context of a group program for the secondary prevention of low back pain. An explorative pilot study. *Europa medicophysica*. 2007;43:359-367.

749.   Seferlis T, Nemeth G, Carlsson AM, Gillstrom P. Conservative treatment in patients sick-listed for acute low-back pain: a prospective randomised study with 12 months' follow-up. *European spine journal : official publication of the European*

Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society. 1998;7:461-470.

750.   Smith D, Bissell G, Bruce-Low S, Wakefield C. The effect of lumbar extension training with and without pelvic stabilization on lumbar strength and low back pain. *Journal of back and musculoskeletal rehabilitation*. 2011;24:241-249.

751.   Hollinghurst S, Sharp D, Ballard K, et al. Randomised controlled trial of Alexander technique lessons, exercise, and massage (ATEAM) for chronic and recurrent back pain: economic evaluation. *BMJ*. 2008;337:a2656.

752.   Rasmussen-Barr E, Nilsson-Wikmar L, Arvidsson I. Stabilizing training compared with manual treatment in sub-acute and chronic low-back pain. *Manual therapy*. 2003;8:233-241.

753.   Yardley L, Dennison L, Coker R, et al. Patients' views of receiving lessons in the Alexander technique and an exercise prescription for managing back pain in the ATEAM trial. *Family practice*. 2010;27:198-204.

754.   Ariyoshi M, Sonoda K, Nagata K, et al. Efficacy of aquatic exercises for patients with low-back pain. *The Kurume medical journal*. 1999;46:91-96.

755.   Dogan M, Sahin O, Elden H, Hayta E, Kaptanoglu E. Additional therapeutic effect of balneotherapy in low back pain. *Southern medical journal*. 2011;104:574-578.

756.   Falagas ME, Zarkadoulia E, Rafailidis PI. The therapeutic effect of balneotherapy: evaluation of the evidence from randomised controlled trials. *International journal of clinical practice*. 2009;63:1068-1084.

757.   Gaal J, Varga J, Szekanecz Z, et al. Balneotherapy in elderly patients: effect on pain from degenerative knee and spine conditions and on quality of life. *The Israel Medical Association journal : IMAJ*. 2008;10:365-369.

758.   Kamioka H, Tsutani K, Okuizumi H, et al. Effectiveness of aquatic exercise and balneotherapy: a summary of systematic reviews based on randomized controlled trials of water immersion therapies. *Journal of epidemiology / Japan Epidemiological Association*. 2010;20:2-12.

759.   Nasermoaddeli A, Kagamimori S. Balneotherapy in medicine: A review. *Environmental health and preventive medicine*. 2005;10:171-179.

760.   Pittler MH, Karagulle MZ, Karagulle M, Ernst E. Spa therapy and balneotherapy for treating low back pain: meta-analysis of randomized trials. *Rheumatology (Oxford)*. 2006;45:880-884.

761.   Waller B, Lambeck J, Daly D. Therapeutic aquatic exercise in the treatment of low back pain: a systematic review. *Clinical rehabilitation*. 2009;23:3-14.

762.   Bender T, Karagulle Z, Balint GP, Gutenbrunner C, Balint PV, Sukenik S. Hydrotherapy, balneotherapy, and spa treatment in pain management. *Rheumatology international*. 2005;25:220-224.

763.   Dundar U, Solak O, Yigit I, Evcik D, Kavuncu V. Clinical effectiveness of aquatic exercise to treat chronic low back pain: a randomized controlled trial. *Spine*. 2009;34:1436-1440.

764.   McIlveen B, Robertson V. A randomised controlled study of the outcome of hydrotherapy for subjects with low back or back and leg pain. *Physiother*. 1998;84:17-26.

765.   Kesiktas N, Karakas S, Gun K, Gun N, Murat S, Uludag M. Balneotherapy for chronic low back pain: a randomized, controlled study. *Rheumatology international*. 2012;32:3193-3199.

766.   Balogh Z, Ordogh J, Gasz A, Nemet L, Bender T. Effectiveness of balneotherapy in chronic low back pain -- a randomized single-blind controlled follow-up study. *Forschende Komplementarmedizin und klassische Naturheilkunde = Research in complementary and natural classical medicine*. 2005;12:196-201.

767.   Tefner IK, Nemeth A, Laszlofi A, Kis T, Gyetvai G, Bender T. The effect of spa therapy in chronic low back pain: a randomized controlled, single-blind, follow-up study. *Rheumatology international*. 2012;32:3163-3169.

768.   Graves JE, Pollock ML, Foster D, et al. Effect of training frequency and specificity on isometric lumbar extension strength. *Spine*. 1990;15:504-509.

769.   Graves JE, Pollock ML, Leggett SH, Carpenter DM, Fix CK, Fulton MN. Limited range-of-motion lumbar extension strength training. *Medicine and science in sports and exercise*. 1992;24:128-133.

770.   Graves JE, Webb DC, Pollock ML, et al. Pelvic stabilization during resistance training: its effect on the development of lumbar extension strength. *Arch Phys Med Rehabil*. 1994;75:210-215.

771.   Holmes B, Leggett S, Mooney V, Nichols J, Negri S, Hoeyberghs A. Comparison of female geriatric lumbar-extension strength: asymptotic versus chronic low back pain patients and their response to active rehabilitation. *Journal of spinal disorders*. 1996;9:17-22.

772.   Nelson BW, O'Reilly E, Miller M, Hogan M, Wegner JA, Kelly C. The clinical effects of intensive, specific exercise on chronic low back pain: a controlled study of 895 consecutive patients with 1-year follow up. *Orthopedics*. 1995;18:971-981.

773.   Choi G, Raiturker PP, Kim MJ, Chung DJ, Chae YS, Lee SH. The effect of early isolated lumbar extension exercise program for patients with herniated disc undergoing lumbar discectomy. *Neurosurgery*. 2005;57:764-772; discussion 764-772.

774.   Christensen FB, Laurberg I, Bunger CE. Importance of the back-cafe concept to rehabilitation after lumbar spinal fusion: a randomized clinical study with a 2-year follow-up. *Spine*. 2003;28:2561-2569.

775.   Friedrich M, Gittler G, Halberstadt Y, Cermak T, Heiller I. Combined exercise and motivation program: effect on the compliance and level of disability of patients with chronic low back pain: a randomized controlled trial. *Archives of physical medicine and rehabilitation*. 1998;79:475-487.

776.   Manniche C, Lundberg E, Christensen I, Bentzen L, Hesselsoe G. Intensive dynamic back exercises for chronic low back pain: a clinical trial. *Pain*. 1991;47:53-63.

777.   Cramer H, Lauche R, Haller H, Dobos G. A systematic review and meta-analysis of yoga for low back pain. *The Clinical journal of pain*. 2013;29:450-460.

778.   Hill C. Is yoga an effective treatment in the management of patients with chronic low back pain compared with other care modalities - a systematic review. *Journal of complementary & integrative medicine*. 2013;10.

779.   Kelly Z. Is yoga an effective treatment for low back pain: a research review. *Int J Yoga Ther*. 2009;19:103-112.

780.   Taimini. The Science of Yoga - the Yoga Sutras of Patanjali in Sankrit with Transliteration in Roman, Translation in English and Commentary. . *Madras, India*. 1986;The Theosophical Publishing House.

781.   Williams KA, Petronis J, Smith D, et al. Effect of Iyengar yoga therapy for chronic low back pain. *Pain*. 2005;115:107-117.

782.   Cox H, Tilbrook H, Aplin J, et al. A randomised controlled trial of yoga for the treatment of chronic low back pain: results of a pilot study. *Complementary therapies in clinical practice*. 2010;16:187-193.

783.   Galantino ML, Bzdewka TM, Eissler-Russo JL, et al. The impact of modified Hatha yoga on chronic low back pain: a pilot study. *Alternative therapies in health and medicine*. 2004;10:56-59.

784.   Saper RB, Sherman KJ, Cullum-Dugan D, Davis RB, Phillips RS, Culpepper L. Yoga for chronic low back pain in a predominantly minority population: a pilot randomized controlled trial. *Alternative therapies in health and medicine*. 2009;15:18-27.

785.   Sherman KJ, Cherkin DC, Erro J, Miglioretti DL, Deyo RA. Comparing yoga, exercise, and a self-care book for chronic low back pain: a randomized, controlled trial. *Ann Intern Med*. 2005;143:849-856.

786.   Tilbrook HE, Cox H, Hewitt CE, et al. Yoga for chronic low back pain: a randomized trial. *Ann Intern Med*. 2011;155:569-578.

787.   Tekur P, Chametcha S, Hongasandra RN, Raghuram N. Effect of yoga on quality of life of CLBP patients: A randomized control study. *International journal of yoga*. 2010;3:10-17.

788.   Tekur P, Nagarathna R, Chametcha S, Hankey A, Nagendra HR. A comprehensive yoga programs improves pain, anxiety and depression in chronic low back pain patients more than exercise: an RCT. *Complementary therapies in medicine*. 2012;20:107-118.

789.   Williams K, Abildso C, Steinberg L, et al. Evaluation of the effectiveness and efficacy of Iyengar yoga therapy on chronic low back pain. *Spine (Phila Pa 1976)*. 2009;34:2066-2076.

790.   Donzelli S, Di Domenica E, Cova AM, Galletti R, Giunta N. Two different techniques in the rehabilitation treatment of low back pain: a randomized controlled trial. *Europa medicophysica*. 2006;42:205-210.

791.   Hall AM, Maher CG, Lam P, Ferreira M, Latimer J. Tai chi exercise for treatment of pain and disability in people with persistent low back pain: a randomized controlled trial. *Arthritis care & research*. 2011;63:1576-1583.

792.   Pushpika Attanayake AM, Somarathna KI, Vyas GH, Dash SC. Clinical evaluation of selected Yogic procedures in individuals with low back pain. *Ayu*. 2010;31:245-250.

793.   Chou R, Huffman LH. Medications for acute and chronic low back pain: a review of the evidence for an American Pain Society/American College of Physicians clinical practice guideline. *Ann Intern Med*. 2007;147:505-514.

794.   Dahners LE, Mullis BH. Effects of nonsteroidal anti-inflammatory drugs on bone formation and soft-tissue healing. *The Journal of the American Academy of Orthopaedic Surgeons*. 2004;12:139-143.

795.   Jirarattanaphochai K, Jung S. Nonsteroidal antiinflammatory drugs for postoperative pain management after lumbar spine surgery: a meta-analysis of randomized controlled trials. *Journal of neurosurgery Spine*. 2008;9:22-31.

796.   Krismer M, van Tulder M. Strategies for prevention and management of musculoskeletal conditions. Low back pain (non-specific). *Best practice & research*. 2007;21:77-91.

797.   Kroenke K, Krebs EE, Bair MJ. Pharmacotherapy of chronic pain: a synthesis of recommendations from systematic reviews. *General hospital psychiatry*. 2009;31:206-219.

798.   Kuijpers T, van Middelkoop M, Rubinstein SM, et al. A systematic review on the effectiveness of pharmacological interventions for chronic non-specific low-back pain. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2011;20:40-50.

799.   Last AR, Hulbert K. Chronic low back pain: evaluation and management. *American family physician*. 2009;79:1067-1074.

800.   Machado LA, Kamper SJ, Herbert RD, Maher CG, McAuley JH. Analgesic effects of treatments for non-specific low back pain: a meta-analysis of placebo-controlled randomized trials. *Rheumatology (Oxford)*. 2009;48:520-527.

801.   Machado GC, Maher CG, Ferreira PH, et al. Efficacy and safety of paracetamol for spinal pain and osteoarthritis: systematic review and meta-analysis of randomised placebo controlled trials. *BMJ*. 2015;350:h1225.

802.   Graham DY, Agrawal NM, Campbell DR, et al. Ulcer prevention in long-term users of nonsteroidal anti-inflammatory drugs: results of a double-blind, randomized, multicenter, active- and placebo-controlled study of misoprostol vs lansoprazole. *Archives of internal medicine*. 2002;162:169-175.

803.   Antman EM, Bennett JS, Daugherty A, Furberg C, Roberts H, Taubert KA. Use of nonsteroidal antiinflammatory drugs: an update for clinicians: a scientific statement from the American Heart Association. *Circulation*. 2007;115:1634-1642.

804.   Dreiser RL, Marty M, Ionescu E, Gold M, Liu JH. Relief of acute low back pain with diclofenac-K 12.5 mg tablets: a flexible dose, ibuprofen 200 mg and placebo-controlled clinical trial. *International journal of clinical pharmacology and therapeutics*. 2003;41:375-385.

805.   Berry H, Bloom B, Hamilton EB, Swinson DR. Naproxen sodium, diflunisal, and placebo in the treatment of chronic back pain. *Ann Rheum Dis*. 1982;41:129-132.

806.   Birbara CA, Puopolo AD, Munoz DR, et al. Treatment of chronic low back pain with etoricoxib, a new cyclo-oxygenase-2 selective inhibitor: improvement in pain and disability--a randomized, placebo-controlled, 3-month trial. *J Pain*. 2003;4:307-315.

807.   Pallay RM, Seger W, Adler JL, et al. Etoricoxib reduced pain and disability and improved quality of life in patients with chronic low back pain: a 3 month, randomized, controlled trial. *Scandinavian journal of rheumatology*. 2004;33:257-266.

808.   Herrmann WA, Geertsen MS. Efficacy and safety of lornoxicam compared with placebo and diclofenac in acute sciatica/lumbo-sciatica: an analysis from a randomised, double-blind, multicentre, parallel-group study. *International journal of clinical practice*. 2009;63:1613-1621.

809.   Evans DP, Burke MS, Newcombe RG. Medicines of choice in low back pain. *Current medical research and opinion*. 1980;6:540-547.

810.   Szpalski M, Hayez JP. Objective functional assessment of the efficacy of tenoxicam in the treatment of acute low back pain. A double-blind placebo-controlled study. *British journal of rheumatology*. 1994;33:74-78.

811.   Videman T, Osterman K. Double-blind parallel study of piroxicam versus indomethacin in the treatment of low back pain. *Annals of clinical research*. 1984;16:156-160.

812.   Hancock MJ, Maher CG, Latimer J, et al. Assessment of diclofenac or spinal manipulative therapy, or both, in addition to recommended first-line treatment for acute low back pain: a randomised controlled trial. *Lancet*. 2007;370:1638-1643.

813.   Boutaud O, Aronoff DM, Richardson JH, Marnett LJ, Oates JA. Determinants of the cellular specificity of acetaminophen as an inhibitor of prostaglandin H(2) synthases. *Proceedings of the National Academy of Sciences of the United States of America*. 2002;99:7130-7135.

814.   Videman T, Heikkila J, Partanen T. Double-blind parallel study of meptazinol versus diflunisal in the treatment of lumbago. *Current medical research and opinion*. 1984;9:246-252.

815.   Ritchie LD. A clinical evaluation of flurbiprofen LAT and piroxicam gel: a multicentre study in general practice. *Clinical rheumatology*. 1996;15:243-247.

816.   Rosenthal M. The efficacy of flurbiprofen versus piroxicam in the treatment of acute soft tissue rheumatism. *Current medical research and opinion*. 1984;9:304-309.

817.   Weber H, Holme I, Amlie E. The natural course of acute sciatica with nerve root symptoms in a double-blind placebo-controlled trial evaluating the effect of piroxicam. *Spine*. 1993;18:1433-1438.

818.   McGettigan P, Henry D. Cardiovascular risk and inhibition of cyclooxygenase: a systematic review of the observational studies of selective and nonselective inhibitors of cyclooxygenase 2. *JAMA : the journal of the American Medical Association*. 2006;296:1633-1644.

819.   McGettigan P, Henry D. Cardiovascular risk with non-steroidal anti-inflammatory drugs: systematic review of population-based controlled observational studies. *PLoS medicine*. 2011;8:e1001098.

820.   Bombardier C, Laine L, Reicin A, et al. Comparison of upper gastrointestinal toxicity of rofecoxib and naproxen in patients with rheumatoid arthritis. VIGOR Study Group. *N Engl J Med*. 2000;343:1520-1528, 1522 p following 1528.

821.   Fosbol EL, Folke F, Jacobsen S, et al. Cause-specific cardiovascular risk associated with nonsteroidal antiinflammatory drugs among healthy individuals. *Circulation Cardiovascular quality and outcomes*. 2010;3:395-405.

822.   Fosbol EL, Kober L, Torp-Pedersen C, Gislason GH. Cardiovascular safety of non-steroidal anti-inflammatory drugs among healthy individuals. *Expert opinion on drug safety*. 2010;9:893-903.

823.   Nussmeier NA, Whelton AA, Brown MT, et al. Complications of the COX-2 inhibitors parecoxib and valdecoxib after cardiac surgery. *The New England journal of medicine*. 2005;352:1081-1091.

824.   Ott E, Nussmeier NA, Duke PC, et al. Efficacy and safety of the cyclooxygenase 2 inhibitors parecoxib and valdecoxib in patients undergoing coronary artery bypass surgery. *The Journal of thoracic and cardiovascular surgery*. 2003;125:1481-1492.

825.   Trelle S, Reichenbach S, Wandel S, et al. Cardiovascular safety of non-steroidal anti-inflammatory drugs: network meta-analysis. *BMJ*. 2011;342:c7086.

826.   Feldman M, McMahon AT. Do cyclooxygenase-2 inhibitors provide benefits similar to those of traditional nonsteroidal anti-inflammatory drugs, with less gastrointestinal toxicity? *Annals of internal medicine*. 2000;132:134-143.

827.   Davies RA, Maher CG, Hancock MJ. A systematic review of paracetamol for non-specific low back pain. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2008;17:1423-1430.

828.   Hickey RF. Chronic low back pain: a comparison of diflunisal with paracetamol. *The New Zealand medical journal*. 1982;95:312-314.

829.   McGuinness BW. A double-blind comparison in general practice of a combination tablet containing orphenadrine citrate and paracetamol ('Norgesic') with paracetamol alone. *The Journal of international medical research*. 1983;11:42-45.

830.   McGuinness BW, Lloyd-Jones M, Fowler PD. A double-blind comparative trial of 'parazolidin' and paracetamol. *The British journal of clinical practice*. 1969;23:452-455.

831.   Valtonen EJ. A controlled clinical trial of chlormezanone, orphenadrine, orphenadrine/paracetamol and placebo in the treatment of painful skeletal muscle spasms. *Annals of clinical research*. 1975;7:85-88.

832.   Doran DM, Newell DJ. Manipulation in treatment of low back pain: a multicentre study. *British medical journal*. 1975;2:161-164.

833.   Hackett GI, Seddon D, Kaminski D. Electroacupuncture compared with paracetamol for acute low back pain. *The Practitioner*. 1988;232:163-164.

834.   Nadler SF, Steiner DJ, Erasala GN, et al. Continuous low-level heat wrap therapy provides more efficacy than Ibuprofen and acetaminophen for acute low back pain. *Spine*. 2002;27:1012-1017.

835.   Vernon WG. A double-blind evaluation of Parafon Forte in the treatment of musculo-skeletal back conditions. *Current therapeutic research, clinical and experimental*. 1972;14:801-806.

836.   Brown FL, Jr., Bodison S, Dixon J, Davis W, Nowoslawski J. Comparison of diflunisal and acetaminophen with codeine in the treatment of initial or recurrent acute low back strain. *Clin Ther*. 1986;9 Suppl C:52-58.

837.   Baraf HS, Fuentealba C, Greenwald M, et al. Gastrointestinal side effects of etoricoxib in patients with osteoarthritis: results of the Etoricoxib versus Diclofenac Sodium Gastrointestinal Tolerability and Effectiveness (EDGE) trial. *The Journal of rheumatology*. 2007;34:408-420.

838.   Bensen WG, Zhao SZ, Burke TA, et al. Upper gastrointestinal tolerability of celecoxib, a COX-2 specific inhibitor, compared to naproxen and placebo. *The Journal of rheumatology*. 2000;27:1876-1883.

839.   Chan FK. NSAID-induced peptic ulcers and Helicobacter pylori infection: implications for patient management. *Drug Saf*. 2005;28:287-300.

840.   FitzGerald GA, Patrono C. The coxibs, selective inhibitors of cyclooxygenase-2. *The New England journal of medicine*. 2001;345:433-442.

841.   Aubrun F, Langeron O, Heitz D, Coriat P, Riou B. Randomised, placebo-controlled study of the postoperative analgesic effects of ketoprofen after spinal fusion surgery. *Acta anaesthesiologica Scandinavica*. 2000;44:934-939.

842.   Fletcher D, Negre I, Barbin C, et al. Postoperative analgesia with i.v. propacetamol and ketoprofen combination after disc surgery. *Canadian journal of anaesthesia = Journal canadien d'anesthesie*. 1997;44:479-485.

843.   Grundmann U, Wornle C, Biedler A, Kreuer S, Wrobel M, Wilhelm W. The efficacy of the non-opioid analgesics parecoxib, paracetamol and metamizol for postoperative pain relief after lumbar microdiscectomy. *Anesthesia and analgesia*. 2006;103:217-222, table of contents.

844.   Pohjolainen T, Jekunen A, Autio L, Vuorela H. Treatment of acute low back pain with the COX-2-selective anti-inflammatory drug nimesulide: results of a randomized, double-blind comparative trial versus ibuprofen. *Spine*. 2000;25:1579-1585.

845.   Pookarnjanamorakot C, Laohacharoensombat W, Jaovisidha S. The clinical efficacy of piroxicam fast-dissolving dosage form for postoperative pain control after simple lumbar spine surgery: a double-blinded randomized study. *Spine (Phila Pa 1976)*. 2002;27:447-451.

846.   Schattenkirchner M, Milachowski KA. A double-blind, multicentre, randomised clinical trial comparing the efficacy and tolerability of aceclofenac with diclofenac resinate in patients with acute low back pain. *Clinical rheumatology*. 2003;22:127-135.

847.   Zerbini C, Ozturk ZE, Grifka J, et al. Efficacy of etoricoxib 60 mg/day and diclofenac 150 mg/day in reduction of pain and disability in patients with chronic low back pain: results of a 4-week, multinational, randomized, double-blind study. *Current medical research and opinion*. 2005;21:2037-2049.

848.   Coats TL, Borenstein DG, Nangia NK, Brown MT. Effects of valdecoxib in the treatment of chronic low back pain: results of a randomized, placebo-controlled trial. *Clin Ther*. 2004;26:1249-1260.

849.   Goldie I. A clinical trial with indomethacin (indomee(R)) in low back pain and sciatica. *Acta orthopaedica Scandinavica*. 1968;39:117-128.

850.   Katz N, Ju WD, Krupa DA, et al. Efficacy and safety of rofecoxib in patients with chronic low back pain: results from two 4-week, randomized, placebo-controlled, parallel-group, double-blind trials. *Spine*. 2003;28:851-858; discussion 859.

851.   Katz N, Rodgers DB, Krupa D, Reicin A. Onset of pain relief with rofecoxib in chronic low back pain: results of two four-week, randomized, placebo-controlled trials. *Current medical research and opinion*. 2004;20:651-658.

852.   Koes BW, Bouter LM, van Mameren H, et al. Randomised clinical trial of manipulative therapy and physiotherapy for persistent back and neck complaints: results of one year follow up. *Bmj*. 1992;304:601-605.

853.   Konstantinovic LM, Kanjuh ZM, Milovanovic AN, et al. Acute low back pain with radiculopathy: a double-blind, randomized, placebo-controlled study. *Photomedicine and laser surgery*. 2010;28:553-560.

854.   Pareek A, Chandurkar N, Chandanwale A, Ambade R, Gupta A, Bartakke G. Aceclofenac-tizanidine in the treatment of acute low back pain: a double-blind, double-dummy, randomized, multicentric, comparative study against aceclofenac alone. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2009;18:1836-1842.

855.   Reuben SS, Connelly NR, Lurie S, Klatt M, Gibson CS. Dose-response of ketorolac as an adjunct to patient-controlled analgesia morphine in patients after spinal fusion surgery. *Anesthesia and analgesia*. 1998;87:98-102.

856.   Reuben SS, Connelly NR, Steinberg R. Ketorolac as an adjunct to patient-controlled morphine in postoperative spine surgery patients. *Regional anesthesia*. 1997;22:343-346.

857.   Veenema KR, Leahey N, Schneider S. Ketorolac versus meperidine: ED treatment of severe musculoskeletal low back pain. *The American journal of emergency medicine*. 2000;18:404-407.

858.   Babej-Dolle R, Freytag S, Eckmeyer J, et al. Parenteral dipyrone versus diclofenac and placebo in patients with acute lumbago or sciatic pain: randomized observer-blind multicenter study. *International journal of clinical pharmacology and therapeutics*. 1994;32:204-209.

859.   Bakshi R, Thumb N, Broll H, al. e. Treatment of acute lumbosacral back pain with diclofenac resinate. Results of a double-blind comparative trial versus piroxicam. *Drug Invest*. 1994;8:288-293.

860.   Blazek M, Keszthelyi B, Varhelyi M, Korosi O. Comparative study of Biarison and Voltaren in acute lumbar pain and lumbo-ischialgia. *Therapia Hungarica (English edition)*. 1986;34:163-166.

861.   Giles LG, Muller R. Chronic spinal pain: a randomized clinical trial comparing medication, acupuncture, and spinal manipulation. *Spine*. 2003;28:1490-1502; discussion 1502-1493.

862.   Matsumo S, Kaneda K, Norhara Y. Clinical evaluation of ketoprofen (Orudis) in lumbago - a double-blind comparison with diclofenac sodium. *The British journal of clinical practice*. 1981;35:266.

863.   Orava S. Medical treatment of acute low back pain. Diflunisal compared with indomethacin in acute lumbago. *International journal of clinical pharmacology research*. 1986;6:45-51.

864.   Pownall R, Pickvance NJ. Does treatment timing matter?--A double blind crossover study of ibuprofen 2400 mg per day in different dosage schedules in treatment of chronic low back pain. *The British journal of clinical practice*. 1985;39:267-275.

865.   Auvinet B, Ziller R, Appelboom T, Velicitat P. Comparison of the onset and intensity of action of intramuscular meloxicam and oral meloxicam in patients with acute sciatica. *Clin Ther*. 1995;17:1078-1098.

866.   Bekker A, Cooper PR, Frempong-Boadu A, Babu R, Errico T, Lebovits A. Evaluation of preoperative administration of the cyclooxygenase-2 inhibitor rofecoxib for the treatment of postoperative pain after lumbar disc surgery. *Neurosurgery*. 2002;50:1053-1057; discussion 1057-1058.

867.   Dincer U, Kiralp MZ, Cakar E, Yasar E, Dursan H. Caudal epidural injection versus non-steroidal anti-inflammatory drugs in the treatment of low back pain accompanied with radicular pain. *Joint Bone Spine*. 2007;74:467-471.

868.   Innes GD, Croskerry P, Worthington J, Beveridge R, Jones D. Ketorolac versus acetaminophen-codeine in the emergency department treatment of acute low back pain. *The Journal of emergency medicine*. 1998;16:549-556.

869.   Mack PF, Hass D, Lavyne MH, Snow RB, Lien CA. Postoperative narcotic requirement after microscopic lumbar discectomy is not affected by intraoperative ketorolac or bupivacaine. *Spine*. 2001;26:658-661.

870.   Nissen I, Jensen KA, Ohrstrom JK. Indomethacin in the management of postoperative pain. *British journal of anaesthesia*. 1992;69:304-306.

871.   O'Donnell JB, Ekman EF, Spalding WM, Bhadra P, McCabe D, Berger MF. The effectiveness of a weak opioid medication versus a cyclo-oxygenase-2 (COX-2) selective non-steroidal anti-inflammatory drug in treating flare-up of chronic low-back pain: results from two randomized, double-blind, 6-week studies. *The Journal of international medical research*. 2009;37:1789-1802.

872.   Romano CL, Romano D, Bonora C, Mineo G. Pregabalin, celecoxib, and their combination for treatment of chronic low-back pain. *Journal of orthopaedics and traumatology : official journal of the Italian Society of Orthopaedics and Traumatology*. 2009;10:185-191.

873.   Sweetman BJ, Baig A, Parsons DL. Mefenamic acid, chlormezanone-paracetamol, ethoheptazine-aspirin-meprobamate: a comparative study in acute low back pain. *The British journal of clinical practice*. 1987;41:619-624.

874.   Thienthong S, Jirarattanaphochai K, Krisanaprakornkit W, Simajareuk S, Tantanatewin W, Sathitkarnmanee A. Treatment of pain after spinal surgery in the recovery room by single dose lornoxicam: a randomized, double blind, placebo-controlled trial. *Journal of the Medical Association of Thailand = Chotmaihet thangphaet*. 2004;87:650-655.

875.   Yamashita K, Fukusaki M, Ando Y, et al. Preoperative administration of intravenous flurbiprofen axetil reduces postoperative pain for spinal fusion surgery. *J Anesth*. 2006;20:92-95.

876.   Le Roux PD, Samudrala S. Postoperative pain after lumbar disc surgery: a comparison between parenteral ketorolac and narcotics. *Acta neurochirurgica*. 1999;141:261-267.

877.   Aghababian RV, Volturo GA, Heifetz IN. Comparison of diflunisal and naproxen in the management of acute low back strain. *Clin Ther*. 1986;9 Suppl C:47-51.

878.   Waterworth RF, Hunter IA. An open study of diflunisal, conservative and manipulative therapy in the management of acute mechanical low back pain. *The New Zealand medical journal*. 1985;98:372-375.

879.   Reuben SS, Ablett D, Kaye R. High dose nonsteroidal anti-inflammatory drugs compromise spinal fusion. *Canadian journal of anaesthesia = Journal canadien d'anesthesie*. 2005;52:506-512.

880.   Reuben SS, Buvanendran A, Kroin JS, Raghunathan K. The analgesic efficacy of celecoxib, pregabalin, and their combination for spinal fusion surgery. *Anesthesia and analgesia*. 2006;103:1271-1277.

881.   Reuben SS, Connelly NR. Postoperative analgesic effects of celecoxib or rofecoxib after spinal fusion surgery. *Anesthesia and analgesia*. 2000;91:1221-1225.

882.   Albert HB, Manniche C, Sorensen JS, Deleuran BW. Antibiotic treatment in patients with low-back pain associated with Modic changes Type 1 (bone oedema): a pilot study. *British journal of sports medicine*. 2008;42:969-973.

883.   Albert HB, Sorensen JS, Christensen BS, Manniche C. Antibiotic treatment in patients with chronic low back pain and vertebral bone edema (Modic type 1 changes): a double-blind randomized clinical controlled trial of efficacy. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2013;22:697-707.

884.   Medrik-Goldberg T, Lifschitz D, Pud D, Adler R, Eisenberg E. Intravenous lidocaine, amantadine, and placebo in the treatment of sciatica: a double-blind, randomized, controlled study. *Regional anesthesia and pain medicine*. 1999;24:534-540.

885.   Atkinson JH, Slater MA, Wahlgren DR, et al. Effects of noradrenergic and serotonergic antidepressants on chronic low back pain intensity. *Pain*. 1999;83:137-145.

886.   Dickens C, Jayson M, Sutton C, Creed F. The relationship between pain and depression in a trial using paroxetine in sufferers of chronic low back pain. *Psychosomatics*. 2000;41:490-499.

887.   Goodkin K, Gullion CM, Agras WS. A randomized, double-blind, placebo-controlled trial of trazodone hydrochloride in chronic low back pain syndrome. *Journal of clinical psychopharmacology*. 1990;10:269-278.

888.   Katz J, Pennella-Vaughan J, Hetzel RD, Kanazi GE, Dworkin RH. A randomized, placebo-controlled trial of bupropion sustained release in chronic low back pain. *J Pain*. 2005;6:656-661.

889.   Alcoff J, Jones E, Rust P, Newman R. Controlled trial of imipramine for chronic low back pain. *The Journal of family practice*. 1982;14:841-846.

890.   Jenkins DG, Ebbutt AF, Evans CD. Tofranil in the treatment of low back pain. *The Journal of international medical research*. 1976;4:28-40.

891.   Pheasant H, Bursk A, Goldfarb J, Azen SP, Weiss JN, Borelli L. Amitriptyline and chronic low-back pain. A randomized double-blind crossover study. *Spine*. 1983;8:552-557.

892.   Atkinson JH, Slater MA, Williams RA, et al. A placebo-controlled randomized clinical trial of nortriptyline for chronic low back pain. *Pain*. 1998;76:287-296.

893.   Hameroff SR, Weiss JL, Lerman JC, et al. Doxepin's effects on chronic pain and depression: a controlled study. *The Journal of clinical psychiatry*. 1984;45:47-53.

894.   Skljarevski V, Ossanna M, Liu-Seifert H, et al. A double-blind, randomized trial of duloxetine versus placebo in the management of chronic low back pain. *Eur J Neurol*. 2009;16:1041-1048.

895.   Skljarevski V, Zhang S, Chappell AS, Walker DJ, Murray I, Backonja M. Maintenance of effect of duloxetine in patients with chronic low back pain: a 41-week uncontrolled, dose-blinded study. *Pain Med*. 2010;11:648-657.

896.   Skljarevski V, Zhang S, Desaiah D, et al. Duloxetine versus placebo in patients with chronic low back pain: a 12-week, fixed-dose, randomized, double-blind trial. *J Pain*. 2010;11:1282-1290.

897.   Khoromi S, Cui L, Nackers L, Max MB. Morphine, nortriptyline and their combination vs. placebo in patients with chronic lumbar root pain. *Pain*. 2007;130:66-75.

898.   Atkinson JH, Slater MA, Capparelli EV, et al. Efficacy of noradrenergic and serotonergic antidepressants in chronic back pain: a preliminary concentration-controlled trial. *Journal of clinical psychopharmacology*. 2007;27:135-142.

899.   Hameroff SR, Cork RC, Scherer K, et al. Doxepin effects on chronic pain, depression and plasma opioids. *The Journal of clinical psychiatry*. 1982;43:22-27.

900.   Kroenke K, Bair MJ, Damush TM, et al. Optimized antidepressant therapy and pain self-management in primary care patients with depression and musculoskeletal pain: a randomized controlled trial. *JAMA*. 2009;301:2099-2110.

901.   Mazza M, Mazza O, Pazzaglia C, Padua L, Mazza S. Escitalopram 20 mg versus duloxetine 60 mg for the treatment of chronic low back pain. *Expert opinion on pharmacotherapy*. 2010;11:1049-1052.

902.   Stein D, Peri T, Edelstein E, Elizur A, Floman Y. The efficacy of amitriptyline and acetaminophen in the management of acute low back pain. *Psychosomatics*. 1996;37:63-70.

903.   Ward N, Bokan JA, Phillips M, Benedetti C, Butler S, Spengler D. Antidepressants in concomitant chronic back pain and depression: doxepin and desipramine compared. *The Journal of clinical psychiatry*. 1984;45:54-59.

904.   Ward NG. Tricyclic antidepressants for chronic low-back pain. Mechanisms of action and predictors of response. *Spine*. 1986;11:661-665.

905.   Beydoun A, Shaibani A, Hopwood M, Wan Y. Oxcarbazepine in painful diabetic neuropathy: results of a dose-ranging study. *Acta neurologica Scandinavica*. 2006;113:395-404.

906.   Beydoun S, Alarcon F, Mangat S, Wan Y. Long-term safety and tolerability of oxcarbazepine in painful diabetic neuropathy. *Acta neurologica Scandinavica*. 2007;115:284-288.

907.   Challapalli V, Tremont-Lukats IW, McNicol ED, Lau J, Carr DB. Systemic administration of local anesthetic agents to relieve neuropathic pain. *Cochrane Database Syst Rev*. 2005:CD003345.

908.   Dogra S, Beydoun S, Mazzola J, Hopwood M, Wan Y. Oxcarbazepine in painful diabetic neuropathy: a randomized, placebo-controlled study. *Eur J Pain*. 2005;9:543-554.

909.   Grosskopf J, Mazzola J, Wan Y, Hopwood M. A randomized, placebo-controlled study of oxcarbazepine in painful diabetic neuropathy. *Acta neurologica Scandinavica*. 2006;114:177-180.

910.   Pandey CK, Raza M, Tripathi M, Navkar DV, Kumar A, Singh UK. The comparative evaluation of gabapentin and carbamazepine for pain management in Guillain-Barre syndrome patients in the intensive care unit. *Anesthesia and analgesia*. 2005;101:220-225.

911.   Pandey CK, Sahay S, Gupta D, et al. Preemptive gabapentin decreases postoperative pain after lumbar discoidectomy. *Canadian journal of anaesthesia = Journal canadien d'anesthesie*. 2004;51:986-989.

912.   Pandey CK, Navkar DV, Giri PJ, et al. Evaluation of the optimal preemptive dose of gabapentin for postoperative pain relief after lumbar diskectomy: a randomized, double-blind, placebo-controlled study. *Journal of neurosurgical anesthesiology*. 2005;17:65-68.

913.   Radhakrishnan M, Bithal PK, Chaturvedi A. Effect of preemptive gabapentin on postoperative pain relief and morphine consumption following lumbar laminectomy and discectomy: a randomized, double-blinded, placebo-controlled study. *Journal of neurosurgical anesthesiology*. 2005;17:125-128.

914.   Turan A, Karamanlioglu B, Memis D, et al. Analgesic effects of gabapentin after spinal surgery. *Anesthesiology*. 2004;100:935-938.

915.   Kim J, Choi Y, Kim K, Shim J, Lee J, Kwak Y. Effective dose of peri-operative oral pregabalin as an adjunct to multimodal analgesic regimen in lumbar spinal fusion surgery. *Spine*. 2011;36:428-433.

916.   Ozgencil E, Yalcin S, Tuna H, Yorukoglu D, Kecik Y. Perioperative administration of gabapentin 1,200 mg day-1 and pregabalin 300 mg day-1 for pain following lumbar laminectomy and discectomy: a randomised, double-blinded, placebo-controlled study. *Singapore medical journal*. 2011;52:883-889.

917.   Muehlbacher M, Nickel MK, Kettler C, et al. Topiramate in treatment of patients with chronic low back pain: a randomized, double-blind, placebo-controlled study. *Clin J Pain*. 2006;22:526-531.

918.   McCleane G. Does gabapentin have an analgesic on background, movement, and referred pain? A randomized, double-blind, placebo controlled study. *Pain Clin*. 2001;13:103-107.

919.   Baron R, Freynhagen R, Tolle TR, et al. The efficacy and safety of pregabalin in the treatment of neuropathic pain associated with chronic lumbosacral radiculopathy. *Pain*. 2010;150:420-427.

920.   Yildirim K. The effectiveness of gabapentin in patients with chronic radiculopathy. *Pain Clin*. 2003;15:213-218.

921.	Harke H, Gretenkort P, Ladleif HU, Rahman S, Harke O. The response of neuropathic pain and pain in complex regional pain syndrome I to carbamazepine and sustained-release morphine in patients pretreated with spinal cord stimulation: a double-blinded randomized study. *Anesthesia and analgesia*. 2001;92:488-495.

922.	Silver M, Blum D, Grainger J, Hammer AE, Quessy S. Double-blind, placebo-controlled trial of lamotrigine in combination with other medications for neuropathic pain. *Journal of pain and symptom management*. 2007;34:446-454.

923.	Yaksi A, Ozgonenel L, Ozgonenel B. The efficiency of gabapentin therapy in patients with lumbar spinal stenosis. *Spine*. 2007;32:939-942.

924.	Backonja M, Beydoun A, Edwards KR, et al. Gabapentin for the symptomatic treatment of painful neuropathy in patients with diabetes mellitus: a randomized controlled trial. *JAMA*. 1998;280:1831-1836.

925.	Barthel HR. Bisphosphonates for RSDS and prostate cancer--more benefit then revealed! *Joint Bone Spine*. 2002;69:521; author reply 522.

926.	Schott GD. Bisphosphonates for pain relief in reflex sympathetic dystrophy? *Lancet*. 1997;350:1117.

927.	Sharma A, Williams K, Raja SN. Advances in treatment of complex regional pain syndrome: recent insights on a perplexing disease. *Current opinion in anaesthesiology*. 2006;19:566-572.

928.	Meek G, McFadden J. Colchicine confirmed as highly effective in disk disorders. Final results of a double-blind study. *J Neuro & Orthop Med & Surg*. 1985;6:211-218.

929.	Schnebel BE, Simmons JW. The use of oral colchicine for low-back pain. A double-blind study. *Spine*. 1988;13:354-357.

930.	Simmons JW, Harris WP, Koulisis CW, Kimmich SJ. Intravenous colchicine for low-back pain: a double-blind study. *Spine*. 1990;15:716-717.

931.	Ketenci A, Ozcan E, Karamursel S. Assessment of efficacy and psychomotor performances of thiocolchicoside and tizanidine in patients with acute low back pain. *International journal of clinical practice*. 2005;59:764-770.

932.	Tuzun F, Unalan H, Oner N, et al. Multicenter, randomized, double-blinded, placebo-controlled trial of thiocolchicoside in acute low back pain. *Joint Bone Spine*. 2003;70:356-361.

933.	Correll GE, Maleki J, Gracely EJ, Muir JJ, Harbut RE. Subanesthetic ketamine infusion therapy: a retrospective analysis of a novel therapeutic approach to complex regional pain syndrome. *Pain Med*. 2004;5:263-275.

934.	Goldberg ME, Domsky R, Scaringe D, et al. Multi-day low dose ketamine infusion for the treatment of complex regional pain syndrome. *Pain Physician*. 2005;8:175-179.

935.	Hocking G, Cousins MJ. Ketamine in chronic pain management: an evidence-based review. *Anesthesia and analgesia*. 2003;97:1730-1739.

936.	Krupitsky E, Burakov A, Romanova T, Dunaevsky I, Strassman R, Grinenko A. Ketamine psychotherapy for heroin addiction: immediate effects and two-year follow-up. *J Subst Abuse Treat*. 2002;23:273-283.

937.	Kvarnstrom A, Karlsten R, Quiding H, Emanuelsson BM, Gordh T. The effectiveness of intravenous ketamine and lidocaine on peripheral neuropathic pain. *Acta anaesthesiologica Scandinavica*. 2003;47:868-877.

938.	Kvarnstrom A, Karlsten R, Quiding H, Gordh T. The analgesic effect of intravenous ketamine and lidocaine on pain after spinal cord injury. *Acta anaesthesiologica Scandinavica*. 2004;48:498-506.

939.	Amr YM. Effect of addition of epidural ketamine to steroid in lumbar radiculitis: one-year follow-up. *Pain Physician*. 2011;14:475-481.

940.	Loftus RW, Yeager MP, Clark JA, et al. Intraoperative ketamine reduces perioperative opiate consumption in opiate-dependent patients with chronic back pain undergoing back surgery. *Anesthesiology*. 2010;113:639-646.

941.	Subramaniam K, Akhouri V, Glazer PA, et al. Intra- and postoperative very low dose intravenous ketamine infusion does not increase pain relief after major spine surgery in patients with preoperative narcotic analgesic intake. *Pain Med*. 2011;12:1276-1283.

942.	Galer BS, Rowbotham MC, Perander J, Friedman E. Topical lidocaine patch relieves postherpetic neuralgia more effectively than a vehicle topical patch: results of an enriched enrollment study. *Pain*. 1999;80:533-538.

943.	Nalamachu S, Crockett RS, Gammaitoni AR, Gould EM. A comparison of the lidocaine patch 5% vs naproxen 500 mg twice daily for the relief of pain associated with carpal tunnel syndrome: a 6-week, randomized, parallel-group study. *MedGenMed*. 2006;8:33.

944.	Hashmi J, Baliki M, Huang L, et al. Lidocaine patch (5%) is no more potent than placebo in treating chronic back pain when tested in a randomised double blind placebo controlled brain imaging study. *Molecular Pain*. 2012;8:doi:10.1186/1744-8069-1188-1129.

945.	Sang CN. NMDA-receptor antagonists in neuropathic pain: experimental methods to clinical trials. *J Pain Symptom Manage*. 2000;19:S21-25.

946.	Weinbroum AA, Rudick V, Paret G, Ben-Abraham R. The role of dextromethorphan in pain control. *Canadian journal of anaesthesia = Journal canadien d'anesthesie*. 2000;47:585-596.

947.	Dudgeon DJ, Bruera E, Gagnon B, et al. A phase III randomized, double-blind, placebo-controlled study evaluating dextromethorphan plus slow-release morphine for chronic cancer pain relief in terminally ill patients. *J Pain Symptom Manage*. 2007;33:365-371.

948.	Mercadante S, Casuccio A, Genovese G. Ineffectiveness of dextromethorphan in cancer pain. *J Pain Symptom Manage*. 1998;16:317-322.

949.	Carlsson KC, Hoem NO, Moberg ER, Mathisen LC. Analgesic effect of dextromethorphan in neuropathic pain. *Acta anaesthesiologica Scandinavica*. 2004;48:328-336.

950.	McQuay HJ, Carroll D, Jadad AR, et al. Dextromethorphan for the treatment of neuropathic pain: a double-blind randomised controlled crossover trial with integral n-of-1 design. *Pain*. 1994;59:127-133.

951.	Heiskanen T, Hartel B, Dahl ML, Seppala T, Kalso E. Analgesic effects of dextromethorphan and morphine in patients with chronic pain. *Pain*. 2002;96:261-267.

952.	Katz NP. MorphiDex (MS:DM) double-blind, multiple-dose studies in chronic pain patients. *J Pain Symptom Manage*. 2000;19:S37-41.

953.	Hasan RA, Kartush JM, Thomas JD, Sigler DL. Oral dextromethorphan reduces perioperative analgesic administration in children undergoing tympanomastoid surgery. *Otolaryngol Head Neck Surg*. 2004;131:711-716.

954.	Atluri S, Sudarshan G. Development of a screening tool to detect the risk of inappropriate prescription opioid use in patients with chronic pain. *Pain physician*. 2004;7:333-338.

955.	Cheng M, Sauer B, Johnson E, Porucznik C, Hegmann K. Comparison of opioid-related deaths by work-related injury. *Am J Industrial Med*. 2013;56:308-316.

956.	Green TC, Grau LE, Carver HW, Kinzly M, Heimer R. Epidemiologic trends and geographic patterns of fatal opioid intoxications in Connecticut, USA: 1997-2007. *Drug and alcohol dependence*. 2011;115:221-228.

957.	Centers for Disease Control and Prevention. Unintentional deaths from drug poisoning by urbanization of area — New Mexico, 1994–2003. *MMWR Morbidity and mortality weekly report*. 2005;54:870-873.

958.	Centers for Disease Control and Prevention. Adult Use of Prescription Opioid Pain Medications - Utah, 2008. *MMWR Morbidity and mortality weekly report*. 2010;59:153-157.

959.	Dean M. Opioids in renal failure and dialysis patients. *Journal of pain and symptom management*. 2004;28:497-504.

960.	Deyo RA, Smith DH, Johnson ES, et al. Opioids for back pain patients: primary care prescribing patterns and use of services. *Journal of the American Board of Family Medicine : JABFM*. 2011;24:717-727.

961.	Dunn KM, Saunders KW, Rutter CM, et al. Opioid prescriptions for chronic pain and overdose: a cohort study. *Annals of internal medicine*. 2010;152:85-92.

962.	Fareed A, Casarella J, Roberts M, et al. High dose versus moderate dose methadone maintenance: is there a better outcome? *Journal of addictive diseases*. 2009;28:399-405.

963.	Goodridge D, Lawson J, Rocker G, Marciniuk D, Rennie D. Factors associated with opioid dispensation for patients with COPD and lung cancer in the last year of life: A retrospective analysis. *Int J Chron Obstruct Pulmon Dis*. 2010;5:99-105.

964.	Grattan A, Sullivan M, Saunders K, Campbell C, Von Korff M. Depression and prescription opioid misuse among chronic opioid therapy recipients with no history of substance abuse. *Annals Fam Med*. 2012;10:304-311.

965.	Hadidi MS, Ibrahim MI, Abdallat IM, Hadidi KA. Current trends in drug abuse associated fatalities - Jordan, 2000-2004. *Forensic science international*. 2009;186:44-47.

966.	Hall A, Logan J, Toblin R, et al. Patterns of abuse among unintentional pharmaceutical overdose fatalities. *JAMA*. 2008;300:2613-2620.

967.	Manchikanti L, Damron KS, McManus CD, Barnhill RC. Patterns of illicit drug use and opioid abuse in patients with chronic pain at initial evaluation: a prospective, observational study. *Pain physician*. 2004;7:431-437.

968.	Mills K, Teesson M, Ross J, Darke S, Shanahan M. The costs and outcomes of treatment for opioid dependence associated with posttraumatic stress disorder. *Psychiatr Serv*. 2005;56:940-945.

969.	Nyhlen A, Fridell M, Backstrom M, Hesse M, Krantz P. Substance abuse and psychiatric co-morbidity as predictors of premature mortality in Swedish drug abusers: a prospective longitudinal study 1970-2006. *BMC psychiatry*. 2011;11:122.

970.	Paulozzi L, Baldwin G, Franklin G, et al. CDC Grand Rounds: Prescription Drug Overdoses-a U.S. Epidemic. *MMWR Morbidity and mortality weekly report*. 2012;61:10-13.

971.	Paulozzi LJ, Logan JE, Hall AJ, McKinstry E, Kaplan JA, Crosby AE. A comparison of drug overdose deaths involving methadone and other opioid analgesics in West Virginia. *Addiction*. 2009;104:1541-1548.

972.	Seal KH, Shi Y, Cohen G, et al. Association of mental health disorders with prescription opioids and high-risk opioid use in US veterans of Iraq and Afghanistan. *JAMA : the journal of the American Medical Association*. 2012;307:940-947.

973.	Shah NG, Lathrop SL, Reichard RR, Landen MG. Unintentional drug overdose death trends in New Mexico, USA, 1990-2005: combinations of heroin, cocaine, prescription opioids and alcohol. *Addiction*. 2008;103:126-136.

974.	Toblin RL, Paulozzi LJ, Logan JE, Hall AJ, Kaplan JA. Mental illness and psychotropic drug use among prescription drug overdose deaths: a medical examiner chart review. *The Journal of clinical psychiatry*. 2010;71:491-496.

975.	Webster LR, Johnson FK, Stauffer J, Setnik B, Ciric S. Impact of intravenous naltrexone on intravenous morphine-induced high, drug liking, and euphoric effects in experienced, nondependent male opioid users. *Drugs R D*. 2011;11:259-275.

976.	Wunsch M, Nakamoto K, Behonick G, Massello W. Opioid deaths in rural Virginia: a description of the high prevalence of accidental fatalities involving prescribed medications. *Am J Addict*. 2009;18.

977.	Wysowski DK. Surveillance of prescription drug-related mortality using death certificate data. *Drug Saf*. 2007;30:533-540.

978.	Wysowski DK, Governale LA, Swann J. Trends in outpatient prescription drug use and related costs in the US: 1998-2003. *PharmacoEconomics*. 2006;24:233-236.

979.	Walter SR, Thein HH, Amin J, et al. Trends in mortality after diagnosis of hepatitis B or C infection: 1992-2006. *Journal of hepatology*. 2011;54:879-886.

980.	Gomes T, Redelmeier DA, Juurlink DN, Dhalla IA, Camacho X, Mamdani MM. Opioid dose and risk of road trauma in Canada: a population-based study. *JAMA Intern Med*. 2013;173:196-201.

981.	Cifuentes M, Webster B, Genevay S, Pransky G. The course of opioid prescribing for a new episode of disabling low back pain: opioid features and dose escalation. *Pain*. 2010;151:22-29.

982.	Volinn E, Fargo JD, Fine PG. Opioid therapy for nonspecific low back pain and the outcome of chronic work loss. *Pain*. 2009;142:194-201.

983.	Dersh J, Mayer T, Gatchel R, Polatin P, Theodore B, Mayer E. Prescription opioid dependence is associated with poorer outcomes in disabling spinal disorders. *Spine*. 2008;33:2219-2227.

984.	Reneman MF, Jorritsma W, Schellekens JM, Goeken LN. Concurrent validity of questionnaire and performance-based disability measurements in patients with chronic nonspecific low back pain. *Journal of occupational rehabilitation*. 2002;12:119-129.

985.	Swinkels-Meewisse IE, Roelofs J, Oostendorp RA, Verbeek AL, Vlaeyen JW. Acute low back pain: pain-related fear and pain catastrophizing influence physical performance and perceived disability. *Pain*. 2006;120:36-43.

986.	Bohnert AS, Valenstein M, Bair MJ, et al. Association between opioid prescribing patterns and opioid overdose-related deaths. *JAMA : the journal of the American Medical Association*. 2011;305:1315-1321.

987.	Church CA, Stewart Ct, TJ OL, Wallace D. Rofecoxib versus hydrocodone/acetaminophen for postoperative analgesia in functional endoscopic sinus surgery. *Laryngoscope*. 2006;116:602-606.

988.	Nussmeier NA, Whelton AA, Brown MT, et al. Safety and efficacy of the cyclooxygenase-2 inhibitors parecoxib and valdecoxib after noncardiac surgery. *Anesthesiology*. 2006;104:518-526.

989.	Dirks J, Fredensborg BB, Christensen D, Fomsgaard JS, Flyger H, Dahl JB. A randomized study of the effects of single-dose gabapentin versus placebo on postoperative pain and morphine consumption after mastectomy. *Anesthesiology*. 2002;97:560-564.

990.	Legeby M, Sandelin K, Wickman M, Olofsson C. Analgesic efficacy of diclofenac in combination with morphine and paracetamol after mastectomy and immediate breast reconstruction. *Acta anaesthesiologica Scandinavica*. 2005;49:1360-1366.

991.	Pettersson PH, Jakobsson J, Owall A. Intravenous acetaminophen reduced the use of opioids compared with oral administration after coronary artery bypass grafting. *Journal of cardiothoracic and vascular anesthesia*. 2005;19:306-309.

992.	Buchler MW, Seiler CM, Monson JR, et al. Clinical trial: alvimopan for the management of post-operative ileus after abdominal surgery: results of an international randomized, double-blind, multicentre, placebo-controlled clinical study. *Alimentary pharmacology & therapeutics*. 2008;28:312-325.

993.	Dierking G, Duedahl TH, Rasmussen ML, et al. Effects of gabapentin on postoperative morphine consumption and pain after abdominal hysterectomy: a randomized, double-blind trial. *Acta anaesthesiologica Scandinavica*. 2004;48:322-327.

994.	Wininger SJ, Miller H, Minkowitz HS, et al. A randomized, double-blind, placebo-controlled, multicenter, repeat-dose study of two intravenous acetaminophen dosing regimens for the treatment of pain after abdominal laparoscopic surgery. *Clinical therapeutics*. 2010;32:2348-2369.

995.	Wolff BG, Michelassi F, Gerkin TM, et al. Alvimopan, a novel, peripherally acting mu opioid antagonist: results of a multicenter, randomized, double-blind, placebo-controlled, phase III trial of major abdominal surgery and postoperative ileus. *Annals of surgery*. 2004;240:728-734; discussion 734-725.

996.	Pizzi LT, Toner R, Foley K, et al. Relationship between potential opioid-related adverse effects and hospital length of stay in patients receiving opioids after orthopedic surgery. *Pharmacotherapy*. 2012;32:502-514.

997.	Christensen KS, Cohen AE, Mermelstein FH, et al. The analgesic efficacy and safety of a novel intranasal morphine formulation (morphine plus chitosan), immediate release oral morphine, intravenous morphine, and placebo in a postsurgical dental pain model. *Anesthesia and analgesia*. 2008;107:2018-2024.

998.   Nader A, Kendall MC, Wixson RL, Chung B, Polakow LM, McCarthy RJ. A randomized trial of epidural analgesia followed by continuous femoral analgesia compared with oral opioid analgesia on short- and long-term functional recovery after total knee replacement. *Pain Med*. 2012;13:937-947.

999.   Belknap SM, Moore H, Lanzotti SA, et al. Application of software design principles and debugging methods to an analgesia prescription reduces risk of severe injury from medical use of opioids. *Clinical pharmacology and therapeutics*. 2008;84:385-392.

1000.   Federation of State Medical Boards. Model Policy on the Use of Opioid Analgesics in the Treatment of Chronic Pain. 2013.

1001.   International Association of Industrial Accident Boards and Commissions. Reducing Inappropriate Opioid Use in Treatment of Injured Workers. A Policy Guide. 2013.

1002.   Buelow AK, Haggard R, Gatchel RJ. Additional validation of the pain medication questionnaire in a heterogeneous sample of chronic pain patients. *Pain practice : the official journal of World Institute of Pain*. 2009;9:428-434.

1003.   Food and Drug Administration. Letter to Dr. Andrew Kolodny in Response to the Citizen Petition Submitted by Physicians for Responsible Opioid Prescribing. 2013.

1004.   Fox CD, Steger HG, Jennison JH. Ratio scaling of pain perception with the submaximum effort tourniquet technique. *Pain*. 1979;7:21-29.

1005.   Hartrick CT, Kovan JP, Shapiro S. The numeric rating scale for clinical pain measurement: a ratio measure? *Pain practice : the official journal of World Institute of Pain*. 2003;3:310-316.

1006.   Lund I, Lundeberg T, Sandberg L, Budh CN, Kowalski J, Svensson E. Lack of interchangeability between visual analogue and verbal rating pain scales: a cross sectional description of pain etiology groups. *BMC Med Res Methodol*. 2005;5:31.

1007.   Mahowald ML, Singh JA, Majeski P. Opioid use by patients in an orthopedics spine clinic. *Arthritis and rheumatism*. 2005;52:312-321.

1008.   Morasco BJ, Cavanagh R, Gritzner S, Dobscha SK. Care management practices for chronic pain in veterans prescribed high doses of opioid medications. *Family practice*. 2013.

1009.   Von Korff M, Merrill JO, Rutter CM, Sullivan M, Campbell CI, Weisner C. Time-scheduled vs. pain-contingent opioid dosing in chronic opioid therapy. *Pain*. 2011;152:1256-1262.

1010.   Cifuentes M, Powell R, Webster B. Shorter time between opioid prescriptions associated with reduced work disability among acute low back pain opioid users. *Journal of occupational and environmental medicine / American College of Occupational and Environmental Medicine*. 2012;54:491-496.

1011.   Hartrick C, Gatchel R, Conroy S. Identification and management of pain medication abuse and misuse: current state and future directions. *Expert review of neurotherapeutics*. 2012;12.

1012.   Kidner CL, Gatchel RJ, Mayer TG. MMPI disability profile is associated with degree of opioid use in chronic work-related musculoskeletal disorders. *The Clinical journal of pain*. 2010;26:9-15.

1013.   Naliboff BD, Wu SM, Schieffer B, et al. A randomized trial of 2 prescription strategies for opioid treatment of chronic nonmalignant pain. *The journal of pain : official journal of the American Pain Society*. 2011;12:288-296.

1014.   Burchman S, Pagel P. Implementation of a formal treatment agreement for outpatient management of chronic nonmalignant pain with opioid analgesics. *Journal of pain and symptom management*. 1995;10:556-563.

1015.   Chelminski PR, Ives TJ, Felix KM, et al. A primary care, multi-disciplinary disease management program for opioid-treated patients with chronic non-cancer pain and a high burden of psychiatric comorbidity. *BMC health services research*. 2005;5:3.

1016.   Chou R, Fanciullo GJ, Fine PG, et al. Clinical guidelines for the use of chronic opioid therapy in chronic noncancer pain. *The journal of pain : official journal of the American Pain Society*. 2009;10:113-130.

1017.   Compton PA, Wu SM, Schieffer B, Pham Q, Naliboff BD. Introduction of a self-report version of the Prescription Drug Use Questionnaire and relationship to medication agreement noncompliance. *Journal of pain and symptom management*. 2008;36:383-395.

1018.   Goldberg K, Simel D, Oddone E. Effect of an opioid management system on opioid prescribing and unscheduled visits in a large primary care clinic. *JCOM*. 2005;12:621-628.

1019.   Hariharan J, Lamb GC, Neuner JM. Long-term opioid contract use for chronic pain management in primary care practice. A five year experience. *Journal of general internal medicine*. 2007;22:485-490.

1020.   Ives TJ, Chelminski PR, Hammett-Stabler CA, et al. Predictors of opioid misuse in patients with chronic pain: a prospective cohort study. *BMC health services research*. 2006;6:46.

1021.   Manchikanti L, Manchukonda R, Damron KS, Brandon D, McManus CD, Cash K. Does adherence monitoring reduce controlled substance abuse in chronic pain patients? *Pain physician*. 2006;9:57-60.

1022.   Manchikanti L, Manchukonda R, Pampati V, et al. Does random urine drug testing reduce illicit drug use in chronic pain patients receiving opioids? *Pain physician*. 2006;9:123-129.

1023.   Starrels JL, Becker WC, Alford DP, Kapoor A, Williams AR, Turner BJ. Systematic review: treatment agreements and urine drug testing to reduce opioid misuse in patients with chronic pain. *Annals of internal medicine*. 2010;152:712-720.

1024.   Vaglienti RM, Huber SJ, Noel KR, Johnstone RE. Misuse of prescribed controlled substances defined by urinalysis. *W V Med J*. 2003;99:67-70.

1025.   Wiedemer N, Harden P, Arndt I, Gallagher R. The opioid renewal clinic: a primary care, managed approach to opioid therapy in chronic pain patients at risk for substance abuse. *Pain Med*. 2007;8:573-584.

1026.   Appenzeller BM, Agirman R, Neuberg P, Yegles M, Wennig R. Segmental determination of ethyl glucuronide in hair: a pilot study. *Forensic science international*. 2007;173:87-92.

1027.   Cooper GA, Kronstrand R, Kintz P. Society of Hair Testing guidelines for drug testing in hair. *Forensic science international*. 2012;218:20-24.

1028.   Kulaga V, Velazquez-Armenta Y, Aleksa K, Vergee Z, Koren G. The effect of hair pigment on the incorporation of fatty acid ethyl esters (FAEE). *Alcohol Alcohol*. 2009;44:287-292.

1029.   Lamoureux F, Gaulier JM, Sauvage FL, Mercerolle M, Vallejo C, Lachatre G. Determination of ethyl-glucuronide in hair for heavy drinking detection using liquid chromatography-tandem mass spectrometry following solid-phase extraction. *Analytical and bioanalytical chemistry*. 2009;394:1895-1901.

1030.   Lees R, Kingston R, Williams TM, Henderson G, Lingford-Hughes A, Hickman M. Comparison of ethyl glucuronide in hair with self-reported alcohol consumption. *Alcohol Alcohol*. 2012;47:267-272.

1031.   Politi L, Zucchella A, Morini L, Stramesi C, Polettini A. Markers of chronic alcohol use in hair: comparison of ethyl glucuronide and cocaethylene in cocaine users. *Forensic science international*. 2007;172:23-27.

1032.   Substance Abuse and Mental Health Services Administration. Federal Guidelines for Opioid Treatment. 2013.

1033.   Auerbach K. Drug testing methods. In: Lessenger J, Roper G, eds. *Drug Courts: A New Approach to Treatment and Rehabilitation*: Springer; 2007.

1034.   Heit H, Gourlay D. Urine drug testing in pain medicine. *Journal of pain and symptom management*. 2004;27:260-267.

1035.   Jortani S, Stauble E, Wong S. Chapter 1. Pharmacogenetics in clinical and forensic toxicology: opioid overdoses and deaths. In: Mozayani A, Raymon L, eds. *Handbook of Drug Interactions A Clinical and Forensic Guide*. New York, NY: Humana Press; 2012:3-22.

1036.   Abbruzzese G. The medical management of spasticity. *Eur J Neurol*. 2002;9 Suppl 1:30-34; discussion 53-61.

1037.   Elenbaas JK. Centrally acting oral skeletal muscle relaxants. *American journal of hospital pharmacy*. 1980;37:1313-1323.

1038.   Browning R, Jackson JL, O'Malley PG. Cyclobenzaprine and back pain: a meta-analysis. *Archives of internal medicine*. 2001;161:1613-1620.

1039.   Cherkin DC. Primary care research on low back pain. The state of the science. *Spine*. 1998;23:1997-2002.

1040.   Deyo RA, Walsh NE, Martin DC, Schoenfeld LS, Ramamurthy S. A controlled trial of transcutaneous electrical nerve stimulation (TENS) and exercise for chronic low back pain. *N Engl J Med*. 1990;322:1627-1634.

1041.   Di Iorio D, Henley E, Doughty A. A survey of primary care physician practice patterns and adherence to acute low back problem guidelines. *Archives of family medicine*. 2000;9:1015-1021.

1042.   Schnitzer TJ, Ferraro A, Hunsche E, Kong SX. A comprehensive review of clinical trials on the efficacy and safety of drugs for the treatment of low back pain. *J Pain Symptom Manage*. 2004;28:72-95.

1043.   Toth PP, Urtis J. Commonly used muscle relaxant therapies for acute low back pain: a review of carisoprodol, cyclobenzaprine hydrochloride, and metaxalone. *Clin Ther*. 2004;26:1355-1367.

1044.   Borenstein DG, Korn S. Efficacy of a low-dose regimen of cyclobenzaprine hydrochloride in acute skeletal muscle spasm: results of two placebo-controlled trials. *Clin Ther*. 2003;25:1056-1073.

1045.   Baratta RR. A double-blind comparative study of carisoprodol, propoxyphene, and placebo in the management of low back syndrome. *Current therapeutic research, clinical and experimental*. 1976;20:233-240.

1046.   Basmajian JV. Cyclobenzaprine hydrochloride effect on skeletal muscle spasm in the lumbar region and neck: two double-blind controlled clinical and laboratory studies. *Arch Phys Med Rehabil*. 1978;59:58-63.

1047.   Boyles W GJ, Soyka J. Management of acute musculoketal conditions: thoracolumbar strain or sprain. Double-blind evaluation comparing the efficacy and safety of carisoprodol with diazepam. *Today's Ther Trends* 1983;1:1-16.

1048.   Casale R. Acute Low Back Pain. Symptomatic Treatment with a Muscle Relaxant Drug. *The Clinical journal of pain*. 1988;4:81-88.

1049.   Lofland JH, Szarlej D, Buttaro T, Shermock S, Jalali S. Cyclobenzaprine hydrochloride is a commonly prescribed centrally acting muscle relaxant, which is structurally similar to tricyclic antidepressants (TCAs) and differs from amitriptyline by only one double bond. *Clin J Pain*. 2001;17:103-104.

1050.   Elder NC. Abuse of skeletal muscle relaxants. *American family physician*. 1991;44:1223-1226.

1051.   Littrell RA, Hayes LR, Stillner V. Carisoprodol (Soma): a new and cautious perspective on an old agent. *Southern medical journal*. 1993;86:753-756.

1052.   Koes BW, van Tulder MW, Ostelo R, Kim Burton A, Waddell G. Clinical guidelines for the management of low back pain in primary care: an international comparison. *Spine*. 2001;26:2504-2513; discussion 2513-2504.

1053.   Bercel NA. Cyclobenzaprine in the treatment of skeletal muscle spasm in osteoarthritis of the cervical and lumbar spine. *Curr Ther Res*. 1977;22:462-468.

1054.   Salzmann E, Pforringer W, Paal G, Gierend M. Treatment of chronic low-back syndrome with tetrazepam in a placebo controlled double-blind trial. *J Drug Development*. 1992;4::219-228.

1055.   Hingorani K. Orphenadrin-paracetamol in backache-a double-blind controlled trial. *The British journal of clinical practice*. 1971;25:227-231.

1056.   Brown BR, Jr., Womble J. Cyclobenzaprine in intractable pain syndromes with muscle spasm. *Jama*. 1978;240:1151-1152.

1057.   Bajaj P, Arendt-Nielsen L, Madeleine P, Svensson P. Prophylactic tolperisone for post-exercise muscle soreness causes reduced isometric force--a double-blind randomized crossover control study. *Eur J Pain*. 2003;7:407-418.

1058.   Basmajian JV. Acute back pain and spasm. A controlled multicenter trial of combined analgesic and antispasm agents. *Spine*. 1989;14:438-439.

1059.   Borman P, Keskin D, Bodur H. The efficacy of lumbar traction in the management of patients with low back pain. *Rheumatology international*. 2003;23:82-86.

1060.   Bragstad A, Blikra G. Evaluation of a new skeletal muscle relaxant in the treatment of lower back pain (a comparison of DS 103-282 with chlorzoxazone). *Curr Ther Res Clin Exp* 1979;26:39-43.

1061.   Brizzi A, Giusti A, Giacchetti P, Stefanelli S, Provinciali L, Ceravolo MG. A randomised controlled trial on the efficacy of hydroelectrophoresis in acute recurrences in chronic low back pain patients. *Europa medicophysica*. 2004;40:303-309.

1062.   Preston EJ, Miller CB, Herbertson RK. A double-blind, multicenter trial of methocarbamol (Robaxin®) and cyclobenzaprine (Flexeril®) in acute musculoskeletal conditions. *Today's Ther Trends* 1984;1::1-11.

1063.   Arbus L, Fajadet B, Aubert D, Morre M, Goldberger E. Activity of tetrazepam (myolastan) in low back pain: a double-blind trial v. placebo. *Clin Trials J*. 1990;27:258-267.

1064.   Berry H, Hutchinson DR. Tizanidine and ibuprofen in acute low-back pain: results of a double-blind multicentre study in general practice. *The Journal of international medical research*. 1988;16:83-91.

1065.   Bouchier-Hayes T. Chlormezanone in low back pain and wry neck--an "analgesic sparing" effect. *The British journal of clinical practice*. 1984;38:259-262.

1066.   Cabitza P, Randelli P. Efficacy and safety of eperisone in patients with low back pain: a double blind randomized study. *European review for medical and pharmacological sciences*. 2008;12:229-235.

1067.   Childers MK, Borenstein D, Brown RL, et al. Low-dose cyclobenzaprine versus combination therapy with ibuprofen for acute neck or back pain with muscle spasm: a randomized trial. *Current medical research and opinion*. 2005;21:1485-1493.

1068.   Dapas F, Hartman SF, Martinez L, et al. Baclofen for the treatment of acute low-back syndrome. A double-blind comparison with placebo. *Spine*. 1985;10:345-349.

1069.   Fryda-Kaurimsky Z, Muller-Fassbender H. Tizanidine (DS 103-282) in the treatment of acute paravertebral muscle spasm: a controlled trial comparing tizanidine and diazepam. *The Journal of international medical research*. 1981;9:501-505.

1070.   Gold R. Orphenadrine citrate: sedative or muscle relaxant? *Clin Ther* 1978;1::451-453.

1071.   Hennies OL. A new skeletal muscle relaxant (DS 103-282) compared to diazepam in the treatment of muscle spasm of local origin. *The Journal of international medical research*. 1981;9:62-68.

1072.   Hoiriis KT, Pfleger B, McDuffie FC, et al. A randomized clinical trial comparing chiropractic adjustments to muscle relaxants for subacute low back pain. *Journal of manipulative and physiological therapeutics*. 2004;27:388-398.

1073.   Klinger NM, Wilson RR, Kanniainen CM, Wagenknecht KA, Re ON, Gold RH. Intravenous orphenadrine for the treatment of lumbar paravertebral muslce strain. *Current Therapeutic research*. 1988;43:247-255.

1074.   Rolling H, Glassman J, Soyka J. Management of acute musculoskeletal conditions – thoracolumbar strain or sprain: a double-blind evaluation comparing the efficacy and safety of carisoprodol with cyclobenzaprine hydrochloride. *Curr Ther Res* 1983;34::917-928.

1075.   Sirdalud Ternelin Asia-Pacific Study Group. Efficacy and gastroprotective effects of tizanidine plus diclofenac versus placebo plus diclofenac in patients with painful muscle spasms *Curr Ther Res*. 1998;59:13-22.

1076.   Tervo T, Petaja L, Lepisto P. A controlled clinical trial of a muscle relaxant analgesic combination in the treatment of acute lumbago. *The British journal of clinical practice*. 1976;30:62-64.

1077.   Meng CF, Wang D, Ngeow J, Lao L, Peterson M, Paget S. Acupuncture for chronic low back pain in older patients: a randomized, controlled trial. *Rheumatology (Oxford, England)*. 2003;42:1508-1517.
1078.   Waagen G, Haldeman, S., Cook, G., Lopez, D., DeBoer, KF. Short term trial of chiropractic adjustments for the relief of chronic low back pain. *Manual Medicine*. 1986;2:63-67.
1079.   Zaringhalam J, Manaheji H, Rastqar A, Zaringhalam M. Reduction of chronic non-specific low back pain: a randomised controlled clinical trial on acupuncture and baclofen. *Chinese medicine*. 2010;5:15.
1080.   Borenstein DG, Lacks S, Wiesel SW. Cyclobenzaprine and naproxen versus naproxen alone in the treatment of acute low back pain and muscle spasm. *Clin Ther*. 1990;12:125-131.
1081.   Middleton RS. A comparison of two analgesic muscle relaxant combinations in acute back pain. *The British journal of clinical practice*. 1984;38:107-109.
1082.   Pipino F, Menarini C, Lombardi G. A direct myotonoyptic (Pridinol Mesilate) for the management of chronic low back pain: a multicentre, comparative clinical evaluation. *Eur J Clin Res*. 1991;1:55-70.
1083.   Weber H. Comparison of the effect of diazepam and levomepromazine on pain in patients with acute lumbago-sciatica. *Journal of the Oslo city hospitals*. 1980;30:65-68.
1084.   Barre L, Lutz GE, Southern D, Cooper G. Fluoroscopically guided caudal epidural steroid injections for lumbar spinal stenosis: a restrospective evaluation of long term efficacy. *Pain physician*. 2004;7:187-193.
1085.   Benny B, Azari P. The efficacy of lumbosacral transforaminal epidural steroid injections: a comprehensive literature review. *Journal of back and musculoskeletal rehabilitation*. 2011;24:67-76.
1086.   Benoist M, Boulu P, Hayem G. Epidural steroid injections in the management of low-back pain with radiculopathy: an update of their efficacy and safety. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2012;21:204-213.
1087.   Benyamin RM, Manchikanti L, Parr AT, et al. The effectiveness of lumbar interlaminar epidural injections in managing chronic low back and lower extremity pain. *Pain Physician*. 2012;15:E363-404.
1088.   Benzon HT. Epidural steroid injections for low back pain and lumbosacral radiculopathy. *Pain*. 1986;24:277-295.
1089.   Boswell MV, Hansen HC, Trescot AM, Hirsch JA. Epidural steroids in the management of chronic spinal pain and radiculopathy. *Pain Physician*. 2003;6:319-334.
1090.   Botwin K, Brown LA, Fishman M, Rao S. Fluoroscopically guided caudal epidural steroid injections in degenerative lumbar spine stenosis. *Pain physician*. 2007;10:547-558.
1091.   Botwin KP, Gruber RD. Lumbar epidural steroid injections in the patient with lumbar spinal stenosis. *Physical medicine and rehabilitation clinics of North America*. 2003;14:121-141.
1092.   Botwin KP, Gruber RD, Bouchlas CG, et al. Fluoroscopically guided lumbar transformational epidural steroid injections in degenerative lumbar stenosis: an outcome study. *American journal of physical medicine & rehabilitation / Association of Academic Physiatrists*. 2002;81:898-905.
1093.   Bresnahan BW, Rundell SD, Dagadakis MC, et al. A systematic review to assess comparative effectiveness studies in epidural steroid injections for lumbar spinal stenosis and to estimate reimbursement amounts. *PM & R : the journal of injury, function, and rehabilitation*. 2013;5:705-714.
1094.   Mandel S, Schilling J, Peterson E, Rao DS, Sanders W. A retrospective analysis of vertebral body fractures following epidural steroid injections. *The Journal of bone and joint surgery American volume*. 2013;95:961-964.
1095.   McGrath JM, Schaefer MP, Malkamaki DM. Incidence and characteristics of complications from epidural steroid injections. *Pain Med*. 2011;12:726-731.
1096.   Mobaleghi J, Allahdini F, Nasseri K, et al. Comparing the effects of epidural methylprednisolone acetate injected in patients with pain due to lumbar spinal stenosis or herniated disks: a prospective study. *International journal of general medicine*. 2011;4:875-878.
1097.   Parr AT, Manchikanti L, Hameed H, et al. Caudal epidural injections in the management of chronic low back pain: a systematic appraisal of the literature. *Pain Physician*. 2012;15:E159-198.
1098.   Radcliff K, Hilibrand A, Lurie JD, et al. The impact of epidural steroid injections on the outcomes of patients treated for lumbar disc herniation: a subgroup analysis of the SPORT trial. *The Journal of bone and joint surgery American volume*. 2012;94:1353-1358.
1099.   Whynes DK, McCahon RA, Ravenscroft A, Hardman J. Cost effectiveness of epidural steroid injections to manage chronic lower back pain. *BMC anesthesiology*. 2012;12:26.
1100.   Wilkinson IM, Cohen SP. Epidural steroid injections. *Current pain and headache reports*. 2012;16:50-59.
1101.   DePalma MJ, Slipman CW. Evidence-informed management of chronic low back pain with epidural steroid injections. *The spine journal : official journal of the North American Spine Society*. 2008;8:45-55.
1102.   Dighe G, Friedman J. Systematic review of caudal epidural injections in the management of chronic back pain. *Rhode Island Med J*. 2013.

1103.   Nelemans PJ, deBie RA, deVet HC, Sturmans F. Injection therapy for subacute and chronic benign low back pain. *Spine*. 2001;26:501-515.
1104.   Pinto RZ, Maher CG, Ferreira ML, et al. Epidural corticosteroid injections in the management of sciatica: a systematic review and meta-analysis. *Ann Intern Med*. 2012;157:865-877.
1105.   Roncoroni C, Baillet A, Durand M, Gaudin P, Juvin R. Efficacy and tolerance of systemic steroids in sciatica: a systematic review and meta-analysis. *Rheumatology (Oxford)*. 2011;50:1603-1611.
1106.   Singh V, Manchikanti L. Role of caudal epidural injections in the management of chronic low back pain. *Pain physician*. 2002;5:133-148.
1107.   Cohen SP, Mao J, Vu TN, et al. Does pain score in response to a standardized subcutaneous local anesthetic injection predict epidural steroid injection outcomes in patients with lumbosacral radiculopathy? A prospective correlational study. *Pain Med*. 2013;14:327-335.
1108.   Cohen SP, Strassels SA, Foster L, et al. Comparison of fluoroscopically guided and blind corticosteroid injections for greater trochanteric pain syndrome: multicentre randomised controlled trial. *BMJ*. 2009;338:b1088.
1109.   Manchikanti L, Cash KA, McManus CD, Pampati V, Benyamin R. Fluoroscopic lumbar interlaminar epidural injections in managing chronic lumbar axial or discogenic pain. *J Pain Res*. 2012;5:301-311.
1110.   Cohen SP, White RL, Kurihara C, et al. Epidural steroids, etanercept, or saline in subacute sciatica: a multicenter, randomized trial. *Ann Intern Med*. 2012;156:551-559.
1111.   Goldberg H, Firtch W, Tyburski M, et al. Oral steroids for acute radiculopathy due to a herniated lumbar disk: a randomized clinical trial. *JAMA*. 2015;313:1915-1923.
1112.   Friedman BW, Holden L, Esses D, et al. Parenteral corticosteroids for Emergency Department patients with non-radicular low back pain. *The Journal of emergency medicine*. 2006;31:365-370.
1113.   Haimovic IC, Beresford HR. Dexamethasone is not superior to placebo for treating lumbosacral radicular pain. *Neurology*. 1986;36:1593-1594.
1114.   Porsman O, Friis H. Prolapsed lumbar disc treated with intramuscularly administered dexamethasonephosphate. A prospectively planned, double-blind, controlled clinical trial in 52 patients. *Scandinavian journal of rheumatology*. 1979;8:142-144.
1115.   Finckh A, Zufferey P, Schurch MA, Balague F, Waldburger M, So AK. Short-term efficacy of intravenous pulse glucocorticoids in acute discogenic sciatica. A randomized controlled trial. *Spine*. 2006;31:377-381.
1116.   Holve RL, Barkan H. Oral steroids in initial treatment of acute sciatica. *Journal of the American Board of Family Medicine : JABFM*. 2008;21:469-474.
1117.   Ching DW, McClintock A, Beswick F. Successful treatment with low-dose thalidomide in a patient with both Behcet's disease and complex regional pain syndrome type I: case report. *Journal of clinical rheumatology : practical reports on rheumatic & musculoskeletal diseases*. 2003;9:96-98.
1118.   Rajkumar SV, Fonseca R, Witzig TE. Complete resolution of reflex sympathetic dystrophy with thalidomide treatment. *Archives of internal medicine*. 2001;161:2502-2503.
1119.   Schwartzman RJ, Chevlen E, Bengtson K. Thalidomide has activity in treating complex regional pain syndrome. *Archives of internal medicine*. 2003;163:1487-1488; author reply 1488.
1120.   Zomas A, Anagnostopoulos N, Dimopoulos MA. Successful treatment of multiple myeloma relapsing after high-dose therapy and autologous transplantation with thalidomide as a single agent. *Bone marrow transplantation*. 2000;25:1319-1320.
1121.   Brandt J, Listing J, Haibel H, et al. Long-term efficacy and safety of etanercept after readministration in patients with active ankylosing spondylitis. *Rheumatology (Oxford, England)*. 2005;44:342-348.
1122.   Tobinick E, Davoodifar S. Efficacy of etanercept delivered by perispinal administration for chronic back and/or neck disc-related pain: a study of clinical observations in 143 patients. *Current medical research and opinion*. 2004;20:1075-1085.
1123.   Zochling J ML, Beardmore J, Boonen A. TNF-alpha inhibitors for ankylosing spondylitis (Protocol). *Cochrane Database of Systematic Reviews* 2005;3.
1124.   Autio RA, Karppinen J, Niinimaki J, et al. The effect of infliximab, a monoclonal antibody against TNF-alpha, on disc herniation resorption: a randomized controlled study. *Spine*. 2006;31:2641-2645.
1125.   Korhonen T, Karppinen J, Paimela L, et al. The treatment of disc-herniation-induced sciatica with infliximab: one-year follow-up results of FIRST II, a randomized controlled trial. *Spine*. 2006;31:2759-2766.
1126.   Korhonen T, Karppinen J, Paimela L, et al. The treatment of disc herniation-induced sciatica with infliximab: results of a randomized, controlled, 3-month follow-up study. *Spine*. 2005;30:2724-2728.
1127.   Chenot JF, Becker A, Leonhardt C, et al. Use of complementary alternative medicine for low back pain consulting in general practice: a cohort study. *BMC complementary and alternative medicine*. 2007;7:42.

1128. Gagnier JJ. Evidence-informed management of chronic low back pain with herbal, vitamin, mineral, and homeopathic supplements. *The spine journal : official journal of the North American Spine Society*. 2008;8:70-79.

1129. Gagnier JJ, van Tulder MW, Berman B, Bombardier C. Herbal medicine for low back pain: a Cochrane review. *Spine*. 2007;32:82-92.

1130. MacPherson H, Gould AJ, Fitter M. Acupuncture for low back pain: results of a pilot study for a randomized controlled trial. *Complementary therapies in medicine*. 1999;7:83-90.

1131. Quinn F, Hughes C, Baxter G. Complementary and alternative medicine in the treatment of low back pain: a systematic review. *Phys Ther Rev*. 2006;11:107-116.

1132. Rubinstein SM, van Middelkoop M, Kuijpers T, et al. A systematic review on the effectiveness of complementary and alternative medicine for chronic non-specific low-back pain. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2010;19:1213-1228.

1133. Sherman KJ, Cherkin DC, Connelly MT, et al. Complementary and alternative medical therapies for chronic low back pain: What treatments are patients willing to try? *BMC complementary and alternative medicine*. 2004;4:9.

1134. Witt CM, Ludtke R, Baur R, Willich SN. Homeopathic treatment of patients with chronic low back pain: A prospective observational study with 2 years' follow-up. *The Clinical journal of pain*. 2009;25:334-339.

1135. Wolsko PM, Eisenberg DM, Davis RB, Kessler R, Phillips RS. Patterns and perceptions of care for treatment of back and neck pain: results of a national survey. *Spine*. 2003;28:292-297; discussion 298.

1136. Sherman KJ, Cherkin DC, Kahn J, et al. A survey of training and practice patterns of massage therapists in two US states. *BMC complementary and alternative medicine*. 2005;5:13.

1137. Kong LJ, Fang M, Zhan HS, et al. Tuina-focused integrative chinese medical therapies for inpatients with low back pain: a systematic review and meta-analysis. *Evidence-based complementary and alternative medicine : eCAM*. 2012;2012:578305.

1138. Kulisch A, Bender T, Nemeth A, Szekeres L. Effect of thermal water and adjunctive electrotherapy on chronic low back pain: a double-blind, randomized, follow-up study. *Journal of rehabilitation medicine*. 2009;41:73-79.

1139. Lauche R, Wubbeling K, Ludtke R, et al. Randomized controlled pilot study: pain intensity and pressure pain thresholds in patients with neck and low back pain before and after traditional East Asian "gua sha" therapy. *The American journal of Chinese medicine*. 2012;40:905-917.

1140. Mehling WE, Hamel KA, Acree M, Byl N, Hecht FM. Randomized, controlled trial of breath therapy for patients with chronic low-back pain. *Alternative therapies in health and medicine*. 2005;11:44-52.

1141. Morone NE, Greco CM, Weiner DK. Mindfulness meditation for the treatment of chronic low back pain in older adults: a randomized controlled pilot study. *Pain*. 2008;134:310-319.

1142. Morone NE, Rollman BL, Moore CG, Li Q, Weiner DK. A mind-body program for older adults with chronic low back pain: results of a pilot study. *Pain Med*. 2009;10:1395-1407.

1143. Szczurko O, Cooley K, Busse JW, et al. Naturopathic care for chronic low back pain: a randomized trial. *PLoS One*. 2007;2:e919.

1144. Yuan WA, Huang SR, Guo K, et al. Integrative TCM Conservative Therapy for Low Back Pain due to Lumbar Disc Herniation: A Randomized Controlled Clinical Trial. *Evidence-based complementary and alternative medicine : eCAM*. 2013;2013:309831.

1145. Chiu CK, Low TH, Tey YS, Singh VA, Shong HK. The efficacy and safety of intramuscular injections of methylcobalamin in patients with chronic nonspecific low back pain: a randomised controlled trial. *Singapore medical journal*. 2011;52:868-873.

1146. Shell WE, Charuvastra EH, DeWood MA, May LA, Bullias DH, Silver DS. A double-blind controlled trial of a single dose naproxen and an amino acid medical food theramine for the treatment of low back pain. *American journal of therapeutics*. 2012;19:108-114.

1147. van Tulder MW, Furlan AD, Gagnier JJ. Complementary and alternative therapies for low back pain. *Best practice & research*. 2005;19:639-654.

1148. Chrubasik S, Junck H, Breitschwerdt H, Conradt C, Zappe H. Effectiveness of Harpagophytum extract WS 1531 in the treatment of exacerbation of low back pain: a randomized, placebo-controlled, double-blind study. *European journal of anaesthesiology*. 1999;16:118-129.

1149. Chrubasik S, Model A, Black A, Pollak S. A randomized double-blind pilot study comparing Doloteffin and Vioxx in the treatment of low back pain. *Rheumatology (Oxford)*. 2003;42:141-148.

1150. Chrubasik S, Eisenberg E, Balan E, Weinberger T, Luzzati R, Conradt C. Treatment of low back pain exacerbations with willow bark extract: a randomized double-blind study. *The American journal of medicine*. 2000;109:9-14.

1151.   Chrubasik S, Kunzel O, Model A, Conradt C, Black A. Treatment of low back pain with a herbal or synthetic anti-rheumatic: a randomized controlled study. Willow bark extract for low back pain. *Rheumatology (Oxford)*. 2001;40:1388-1393.

1152.   Krivoy N, Pavlotzky E, Chrubasik S, Eisenberg E, Brook G. Effect of salicis cortex extract on human platelet aggregation. *Planta medica*. 2001;67:209-212.

1153.   Giannetti BM, Staiger C, Bulitta M, Predel HG. Efficacy and safety of comfrey root extract ointment in the treatment of acute upper or lower back pain: results of a double-blind, randomised, placebo controlled, multicentre trial. *British journal of sports medicine*. 2010;44:637-641.

1154.   Pabst H, Schaefer A, Staiger C, Junker-Samek M, Predel HG. Combination of comfrey root extract plus methyl nicotinate in patients with conditions of acute upper or low back pain: a multicentre randomised controlled trial. *Phytotherapy research : PTR*. 2013;27:811-817.

1155.   Hiura A. Neuroanatomical effects of capsaicin on the primary afferent neurons. *Arch Histol Cytol*. 2000;63:199-215.

1156.   Czaja K, Burns GA, Ritter RC. Capsaicin-induced neuronal death and proliferation of the primary sensory neurons located in the nodose ganglia of adult rats. *Neuroscience*. 2008;154:621-630.

1157.   Frerick H, Keitel W, Kuhn U, Schmidt S, Bredehorst A, Kuhlmann M. Topical treatment of chronic low back pain with a capsicum plaster. *Pain*. 2003;106:59-64.

1158.   Stam C, Bonnet MS, van Haselen RA. The efficacy and safety of a homeopathic gel in the treatment of acute low back pain: a multi-centre, randomised, double-blind comparative clinical trial. *The British homoeopathic journal*. 2001;90:21-28.

1159.   Ginsberg F, Famaey JP. A double-blind study of topical massage with Rado-Salil ointment in mechanical low-back pain. *The Journal of international medical research*. 1987;15:148-153.

1160.   Keitel W, Frerick H, Kuhn U, Schmidt U, Kuhlmann M, Bredehorst A. Capsicum pain plaster in chronic non-specific low back pain. *Arzneimittel-Forschung*. 2001;51:896-903.

1161.   Fu PP, Xia Q, Yin JJ, et al. Photodecomposition of vitamin A and photobiological implications for the skin. *Photochemistry and photobiology*. 2007;83:409-424.

1162.   Poljsak B, Raspor P. The antioxidant and pro-oxidant activity of vitamin C and trolox in vitro: a comparative study. *J Appl Toxicol*. 2008;28:183-188.

1163.   Hayden KM, Welsh-Bohmer KA, Wengreen HJ, et al. Risk of mortality with vitamin E supplements: the Cache County study. *The American journal of medicine*. 2007;120:180-184.

1164.   Lonn E, Bosch J, Yusuf S, et al. Effects of long-term vitamin E supplementation on cardiovascular events and cancer: a randomized controlled trial. *Jama*. 2005;293:1338-1347.

1165.   Marchioli R, Levantesi G, Macchia A, et al. Vitamin E increases the risk of developing heart failure after myocardial infarction: Results from the GISSI-Prevenzione trial. *Journal of cardiovascular medicine (Hagerstown, Md*. 2006;7:347-350.

1166.   Heidari B, Shirvani JS, Firouzjahi A, Heidari P, Hajian-Tilaki KO. Association between nonspecific skeletal pain and vitamin D deficiency. *International journal of rheumatic diseases*. 2010;13:340-346.

1167.   Johansen JV, Manniche C, Kjaer P. Vitamin D levels appear to be normal in Danish patients attending secondary care for low back pain and a weak positive correlation between serum level Vitamin D and Modic changes was demonstrated: a cross-sectional cohort study of consecutive patients with non-specific low back pain. *BMC Musculoskelet Disord*. 2013;14:78.

1168.   Al Faraj S, Al Mutairi K. Vitamin D deficiency and chronic low back pain in Saudi Arabia. *Spine*. 2003;28:177-179.

1169.   Schwalfenberg G. Improvement of chronic back pain or failed back surgery with vitamin D repletion: a case series. *Journal of the American Board of Family Medicine : JABFM*. 2009;22:69-74.

1170.   Mibielli MA, Geller M, Cohen JC, et al. Diclofenac plus B vitamins versus diclofenac monotherapy in lumbago: the DOLOR study. *Current medical research and opinion*. 2009;25:2589-2599.

1171.   Kuhlwein A, Meyer HJ, Koehler CO. Reduced diclofenac administration by B vitamins: results of a randomized double-blind study with reduced daily doses of diclofenac (75 mg diclofenac versus 75 mg diclofenac plus B vitamins) in acute lumbar vertebral syndromes. *Klin Wochenschr*. 1990;68:107-115.

1172.   Vetter G, Bruggemann G, Lettko M, et al. Shortening diclofenac therapy by B vitamins. Results of a randomized double-blind study, diclofenac 50 mg versus diclofenac 50 mg plus B vitamins, in painful spinal diseases with degenerative changes. *Z Rheumatol*. 1988;47:351-362.

1173.   Cairns MC, Foster NE, Wright CC, Pennington D. Level of distress in a recurrent low back pain population referred for physical therapy. *Spine*. 2003;28:953-959.

1174.   Cecchi F, Negrini S, Pasquini G, et al. Predictors of functional outcome in patients with chronic low back pain undergoing back school, individual physiotherapy or spinal manipulation. *European journal of physical and rehabilitation medicine*. 2012;48:371-378.

1175.   Freburger JK, Carey TS, Holmes GM. Physical therapy for chronic low back pain in North Carolina: overuse, underuse, or misuse? *Phys Ther*. 2011;91:484-495.

1176.   Freburger JK, Holmes GM, Carey TS. Physician referrals to physical therapy for the treatment of musculoskeletal conditions. *Archives of physical medicine and rehabilitation*. 2003;84:1839-1849.

1177.   Gellhorn AC, Chan L, Martin B, Friedly J. Management patterns in acute low back pain: the role of physical therapy. *Spine (Phila Pa 1976)*. 2012;37:775-782.

1178.   Hahne AJ, Ford JJ, Hinman RS, et al. Outcomes and adverse events from physiotherapy functional restoration for lumbar disc herniation with associated radiculopathy. *Disability and rehabilitation*. 2011;33:1537-1547.

1179.   Haskins R, Rivett DA, Osmotherly PG. Clinical prediction rules in the physiotherapy management of low back pain: a systematic review. *Manual therapy*. 2012;17:9-21.

1180.   Hurley MV, Walsh N, Bhavnani V, Britten N, Stevenson F. Health beliefs before and after participation on an exercised-based rehabilitation programme for chronic knee pain: doing is believing. *BMC musculoskeletal disorders*. 2010;11:31.

1181.   Lewis A, Morris M, Walsh C. Are physiotherapy exercises effective in reducing chronic low back pain? *Phys Ther Rev*. 2008;13:37-44.

1182.   McNeely ML, Torrance G, Magee DJ. A systematic review of physiotherapy for spondylolysis and spondylolisthesis. *Manual therapy*. 2003;8:80-91.

1183.   Peiris CL, Taylor NF, Shields N. Extra physical therapy reduces patient length of stay and improves functional outcomes and quality of life in people with acute or subacute conditions: a systematic review. *Arch Phys Med Rehabil*. 2011;92:1490-1500.

1184.   Pillastrini P, Gardenghi I, Bonetti F, et al. An updated overview of clinical guidelines for chronic low back pain management in primary care. *Joint Bone Spine*. 2012;79:176-185.

1185.   Snodgrass J. Effective occupational therapy interventions in the rehabilitation of individuals with work-related low back injuries and illnesses: a systematic review. *The American journal of occupational therapy : official publication of the American Occupational Therapy Association*. 2011;65:37-43.

1186.   Apeldoorn AT, Bosmans JE, Ostelo RW, de Vet HC, van Tulder MW. Cost-effectiveness of a classification-based system for sub-acute and chronic low back pain. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2012;21:1290-1300.

1187.   Apeldoorn AT, Ostelo RW, van Helvoirt H, et al. A randomized controlled trial on the effectiveness of a classification-based system for subacute and chronic low back pain. *Spine (Phila Pa 1976)*. 2012;37:1347-1356.

1188.   Ostelo RW, de Vet HC, Berfelo MW, et al. Effectiveness of behavioral graded activity after first-time lumbar disc surgery: short term results of a randomized controlled trial. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2003;12:637-644.

1189.   Ostelo RW, de Vet HC, Vlaeyen JW, et al. Behavioral graded activity following first-time lumbar disc surgery: 1-year results of a randomized clinical trial. *Spine (Phila Pa 1976)*. 2003;28:1757-1765.

1190.   Albaladejo C, Kovacs FM, Royuela A, del Pino R, Zamora J. The efficacy of a short education program and a short physiotherapy program for treating low back pain in primary care: a cluster randomized trial. *Spine (Phila Pa 1976)*. 2010;35:483-496.

1191.   Bronfort G, Maiers MJ, Evans RL, et al. Supervised exercise, spinal manipulation, and home exercise for chronic low back pain: a randomized clinical trial. *The spine journal : official journal of the North American Spine Society*. 2011;11:585-598.

1192.   Casserley-Feeney SN, Daly L, Hurley DA. The access randomized clinical trial of public versus private physiotherapy for low back pain. *Spine (Phila Pa 1976)*. 2012;37:85-96.

1193.   Cecchi F, Molino-Lova R, Chiti M, et al. Spinal manipulation compared with back school and with individually delivered physiotherapy for the treatment of chronic low back pain: a randomized trial with one-year follow-up. *Clin Rehabil*. 2010;24:26-36.

1194.   Chiradejnant A, Maher CG, Latimer J, Stepkovitch N. Efficacy of "therapist-selected" versus "randomly selected" mobilisation techniques for the treatment of low back pain: a randomised controlled trial. *The Australian journal of physiotherapy*. 2003;49:233-241.

1195.   Critchley DJ, Ratcliffe J, Noonan S, Jones RH, Hurley MV. Effectiveness and cost-effectiveness of three types of physiotherapy used to reduce chronic low back pain disability: a pragmatic randomized trial with economic evaluation. *Spine*. 2007;32:1474-1481.

1196.   Gudavalli MR, Cambron JA, McGregor M, et al. A randomized clinical trial and subgroup analysis to compare flexion-distraction with active exercise for chronic low back pain. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2006;15:1070-1082.

1197. Hurwitz EL, Morgenstern H, Kominski GF, Yu F, Chiang LM. A randomized trial of chiropractic and medical care for patients with low back pain: eighteen-month follow-up outcomes from the UCLA low back pain study. *Spine*. 2006;31:611-621; discussion 622.

1198. Hay EM, Mullis R, Lewis M, et al. Comparison of physical treatments versus a brief pain-management programme for back pain in primary care: a randomised clinical trial in physiotherapy practice. *Lancet*. 2005;365:2024-2030.

1199. Kaapa EH, Frantsi K, Sarna S, Malmivaara A. Multidisciplinary group rehabilitation versus individual physiotherapy for chronic nonspecific low back pain: a randomized trial. *Spine*. 2006;31:371-376.

1200. Marshall PW, Murphy BA. Muscle activation changes after exercise rehabilitation for chronic low back pain. *Archives of physical medicine and rehabilitation*. 2008;89:1305-1313.

1201. Moseley L. Combined physiotherapy and education is efficacious for chronic low back pain. *The Australian journal of physiotherapy*. 2002;48:297-302.

1202. Nordeman L, Nilsson B, Moller M, Gunnarsson R. Early access to physical therapy treatment for subacute low back pain in primary health care: a prospective randomized clinical trial. *Clin J Pain*. 2006;22:505-511.

1203. Rasmussen-Barr E, Ang B, Arvidsson I, Nilsson-Wikmar L. Graded exercise for recurrent low-back pain: a randomized, controlled trial with 6-, 12-, and 36-month follow-ups. *Spine*. 2009;34:221-228.

1204. Reme SE, Hagen EM, Eriksen HR. Expectations, perceptions, and physiotherapy predict prolonged sick leave in subacute low back pain. *BMC Musculoskelet Disord*. 2009;10:139.

1205. Roche G, Ponthieux A, Parot-Shinkel E, et al. Comparison of a functional restoration program with active individual physical therapy for patients with chronic low back pain: a randomized controlled trial. *Arch Phys Med Rehabil*. 2007;88:1229-1235.

1206. Roche-Leboucher G, Petit-Lemanac'h A, Bontoux L, et al. Multidisciplinary intensive functional restoration versus outpatient active physiotherapy in chronic low back pain: a randomized controlled trial. *Spine (Phila Pa 1976)*. 2011;36:2235-2242.

1207. Schenkman ML, Jordan S, Akuthota V, et al. Functional movement training for recurrent low back pain: lessons from a pilot randomized controlled trial. *PM & R : the journal of injury, function, and rehabilitation*. 2009;1:137-146.

1208. Sertpoyraz F, Eyigor S, Karapolat H, Capaci K, Kirazli Y. Comparison of isokinetic exercise versus standard exercise training in patients with chronic low back pain: a randomized controlled study. *Clin Rehabil*. 2009;23:238-247.

1209. Sorensen PH, Bendix T, Manniche C, Korsholm L, Lemvigh D, Indahl A. An educational approach based on a non-injury model compared with individual symptom-based physical training in chronic LBP. A pragmatic, randomised trial with a one-year follow-up. *BMC Musculoskelet Disord*. 2010;11:212.

1210. van der Roer N, van Tulder M, Barendse J, Knol D, van Mechelen W, de Vet H. Intensive group training protocol versus guideline physiotherapy for patients with chronic low back pain: a randomised controlled trial. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2008;17:1193-1200.

1211. van der Roer N, van Tulder M, van Mechelen W, de Vet H. Economic evaluation of an intensive group training protocol compared with usual care physiotherapy in patients with chronic low back pain. *Spine (Phila Pa 1976)*. 2008;33:445-451.

1212. Vong SK, Cheing GL, Chan F, So EM, Chan CC. Motivational enhancement therapy in addition to physical therapy improves motivational factors and treatment outcomes in people with low back pain: a randomized controlled trial. *Arch Phys Med Rehabil*. 2011;92:176-183.

1213. Wand BM, Bird C, McAuley JH, Dore CJ, MacDowell M, De Souza LH. Early intervention for the management of acute low back pain: a single-blind randomized controlled trial of biopsychosocial education, manual therapy, and exercise. *Spine*. 2004;29:2350-2356.

1214. Wand BM, Tulloch VM, George PJ, et al. Seeing it helps: movement-related back pain is reduced by visualization of the back during movement. *Clin J Pain*. 2012;28:602-608.

1215. Whitman JM, Fritz JM, Childs JD. The influence of experience and specialty certifications on clinical outcomes for patients with low back pain treated within a standardized physical therapy management program. *The Journal of orthopaedic and sports physical therapy*. 2004;34:662-672; discussion 672-665.

1216. Gohner W, Schlicht W. Preventing chronic back pain: evaluation of a theory-based cognitive-behavioural training programme for patients with subacute back pain. *Patient Educ Couns*. 2006;64:87-95.

1217. Helmhout PH, Harts CC, Viechtbauer W, Staal JB, de Bie RA. Isolated lumbar extensor strengthening versus regular physical therapy in an army working population with nonacute low back pain: a randomized controlled trial. *Arch Phys Med Rehabil*. 2008;89:1675-1685.

1218.   Thackeray A, Fritz JM, Brennan GP, Zaman FM, Willick SE. A pilot study examining the effectiveness of physical therapy as an adjunct to selective nerve root block in the treatment of lumbar radicular pain from disk herniation: a randomized controlled trial. *Phys Ther*. 2010;90:1717-1729.
1219.   Erdogmus CB, Resch KL, Sabitzer R, et al. Physiotherapy-based rehabilitation following disc herniation operation: results of a randomized clinical trial. *Spine (Phila Pa 1976)*. 2007;32:2041-2049.
1220.   Iles R, Taylor NF, Davidson M, O'Halloran P. Telephone coaching can increase activity levels for people with non-chronic low back pain: a randomised trial. *Journal of physiotherapy*. 2011;57:231-238.
1221.   Kamioka H, Okuizumi H, Okada S, et al. Effectiveness of intervention for low back pain in female caregivers in nursing homes: a pilot trial based on multicenter randomization. *Environmental health and preventive medicine*. 2011;16:97-105.
1222.   Kell RT, Risi AD, Barden JM. The response of persons with chronic nonspecific low back pain to three different volumes of periodized musculoskeletal rehabilitation. *Journal of strength and conditioning research / National Strength & Conditioning Association*. 2011;25:1052-1064.
1223.   Lau PM, Chow DH, Pope MH. Early physiotherapy intervention in an Accident and Emergency Department reduces pain and improves satisfaction for patients with acute low back pain: a randomised trial. *The Australian journal of physiotherapy*. 2008;54:243-249.
1224.   McGregor AH, Dore CJ, Morris TP, Morris S, Jamrozik K. ISSLS prize winner: Function After Spinal Treatment, Exercise, and Rehabilitation (FASTER): a factorial randomized trial to determine whether the functional outcome of spinal surgery can be improved. *Spine (Phila Pa 1976)*. 2011;36:1711-1720.
1225.   Overman SS, Larson JW, Dickstein DA, Rockey PH. Physical therapy care for low back pain. Monitored program of first-contact nonphysician care. *Phys Ther*. 1988;68:199-207.
1226.   Bekkering GE, van Tulder MW, Hendriks EJM, et al. Implementation of clinical guidelines on physical therapy for patients with low back pain: randomized trial comparing patient outcomes after a standard and active implementation strategy. *Physical Therapy*. 2005;85:544-555.
1227.   Morris S, Morris TP, McGregor AH, Dore CJ, Jamrozik K. Function after spinal treatment, exercise, and rehabilitation: cost-effectiveness analysis based on a randomized controlled trial. *Spine (Phila Pa 1976)*. 2011;36:1807-1814.
1228.   Rivero-Arias O, Gray A, Frost H, Lamb SE, Stewart-Brown S. Cost-utility analysis of physiotherapy treatment compared with physiotherapy advice in low back pain. *Spine (Phila Pa 1976)*. 2006;31:1381-1387.
1229.   Underwood MR, Harding G, Klaber Moffett J, team UBt. Patient perceptions of physical therapy within a trial for back pain treatments (UK BEAM) [ISRCTN32683578]. *Rheumatology (Oxford)*. 2006;45:751-756.
1230.   Bruce-Low S, Smith D, Burnet S, Fisher J, Bissell G, Webster L. One lumbar extension training session per week is sufficient for strength gains and reductions in pain in patients with chronic low back pain ergonomics. *Ergonomics*. 2012;55:500-507.
1231.   Kofotolis N, Kellis E. Effects of two 4-week proprioceptive neuromuscular facilitation programs on muscle endurance, flexibility, and functional performance in women with chronic low back pain. *Phys Ther*. 2006;86:1001-1012.
1232.   Domenech J, Sanchez-Zuriaga D, Segura-Orti E, Espejo-Tort B, Lison JF. Impact of biomedical and biopsychosocial training sessions on the attitudes, beliefs, and recommendations of health care providers about low back pain: a randomised clinical trial. *Pain*. 2011;152:2557-2563.
1233.   Garcia AN, Gondo FL, Costa RA, Cyrillo FN, Costa LO. Effects of two physical therapy interventions in patients with chronic non-specific low back pain: feasibility of a randomized controlled trial. *Rev Bras Fisioter*. 2011;15:420-427.
1234.   Verra ML, Angst F, Beck T, et al. Horticultural therapy for patients with chronic musculoskeletal pain: results of a pilot study. *Alternative therapies in health and medicine*. 2012;18:44-50.
1235.   Shabat S, Gefen T, Nyska M, Folman Y, Gepstein R. The effect of insoles on the incidence and severity of low back pain among workers whose job involves long-distance walking. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2005;14:546-550.
1236.   Basford JR, Smith MA. Shoe insoles in the workplace. *Orthopedics*. 1988;11:285-288.
1237.   Larsen K, Weidich F, Leboeuf-Yde C. Can custom-made biomechanic shoe orthoses prevent problems in the back and lower extremities? A randomized, controlled intervention trial of 146 military conscripts. *Journal of manipulative and physiological therapeutics*. 2002;25:326-331.
1238.   Defrin R, Ben Benyamin S, Aldubi RD, Pick CG. Conservative correction of leg-length discrepancies of 10mm or less for the relief of chronic low back pain. *Arch Phys Med Rehabil*. 2005;86:2075-2080.
1239.   Milgrom C, Finestone A, Lubovsky O, Zin D, Lahad A. A controlled randomized study of the effect of training with orthoses on the incidence of weight bearing induced back pain among infantry recruits. *Spine*. 2005;30:272-275.
1240.   Tooms RE, Griffin JW, Green S, Cagle K. Effect of viscoelastic insoles on pain. *Orthopedics*. 1987;10:1143-1147.

1241.   Alvarez-Alvarez S, Jose FG, Rodriguez-Fernandez AL, Gueita-Rodriguez J, Waller BJ. Effects of Kinesio(R) Tape in low back muscle fatigue: randomized, controlled, doubled-blinded clinical trial on healthy subjects. *Journal of back and musculoskeletal rehabilitation*. 2014;27:203-212.

1242.   Castro-Sanchez AM, Lara-Palomo IC, Mataran-Penarrocha GA, Fernandez-Sanchez M, Sanchez-Labraca N, Arroyo-Morales M. Kinesio Taping reduces disability and pain slightly in chronic non-specific low back pain: a randomised trial. *Journal of physiotherapy*. 2012;58:89-95.

1243.   Chen SM, Alexander R, Lo SK, Cook J. Effects of Functional Fascial Taping on pain and function in patients with non-specific low back pain: a pilot randomized controlled trial. *Clin Rehabil*. 2012;26:924-933.

1244.   Paoloni M, Bernetti A, Fratocchi G, et al. Kinesio Taping applied to lumbar muscles influences clinical and electromyographic characteristics in chronic low back pain patients. *European journal of physical and rehabilitation medicine*. 2011;47:237-244.

1245.   Enciso G. Does kinesio taping improve the functionality and pain relief of people with non specific low back pain? *Evidence Based Practice III-2*. 2009.

1246.   Karatas N, Bicici S, Baltaci G, Caner H. The effect of Kinesiotape application on functional performance in surgeons who have musculo-skeletal pain after performing surgery. *Turkish neurosurgery*. 2012;22:83-89.

1247.   Centre for Reviews and Dissemination. Effective Health Care Bulletin: Acute and Chronic Low Back Pain. University of York, Royal Society of Medicine Press: York, UK; November 2000, Volume 6, Number 5.

1248.   Dillingham TR. Lumbar supports for prevention of low back pain in the workplace. *JAMA : the journal of the American Medical Association*. 1998;279:1826-1828.

1249.   Fischbacher C. Outpatient physiotherapy services for low back pain. In: Foxcroft D, Muthu V, eds. *STEER: Succinct and Timely Evaluated Evidence Reviews* London, England: Wessex Institute for Health Research & Development, University of Southampton; 2002.

1250.   Hsieh CY, Phillips RB, Adams AH, Pope MH. Functional outcomes of low back pain: comparison of four treatment groups in a randomized controlled trial. *Journal of manipulative and physiological therapeutics*. 1992;15:4-9.

1251.   Maher CG. Effective physical treatment for chronic low back pain. *The Orthopedic clinics of North America*. 2004;35:57-64.

1252.   Ammendolia C, Kerr MS, Bombardier C. Back belt use for prevention of occupational low back pain: a systematic review. *Journal of manipulative and physiological therapeutics*. 2005;28:128-134.

1253.   Jellema P, van Tulder MW, van Poppel MN, Nachemson AL, Bouter LM. Lumbar supports for prevention and treatment of low back pain: a systematic review within the framework of the Cochrane Back Review Group. *Spine*. 2001;26:377-386.

1254.   National Institute for Occupational Safety and Health. Publication Number 94-127. Back Belts: Do They Prevent Injury? : Centers for Disease Control and Prevention, U.S. Department of Health and Human Services; 1996.

1255.   Walsh NE, Schwartz RK. The influence of prophylactic orthoses on abdominal strength and low back injury in the workplace. *American journal of physical medicine & rehabilitation / Association of Academic Physiatrists*. 1990;69:245-250.

1256.   Linton SJ, van Tulder MW. Preventive interventions for back and neck pain problems: what is the evidence? *Spine*. 2001;26:778-787.

1257.   Reddell CR, Congleton JJ, Dale Huchingson R, Montgomery JF. An evaluation of a weightlifting belt and back injury prevention training class for airline baggage handlers. *Applied ergonomics*. 1992;23:319-329.

1258.   Coxhead CE, Inskip H, Meade TW, North WR, Troup JD. Multicentre trial of physiotherapy in the management of sciatic symptoms. *Lancet*. 1981;1:1065-1068.

1259.   Oleske DM, Lavender SA, Andersson GB, Kwasny MM. Are back supports plus education more effective than education alone in promoting recovery from low back pain?: Results from a randomized clinical trial. *Spine (Phila Pa 1976)*. 2007;32:2050-2057.

1260.   Roelofs PD, Bierma-Zeinstra SM, van Poppel MN, et al. Lumbar supports to prevent recurrent low back pain among home care workers: a randomized trial. *Annals of internal medicine*. 2007;147:685-692.

1261.   Roelofs PD, Bierma-Zeinstra SM, van Poppel MN, van Mechelen W, Koes BW, van Tulder MW. Cost-effectiveness of lumbar supports for home care workers with recurrent low back pain: an economic evaluation alongside a randomized-controlled trial. *Spine*. 2010;35:E1619-1626.

1262.   Alexander A, Woolley SM, Bisesi M, Schaub E. The effectiveness of back belts on occupational back injuries and worker perception. *Profess Safety*. 1995;40:22-26.

1263.   Kraus JF, Schaffer KB, Rice T, Maroosis J, Harper J. A field trial of back belts to reduce the incidence of acute low back injuries in New York City home attendants. *International journal of occupational and environmental health*. 2002;8:97-104.

1264.   Million R, Nilsen KH, Jayson MI, Baker RD. Evaluation of low back pain and assessment of lumbar corsets with and without back supports. *Ann Rheum Dis*. 1981;40:449-454.

1265.   Valle-Jones JC, Walsh H, O'Hara J, O'Hara H, Davey NB, Hopkin-Richards H. Controlled trial of a back support ('Lumbotrain') in patients with non-specific low back pain. *Current medical research and opinion*. 1992;12:604-613.

1266.   Collacott EA, Zimmerman JT, White DW, Rindone JP. Bipolar permanent magnets for the treatment of chronic low back pain: a pilot study. *Jama*. 2000;283:1322-1325.

1267.   Khoromi S, Blackman MR, Kingman A, et al. Low intensity permanent magnets in the treatment of chronic lumbar radicular pain. *J Pain Symptom Manage*. 2007;34:434-445.

1268.   Brosseau L, Wells GA, Poitras S, et al. Ottawa Panel evidence-based clinical practice guidelines on therapeutic massage for low back pain. *J Bodyw Mov Ther*. 2012;16:424-455.

1269.   Chambers H. Physiotherapy and lumbar facet joint injections as a combination treatment for chronic low back pain. A narrative review of lumbar facet joint injections, lumbar spinal mobilizations, soft tissue massage and lower back mobility exercises. *Musculoskeletal care*. 2013;11:106-120.

1270.   Cherkin DC, Sherman KJ, Deyo RA, Shekelle PG. A review of the evidence for the effectiveness, safety, and cost of acupuncture, massage therapy, and spinal manipulation for back pain. *Ann Intern Med*. 2003;138:898-906.

1271.   Furlan AD, Imamura M, Dryden T, Irvin E. Massage for low-back pain. *Cochrane Database Syst Rev*. 2008:Cd001929.

1272.   Kumar S, Beaton K, Hughes T. The effectiveness of massage therapy for the treatment of nonspecific low back pain: a systematic review of systematic reviews. *International journal of general medicine*. 2013;6:733-741.

1273.   Melancon B, Miller LH. Massage therapy versus traditional therapy for low back pain relief: implications for holistic nursing practice. *Holistic nursing practice*. 2005;19:116-121.

1274.   Romanowski M, Romanowska J, Grzeskowiak M. A comparison of the effects of deep tissue massage and therapeutic massage on chronic low back pain. *Studies in health technology and informatics*. 2012;176:411-414.

1275.   Furlan AD, Yazdi F, Tsertsvadze A, et al. Complementary and alternative therapies for back pain II. *Evidence report/technology assessment*. 2010:1-764.

1276.   Ernst E. Massage therapy for low back pain: a systematic review. *J Pain Symptom Manage*. 1999;17:65-69.

1277.   Imamura M, Furlan AD, Dryden T, Irvin E. Evidence-informed management of chronic low back pain with massage. *The spine journal : official journal of the North American Spine Society*. 2008;8:121-133.

1278.   Netchanok S, Wendy M, Marie C, Siobhan O. The effectiveness of Swedish massage and traditional Thai massage in treating chronic low back pain: a review of the literature. *Complementary therapies in clinical practice*. 2012;18:227-234.

1279.   Furlan AD, Brosseau L, Imamura M, Irvin E. Massage for low-back pain: a systematic review within the framework of the Cochrane Collaboration Back Review Group. *Spine*. 2002;27:1896-1910.

1280.   Furlan AD, Brosseau L, Imamura M, Irvin E. Massage for low back pain. *Cochrane Database Syst Rev*. 2002:CD001929.

1281.   Melzack R, Vetere P, Finch L. Transcutaneous electrical nerve stimulation for low back pain. A comparison of TENS and massage for pain and range of motion. *Phys Ther*. 1983;63:489-493.

1282.   Werners R, Pynsent PB, Bulstrode CJ. Randomized trial comparing interferential therapy with motorized lumbar traction and massage in the management of low back pain in a primary care setting. *Spine*. 1999;24:1579-1584.

1283.   Albright J, Allman R, Bonfiglio R. Philadelphia Panel Evidence-Based Clinical Practice Guidelines on Selected Rehabilitation Interventions for Low Back Pain. *Phys Ther*. 2001;81:1641-1674.

1284.   Cherkin DC, Eisenberg D, Sherman KJ, et al. Randomized trial comparing traditional Chinese medical acupuncture, therapeutic massage, and self-care education for chronic low back pain. *Archives of internal medicine*. 2001;161:1081-1088.

1285.   Preyde M. Effectiveness of massage therapy for subacute low-back pain: a randomized controlled trial. *Cmaj*. 2000;162:1815-1820.

1286.   Buselli. Effectiveness evaluation of an integrated automatic termomechanic massage system (SMATH® system) in nonspecific sub-acute and chronic low back pain - a randomized doubleblinded controlled trial, comparing SMATH therapy versus sham therapy: study protocol for a randomized controlled trial. 2011;12.

1287.   Chatchawan U, Thinkhamrop B, Kharmwan S, Knowles J, et al. Effectiveness of traditional Thai massage versus Swedish massage among patients with back pain associated with myofascial trigger points. *J Bodywork and Movement Ther*. 2005;9:298-309.

1288.   Cherkin DC, Sherman KJ, Kahn J, et al. A comparison of the effects of 2 types of massage and usual care on chronic low back pain: a randomized, controlled trial. *Annals of internal medicine*. 2011;155:1-9.

1289.   Gam AN, Warming S, Larsen LH, et al. Treatment of myofascial trigger-points with ultrasound combined with massage and exercise--a randomised controlled trial. *Pain*. 1998;77:73-79.

1290.   Kalauokalani D, Cherkin DC, Sherman KJ, Koepsell TD, Deyo RA. Lessons from a trial of acupuncture and massage for low back pain: patient expectations and treatment effects. *Spine*. 2001;26:1418-1424.

1291.   Little P, Lewith G, Webley F, et al. Randomised controlled trial of Alexander technique lessons, exercise, and massage (ATEAM) for chronic and recurrent back pain. *BMJ*. 2008;337:a884.

1292.   Hernandez-Reif M, Field T, Krasnegor J, Theakston H. Lower back pain is reduced and range of motion increased after massage therapy. *The International journal of neuroscience*. 2001;106:131-145.

1293.   Mackawan S, Eungpinichpong W, Pantumethakul R, et al. Effects of traditional Thai massage versus joint mobilization on substance P and pain perception in patients with non-specific low back pain. *J Bodywork Movement Ther*. 2007;11:9-16.

1294.   Walach H, Guthlin C, Konig M. Efficacy of massage therapy in chronic pain: a pragmatic randomized trial. *J Altern Complement Med*. 2003;9:837-846.

1295.   Zheng Z, Wang J, Gao Q, et al. Therapeutic evaluation of lumbar tender point deep massage for chronic non-specific low back pain. *J Tradit Chin Med*. 2012;32:534-537.

1296.   Ernst E. Is reflexology an effective intervention? A systematic review of randomised controlled trials. *The Medical journal of Australia*. 2009;191:263-266.

1297.   Poole H, Glenn S, Murphy P. A randomised controlled study of reflexology for the management of chronic low back pain. *Eur J Pain*. 2007;11:878-887.

1298.   Quinn F, Hughes CM, Baxter GD. Reflexology in the management of low back pain: a pilot randomised controlled trial. *Complementary therapies in medicine*. 2008;16:3-8.

1299.   Eghbali M, Safari R, Nazari F, Abdoli S. The effects of reflexology on chronic low back pain intensity in nurses employed in hospitals affiliated with Isfahan University of Medical Sciences. *Iranian journal of nursing and midwifery research*. 2012;17:239-243.

1300.   Hawk C, Long CR, Rowell RM, Gudavalli MR, Jedlicka J. A randomized trial investigating a chiropractic manual placebo: a novel design using standardized forces in the delivery of active and control treatments. *J Altern Complement Med*. 2005;11:109-117.

1301.   Hsieh CY, Adams AH, Tobis J, et al. Effectiveness of four conservative treatments for subacute low back pain: a randomized clinical trial. *Spine*. 2002;27:1142-1148.

1302.   Cai C, Pua Y, Lim K. A clinical prediction rule for classifying patients with low back pain who demonstrate short-term improvement with mechanical lumbar traction. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2009;18:554-561.

1303.   Gay RE, Brault JS. Evidence-informed management of chronic low back pain with traction therapy. *The spine journal : official journal of the North American Spine Society*. 2008;8:234-242.

1304.   Harte AA, Baxter GD, Gracey JH. The efficacy of traction for back pain: a systematic review of randomized controlled trials. *Archives of physical medicine and rehabilitation*. 2003;84:1542-1553.

1305.   Krause M, Refshauge KM, Dessen M, Boland R. Lumbar spine traction: evaluation of effects and recommended application for treatment. *Manual therapy*. 2000;5:72-81.

1306.   Letchuman R, Deusinger RH. Comparison of sacrospinalis myoelectric activity and pain levels in patients undergoing static and intermittent lumbar traction. *Spine*. 1993;18:1361-1365.

1307.   Ljunggren A. Manual traction versus ismetric exercises in patients with herniated inveertebral lumbar discs. *Phiysiotherapy theory and practice* 1992;8:207-213.

1308.   Macario A, Pergolizzi JV. Systematic literature review of spinal decompression via motorized traction for chronic discogenic low back pain. *Pain Pract*. 2006;6:171-178.

1309.   Vroomen PC, de Krom MC, Slofstra PD, Knottnerus JA. Conservative treatment of sciatica: a systematic review. *J Spinal Disord*. 2000;13:463-469.

1310.   Wegner I, Widyahening IS, van Tulder MW, et al. Traction for low-back pain with or without sciatica. *The Cochrane database of systematic reviews*. 2013;8:CD003010.

1311.   Beurskens AJ, de Vet HC, Koke AJ, et al. Efficacy of traction for non-specific low back pain: a randomised clinical trial. *Lancet*. 1995;346:1596-1600.

1312.   Beurskens AJ, de Vet HC, Koke AJ, et al. Efficacy of traction for nonspecific low back pain. 12-week and 6-month results of a randomized clinical trial. *Spine*. 1997;22:2756-2762.

1313.   Beurskens AJ, van der Heijden GJ, de Vet HC, et al. The efficacy of traction for lumbar back pain: design of a randomized clinical trial. *Journal of manipulative and physiological therapeutics*. 1995;18:141-147.

1314.   Schimmel. No effect of traction in patients with low back pain: a single centre, single blind, randomized controlled trial of Intervertebral Differential Dynamics Therapy. *Spine*. 2009;18:1843-1850.

1315.   Sweetman BJ, Heinrich I, Anderson JAD. A randomized controlled trial of exercises, short wave diathermy, and traction for low back pain, with evidence of diagnosis-related response to treatment *J Orthop Rheumatology*. 1993;6:159-166.

1316.   van der Heijden GJ, Beurskens, A.J., Dirx M.J., Bouter L.M., Lindeman, E. Efficacy of lumbar traction: a randomised clinical trial. *Physiotherapy*. 1995;81:29-35

1317.   Mathews JA, Hickling J. Lumbar traction: a double-blind controlled study for sciatica. *Rheumatology and rehabilitation*. 1975;14:222-225.

1318.   Mathews W, Morkel M, Mathews J. Manipulation and traction for lumbago and sciatica: physiotherapeutic techniques used in two controlled trials. *Physiother Prac*. 1988;4::201-206.

1319.   Pal B, Mangion P, Hossain MA, Diffey BL. A controlled trial of continuous lumbar traction in the treatment of back pain and sciatica. *British journal of rheumatology*. 1986;25:181-183.

1320.   Larsson U, Choler U, Lidstrom A, et al. Auto-traction for treatment of lumbago-sciatica. A multicentre controlled investigation. *Acta orthopaedica Scandinavica*. 1980;51:791-798.

1321.   Mathews JA, Mills SB, Jenkins VM, et al. Back pain and sciatica: controlled trials of manipulation, traction, sclerosant and epidural injections. *British journal of rheumatology*. 1987;26:416-423.

1322.   Weber H. Traction therapy in sciatica due to disc prolapse (does traction treatment have any positive effect on patients suffering from sciatica caused by disc prolapse?). *Journal of the Oslo city hospitals*. 1973;23:167-176.

1323.   Weber H, Ljunggren AE, Walker L. Traction therapy in patients with herniated lumbar intervertebral discs. *Journal of the Oslo city hospitals*. 1984;34:61-70.

1324.   Guvenol K, Tuzun C, Peker O, Goktay Y. A comparison of inverted spinal traction and conventional traction in the treatment of lumbar disc herniations. *Physiotherapy Theory and Practice*. 2000;16:151-160.

1325.   Konrad K, Tatrai T, Hunka A, Vereckei E, Korondi I. Controlled trial of balneotherapy in treatment of low back pain. *Ann Rheum Dis*. 1992;51:820-822.

1326.   Ljunggren AE, Weber H, Larsen S. Autotraction versus manual traction in patients with prolapsed lumbar intervertebral discs. *Scandinavian journal of rehabilitation medicine*. 1984;16:117-124.

1327.   Lidstrom A, Zachrisson M. Physical therapy on low back pain and sciatica. An attempt at evaluation. *Scandinavian journal of rehabilitation medicine*. 1970;2:37-42.

1328.   Mirovsky Y. The effect of ambulatory lumbar traction combined with treadmill on patients with chronic low back pain. *J Back Musculoskeletal Rehab*. 2006;19:73-78.

1329.   Tesio L, Merlo A. Autotraction versus passive traction: an open controlled study in lumbar disc herniation. *Arch Phys Med Rehabil*. 1993;74:871-876.

1330.   Sherry E, Kitchener P, Smart R. A prospective randomized controlled study of VAX-D and TENS for the treatment of chronic low back pain. *Neurological research*. 2001;23:780-784.

1331.   Bronfort G, Hondras MA, Schulz CA, Evans RL, Long CR, Grimm R. Spinal manipulation and home exercise with advice for subacute and chronic back-related leg pain: a trial with adaptive allocation. *Ann Intern Med*. 2014;161:381-391.

1332.   Ernst E, Harkness E. Spinal manipulation: a systematic review of sham-controlled, double-blind, randomized clinical trials. *J Pain Symptom Manage*. 2001;22:879-889.

1333.   Rubinstein S, Terwee C, Assendelft W, de Boer M, van Tulder M. Spinal manipulative therapy for acute low back pain: an update of the cochrane review. *Spine*. 2013;38:E158-177.

1334.   Schneider M, Haas M, Glick R, Stevans J, Landsittel D. Comparison of spinal manipulation methods and usual medical care for acute and subacute low back pain: a randomized clinical trial. *Spine*. 2015;40:209-217.

1335.   Strauss S. Myofascial pain syndrome: a short review. *Web J Acupuncture*; 2002.

1336.   Andersson GB, Lucente T, Davis AM, Kappler RE, Lipton JA, Leurgans S. A comparison of osteopathic spinal manipulation with standard care for patients with low back pain. *N Engl J Med*. 1999;341:1426-1431.

1337.   Cleland JA, Fritz JM, Kulig K, et al. Comparison of the effectiveness of three manual physical therapy techniques in a subgroup of patients with low back pain who satisfy a clinical prediction rule: a randomized clinical trial. *Spine*. 2009;34:2720-2729.

1338.   Juni P, Battaglia M, Nuesch E, et al. A randomised controlled trial of spinal manipulative therapy in acute low back pain. *Ann Rheum Dis*. 2009;68:1420-1427.

1339.   Fernandez-De-Las-Penas C, Cleland JA, Huijbregts P, Palomeque-Del-Cerro L, Gonzalez-Iglesias J. Repeated applications of thoracic spine thrust manipulation do not lead to tolerance in patients presenting with acute mechanical neck pain: a secondary analysis. *The Journal of manual & manipulative therapy*. 2009;17:154-162.

1340.   Gross A, Miller J, D'Sylva J, et al. Manipulation or mobilisation for neck pain: a Cochrane Review. *Manual therapy*. 2010;15:315-333.

1341.   Wood TG, Colloca CJ, Matthews R. A pilot randomized clinical trial on the relative effect of instrumental (MFMA) versus manual (HVLA) manipulation in the treatment of cervical spine dysfunction. *Journal of manipulative and physiological therapeutics*. 2001;24:260-271.

1342.   Bialosky JE, Bishop MD, Robinson ME, Zeppieri G, Jr., George SZ. Spinal manipulative therapy has an immediate effect on thermal pain sensitivity in people with low back pain: a randomized controlled trial. *Phys Ther*. 2009;89:1292-1303.

1343. McMorland G, Suter E, Casha S, du Plessis SJ, Hurlbert RJ. Manipulation or microdiskectomy for sciatica? A prospective randomized clinical study. *Journal of manipulative and physiological therapeutics*. 2010;33:576-584.

1344. Paatelma M, Kilpikoski S, Simonen R, Heinonen A, Alen M, Videman T. Orthopaedic manual therapy, McKenzie method or advice only for low back pain in working adults: a randomized controlled trial with one year follow-up. *Journal of rehabilitation medicine*. 2008;40:858-863.

1345. Santilli V, Beghi E, Finucci S. Chiropractic manipulation in the treatment of acute back pain and sciatica with disc protrusion: a randomized double-blind clinical trial of active and simulated spinal manipulations. *The spine journal : official journal of the North American Spine Society*. 2006;6:131-137.

1346. Triano JJ, McGregor M, Hondras MA, Brennan PC. Manipulative therapy versus education programs in chronic low back pain. *Spine*. 1995;20:948-955.

1347. Assendelft WJ, Koes BW, Knipschild PG, Bouter LM. The relationship between methodological quality and conclusions in reviews of spinal manipulation. *Jama*. 1995;274:1942-1948.

1348. Avery S, O'Driscoll M. Randomised controlled trials on the efficacy of spinal manipulation therapy in the treatment of low back pain. *Phys Ther Reviews*. 2004;9:146-152.

1349. Koes BW, Assendelft WJ, van der Heijden GJ, Bouter LM. Spinal manipulation for low back pain. An updated systematic review of randomized clinical trials. *Spine*. 1996;21:2860-2871; discussion 2872-2863.

1350. Hoehler FK, Tobis JS. Appropriate statistical methods for clinical trials of spinal manipulation. *Spine*. 1987;12:409-411.

1351. Rubinstein SM, Terwee CB, Assendelft WJ, de Boer MR, van Tulder MW. Spinal manipulative therapy for acute low-back pain. *The Cochrane database of systematic reviews*. 2012;9:CD008880.

1352. Childs JD, Flynn TW, Fritz JM. A perspective for considering the risks and benefits of spinal manipulation in patients with low back pain. *Manual therapy*. 2006;11:316-320.

1353. Whedon JM, Song Y, Mackenzie TA, Phillips RB, Lukovits TG, Lurie JD. Risk of stroke after chiropractic spinal manipulation in medicare B beneficiaries aged 66 to 99 years with neck pain. *Journal of manipulative and physiological therapeutics*. 2015;38:93-101.

1354. Assendelft WJ, Bouter LM, Knipschild PG. Complications of spinal manipulation: a comprehensive review of the literature. *The Journal of family practice*. 1996;42:475-480.

1355. Legorreta AP, Metz RD, Nelson CF, Ray S, Chernicoff HO, Dinubile NA. Comparative analysis of individuals with and without chiropractic coverage: patient characteristics, utilization, and costs. *Archives of internal medicine*. 2004;164:1985-1992.

1356. Liliedahl RL, Finch MD, Axene DV, Goertz CM. Cost of care for common back pain conditions initiated with chiropractic doctor vs medical doctor/doctor of osteopathy as first physician: experience of one Tennessee-based general health insurer. *Journal of manipulative and physiological therapeutics*. 2010;33:640-643.

1357. Lin CW, Haas M, Maher CG, Machado LA, van Tulder MW. Cost-effectiveness of guideline-endorsed treatments for low back pain: a systematic review. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2011;20:1024-1038.

1358. Martin BI, Gerkovich MM, Deyo RA, et al. The association of complementary and alternative medicine use and health care expenditures for back and neck problems. *Med Care*. 2012;50:1029-1036.

1359. UK BEAM Trial Team. United Kingdom back pain exercise and manipulation (UK BEAM) randomised trial: cost effectiveness of physical treatments for back pain in primary care. *BMJ*. 2004;329:1381.

1360. Glover JR, Morris JG, Khosla T. Back pain: a randomized clinical trial of rotational manipulation of the trunk. *British journal of industrial medicine*. 1974;31:59-64.

1361. Skargren EI, Carlsson PG, Oberg BE. One-year follow-up comparison of the cost and effectiveness of chiropractic and physiotherapy as primary management for back pain. Subgroup analysis, recurrence, and additional health care utilization. *Spine*. 1998;23:1875-1883; discussion 1884.

1362. Skargren EI, Oberg BE. Predictive factors for 1-year outcome of low-back and neck pain in patients treated in primary care: comparison between the treatment strategies chiropractic and physiotherapy. *Pain*. 1998;77:201-207.

1363. Skargren EI, Oberg BE, Carlsson PG, Gade M. Cost and effectiveness analysis of chiropractic and physiotherapy treatment for low back and neck pain. Six-month follow-up. *Spine*. 1997;22:2167-2177.

1364. Sutlive TG, Mabry LM, Easterling EJ, et al. Comparison of short-term response to two spinal manipulation techniques for patients with low back pain in a military beneficiary population. *Military medicine*. 2009;174:750-756.

1365. von Heymann W, Schloemer P, Timm J, Muehlbauer B. Spinal high-velocity low amplitude manipulation in acute nonspecific low back pain: a double-blinded randomized controlled trial in comparison with diclofenac and placebo. *Spine*. 2013;38:540-548.

1366.   Hadler NM, Curtis P, Gillings DB, Stinnett S. A benefit of spinal manipulation as adjunctive therapy for acute low-back pain: a stratified controlled trial. *Spine*. 1987;12:702-706.

1367.   Hondras MA, Long CR, Cao Y, Rowell RM, Meeker WC. A randomized controlled trial comparing 2 types of spinal manipulation and minimal conservative medical care for adults 55 years and older with subacute or chronic low back pain. *Journal of manipulative and physiological therapeutics*. 2009;32:330-343.

1368.   Sanders GE, Reinert O, Tepe R, Maloney P. Chiropractic adjustive manipulation on subjects with acute low back pain: visual analog pain scores and plasma beta-endorphin levels. *Journal of manipulative and physiological therapeutics*. 1990;13:391-395.

1369.   Sims-Williams H, Jayson MI, Young SM, Baddeley H, Collins E. Controlled trial of mobilisation and manipulation for patients with low back pain in general practice. *British medical journal*. 1978;2:1338-1340.

1370.   Sims-Williams H, Jayson MI, Young SM, Baddeley H, Collins E. Controlled trial of mobilisation and manipulation for low back pain: hospital patients. *British medical journal*. 1979;2:1318-1320.

1371.   Farrell JP, Twomey LT. Acute low back pain. Comparison of two conservative treatment approaches. *The Medical journal of Australia*. 1982;1:160-164.

1372.   Jayson MI, Sims-Williams H, Young S, Baddeley H, Collins E. Mobilization and manipulation for low-back pain. *Spine*. 1981;6:409-416.

1373.   Learman KE, Myers JB, Lephart SM, Sell TC, Kerns GJ, Cook CE. Effects of spinal manipulation on trunk proprioception in subjects with chronic low back pain during symptom remission. *Journal of manipulative and physiological therapeutics*. 2009;32:118-126.

1374.   MacDonald RS, Bell CM. An open controlled assessment of osteopathic manipulation in nonspecific low-back pain. *Spine*. 1990;15:364-370.

1375.   Pope MH, Phillips RB, Haugh LD, Hsieh CY, MacDonald L, Haldeman S. A prospective randomized three-week trial of spinal manipulation, transcutaneous muscle stimulation, massage and corset in the treatment of subacute low back pain. *Spine*. 1994;19:2571-2577.

1376.   Rasmussen J, Laetgaard J, Lindecrona AL, Qvistgaard E, Bliddal H. Manipulation does not add to the effect of extension exercises in chronic low-back pain (LBP). A randomized, controlled, double blind study. *Joint Bone Spine*. 2008;75:708-713.

1377.   Wreje U, Nordgren B, Aberg H. Treatment of pelvic joint dysfunction in primary care--a controlled study. *Scandinavian journal of primary health care*. 1992;10:310-315.

1378.   Arkuszewski Z. The efficacy of manual treatment in low back pain: a clinical trial. *Man Med*. 1986;2:68-71.

1379.   Bicalho E, Setti JA, Macagnan J, Cano JL, Manffra EF. Immediate effects of a high-velocity spine manipulation in paraspinal muscles activity of nonspecific chronic low-back pain subjects. *Manual therapy*. 2010;15:469-475.

1380.   Cibulka MT, Delitto A, Koldehoff RM. Changes in innominate tilt after manipulation of the sacroiliac joint in patients with low back pain. An experimental study. *Phys Ther*. 1988;68:1359-1363.

1381.   Cramer GD, Humphreys CR, Hondras MA, McGregor M, Triano JJ. The Hmax/Mmax ratio as an outcome measure for acute low back pain. *Journal of manipulative and physiological therapeutics*. 1993;16:7-13.

1382.   Evans DP, Burke MS, Lloyd KN, Roberts EE, Roberts GM. Lumbar spinal manipulation on trial. Part I--clinical assessment. *Rheumatology and rehabilitation*. 1978;17:46-53.

1383.   Herzog W, Conway PJ, Willcox BJ. Effects of different treatment modalities on gait symmetry and clinical measures for sacroiliac joint patients. *Journal of manipulative and physiological therapeutics*. 1991;14:104-109.

1384.   Hoehler FK, Tobis JS, Buerger AA. Spinal manipulation for low back pain. *Jama*. 1981;245:1835-1838.

1385.   Kinalski R, Kuwik W, Pietrzak D. The comparison of the results of manual therapy versus physiotherapy methods used in treatment of patients with low back pain. *J Man Med*. 1989;4:44-46.

1386.   Nwuga VC. Relative therapeutic efficacy of vertebral manipulation and conventional treatment in back pain management. *American journal of physical medicine*. 1982;61:273-278.

1387.   Postacchini F, Facchini M, Palieri P. Efficacy of various forms of conservative treatment in low back pain. A comparative study. *Neuro-Orthopedics*. 1988;6:28-35.

1388.   Rupert RL, Wagnon R, Thompson P, Ezzeldin MT. Chiropractic adjustments: Results of a controlled clinical trial in Egypt. *ICA International Review of Chiropractic*. 1985;Winter.

1389.   Zylbergold RS, Piper MC. Lumbar disc disease: comparative analysis of physical therapy treatments. *Arch Phys Med Rehabil*. 1981;62:176-179.

1390.   Godfrey CM, Morgan PP, Schatzker J. A randomized trial of manipulation for low-back pain in a medical setting. *Spine*. 1984;9:301-304.

1391.   Digiorgi D. Spinal manipulation under anesthesia: a narrative review of the literature and commentary. *Chiropractic & manual therapies*. 2013;21:14.

1392.   Morningstar MW, Strauchman MN. Manipulation under anesthesia for patients with failed back surgery: retrospective report of 3 cases with 1-year follow-up. *Journal of chiropractic medicine*. 2012;11:30-35.

1393.   Palmieri NF, Smoyak S. Chronic low back pain: a study of the effects of manipulation under anesthesia. *Journal of manipulative and physiological therapeutics*. 2002;25:E8-E17.

1394.   Ross HE, Siehl D. Evaluation of manipulation of the lumbar spine under general anesthesia for lumbar nerve root compression syndrome, utilizing electromyographic and clinical neurologic examinations. *The Journal of the American Osteopathic Association*. 1968;67:1027.

1395.   Siehl D, Olson DR, Ross HE, Rockwood EE. Manipulation of the lumbar spine with the patient under general anesthesia: evaluation by electromyography and clinical-neurologic examination of its use for lumbar nerve root compression syndrome. *The Journal of the American Osteopathic Association*. 1971;70:433-440.

1396.   West DT, Mathews RS, Miller MR, Kent GM. Effective management of spinal pain in one hundred seventy-seven patients evaluated for manipulation under anesthesia. *Journal of manipulative and physiological therapeutics*. 1999;22:299-308.

1397.   Silver JR. The earliest case of cauda equina syndrome caused by manipulation of the lumbar spine under a general anaesthetic. *Spinal cord*. 2001;39:51-53.

1398.   Ongley MJ, Klein RG, Dorman TA, Eek BC, Hubert LJ. A new approach to the treatment of chronic low back pain. *Lancet*. 1987;2:143-146.

1399.   Dan NG, Saccasan PA. Serious complications of lumbar spinal manipulation. *The Medical journal of Australia*. 1983;2:672-673.

1400.   Kohlbeck FJ, Haldeman S. Medication-assisted spinal manipulation. *The spine journal : official journal of the North American Spine Society*. 2002;2:288-302.

1401.   Grana WA. Physical agents in musculoskeletal problems: heat and cold therapy modalities. *Instructional course lectures*. 1993;42::439-442.

1402.   Magyarosy I, Krause K, Resch K, Guggemos W, Utzschneider I, Gall H, et al. Surface EMG response to heat and cold applicationon back muscles: implications for the therapy of low back pain. *Eur J Phys Med Rehabil*. 1996;6:39-42.

1403.   Michlovitz SL. Thermal Agents in Rehabilitation. *Philadelphia, Pa: FA Davis*. 1996;3rd edition.

1404.   Roberts D, Walls C, Carlile J, Wheaton C, Aronoff G. Relief of chronic low back pain: heat versus cold
In: Aronoff G, ed. *Chapter 13, Evaluation and Treatment of Chronic Pain, 2nd Edition*. Baltimore: Urban & Schwarzenberg; 1992:263-266.

1405.   French SD, Cameron M, Walker BF, Reggars JW, Esterman AJ. Superficial heat or cold for low back pain. *Cochrane Database Syst Rev*. 2006:CD004750.

1406.   Melzack R, Jeans ME, Stratford JG, Monks RC. Ice massage and transcutaneous electrical stimulation: comparison of treatment for low-back pain. *Pain*. 1980;9:209-217.

1407.   Vasudevan SV. Physical rehabilitation in managing pain. *Pain Clin Updates*. 1997;5.

1408.   Draper D, Trowbridge C. Continuous low-level heat therapy: what works, what doesn't. *Injury Manag Update*. 2003;8:46-48.

1409.   Lurie-Luke E, Neubauer G, Lindl C, Breitkreutz H, Fischer P, Hitzeroth S. An exploratory workplace study to investigate the perceived value of continuous low-level heatwrap therapy in manual workers. *Occup Med* 2003;53:173-178.

1410.   Nuhr M, Hoerauf K, Bertalanffy A, et al. Active warming during emergency transport relieves acute low back pain. *Spine*. 2004;29:1499-1503.

1411.   Lloyd A, Scott DA, Akehurst RL, Lurie-Luke E, Jessen G. Cost-effectiveness of low-level heat wrap therapy for low back pain. *Value Health*. 2004;7:413-422.

1412.   Mayer JM, Ralph L, Look M, et al. Treating acute low back pain with continuous low-level heat wrap therapy and/or exercise: a randomized controlled trial. *The spine journal : official journal of the North American Spine Society*. 2005;5:395-403.

1413.   Nadler SF, Steiner DJ, Erasala GN, Hengehold DA, Abeln SB, Weingand KW. Continuous low-level heatwrap therapy for treating acute nonspecific low back pain. *Archives of physical medicine and rehabilitation*. 2003;84:329-334.

1414.   Nadler SF, Steiner DJ, Petty SR, Erasala GN, Hengehold DA, Weingand KW. Overnight use of continuous low-level heatwrap therapy for relief of low back pain. *Arch Phys Med Rehabil*. 2003;84:335-342.

1415.   Tao XG, Bernacki EJ. A randomized clinical trial of continuous low-level heat therapy for acute muscular low back pain in the workplace. *Journal of occupational and environmental medicine / American College of Occupational and Environmental Medicine*. 2005;47:1298-1306.

1416.   Gale GD, Rothbart PJ, Li Y. Infrared therapy for chronic low back pain: a randomized, controlled trial. *Pain Res Manag*. 2006;11:193-196.

1417.	Garra G, Singer AJ, Leno R, et al. Heat or cold packs for neck and back strain: a randomized controlled trial of efficacy. *Academic emergency medicine : official journal of the Society for Academic Emergency Medicine*. 2010;17:484-489.
1418.	Constant F, Guillemin F, Collin JF, Boulange M. Use of spa therapy to improve the quality of life of chronic low back pain patients. *Med Care*. 1998;36:1309-1314.
1419.	Durmus D, Durmaz Y, Canturk F. Effects of therapeutic ultrasound and electrical stimulation program on pain, trunk muscle strength, disability, walking performance, quality of life, and depression in patients with low back pain: a randomized-controlled trial. *Rheumatology international*. 2010;30:901-910.
1420.	Kumar S, Negi MP, Sharma VP, Shukla R, Dev R, Mishra UK. Efficacy of two multimodal treatments on physical strength of occupationally subgrouped male with low back pain. *Journal of back and musculoskeletal rehabilitation*. 2009;22:179-188.
1421.	Mohseni-Bandpei MA, Fakhri M, Bagheri-Nesami M, Ahmad-Shirvani M, Khalilian AR, Shayesteh-Azar M. Occupational back pain in Iranian nurses: an epidemiological study. *Br J Nurs*. 2006;15:914-917.
1422.	Siems W, Bresgen N, Brenke R, et al. Pain and mobility improvement and MDA plasma levels in degenerative osteoarthritis, low back pain, and rheumatoid arthritis after infrared A-irradiation. *Acta biochimica Polonica*. 2010;57:313-319.
1423.	Cacolice P, Scibek J, Martin R. Diathermy: A literature review of current research and practices. *OPTP*. 2013;25:155-161.
1424.	Koes BW, Bouter LM, van Mameren H, et al. A randomized clinical trial of manual therapy and physiotherapy for persistent back and neck complaints: subgroup analysis and relationship between outcome measures. *Journal of manipulative and physiological therapeutics*. 1993;16:211-219.
1425.	Koes BW, Bouter LM, van Mameren H, et al. A blinded randomized clinical trial of manual therapy and physiotherapy for chronic back and neck complaints: physical outcome measures. *Journal of manipulative and physiological therapeutics*. 1992;15:16-23.
1426.	Koes BW, Bouter LM, van Mameren H, et al. The effectiveness of manual therapy, physiotherapy, and treatment by the general practitioner for nonspecific back and neck complaints. A randomized clinical trial. *Spine*. 1992;17:28-35.
1427.	Ahmed MS, Shakoor MA, Khan AA. Evaluation of the effects of shortwave diathermy in patients with chronic low back pain. *Bangladesh Medical Research Council bulletin*. 2009;35:18-20.
1428.	Beyerman KL, Palmerino MB, Zohn LE, Kane GM, Foster KA. Efficacy of treating low back pain and dysfunction secondary to osteoarthritis: chiropractic care compared with moist heat alone. *Journal of manipulative and physiological therapeutics*. 2006;29:107-114.
1429.	Gibson T, Grahame R, Harkness J, Woo P, Blagrave P, Hills R. Controlled comparison of short-wave diathermy treatment with osteopathic treatment in non-specific low back pain. *Lancet*. 1985;1:1258-1261.
1430.	Kettenmann B, Wille C, Lurie-Luke E, Walter D, Kobal G. Impact of continuous low level heatwrap therapy in acute low back pain patients: subjective and objective measurements. *Clin J Pain*. 2007;23:663-668.
1431.	Shakoor MA, Rahman MS, Moyeenuzzaman M. Effects of deep heat therapy on the patients with chronic low back pain. *Mymensingh medical journal : MMJ*. 2008;17:S32-38.
1432.	Glazov G, Schattner P, Lopez D, Shandley K. Laser acupuncture for chronic non-specific low back pain: a controlled clinical trial. *Acupunct Med*. 2009;27:94-100.
1433.	Güevenol K, Tüzün Ç, Peker Ö, Göktay Y. A comparison of inverted spinal traction and conventional traction in the treatment of lumbar disc herniations. *Physiother Theory Prac*. 2000;16:151-160.
1434.	Chou R, Huffman LH. Nonpharmacologic therapies for acute and chronic low back pain: a review of the evidence for an American Pain Society/American College of Physicians clinical practice guideline. *Ann Intern Med*. 2007;147:492-504.
1435.	Hicks GS, Duddleston DN, Russell LD, Holman HE, Shepherd JM, Brown CA. Low back pain. *The American journal of the medical sciences*. 2002;324:207-211.
1436.	Poitras S, Brosseau L. Evidence-informed management of chronic low back pain with transcutaneous electrical nerve stimulation, interferential current, electrical muscle stimulation, ultrasound, and thermotherapy. *The spine journal : official journal of the North American Spine Society*. 2008;8:226-233.
1437.	Seco J, Kovacs FM, Urrutia G. The efficacy, safety, effectiveness, and cost-effectiveness of ultrasound and shock wave therapies for low back pain: a systematic review. *The spine journal : official journal of the North American Spine Society*. 2011;11:966-977.
1438.	Morrisette DC, Brown D, Saladin ME. Temperature change in lumbar periarticular tissue with continuous ultrasound. *The Journal of orthopaedic and sports physical therapy*. 2004;34:754-760.
1439.	Robertson VJ, Baker KG. A review of therapeutic ultrasound: effectiveness studies. *Phys Ther*. 2001;81:1339-1350.
1440.	Ansari NN, Ebadi S, Talebian S, et al. A randomized, single blind placebo controlled clinical trial on the effect of continuous ultrasound on low back pain. *Electromyography and clinical neurophysiology*. 2006;46:329-336.

1441.   Licciardone JC, Minotti DE, Gatchel RJ, Kearns CM, Singh KP. Osteopathic manual treatment and ultrasound therapy for chronic low back pain: a randomized controlled trial. *Annals of family medicine*. 2013;11:122-129.

1442.   Blomberg S, Svardsudd K, Mildenberger F. A controlled, multicentre trial of manual therapy in low-back pain. Initial status, sick-leave and pain score during follow-up. *Scandinavian journal of primary health care*. 1992;10:170-178.

1443.   Ebadi S, Ansari NN, Naghdi S, et al. The effect of continuous ultrasound on chronic non-specific low back pain: a single blind placebo-controlled randomized trial. *BMC Musculoskelet Disord*. 2012;13:192.

1444.   Galiano K, Obwegeser A, Walch C, Schatzer R, Ploner F, Gruber H. Ultrasound-guided versus computed tomography-controlled facet joint injections in the lumbar spine: a prospective randomized clinical trial. *Regional anesthesia and pain medicine*. 2007;32:317-322.

1445.   Chon S-C, Chang K-Y, You J. Effect of the abdominal draw-in manoeuvre in combination with ankle dorsiflexion in strengthening the transverse abdominal muscle in healthy young adults: a preliminary, randomised, controlled study. *Physiother*. 2010;96:130-136.

1446.   Fitz-Ritson D. Lasers and their therapeutic applications in chiropractic. *J Canadian Chiropractic Assoc*. 2001;45:26-34.

1447.   Ay S, Dogan SK, Evcik D. Is low-level laser therapy effective in acute or chronic low back pain? *Clinical rheumatology*. 2010;29:905-910.

1448.   Basford JR, Sheffield CG, Harmsen WS. Laser therapy: a randomized, controlled trial of the effects of low-intensity Nd:YAG laser irradiation on musculoskeletal back pain. *Arch Phys Med Rehabil*. 1999;80:647-652.

1449.   Klein RG, Eek BC. Low-energy laser treatment and exercise for chronic low back pain: double-blind controlled trial. *Arch Phys Med Rehabil*. 1990;71:34-37.

1450.   Soriano F, Rios R. Gallium arsenide laser treatment of chronic low back pain: a prospective, randomized and double blind study. *Laser Ther* 1998;10::175-180.

1451.   Toya S. Report on a computer-randomized double blind clinical trial to determine the eddectiveness of the GaAlAs (830 nm) diode laser for pain attenuation in selected pain groups. *Laser Ther*. 1994;6:143-148.

1452.   Djavid GE, Mehrdad R, Ghasemi M, Hasan-Zadeh H, Sotoodeh-Manesh A, Pouryaghoub G. In chronic low back pain, low level laser therapy combined with exercise is more beneficial than exercise alone in the long term: a randomised trial. *The Australian journal of physiotherapy*. 2007;53:155-160.

1453.   Gur A, Karakoc M, Cevik R, Nas K, Sarac AJ, Karakoc M. Efficacy of low power laser therapy and exercise on pain and functions in chronic low back pain. *Lasers in surgery and medicine*. 2003;32:233-238.

1454.   Jovicic M, Konstantinovic L, Lazovic M, Jovicic V. Clinical and functional evaluation of patients with acute low back pain and radiculopathy treated with different energy doses of low level laser therapy. *Vojnosanitetski pregled Military-medical and pharmaceutical review*. 2012;69:656-662.

1455.   Berman BM, Langevin HM, Witt CM, Dubner R. Acupuncture for chronic low back pain. *The New England journal of medicine*. 2010;363:454-461.

1456.   Furlan AD, Yazdi F, Tsertsvadze A, et al. A systematic review and meta-analysis of efficacy, cost-effectiveness, and safety of selected complementary and alternative medicine for neck and low-back pain. *Evidence-based complementary and alternative medicine : eCAM*. 2012;2012:953139.

1457.   Henderson H. Acupuncture: evidence for its use in chronic low back pain. *Br J Nurs*. 2002;11:1395-1403.

1458.   Lewis C, Souvlis T, Sterling M. Sensory characteristics of tender points in the lower back. *Manual therapy*. 2010;15:451-456.

1459.   Sherman KJ, Cherkin DC, Hogeboom CJ. The diagnosis and treatment of patients with chronic low-back pain by traditional Chinese medical acupuncturists. *J Altern Complement Med*. 2001;7:641-650.

1460.   Strauss A. Acupuncture and the treatment of chronic low back pain: a review of the literature. *Chiro J Austral* 1999;29:213-218.

1461.   Xu M, Yan S, Yin X, et al. Acupuncture for chronic low back pain in long-term follow-up: a meta-analysis of 13 randomized controlled trials. *The American journal of Chinese medicine*. 2013;41:1-19.

1462.   Yuan J, Kerr D, Park J, Liu XH, McDonough S. Treatment regimens of acupuncture for low back pain--a systematic review. *Complementary therapies in medicine*. 2008;16:295-304.

1463.   Yuan J, Purepong N, Kerr DP, Park J, Bradbury I, McDonough S. Effectiveness of acupuncture for low back pain: a systematic review. *Spine*. 2008;33:E887-900.

1464.   MacPherson H, Mercer SW, Scullion T, Thomas KJ. Empathy, enablement, and outcome: an exploratory study on acupuncture patients' perceptions. *J Altern Complement Med*. 2003;9:869-876.

1465.   Brinkhaus B, Witt CM, Jena S, et al. Acupuncture in patients with chronic low back pain: a randomized controlled trial. *Archives of internal medicine*. 2006;166:450-457.

1466.   Furlan AD, van Tulder M, Cherkin D, et al. Acupuncture and dry-needling for low back pain: an updated systematic review within the framework of the cochrane collaboration. *Spine*. 2005;30:944-963.

1467.   van Tulder M, Cherkin D, Berman B, Lao L, Koes B. The effectiveness of acupuncture in the management of acute and chronic low back pain. A systematic review within the framework of the Cochrane Collaboration Back Review Group. *Spine*. 1999;24:1113-1123.

1468.   Cherkin DC, Sherman KJ, Avins AL, et al. A randomized trial comparing acupuncture, simulated acupuncture, and usual care for chronic low back pain. *Archives of internal medicine*. 2009;169:858-866.

1469.   Haake M, Muller HH, Schade-Brittinger C, et al. German Acupuncture Trials (GERAC) for chronic low back pain: randomized, multicenter, blinded, parallel-group trial with 3 groups. *Archives of internal medicine*. 2007;167:1892-1898.

1470.   Brinkhaus B, Witt CM, Jena S, et al. Interventions and physician characteristics in a randomized multicenter trial of acupuncture in patients with low-back pain. *J Altern Complement Med*. 2006;12:649-657.

1471.   Cho YJ, Song YK, Cha YY, et al. Acupuncture for chronic low back pain: a multicenter, randomized, patient-assessor blind, sham-controlled clinical trial. *Spine*. 2013;38:549-557.

1472.   Di Cesare A, Giombini A, Di Cesare M, Ripani M, Vulpiani MC, Saraceni VM. Comparison between the effects of trigger point mesotherapy versus acupuncture points mesotherapy in the treatment of chronic low back pain: a short term randomized controlled trial. *Complementary therapies in medicine*. 2011;19:19-26.

1473.   Inoue M, Kitakoji H, Ishizaki N, et al. Relief of low back pain immediately after acupuncture treatment--a randomised, placebo controlled trial. *Acupuncture in medicine : journal of the British Medical Acupuncture Society*. 2006;24:103-108.

1474.   Kennedy S, Baxter GD, Kerr DP, Bradbury I, Park J, McDonough SM. Acupuncture for acute non-specific low back pain: a pilot randomised non-penetrating sham controlled trial. *Complementary therapies in medicine*. 2008;16:139-146.

1475.   Leibing E, Leonhardt U, Koster G, et al. Acupuncture treatment of chronic low-back pain -- a randomized, blinded, placebo-controlled trial with 9-month follow-up. *Pain*. 2002;96:189-196.

1476.   Sherman KJ, Cherkin DC, Ichikawa L, et al. Treatment expectations and preferences as predictors of outcome of acupuncture for chronic back pain. *Spine (Phila Pa 1976)*. 2010;35:1471-1477.

1477.   Yuan J, Purepong N, Hunter RF, et al. Different frequencies of acupuncture treatment for chronic low back pain: an assessor-blinded pilot randomised controlled trial. *Complementary therapies in medicine*. 2009;17:131-140.

1478.   Garvey TA, Marks MR, Wiesel SW. A prospective, randomized, double-blind evaluation of trigger-point injection therapy for low-back pain. *Spine*. 1989;14:962-964.

1479.   Inoue M, Hojo T, Nakajima M, Kitakoji H, Itoi M. Comparison of the effectiveness of acupuncture treatment and local anaesthetic injection for low back pain: a randomised controlled clinical trial. *Acupuncture in medicine : journal of the British Medical Acupuncture Society*. 2009;27:174-177.

1480.   Itoh K, Itoh S, Katsumi Y, Kitakoji H. A pilot study on using acupuncture and transcutaneous electrical nerve stimulation to treat chronic non-specific low back pain. *Complementary therapies in clinical practice*. 2009;15:22-25.

1481.   Sator-Katzenschlager SM, Scharbert G, Kozek-Langenecker SA, et al. The short- and long-term benefit in chronic low back pain through adjuvant electrical versus manual auricular acupuncture. *Anesthesia and analgesia*. 2004;98:1359-1364, table of contents.

1482.   Thomas KJ, MacPherson H, Ratcliffe J, et al. Longer term clinical and economic benefits of offering acupuncture care to patients with chronic low back pain. *Health Technol Assess*. 2005;9:iii-iv, ix-x, 1-109.

1483.   Thomas KJ, MacPherson H, Thorpe L, et al. Randomised controlled trial of a short course of traditional acupuncture compared with usual care for persistent non-specific low back pain. *Bmj*. 2006;333:623.

1484.   Tsukayama H, Yamashita H, Amagai H, Tanno Y. Randomised controlled trial comparing the effectiveness of electroacupuncture and TENS for low back pain: a preliminary study for a pragmatic trial. *Acupunct Med*. 2002;20:175-180.

1485.   Wasan AD, Kong J, Pham LD, Kaptchuk TJ, Edwards R, Gollub RL. The impact of placebo, psychopathology, and expectations on the response to acupuncture needling in patients with chronic low back pain. *J Pain*. 2010;11:555-563.

1486.   Witt CM, Jena S, Selim D, et al. Pragmatic randomized trial evaluating the clinical and economic effectiveness of acupuncture for chronic low back pain. *American journal of epidemiology*. 2006;164:487-496.

1487.   Lehmann TR, Russell DW, Spratt KF. The impact of patients with nonorganic physical findings on a controlled trial of transcutaneous electrical nerve stimulation and electroacupuncture. *Spine*. 1983;8:625-634.

1488.   Lehmann TR, Russell DW, Spratt KF, et al. Efficacy of electroacupuncture and TENS in the rehabilitation of chronic low back pain patients. *Pain*. 1986;26:277-290.

1489.   Macdonald AJ, Macrae KD, Master BR, Rubin AP. Superficial acupuncture in the relief of chronic low back pain. *Annals of the Royal College of Surgeons of England*. 1983;65:44-46.

1490.   Mendelson G, Selwood TS, Kranz H, Loh TS, Kidson MA, Scott DS. Acupuncture treatment of chronic back pain. A double-blind placebo-controlled trial. *The American journal of medicine*. 1983;74:49-55.

1491.   Molsberger AF, Mau J, Pawelec DB, Winkler J. Does acupuncture improve the orthopedic management of chronic low back pain--a randomized, blinded, controlled trial with 3 months follow up. *Pain*. 2002;99:579-587.

1492. Vas J, Aranda JM, Modesto M, et al. Acupuncture in patients with acute low back pain: a multicentre randomised controlled clinical trial. *Pain*. 2012;153:1883-1889.
1493. Yeung CK, Leung MC, Chow DH. The use of electro-acupuncture in conjunction with exercise for the treatment of chronic low-back pain. *J Altern Complement Med*. 2003;9:479-490.
1494. Yun M. Hegu acupuncture for chronic low-back pain: a randomized controlled trial. *J Altern Complement Med* 2012;18:7.
1495. Carlsson CP, Sjolund BH. Acupuncture for chronic low back pain: a randomized placebo-controlled study with long-term follow-up. *Clin J Pain*. 2001;17:296-305.
1496. Grant DJ, Bishop-Miller J, Winchester DM, Anderson M, Faulkner S. A randomized comparative trial of acupuncture versus transcutaneous electrical nerve stimulation for chronic back pain in the elderly. *Pain*. 1999;82:9-13.
1497. Itoh K, Katsumi Y, Hirota S, Kitakoji H. Effects of trigger point acupuncture on chronic low back pain in elderly patients--a sham-controlled randomised trial. *Acupunct Med*. 2006;24:5-12.
1498. Itoh K, Katsumi Y, Kitakoji H. Trigger point acupuncture treatment of chronic low back pain in elderly patients--a blinded RCT. *Acupunct Med*. 2004;22:170-177.
1499. Coan RM, Wong G, Ku SL, et al. The acupuncture treatment of low back pain: a randomized controlled study. *The American journal of Chinese medicine*. 1980;8:181-189.
1500. Kerr DP, Walsh DM, Baxter D. Acupuncture in the management of chronic low back pain: a blinded randomized controlled trial. *Clin J Pain*. 2003;19:364-370.
1501. Lin ML, Lin MH, Fen JJ, Lin WT, Lin CW, Chen PQ. A comparison between pulsed radiofrequency and electro-acupuncture for relieving pain in patients with chronic low back pain. *Acupuncture & electro-therapeutics research*. 2010;35:133-146.
1502. Shankar N, Thakur M, Tandon OP, Saxena AK, Arora S, Bhattacharya N. Autonomic status and pain profile in patients of chronic low back pain and following electro acupuncture therapy: a randomized control trial. *Indian journal of physiology and pharmacology*. 2011;55:25-36.
1503. Glazov G. The influence of baseline characteristics on response to a laser acupuncture intervention: an exploratory analysis. *Acupunct Med*. 2010;28:6-11.
1504. Kovacs FM, Abraira V, Pozo F, et al. Local and remote sustained trigger point therapy for exacerbations of chronic low back pain. A randomized, double-blind, controlled, multicenter trial. *Spine*. 1997;22:786-797.
1505. Urrutia G, Burton AK, Morral A, Bonfill X, Zanoli G. Neuroreflexotherapy for non-specific low-back pain. *Cochrane Database Syst Rev*. 2004:CD003009.
1506. Urrutia G, Burton K, Morral A, Bonfill X, Zanoli G. Neuroreflexotherapy for nonspecific low back pain: a systematic review. *Spine*. 2005;30:E148-153.
1507. Corcoll J, Orfila J, Tobajas P, Alegre L. Implementation of neuroreflexotherapy for subacute and chronic neck and back pain within the Spanish public health system: audit results after one year. *Health policy (Amsterdam, Netherlands)*. 2006;79:345-357.
1508. Kovacs FM, Llobera J, Abraira V, Lazaro P, Pozo F, Kleinbaum D. Effectiveness and cost-effectiveness analysis of neuroreflexotherapy for subacute and chronic low back pain in routine general practice: a cluster randomized, controlled trial. *Spine*. 2002;27:1149-1159.
1509. Engers A, Jellema P, Wensing M, van der Windt DA, Grol R, van Tulder MW. Individual patient education for low back pain. *Cochrane Database Syst Rev*. 2008:CD004057.
1510. Facci LM, Nowotny JP, Tormem F, Trevisani VF. Effects of transcutaneous electrical nerve stimulation (TENS) and interferential currents (IFC) in patients with nonspecific chronic low back pain: randomized clinical trial. *Sao Paulo medical journal = Revista paulista de medicina*. 2011;129:206-216.
1511. Hurley DA, McDonough SM, Dempster M, Moore AP, Baxter GD. A randomized clinical trial of manipulative therapy and interferential therapy for acute low back pain. *Spine*. 2004;29:2207-2216.
1512. Hurley DA, Minder PM, McDonough SM, Walsh DM, Moore AP, Baxter DG. Interferential therapy electrode placement technique in acute low back pain: a preliminary investigation. *Arch Phys Med Rehabil*. 2001;82:485-493.
1513. Lara-Palomo IC, Aguilar-Ferrandiz ME, Mataran-Penarrocha GA, et al. Short-term effects of interferential current electro-massage in adults with chronic non-specific low back pain: a randomized controlled trial. *Clin Rehabil*. 2013;27:439-449.
1514. Zambito A, Bianchini D, Gatti D, Viapiana O, Rossini M, Adami S. Interferential and horizontal therapies in chronic low back pain: a randomized, double blind, clinical study. *Clinical and experimental rheumatology*. 2006;24:534-539.
1515. Zambito A, Bianchini D, Gatti D, Rossini M, Adami S, Viapiana O. Interferential and horizontal therapies in chronic low back pain due to multiple vertebral fractures: a randomized, double blind, clinical study. *Osteoporosis international : a journal*

established as result of cooperation between the European Foundation for Osteoporosis and the National Osteoporosis Foundation of the USA. 2007;18:1541-1545.

1516.   Brosseau L, Milne S, Robinson V, et al. Efficacy of the transcutaneous electrical nerve stimulation for the treatment of chronic low back pain: a meta-analysis. *Spine*. 2002;27:596-603.

1517.   Diamond S, Borenstein D. Chronic low back pain in a working-age adult. *Best practice & research*. 2006;20:707-720.

1518.   Dubinsky RM, Miyasaki J. Assessment: efficacy of transcutaneous electric nerve stimulation in the treatment of pain in neurologic disorders (an evidence-based review): report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. *Neurology*. 2010;74:173-176.

1519.   Gaid C. The role of transcutaneous electric nerve stimulation (TENS) for the management of chronic low back pain. *International Musculoskeletal Medicine*. 2009;31.

1520.   Khadilkar A, Odebiyi DO, Brosseau L, Wells GA. Transcutaneous electrical nerve stimulation (TENS) versus placebo for chronic low-back pain. *Cochrane Database Syst Rev*. 2008:CD003008.

1521.   Milne S, Welch V, Brosseau L, et al. Transcutaneous electrical nerve stimulation (TENS) for chronic low back pain. *Cochrane Database Syst Rev*. 2001:CD003008.

1522.   Pengel HM, Maher CG, Refshauge KM. Systematic review of conservative interventions for subacute low back pain. *Clin Rehabil*. 2002;16:811-820.

1523.   Gemignani G, Olivieri I, Ruju G, Pasero G. Transcutaneous electrical nerve stimulation in ankylosing spondylitis: a double-blind study. *Arthritis and rheumatism*. 1991;34:788-789.

1524.   Khadilkar A, Milne S, Brosseau L, et al. Transcutaneous electrical nerve stimulation (TENS) for chronic low-back pain. *Cochrane Database Syst Rev*. 2005:CD003008.

1525.   Richardson RR, Arbit J, Siqueira EB, Zagar R. Transcutaneous electrical neurostimulation in functional pain. *Spine*. 1981;6:185-188.

1526.   van Tulder MW, Koes B, Malmivaara A. Outcome of non-invasive treatment modalities on back pain: an evidence-based review. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2006;15 Suppl 1:S64-81.

1527.   Lake DA. Neuromuscular electrical stimulation. An overview and its application in the treatment of sports injuries. *Sports Med*. 1992;13:320-336.

1528.   Robinson AJ. Transcutaneous electrical nerve stimulation for the control of pain in musculoskeletal disorders. *The Journal of orthopaedic and sports physical therapy*. 1996;24:208-226.

1529.   Hsieh RL, Lee WC. One-shot percutaneous electrical nerve stimulation vs. transcutaneous electrical nerve stimulation for low back pain: comparison of therapeutic effects. *American journal of physical medicine & rehabilitation / Association of Academic Physiatrists*. 2002;81:838-843.

1530.   Bertalanffy A, Kober A, Bertalanffy P, et al. Transcutaneous electrical nerve stimulation reduces acute low back pain during emergency transport. *Academic emergency medicine : official journal of the Society for Academic Emergency Medicine*. 2005;12:607-611.

1531.   Jarzem PF, Harvey EJ, Arcaro N, Kaczorowski J. Transcutaneous Electrical Nerve Stimulation [TENS] for Short-Term Treatment of Low Back Pain-Randomized Double Blind Crossover Study of Sham versus Conventional TENS. *J Musculoskeletal Pain* 2005;13:11-17.

1532.   Bloodworth DM, Nguyen BN, Garver W, et al. Comparison of stochastic vs. conventional transcutaneous electrical stimulation for pain modulation in patients with electromyographically documented radiculopathy. *American journal of physical medicine & rehabilitation / Association of Academic Physiatrists*. 2004;83:584-591.

1533.   Al-Smadi J, Warke K, Wilson I, et al. A pilot investigation of the hypoalgesic effects of transcutaneous electrical nerve stimulation upon low back pain in people with multiple sclerosis. *Clin Rehabil*. 2003;17:742-749.

1534.   Buchmuller A, Navez M, Milletre-Bernardin M, et al. Value of TENS for relief of chronic low back pain with or without radicular pain. *Eur J Pain*. 2012;16:656-665.

1535.   Cheing GL, Hui-Chan CW. Transcutaneous electrical nerve stimulation: nonparallel antinociceptive effects on chronic clinical pain and acute experimental pain. *Arch Phys Med Rehabil*. 1999;80:305-312.

1536.   Gabis L, Shklar B, Baruch YK, Raz R, Gabis E, Geva D. Pain reduction using transcranial electrostimulation: a double blind "active placebo" controlled trial. *Journal of rehabilitation medicine*. 2009;41:256-261.

1537.   Gabis L, Shklar B, Geva D. Immediate influence of transcranial electrostimulation on pain and beta-endorphin blood levels: an active placebo-controlled study. *American journal of physical medicine & rehabilitation / Association of Academic Physiatrists*. 2003;82:81-85.

1538.   Ghoname ES, Craig WF, White PF, et al. The effect of stimulus frequency on the analgesic response to percutaneous electrical nerve stimulation in patients with chronic low back pain. *Anesthesia and analgesia*. 1999;88:841-846.

1539.   Herman E, Williams R, Stratford P, Fargas-Babjak A, Trott M. A randomized controlled trial of transcutaneous electrical nerve stimulation (CODETRON) to determine its benefits in a rehabilitation program for acute occupational low back pain. *Spine*. 1994;19:561-568.

1540.   Marchand S, Charest J, Li J, Chenard JR, Lavignolle B, Laurencelle L. Is TENS purely a placebo effect? A controlled study on chronic low back pain. *Pain*. 1993;54:99-106.

1541.   Thompson JW, Bower S, Tyrer SP. A double blind randomised controlled clinical trial on the effect of transcutaneous spinal electroanalgesia (TSE) on low back pain. *Eur J Pain*. 2008;12:371-377.

1542.   Thorsteinsson G, Stonnington HH, Stillwell GK, Elveback LR. Transcutaneous electrical stimulation: a double-blind trial of its efficacy for pain. *Arch Phys Med Rehabil*. 1977;58:8-13.

1543.   Barker KL, Elliott CJ, Sackley CM, Fairbank JC. Treatment of chronic back pain by sensory discrimination training. A Phase I RCT of a novel device (FairMed) vs. TENS. *BMC Musculoskelet Disord*. 2008;9:97.

1544.   Fox EJ, Melzack R. Transcutaneous electrical stimulation and acupuncture: comparison of treatment for low-back pain. *Pain*. 1976;2:141-148.

1545.   Jarzem P, et al. . Transcutaneous electrical nerve stimulation [TENS] for chronic low back pain. *J Musculoskel Pain*. 2005;13:3-9.

1546.   Kofotolis ND, Vlachopoulos SP, Kellis E. Sequentially allocated clinical trial of rhythmic stabilization exercises and TENS in women with chronic low back pain. *Clin Rehabil*. 2008;22:99-111.

1547.   Moore SR, Shurman J. Combined neuromuscular electrical stimulation and transcutaneous electrical nerve stimulation for treatment of chronic back pain: a double-blind, repeated measures comparison. *Arch Phys Med Rehabil*. 1997;78:55-60.

1548.   Shimoji K, Takahashi N, Nishio Y, Koyanagi M, Aida S. Pain relief by transcutaneous electric nerve stimulation with bidirectional modulated sine waves in patients with chronic back pain: a randomized, double-blind, sham-controlled study. *Neuromodulation*. 2007;10:42-51.

1549.   Yip YB, Tse HM, Wu KK. An experimental study comparing the effects of combined transcutaneous acupoint electrical stimulation and electromagnetic millimeter waves for spinal pain in Hong Kong. *Complementary therapies in clinical practice*. 2007;13:4-14.

1550.   Glaser JA, Baltz MA, Nietert PJ, Bensen CV. Electrical muscle stimulation as an adjunct to exercise therapy in the treatment of nonacute low back pain: a randomized trial. *J Pain*. 2001;2:295-300.

1551.   Presser M BJ, Aqdler R, Hanani A, Eisenberg E. Transcutaneous electrical nerve stimulation (TENS) during epidural steroids injection; A rondom. *The pain clinic* 2000;12:77-80.

1552.   Puranik S FJ, Paremain G, Kilminster S, Hughes D, Williams E. A randomized, single blind study to evaluate the effects of action potential stim. *Pain Clinic*. 2002;14:69-73.

1553.   Topuz O OE, Ozgen M, Ardic F. Efficacy of transcutaneous electrical nerve stimulation and percutaneous neuromodulation therapy in chronic low back pain. *J Back Musculoskeletal Rehabil*. 2004;17:127-133.

1554.   Warke K, Al-Smadi J, Baxter D, Walsh DM, Lowe-Strong AS. Efficacy of transcutaneous electrical nerve stimulation (tens) for chronic low-back pain in a multiple sclerosis population: a randomized, placebo-controlled clinical trial. *Clin J Pain*. 2006;22:812-819.

1555.   Yokoyama M, Sun X, Oku S, et al. Comparison of percutaneous electrical nerve stimulation with transcutaneous electrical nerve stimulation for long-term pain relief in patients with chronic low back pain. *Anesthesia and analgesia*. 2004;98:1552-1556, table of contents.

1556.   Ghoname EA, Craig WF, White PF, et al. Percutaneous electrical nerve stimulation for low back pain: a randomized crossover study. *Jama*. 1999;281:818-823.

1557.   Hamza MA, Ghoname EA, White PF, et al. Effect of the duration of electrical stimulation on the analgesic response in patients with low back pain. *Anesthesiology*. 1999;91:1622-1627.

1558.   White PF, Ghoname EA, Ahmed HE, Hamza MA, Craig WF, Vakharia AS. The effect of montage on the analgesic response to percutaneous neuromodulation therapy. *Anesthesia and analgesia*. 2001;92:483-487.

1559.   Pérez-Palomares S, Oliván-Blázquez B, Magallón-Botaya R, et al. Percutaneous electrical nerve stimulation versus dry needling: effectiveness in the treatment of chronic low back pain. *J Musculoskel Pain*. 2010;18:23-30.

1560.   Koopman JS, Vrinten DH, van Wijck AJ. Efficacy of microcurrent therapy in the treatment of chronic nonspecific back pain: a pilot study. *Clin J Pain*. 2009;25:495-499.

1561.   Prasad KS, Gregson BA, Hargreaves G, Byrnes T, Winburn P, Mendelow AD. Inversion therapy in patients with pure single level lumbar discogenic disease: a pilot randomized trial. *Disability and rehabilitation*. 2012;34:1473-1480.

1562.   Kim J-D, Oh H-W, Lee J-H, et al. The effect of inversion traction on pain sensation, lumbar flexibility and trunk muscles strength in patients with chronic low back pin. *Isokinetics and Exercise Science* 2013;21:237-246.

1563.   Quraishi NA. Transforaminal injection of corticosteroids for lumbar radiculopathy: systematic review and meta-analysis. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2012;21:214-219.

1564.   Radcliff K, Kepler C, Hilibrand A, et al. Epidural steroid injections are associated with less improvement in patients with lumbar spinal stenosis: a subgroup analysis of the Spine Patient Outcomes Research Trial. *Spine*. 2013;38:279-291.

1565.   Ranguis SC, Li D, Webster AC. Perioperative epidural steroids for lumbar spine surgery in degenerative spinal disease. A review. *Journal of neurosurgery Spine*. 2010;13:745-757.

1566.   Spaccarelli KC. Lumbar and caudal epidural corticosteroid injections. *Mayo Clinic proceedings Mayo Clinic*. 1996;71:169-178.

1567.   van Helvoirt H, Apeldoorn AT, Ostelo RW, et al. Transforaminal epidural steroid injections followed by mechanical diagnosis and therapy to prevent surgery for lumbar disc herniation. *Pain Med*. 2014;15:1100-1108.

1568.   Young IA, Hyman GS, Packia-Raj LN, Cole AJ. The use of lumbar epidural/transforaminal steroids for managing spinal disease. *The Journal of the American Academy of Orthopaedic Surgeons*. 2007;15:228-238.

1569.   Ammendolia C, Stuber KJ, Rok E, et al. Nonoperative treatment for lumbar spinal stenosis with neurogenic claudication. *Cochrane Database Syst Rev*. 2013;8:CD010712.

1570.   Banaszkiewicz PA, Kader D, Wardlaw D. The role of caudal epidural injections in the management of low back pain. *Bulletin*. 2003;61:127-131.

1571.   Bellini M, Barbieri M. Systemic effects of epidural steroid injections. *Anaesthesiology intensive therapy*. 2013;45:93-98.

1572.   Manchikanti L, Cash KA, McManus CD, Pampati V, Fellows B. Fluoroscopic caudal epidural injections with or without steroids in managing pain of lumbar spinal stenosis: one-year results of randomized, double-blind, active-controlled trial. *Journal of spinal disorders & techniques*. 2012;25:226-234.

1573.   May S, Comer C. Is surgery more effective than non-surgical treatment for spinal stenosis, and which non-surgical treatment is more effective? A systematic review. *Physiotherapy*. 2013;99:12-20.

1574.   McLain RF, Kapural L, Mekhail NA. Epidural steroids for back and leg pain: mechanism of action and efficacy. *Cleveland Clinic journal of medicine*. 2004;71:961-970.

1575.   Choi HJ, Hahn S, Kim CH, et al. Epidural steroid injection therapy for low back pain: a meta-analysis. *International journal of technology assessment in health care*. 2013;29:244-253.

1576.   Galhom AE, al-Shatouri MA. Efficacy of therapeutic fluoroscopy-guided lumbar spine interventional procedures. *Clinical imaging*. 2013;37:649-656.

1577.   Golish SR, Hanna LS, Bowser RP, Montesano PX, Carragee EJ, Scuderi GJ. Outcome of lumbar epidural steroid injection is predicted by assay of a complex of fibronectin and aggrecan from epidural lavage. *Spine*. 2011;36:1464-1469.

1578.   Slipman CW, Chow DW, Lenrow DA, Blaugrund JE, Chou LH. Dysphonia associated with epidural steroid injection: a case report. *Archives of physical medicine and rehabilitation*. 2002;83:1309-1310.

1579.   Spijker-Huiges A, Vermeulen K, Winters JC, van Wijhe M, van der Meer K. Epidural steroids for lumbosacral radicular syndrome compared to usual care: quality of life and cost utility in general practice. *Archives of physical medicine and rehabilitation*. 2015;96:381-387.

1580.   Stafford MA, Peng P, Hill DA. Sciatica: a review of history, epidemiology, pathogenesis, and the role of epidural steroid injection in management. *British journal of anaesthesia*. 2007;99:461-473.

1581.   Stout A. Discography. *Physical medicine and rehabilitation clinics of North America*. 2010;21:859-867.

1582.   Abdi S, Datta S, Trescot AM, et al. Epidural steroids in the management of chronic spinal pain: a systematic review. *Pain Physician*. 2007;10:185-212.

1583.   Boswell MV, Trescot AM, Datta S, et al. Interventional techniques: evidence-based practice guidelines in the management of chronic spinal pain. *Pain Physician*. 2007;10:7-111.

1584.   Gordon J. Caudal extradural injection for the treatment of low back pain. *Anaesthesia*. 1980;35:515-516.

1585.   Manchikanti L, Staats PS, Singh V, et al. Evidence-based practice guidelines for interventional techniques in the management of chronic spinal pain. *Pain Physician*. 2003;6:3-81.

1586.   Cannon DT, Aprill CN. Lumbosacral epidural steroid injections. *Arch Phys Med Rehabil*. 2000;81:S87-98; quiz S99-100.

1587.   Manchikanti L, Cash KA, Pampati V, McManus CD, Damron KS. Evaluation of fluoroscopically guided caudal epidural injections. *Pain Physician*. 2004;7:81-92.

1588.   Geurts JW, Kallewaard JW, Richardson J, Groen GJ. Targeted methylprednisolone acetate/hyaluronidase/clonidine injection after diagnostic epiduroscopy for chronic sciatica: a prospective, 1-year follow-up study. *Regional anesthesia and pain medicine*. 2002;27:343-352.

1589.   Botwin KP, Thomas S, Gruber RD, et al. Radiation exposure of the spinal interventionalist performing fluoroscopically guided lumbar transforaminal epidural steroid injections. *Arch Phys Med Rehabil*. 2002;83:697-701.

1590.   Lutze M, Stendel R, Vesper J, Brock M. Periradicular therapy in lumbar radicular syndromes: methodology and results. *Acta neurochirurgica*. 1997;139:719-724.

1591.   Haynsworth RF, Jr. Selective nerve root blocks: a new technique using electrical stimulation. *Pain Physician*. 2003;6:517-520.

1592.   Samanta A, Samanta J. Is epidural injection of steroids effective for low back pain? *BMJ*. 2004;328:1509-1510.

1593.   Arden NK, Price C, Reading I, et al. A multicentre randomized controlled trial of epidural corticosteroid injections for sciatica: the WEST study. *Rheumatology (Oxford)*. 2005;44:1399-1406.

1594.   Price C, Arden N, Coglan L, Rogers P. Cost-effectiveness and safety of epidural steroids in the management of sciatica. *Health Technol Assess*. 2005;9:1-58, iii.

1595.   Watts RW, Silagy CA. A meta-analysis on the efficacy of epidural corticosteroids in the treatment of sciatica. *Anaesthesia and intensive care*. 1995;23:564-569.

1596.   Karppinen J, Malmivaara A, Kurunlahti M, et al. Periradicular infiltration for sciatica: a randomized controlled trial. *Spine*. 2001;26:1059-1067.

1597.   Carette S, Leclaire R, Marcoux S, et al. Epidural corticosteroid injections for sciatica due to herniated nucleus pulposus. *N Engl J Med*. 1997;336:1634-1640.

1598.   Friedly JL, Comstock BA, Turner JA, et al. A randomized trial of epidural glucocorticoid injections for spinal stenosis. *The New England journal of medicine*. 2014;371:11-21.

1599.   Bush K, Cowan N, Katz DE, Gishen P. The natural history of sciatica associated with disc pathology. A prospective study with clinical and independent radiologic follow-up. *Spine*. 1992;17:1205-1212.

1600.   American Academy of Neurology. Guideline: Use of Epidural Steroid Injections to Treat Radicular Lumbosacral Pain. 2007.

1601.   Bogduk N, Cherry D. Epidural corticosteroid agents for sciatica. *The Medical journal of Australia*. 1985;143:402-406.

1602.   Botwin KP, Castellanos R, Rao S, et al. Complications of fluoroscopically guided interlaminar cervical epidural injections. *Archives of physical medicine and rehabilitation*. 2003;84:627-633.

1603.   Butler SH. Primum non nocere--first do no harm. *Pain*. 2005;116:175-176.

1604.   Gaul C, Neundorfer B, Winterholler M. Iatrogenic (para-) spinal abscesses and meningitis following injection therapy for low back pain. *Pain*. 2005;116:407-410.

1605.   Nelemans PJ, de Bie RA, de Vet HC, Sturmans F. Injection therapy for subacute and chronic benign low back pain. *The Cochrane database of systematic reviews*. 2000:CD001824.

1606.   Weinstein SM, Herring SA, Nass. Lumbar epidural steroid injections. *The spine journal : official journal of the North American Spine Society*. 2003;3:37S-44S.

1607.   Kay J, Findling J, Raff H. Epidural triamcinolone suppresses the pituitary-adrenal axis in human subjects. *Anesthesia and analgesia*. 1994;79:501-505.

1608.   Hopwood MB, Abram SE. Factors associated with failure of lumbar epidural steroids. *Regional anesthesia*. 1993;18:238-243.

1609.   Fukusaki M, Kobayashi I, Hara T, Sumikawa K. Symptoms of spinal stenosis do not improve after epidural steroid injection. *The Clinical journal of pain*. 1998;14:148-151.

1610.   Wilson-MacDonald J, Burt G, Griffin D, Glynn C. Epidural steroid injection for nerve root compression. A randomised, controlled trial. *J Bone Joint Surg Br*. 2005;87:352-355.

1611.   Dashfield AK, Taylor MB, Cleaver JS, Farrow D. Comparison of caudal steroid epidural with targeted steroid placement during spinal endoscopy for chronic sciatica: a prospective, randomized, double-blind trial. *British journal of anaesthesia*. 2005;94:514-519.

1612.   Jamison RN, VadeBoncouer T, Ferrante FM. Low back pain patients unresponsive to an epidural steroid injection: identifying predictive factors. *The Clinical journal of pain*. 1991;7:311-317.

1613.   Inman SL, Faut-Callahan M, Swanson BA, Fillingim RB. Sex differences in responses to epidural steroid injection for low back pain. *The journal of pain : official journal of the American Pain Society*. 2004;5:450-457.

1614.   Buttermann GR. Lumbar disc herniation regression after successful epidural steroid injection. *Journal of spinal disorders & techniques*. 2002;15:469-476.

1615.   Kimura S, Ohtori S, Orita S, et al. Injection of bupivacaine into disc space to detect painful nonunion after anterior lumbar interbody fusion (ALIF) surgery in patients with discogenic low back pain. *Yonsei medical journal*. 2014;55:487-492.

1616.   Ohtori S, Miyagi M, Eguchi Y, et al. Epidural administration of spinal nerves with the tumor necrosis factor-alpha inhibitor, etanercept, compared with dexamethasone for treatment of sciatica in patients with lumbar spinal stenosis: a prospective randomized study. *Spine (Phila Pa 1976)*. 2012;37:439-444.

1617.   Park Y, Lee JH, Park KD, Ahn JK, Park J, Jee H. Ultrasound-guided vs. fluoroscopy-guided caudal epidural steroid injection for the treatment of unilateral lower lumbar radicular pain: a prospective, randomized, single-blind clinical study. *American journal of physical medicine & rehabilitation / Association of Academic Physiatrists*. 2013;92:575-586.

1618.   Gallucci M, Limbucci N, Zugaro L, et al. Sciatica: treatment with intradiscal and intraforaminal injections of steroid and oxygen-ozone versus steroid only. *Radiology*. 2007;242:907-913.

1619.   Khot A, Bowditch M, Powell J, Sharp D. The use of intradiscal steroid therapy for lumbar spinal discogenic pain: a randomized controlled trial. *Spine*. 2004;29:833-836; discussion 837.

1620.   Simmons JW, McMillin JN, Emery SF, Kimmich SJ. Intradiscal steroids. A prospective double-blind clinical trial. *Spine*. 1992;17:S172-175.

1621.   Cao P, Jiang L, Zhuang C, et al. Intradiscal injection therapy for degenerative chronic discogenic low back pain with end plate Modic changes. *The spine journal : official journal of the North American Spine Society*. 2011;11:100-106.

1622.   Candido KD, Raghavendra MS, Chinthagada M, Badiee S, Trepashko DW. A prospective evaluation of iodinated contrast flow patterns with fluoroscopically guided lumbar epidural steroid injections: the lateral parasagittal interlaminar epidural approach versus the transforaminal epidural approach. *Anesthesia and analgesia*. 2008;106:638-644, table of contents.

1623.   Rauck RL, Eisenach JC, Jackson K, Young LD, Southern J. Epidural clonidine treatment for refractory reflex sympathetic dystrophy. *Anesthesiology*. 1993;79:1163-1169; discussion 1127A.

1624.   Burgher AH, Hoelzer BC, Schroeder DR, Wilson GA, Huntoon MA. Transforaminal epidural clonidine versus corticosteroid for acute lumbosacral radiculopathy due to intervertebral disc herniation. *Spine (Phila Pa 1976)*. 2011;36:E293-300.

1625.   Naja Z, Al-Tannir M, El-Rajab M, et al. The effectiveness of clonidine-bupivacaine repeated nerve stimulator-guided injection in piriformis syndrome. *Clin J Pain*. 2009;25:199-205.

1626.   Reuben SS, Rosenthal EA, Steinberg RB, Faruqi S, Kilaru PA. Surgery on the affected upper extremity of patients with a history of complex regional pain syndrome: the use of intravenous regional anesthesia with clonidine. *Journal of clinical anesthesia*. 2004;16:517-522.

1627.   Hoogland T, Schubert M, Miklitz B, Ramirez A. Transforaminal posterolateral endoscopic discectomy with or without the combination of a low-dose chymopapain: a prospective randomized study in 280 consecutive cases. *Spine*. 2006;31:E890-897.

1628.   Kim YS, Chin DK, Yoon DH, Jin BH, Cho YE. Predictors of successful outcome for lumbar chemonucleolysis: analysis of 3000 cases during the past 14 years. *Neurosurgery*. 2002;51:S123-128.

1629.   Revel M, Payan C, Vallee C, et al. Automated percutaneous lumbar discectomy versus chemonucleolysis in the treatment of sciatica. A randomized multicenter trial. *Spine*. 1993;18:1-7.

1630.   Bromley JW, Varma AO, Santoro AJ, Cohen P, Jacobs R, Berger L. Double-blind evaluation of collagenase injections for herniated lumbar discs. *Spine*. 1984;9:486-488.

1631.   Gibson JN, Waddell G. Surgical interventions for lumbar disc prolapse: updated Cochrane Review. *Spine*. 2007;32:1735-1747.

1632.   Han SC, Harrison P. Myofascial pain syndrome and trigger-point management. *Regional anesthesia*. 1997;22:89-101.

1633.   Klippel J, (ed). *Primer on the Rheumatic Diseases, 12th edition*. Atlanta: Arthritis Foundation; 2001.

1634.   Centre for Reviews and Dissemination. *Effective Health Care Bulletin: Acute and chronic low back pain*; 2000.

1635.   Porta M. A comparative trial of botulinum toxin type A and methylprednisolone for the treatment of myofascial pain syndrome and pain from chronic muscle spasm. *Pain*. 2000;85:101-105.

1636.   Hameroff SR, Crago BR, Blitt CD, Womble J, Kanel J. Comparison of bupivacaine, etidocaine, and saline for trigger-point therapy. *Anesthesia and analgesia*. 1981;60:752-755.

1637.   Collee G, Dijkmans BA, Vandenbroucke JP, Cats A. Iliac crest pain syndrome in low back pain. A double blind, randomized study of local injection therapy. *The Journal of rheumatology*. 1991;18:1060-1063.

1638.   Sonne M, Christensen K, Hansen SE, Jensen EM. Injection of steroids and local anaesthetics as therapy for low-back pain. *Scandinavian journal of rheumatology*. 1985;14:343-345.

1639.   Ikegami S, Kamimura M, Uchiyama S, et al. Anti-nociceptive effects of elcatonin injection for postmenopausal women with back pain: a randomized controlled trial. *The open orthopaedics journal*. 2010;4:132-136.

1640.   Vad VB, Bhat AL, Lutz GE, Cammisa F. Transforaminal epidural steroid injections in lumbosacral radiculopathy: a prospective randomized study. *Spine (Phila Pa 1976)*. 2002;27:11-16.

1641.   Datta S, Lee M, Falco FJ, Bryce DA, Hayek SM. Systematic assessment of diagnostic accuracy and therapeutic utility of lumbar facet joint interventions. *Pain Physician*. 2009;12:437-460.

1642.   Falco FJ, Manchikanti L, Datta S, et al. An update of the effectiveness of therapeutic lumbar facet joint interventions. *Pain Physician*. 2012;15:E909-953.

1643.	Jackson RP. The facet syndrome. Myth or reality? *Clinical orthopaedics and related research*. 1992:110-121.

1644.	Kuukkanen TM, Malkia EA. An experimental controlled study on postural sway and therapeutic exercise in subjects with low back pain. *Clinical rehabilitation*. 2000;14:192-202.

1645.	Chou R, Atlas SJ, Stanos SP, Rosenquist RW. Nonsurgical interventional therapies for low back pain: a review of the evidence for an American Pain Society clinical practice guideline. *Spine*. 2009;34:1078-1093.

1646.	Pampati S, Cash KA, Manchikanti L. Accuracy of diagnostic lumbar facet joint nerve blocks: a 2-year follow-up of 152 patients diagnosed with controlled diagnostic blocks. *Pain physician*. 2009;12:855-866.

1647.	Wynne KA. Facet joint injections in the management of chronic low back pain: a review. *Pain Reviews*. 2002;9:81-86.

1648.	Manchikanti L, Singh V, Falco FJ, Cash KA, Fellows B. Comparative outcomes of a 2-year follow-up of cervical medial branch blocks in management of chronic neck pain: a randomized, double-blind controlled trial. *Pain physician*. 2010;13:437-450.

1649.	Manchikanti L, Singh V, Falco FJ, Cash KA, Pampati V. Evaluation of lumbar facet joint nerve blocks in managing chronic low back pain: a randomized, double-blind, controlled trial with a 2-year follow-up. *Int J Med Sci*. 2010;7:124-135.

1650.	Datta S, Manchikanti L, Falco FJ, et al. Diagnostic utility of selective nerve root blocks in the diagnosis of lumbosacral radicular pain: systematic review and update of current evidence. *Pain physician*. 2013;16:SE97-124.

1651.	Dreyfuss PH, Dreyer SJ. Lumbar zygapophysial (facet) joint injections. *The spine journal : official journal of the North American Spine Society*. 2003;3:50S-59S.

1652.	Dreyer SJ, Dreyfuss PH. Low back pain and the zygapophysial (facet) joints. *Arch Phys Med Rehabil*. 1996;77:290-300.

1653.	Huston CW, Slipman CW. Diagnostic selective nerve root blocks: indications and usefulness. *Physical medicine and rehabilitation clinics of North America*. 2002;13:545-565.

1654.	el-Khoury GY, Renfrew DL. Percutaneous procedures for the diagnosis and treatment of lower back pain: diskography, facet-joint injection, and epidural injection. *AJR American journal of roentgenology*. 1991;157:685-691.

1655.	Falco FJ, Irwin L, Zhu J. Lumbar spine injection and interventional procedures in the management of low back pain. *Clinics in occupational and environmental medicine*. 2006;5:655-702, vii-viii.

1656.	Birkenmaier C, Veihelmann A, Trouillier HH, Hausdorf J, von Schulze Pellengahr C. Medial branch blocks versus pericapsular blocks in selecting patients for percutaneous cryodenervation of lumbar facet joints. *Reg Anesth Pain Med*. 2007;32:27-33.

1657.	Lilius G, Laasonen EM, Myllynen P, Harilainen A, Gronlund G. Lumbar facet joint syndrome. A randomised clinical trial. *The Journal of bone and joint surgery*. 1989;71:681-684.

1658.	Marks RC, Houston T, Thulbourne T. Facet joint injection and facet nerve block: a randomised comparison in 86 patients with chronic low back pain. *Pain*. 1992;49:325-328.

1659.	Schutz U, Cakir B, Dreinhofer K, Richter M, Koepp H. Diagnostic value of lumbar facet joint injection: a prospective triple cross-over study. *PLoS One*. 2011;6:e27991.

1660.	Revel M, Poiraudeau S, Auleley GR, et al. Capacity of the clinical picture to characterize low back pain relieved by facet joint anesthesia. Proposed criteria to identify patients with painful facet joints. *Spine*. 1998;23:1972-1976; discussion 1977.

1661.	North RB, Kidd DH, Zahurak M, Piantadosi S. Specificity of diagnostic nerve blocks: a prospective, randomized study of sciatica due to lumbosacral spine disease. *Pain*. 1996;65:77-85.

1662.	Manchikanti L, Damron K, Cash K, Manchukonda R, Pampati V. Therapeutic cervical medial branch blocks in managing chronic neck pain: a preliminary report of a randomized, double-blind, controlled trial: clinical trial NCT0033272. *Pain Physician*. 2006;9:333-346.

1663.	Schulte TL, Pietila TA, Heidenreich J, Brock M, Stendel R. Injection therapy of lumbar facet syndrome: a prospective study. *Acta neurochirurgica*. 2006;148:1165-1172; discussion 1172.

1664.	Bogduk N. A narrative review of intra-articular corticosteroid injections for low back pain. *Pain Med*. 2005;6:287-296.

1665.	Stojanovic MP, Dey D, Hord ED, Zhou Y, Cohen SP. A prospective crossover comparison study of the single-needle and multiple-needle techniques for facet-joint medial branch block. *Reg Anesth Pain Med*. 2005;30:484-490.

1666.	Manchikanti L, Singh V, Falco FJ, Cash KM, Fellows B. Cervical medial branch blocks for chronic cervical facet joint pain: a randomized, double-blind, controlled trial with one-year follow-up. *Spine*. 2008;33:1813-1820.

1667.	Murata Y, Kato Y, Miyamoto K, Takahashi K. Clinical study of low back pain and radicular pain pathways by using l2 spinal nerve root infiltration: a randomized, controlled, clinical trial. *Spine*. 2009;34:2008-2013.

1668.	Manchikanti L, Singh V, Falco FJ, Cash KA, Pampati V, Fellows B. The role of thoracic medial branch blocks in managing chronic mid and upper back pain: a randomized, double-blind, active-control trial with a 2-year followup. *Anesthesiology research and practice*. 2012.

1669.	Carette S, Marcoux S, Truchon R, et al. A controlled trial of corticosteroid injections into facet joints for chronic low back pain. *N Engl J Med*. 1991;325:1002-1007.

1670.   de Campos TF. Low back pain and sciatica in over 16s: assessment and management NICE Guideline [NG59]. *Journal of physiotherapy*. 2017;63:120.

1671.   Chou R, Loeser JD, Owens DK, et al. Interventional therapies, surgery, and interdisciplinary rehabilitation for low back pain: an evidence-based clinical practice guideline from the American Pain Society. *Spine (Phila Pa 1976)*. 2009;34:1066-1077.

1672.   Fuchs S, Erbe T, Fischer HL, Tibesku CO. Intraarticular hyaluronic acid versus glucocorticoid injections for nonradicular pain in the lumbar spine. *J Vasc Interv Radiol*. 2005;16:1493-1498.

1673.   Rupert MP, Lee M, Manchikanti L, Datta S, Cohen SP. Evaluation of sacroiliac joint interventions: a systematic appraisal of the literature. *Pain physician*. 2009;12:399-418.

1674.   Fortin JD, Aprill CN, Ponthieux B, Pier J. Sacroiliac joint: pain referral maps upon applying a new injection/arthrography technique. Part II: Clinical evaluation. *Spine*. 1994;19:1483-1489.

1675.   Bollow M, Braun J, Taupitz M, et al. CT-guided intraarticular corticosteroid injection into the sacroiliac joints in patients with spondyloarthropathy: indication and follow-up with contrast-enhanced MRI. *Journal of computer assisted tomography*. 1996;20:512-521.

1676.   Levin JH. Prospective, double-blind, randomized placebo-controlled trials in interventional spine: what the highest quality literature tells us. *The spine journal : official journal of the North American Spine Society*. 2009;9:690-703.

1677.   Sadreddini S, Noshad H, Molaeefard M, Ardalan MR, Ghojazadeh M, Shakouri SK. Unguided sacroiliac injection: effect on refractory buttock pain in patients with spondyloarthropathies. *Presse Med*. 2009;38:710-716.

1678.   Hanly JG, Mitchell M, MacMillan L, Mosher D, Sutton E. Efficacy of sacroiliac corticosteroid injections in patients with inflammatory spondyloarthropathy: results of a 6 month controlled study. *The Journal of rheumatology*. 2000;27:719-722.

1679.   Hansen H, Manchikanti L, Simopoulos TT, et al. A systematic evaluation of the therapeutic effectiveness of sacroiliac joint interventions. *Pain physician*. 2012;15:E247-278.

1680.   Luukkainen RK, Wennerstrand PV, Kautiainen HH, Sanila MT, Asikainen EL. Efficacy of periarticular corticosteroid treatment of the sacroiliac joint in non-spondylarthropathic patients with chronic low back pain in the region of the sacroiliac joint. *Clinical and experimental rheumatology*. 2002;20:52-54.

1681.   Hansen HC. Is fluoroscopy necessary for sacroiliac joint injections? *Pain Physician*. 2003;6:155-158.

1682.   Boezaart AP, Eksteen JA, Spuy GV, Rossouw P, Knipe M. Intrathecal morphine. Double-blind evaluation of optimal dosage for analgesia after major lumbar spinal surgery. *Spine*. 1999;24:1131-1137.

1683.   Chan JH, Heilpern GN, Packham I, Trehan RK, Marsh GD, Knibb AA. A prospective randomized double-blind trial of the use of intrathecal fentanyl in patients undergoing lumbar spinal surgery. *Spine*. 2006;31:2529-2533.

1684.   France JC, Jorgenson SS, Lowe TG, Dwyer AP. The use of intrathecal morphine for analgesia after posterolateral lumbar fusion: a prospective, double-blind, randomized study. *Spine*. 1997;22:2272-2277.

1685.   O'Neill P, Knickenberg C, Bogahalanda S, Booth AE. Use of intrathecal morphine for postoperative pain relief following lumbar spine surgery. *J Neurosurg*. 1985;63:413-416.

1686.   Coffey RJ, Owens ML, Broste SK, et al. Mortality associated with implantation and management of intrathecal opioid drug infusion systems to treat noncancer pain. *Anesthesiology*. 2009;111:881-891.

1687.   Deer T, Chapple I, Classen A, et al. Intrathecal drug delivery for treatment of chronic low back pain: report from the National Outcomes Registry for Low Back Pain. *Pain Med*. 2004;5:6-13.

1688.   Lemming D, Sorensen J, Graven-Nielsen T, Lauber R, Arendt-Nielsen L, Gerdle B. Managing chronic whiplash associated pain with a combination of low-dose opioid (remifentanil) and NMDA-antagonist (ketamine). *Eur J Pain*. 2007;11:719-732.

1689.   Turner JA, Sears JM, Loeser JD. Programmable intrathecal opioid delivery systems for chronic noncancer pain: a systematic review of effectiveness and complications. *Clin J Pain*. 2007;23:180-195.

1690.   Lemming D, Sorensen J, Graven-Nielsen T, Arendt-Nielsen L, Gerdle B. The responses to pharmacological challenges and experimental pain in patients with chronic whiplash-associated pain. *The Clinical journal of pain*. 2005;21:412-421.

1691.   Deer TR, Levy R, Prager J, et al. Polyanalgesic Consensus Conference--2012: recommendations to reduce morbidity and mortality in intrathecal drug delivery in the treatment of chronic pain. *Neuromodulation : journal of the International Neuromodulation Society*. 2012;15:467-482; discussion 482.

1692.   Miele VJ, Price KO, Bloomfield S, Hogg J, Bailes JE. A review of intrathecal morphine therapy related granulomas. *Eur J Pain*. 2006;10:251-261.

1693.   Rauck RL, Wallace MS, Leong MS, et al. A randomized, double-blind, placebo-controlled study of intrathecal ziconotide in adults with severe chronic pain. *J Pain Symptom Manage*. 2006;31:393-406.

1694.   Hayek SM, Deer TR, Pope JE, Panchal SJ, Patel VB. Intrathecal therapy for cancer and non-cancer pain. *Pain physician*. 2011;14:219-248.

1695.   Raffaeli W, Marconi G, Fanelli G, Taddei S, Borghi GB, Casati A. Opioid-related side-effects after intrathecal morphine: a prospective, randomized, double-blind dose-response study. *European journal of anaesthesiology*. 2006;23:605-610.

1696.   Dechow E, Davies RK, Carr AJ, Thompson PW. A randomized, double-blind, placebo-controlled trial of sclerosing injections in patients with chronic low back pain. *Rheumatology (Oxford)*. 1999;38:1255-1259.

1697.   Feldman JB. The prevention of occupational low back pain disability: evidence-based reviews point in a new direction. *Journal of surgical orthopaedic advances*. 2004;13:1-14.

1698.   Hooper RA, Ding M. Retrospective case series on patients with chronic spinal pain treated with dextrose prolotherapy. *J Altern Complement Med*. 2004;10:670-674.

1699.   Kim SR, Stitik TP, Foye PM, Greenwald BD, Campagnolo DI. Critical review of prolotherapy for osteoarthritis, low back pain, and other musculoskeletal conditions: a physiatric perspective. *American journal of physical medicine & rehabilitation / Association of Academic Physiatrists*. 2004;83:379-389.

1700.   Rabago D, Slattengren A, Zgierska A. Prolotherapy in primary care practice. *Primary care*. 2010;37:65-80.

1701.   van Tulder MW, Koes B, Seitsalo S, Malmivaara A. Outcome of invasive treatment modalities on back pain and sciatica: an evidence-based review. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2006;15 Suppl 1:S82-92.

1702.   Dagenais S, Haldeman S, Wooley JR. Intraligamentous injection of sclerosing solutions (prolotherapy) for spinal pain: a critical review of the literature. *The spine journal : official journal of the North American Spine Society*. 2005;5:310-328.

1703.   Fonstad P. Prolotherapy in the treatment of chronic low back pain: a literature review. *J Manual Manipulative Ther*. 2005;13:27-34.

1704.   Dagenais S, Yelland MJ, Del Mar C, Schoene ML. Prolotherapy injections for chronic low-back pain. *Cochrane Database Syst Rev*. 2007:CD004059.

1705.   Rabago D, Best TM, Beamsley M, Patterson J. A systematic review of prolotherapy for chronic musculoskeletal pain. *Clin J Sport Med*. 2005;15:376-380.

1706.   Yelland MJ, Glasziou P, Bogduk N, Schluter PJ, McKernon M. Prolotherapy injections, saline injections, and exercises for chronic low-back pain: a randomized trial. *Spine*. 2003;29:9-16.

1707.   Pach D, Brinkhaus B, Roll S, et al. Efficacy of injections with Disci/Rhus toxicodendron compositum for chronic low back pain--a randomized placebo-controlled trial. *PLoS One*. 2011;6:e26166.

1708.   Klein RG, Eek BC, DeLong WB, Mooney V. A randomized double-blind trial of dextrose-glycerine-phenol injections for chronic, low back pain. *Journal of spinal disorders*. 1993;6:23-33.

1709.   Yelland MJ, Mar C, Pirozzo S, Schoene ML, Vercoe P. Prolotherapy injections for chronic low-back pain. *Cochrane Database Syst Rev*. 2004:CD004059.

1710.   Kim WM, Lee HG, Jeong CW, Kim CM, Yoon MH. A randomized controlled trial of intra-articular prolotherapy versus steroid injection for sacroiliac joint pain. *J Altern Complement Med*. 2010;16:1285-1290.

1711.   Gobel H, Heinze A, Reichel G, Hefter H, Benecke R. Efficacy and safety of a single botulinum type A toxin complex treatment (Dysport) for the relief of upper back myofascial pain syndrome: results from a randomized double-blind placebo-controlled multicentre study. *Pain*. 2006;125:82-88.

1712.   Difazio M, Jabbari B. A focused review of the use of botulinum toxins for low back pain. *Clin J Pain*. 2002;18:S155-162.

1713.   Ney JP, Difazio M, Sichani A, Monacci W, Foster L, Jabbari B. Treatment of chronic low back pain with successive injections of botulinum toxin a over 6 months: a prospective trial of 60 patients. *Clin J Pain*. 2006;22:363-369.

1714.   De Andres J, Cerda-Olmedo G, Valia JC, Monsalve V, Lopez A, Minguez A. Use of botulinum toxin in the treatment of chronic myofascial pain. *Clin J Pain*. 2003;19:269-275.

1715.   Porta M, Maggioni G. Botulinum toxin (BoNT) and back pain. *J Neurol*. 2004;251 Suppl 1:I15-18.

1716.   Gallien P, Nicolas B, Petrilli S, et al. Role for botulinum toxin in back pain treatment in adults with cerebral palsy: report of a case. *Joint, bone, spine : revue du rhumatisme*. 2004;71:76-78.

1717.   Jabbari B, Ney J, Sichani A, Monacci W, Foster L, Difazio M. Treatment of refractory, chronic low back pain with botulinum neurotoxin A: an open-label, pilot study. *Pain Med*. 2006;7:260-264.

1718.   Nagarajan V, Al-Shubaili A, Ayad YM, Alexander J, Al-Ramezi K. Low back ache treatment with botulinum neurotoxin type A. Local experience in Kuwait. *Med Princ Pract*. 2007;16:181-186.

1719.   Argoff CE. The use of botulinum toxins for chronic pain and headaches. *Current treatment options in neurology*. 2003;5:483-492.

1720.   De Andres J, Adsuara VM, Palmisani S, Villanueva V, Lopez-Alarcon MD. A double-blind, controlled, randomized trial to evaluate the efficacy of botulinum toxin for the treatment of lumbar myofascial pain in humans. *Reg Anesth Pain Med*. 2010;35:255-260.

1721.	Herskowitz A, Herskowitz B. Treatment of neck and shoulder pain with botulinum neurotoxins. *Pain Pract*. 2004;4 Suppl 1:S27-37.

1722.	Jabbari B. Treatment of chronic low back pain with botulinum neurotoxins. *Current pain and headache reports*. 2007;11:352-358.

1723.	Lang AM. Botulinum toxin type B in piriformis syndrome. *American journal of physical medicine & rehabilitation / Association of Academic Physiatrists*. 2004;83:198-202.

1724.	Waseem Z, Boulias C, Gordon A, Ismail F, Sheean G, Furlan AD. Botulinum toxin injections for low-back pain and sciatica. *The Cochrane database of systematic reviews*. 2011:CD008257.

1725.	Foster L, Clapp L, Erickson M, Jabbari B. Botulinum toxin A and chronic low back pain: a randomized, double-blind study. *Neurology*. 2001;56:1290-1293.

1726.	Moghtaderi A, Loghmani A. Efficacy of botulinum toxin type A for treating chronic low back pain. *Anesth Pain*. 2011;1:77-80.

1727.	Fishman LM, Anderson C, Rosner B. BOTOX and physical therapy in the treatment of piriformis syndrome. *American journal of physical medicine & rehabilitation / Association of Academic Physiatrists*. 2002;81:936-942.

1728.	Chua NH, Vissers KC, Sluijter ME. Pulsed radiofrequency treatment in interventional pain management: mechanisms and potential indications-a review. *Acta neurochirurgica*. 2011;153:763-771.

1729.	Dobrogowski J, Wrzosek A, Wordliczek J. Radiofrequency denervation with or without addition of pentoxifylline or methylprednisolone for chronic lumbar zygapophysial joint pain. *Pharmacological reports : PR*. 2005;57:475-480.

1730.	Niemisto L, Jousimaa J, Hurri H, Kalso E, Malmivaara A. Radiofrequency denervation for chronic low-back pain (Protocol). *Cochrane Database of Systematic Reviews*. 2010:Art. No.: CD008572.

1731.	Racz GB, Ruiz-Lopez R. Radiofrequency procedures. *Pain practice : the official journal of World Institute of Pain*. 2006;6:46-50.

1732.	van Boxem K, van Eerd M, Brinkhuizen T, Patijn J, van Kleef M, van Zundert J. Radiofrequency and pulsed radiofrequency treatment of chronic pain syndromes: the available evidence. *Pain practice : the official journal of World Institute of Pain*. 2008;8:385-393.

1733.	Van Zundert J, Raj P, Erdine S, Van Kleef M. Application of radiofrequency treatment in practical pain management: state of the art. *Pain Prac*. 2002;2:269-278.

1734.	Slipman CW, Bhat AL, Gilchrist RV, Issac Z, Chou L, Lenrow DA. A critical review of the evidence for the use of zygapophysial injections and radiofrequency denervation in the treatment of low back pain. *The spine journal : official journal of the North American Spine Society*. 2003;3:310-316.

1735.	Bogduk N. Evidence-informed management of chronic low back pain with facet injections and radiofrequency neurotomy. *The spine journal : official journal of the North American Spine Society*. 2008;8:56-64.

1736.	Cohen S, Strassels S, Kurihara C, et al. Randomized study assessing the accuracy of cervical facet joint nerve (medial branch) blocks using different injectate volumes. *Anesthesiology*. 2010;112:144-152.

1737.	Lord SM, Barnsley L, Wallis BJ, McDonald GJ, Bogduk N. Percutaneous radio-frequency neurotomy for chronic cervical zygapophyseal-joint pain. *The New England journal of medicine*. 1996;335:1721-1726.

1738.	Leclaire R, Fortin L, Lambert R, Bergeron YM, Rossignol M. Radiofrequency facet joint denervation in the treatment of low back pain: a placebo-controlled clinical trial to assess efficacy. *Spine*. 2001;26:1411-1416; discussion 1417.

1739.	van Wijk RM, Geurts JW, Wynne HJ, et al. Radiofrequency denervation of lumbar facet joints in the treatment of chronic low back pain: a randomized, double-blind, sham lesion-controlled trial. *Clin J Pain*. 2005;21:335-344.

1740.	van Kleef M, Barendse GA, Kessels A, Voets HM, Weber WE, de Lange S. Randomized trial of radiofrequency lumbar facet denervation for chronic low back pain. *Spine*. 1999;24:1937-1942.

1741.	Nath S, Nath CA, Pettersson K. Percutaneous lumbar zygapophysial (Facet) joint neurotomy using radiofrequency current, in the management of chronic low back pain: a randomized double-blind trial. *Spine (Phila Pa 1976)*. 2008;33:1291-1297; discussion 1298.

1742.	Gallagher J. Radiofrequency facet joint denervation in the treatment of low back pain: a prospective controlled double-blind study to assess its efficacy. *Pain Clin*. 1994;7:193-198.

1743.	North RB, Kidd DH, Campbell JN, Long DM. Dorsal root ganglionectomy for failed back surgery syndrome: a 5-year follow-up study. *Journal of neurosurgery*. 1991;74:236-242.

1744.	Malik K, Benzon HT. Radiofrequency applications to dorsal root ganglia: a literature review. *Anesthesiology*. 2008;109:527-542.

1745.	Geurts JW, van Wijk RM, Wynne HJ, et al. Radiofrequency lesioning of dorsal root ganglia for chronic lumbosacral radicular pain: a randomised, double-blind, controlled trial. *Lancet*. 2003;361:21-26.

1746.	Chou LH, Lew HL, Coelho PC, Slipman CW. Intradiscal electrothermal annuloplasty. *American journal of physical medicine & rehabilitation / Association of Academic Physiatrists*. 2005;84:538-549.

1747.   Heary RF. Intradiscal electrothermal annuloplasty: the IDET procedure. *Journal of spinal disorders*. 2001;14:353-360.

1748.   Wetzel FT, McNally TA, Phillips FM. Intradiscal electrothermal therapy used to manage chronic discogenic low back pain: new directions and interventions. *Spine*. 2002;27:2621-2626.

1749.   Freeman BJ, Fraser RD, Cain CM, Hall DJ, Chapple DC. A randomized, double-blind, controlled trial: intradiscal electrothermal therapy versus placebo for the treatment of chronic discogenic low back pain. *Spine*. 2005;30:2369-2377; discussion 2378.

1750.   Pauza KJ, Howell S, Dreyfuss P, Peloza JH, Dawson K, Bogduk N. A randomized, placebo-controlled trial of intradiscal electrothermal therapy for the treatment of discogenic low back pain. *The spine journal : official journal of the North American Spine Society*. 2004;4:27-35.

1751.   Saal JA, Saal JS. Intradiscal electrothermal therapy for the treatment of chronic discogenic low back pain. *Clinics in sports medicine*. 2002;21:167-187.

1752.   Barendse GA, van Den Berg SG, Kessels AH, Weber WE, van Kleef M. Randomized controlled trial of percutaneous intradiscal radiofrequency thermocoagulation for chronic discogenic back pain: lack of effect from a 90-second 70 C lesion. *Spine*. 2001;26:287-292.

1753.   Fukui S, Nitta K, Iwashita N, Tomie H, Nosaka S, Rohof O. Intradiscal pulsed radiofrequency for chronic lumbar discogenic low back pain: a one year prospective outcome study using discoblock for diagnosis. *Pain physician*. 2013;16:E435-442.

1754.   Gautam S, Rastogi V, Jain A, Singh AP. Comparative evaluation of oxygen-ozone therapy and combined use of oxygen-ozone therapy with percutaneous intradiscal radiofrequency thermocoagulation for the treatment of lumbar disc herniation. *Pain Pract*. 2011;11:160-166.

1755.   Ercelen O, Bulutcu E, Oktenoglu T, et al. Radiofrequency lesioning using two different time modalities for the treatment of lumbar discogenic pain: a randomized trial. *Spine*. 2003;28:1922-1927.

1756.   Peul WC, van Houwelingen HC, van den Hout WB, et al. Surgery versus prolonged conservative treatment for sciatica. *N Engl J Med*. 2007;356:2245-2256.

1757.   Koes BW, van Tulder MW, Peul WC. Diagnosis and treatment of sciatica. *BMJ (Clinical research ed*. 2007;334:1313-1317.

1758.   Rhee JM, Schaufele M, Abdu WA. Radiculopathy and the herniated lumbar disc. Controversies regarding pathophysiology and management. *The Journal of bone and joint surgery American volume*. 2006;88:2070-2080.

1759.   Horng S, Miller FG. Is placebo surgery unethical? *N Engl J Med*. 2002;347:137-139.

1760.   Hu RW, Jaglal S, Axcell T, Anderson G. A population-based study of reoperations after back surgery. *Spine (Phila Pa 1976)*. 1997;22:2265-2270; discussion 2271.

1761.   Malter AD, McNeney B, Loeser JD, Deyo RA. 5-year reoperation rates after different types of lumbar spine surgery. *Spine (Phila Pa 1976)*. 1998;23:814-820.

1762.   Martin CR, Gruszczynski AT, Braunsfurth HA, Fallatah SM, O'Neil J, Wai EK. The surgical management of degenerative lumbar spondylolisthesis: a systematic review. *Spine (Phila Pa 1976)*. 2007;32:1791-1798.

1763.   Osterman H, Sund R, Seitsalo S, Keskimaki I. Risk of multiple reoperations after lumbar discectomy: a population-based study. *Spine (Phila Pa 1976)*. 2003;28:621-627.

1764.   Ciol MA, Deyo RA, Howell E, Kreif S. An assessment of surgery for spinal stenosis: time trends, geographic variations, complications, and reoperations. *J Am Geriatr Soc*. 1996;44:285-290.

1765.   Deyo RA, Ciol MA, Cherkin DC, Loeser JD, Bigos SJ. Lumbar spinal fusion. A cohort study of complications, reoperations, and resource use in the Medicare population. *Spine (Phila Pa 1976)*. 1993;18:1463-1470.

1766.   Minnesota Multiphasic Personality Inventory (MMPI-2). *Manual for Administration and Scoring*. Minneapolis, MN: University of Minnesota Press; 1989.

1767.   Haines SJ, Jordan N, Boen JR, Nyman JA, Oldridge NB, Lindgren BR. Discectomy strategies for lumbar disc herniation: results of the LAPDOG trial. *J Clin Neuroscience* 2002;9:411-417.

1768.   McCulloch JA. Focus issue on lumbar disc herniation: macro- and microdiscectomy. *Spine*. 1996;21:45S-56S.

1769.   Memmo PA, Nadler S, Malanga G. Lumbar disc herniations: A review of surgical and non-surgical indications and outcomes. *J Back Musculoskeletal Rehabil*. 2000;14:79.

1770.   Patel N, Pople IK, Cummins BH. Revisional lumbar microdiscectomy: an analysis of operative findings and clinical outcome. *British journal of neurosurgery*. 1995;9:733-737.

1771.   Ranjan A, Lath R. Microendoscopic discectomy for prolapsed lumbar intervertebral disc. *Neurology India*. 2006;54:190-194.

1772.   Dasenbrock HH, Juraschek SP, Schultz LR, et al. The efficacy of minimally invasive discectomy compared with open discectomy: a meta-analysis of prospective randomized controlled trials. *Journal of neurosurgery Spine*. 2012;16:452-462.

1773.   Gotfryd A, Avanzi O. A systematic review of randomised clinical trials using posterior discectomy to treat lumbar disc herniations. *International orthopaedics*. 2009;33:11-17.

1774.   Jacobs W, Willems PC, van Limbeek J, et al. Single or double-level anterior interbody fusion techniques for cervical degenerative disc disease. *Cochrane Database Syst Rev*. 2011:CD004958.

1775.   Jacobs WC, Rubinstein SM, Willems PC, et al. The evidence on surgical interventions for low back disorders, an overview of systematic reviews. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2013;22:1936-1949.

1776.   Smith N, Masters J, Jensen C, Khan A, Sprowson A. Systematic review of microendoscopic discectomy for lumbar disc herniation. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2013;22:2458-2465.

1777.   Watters WC, 3rd, McGirt MJ. An evidence-based review of the literature on the consequences of conservative versus aggressive discectomy for the treatment of primary disc herniation with radiculopathy. *The spine journal : official journal of the North American Spine Society*. 2009;9:240-257.

1778.   Manchikanti L, Singh V, Calodney AK, et al. Percutaneous lumbar mechanical disc decompression utilizing Dekompressor(R): an update of current evidence. *Pain physician*. 2013;16:SE1-24.

1779.   Manchikanti L, Singh V, Falco FJ, et al. An updated review of automated percutaneous mechanical lumbar discectomy for the contained herniated lumbar disc. *Pain physician*. 2013;16:SE151-184.

1780.   Hirsch JA, Singh V, Falco FJ, Benyamin RM, Manchikanti L. Automated percutaneous lumbar discectomy for the contained herniated lumbar disc: a systematic assessment of evidence. *Pain physician*. 2009;12:601-620.

1781.   Singh V, Benyamin RM, Datta S, Falco FJ, Helm S, 2nd, Manchikanti L. Systematic review of percutaneous lumbar mechanical disc decompression utilizing Dekompressor. *Pain physician*. 2009;12:589-599.

1782.   Griffith HB, Mathew BG. Microdiscectomy. *British journal of hospital medicine*. 1990;44:410-412.

1783.   Kambin P. Arthroscopic microdiscectomy. *The spine journal : official journal of the North American Spine Society*. 2003;3:60S-64S.

1784.   Nellensteijn J, Ostelo R, Bartels R, Peul W, van Royen B, van Tulder M. Transforaminal endoscopic surgery for symptomatic lumbar disc herniations: a systematic review of the literature. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2010;19:181-204.

1785.   Schizas C, Tsiridis E, Saksena J. Microendoscopic discectomy compared with standard microsurgical discectomy for treatment of uncontained or large contained disc herniations. *Neurosurgery*. 2005;57:357-360; discussion 357-360.

1786.   Williams RW. Microlumbar discectomy: a conservative surgical approach to the virgin herniated lumbar disc. *Spine*. 1978;3:175-182.

1787.   Schenk B, Brouwer PA, Peul WC, van Buchem MA. Percutaneous laser disk decompression: a review of the literature. *Am J Neuroradiol*. 2006;27:232-235.

1788.   Hanley E, Green NE, Spengler DM. An AOA critical issue. Less invasive procedures in spine surgery. *The Journal of bone and joint surgery*. 2003;85-A:956-961.

1789.   Marin FZ. CAM versus nucleoplasty. *Acta neurochirurgica*. 2005;92:111-114.

1790.   Knight MT, Goswami A, Patko JT, Buxton N. Endoscopic foraminoplasty: a prospective study on 250 consecutive patients with independent evaluation. *Journal of clinical laser medicine & surgery*. 2001;19:73-81.

1791.   Osterman H, Seitsalo S, Karppinen J, Malmivaara A. Effectiveness of microdiscectomy for lumbar disc herniation: a randomized controlled trial with 2 years of follow-up. *Spine*. 2006;31:2409-2414.

1792.   Weber H. Lumbar disc herniation. A controlled, prospective study with ten years of observation. *Spine*. 1983;8:131-140.

1793.   Weinstein JN, Tosteson TD, Lurie JD, et al. Surgical vs nonoperative treatment for lumbar disk herniation: the Spine Patient Outcomes Research Trial (SPORT): a randomized trial. *Jama*. 2006;296:2441-2450.

1794.   Thomas KC, Fisher CG, Boyd M, Bishop P, Wing P, Dvorak MF. Outcome evaluation of surgical and nonsurgical management of lumbar disc protrusion causing radiculopathy. *Spine*. 2007;32:1414-1422.

1795.   Singh V, Manchikanti L, Calodney AK, et al. Percutaneous lumbar laser disc decompression: an update of current evidence. *Pain physician*. 2013;16:SE229-260.

1796.   Gambardella G, Gervasio O, Zaccone C, Puglisi E. Prevention of recurrent radicular pain after lumbar disc surgery: a prospective study. *Acta neurochirurgica*. 2005;92:151-154.

1797.   Manchikanti L, Pampati V, Fellows B, Rivera J, Beyer CD, Damron KS. Role of one day epidural adhesiolysis in management of chronic low back pain: a randomized clinical trial. *Pain Physician*. 2001;4:153-166.

1798.   Hayek SM, Helm S, Benyamin RM, Singh V, Bryce DA, Smith HS. Effectiveness of spinal endoscopic adhesiolysis in post lumbar surgery syndrome: a systematic review. *Pain physician*. 2009;12:419-435.

1799.   Epter RS, Helm S, 2nd, Hayek SM, Benyamin RM, Smith HS, Abdi S. Systematic review of percutaneous adhesiolysis and management of chronic low back pain in post lumbar surgery syndrome. *Pain Physician*. 2009;12:361-378.

1800.   Helm S. A review of the role of epidural percutaneous adhesiolysis. *Pain management*. 2012;2:609-616.

1801.   Manchikanti L, Pampati V, Bakhit CE, Pakanati RR. Non-endoscopic and endoscopic adhesiolysis in post-lumbar laminectomy syndrome: a one-year outcome study and cost effectiveness analysis. *Pain Physician*. 1999;2:52-58.

1802.   Trescot AM, Chopra P, Abdi S, Datta S, Schultz DM. Systematic review of effectiveness and complications of adhesiolysis in the management of chronic spinal pain: an update. *Pain Physician*. 2007;10:129-146.

1803.   Belozer M, Wang G. *Health Technology Assessment: Epidural Adhesiolysis for the Treatment of Back Pain*. Office of the Medical Director,
Washington State Department of Labor and Industries; 2004.

1804.   Veihelmann A, Devens C, Trouillier H, Birkenmaier C, Gerdesmeyer L, Refior HJ. Epidural neuroplasty versus physiotherapy to relieve pain in patients with sciatica: a prospective randomized blinded clinical trial. *J Orthop Sci*. 2006;11:365-369.

1805.   Cahana A, Mavrocordatos P, Geurts JW, Groen GJ. Do minimally invasive procedures have a place in the treatment of chronic low back pain? *Expert review of neurotherapeutics*. 2004;4:479-490.

1806.   Heavner JE, Racz GB, Raj P. Percutaneous epidural neuroplasty: prospective evaluation of 0.9% NaCl versus 10% NaCl with or without hyaluronidase. *Regional anesthesia and pain medicine*. 1999;24:202-207.

1807.   Manchikanti L, Bakhit CE. Percutaneous lysis of epidural adhesions. *Pain Physician*. 2000;3:46-64.

1808.   Manchikanti L, Rivera JJ, Pampati V, et al. One day lumbar epidural adhesiolysis and hypertonic saline neurolysis in treatment of chronic low back pain: a randomized, double-blind trial. *Pain Physician*. 2004;7:177-186.

1809.   Manchikanti L, Boswell M, Rivera J, et al. A randomized, controlled trial of spinal endoscopic adhesiolysis in chronic refractory low back and lower extremity pain [ISRCTN 16558617]. *BMC anesthesiology*. 2005;5.

1810.   Manchikanti L, Cash K, McManus C, Pampati V, Singh V, Benyamin R. The preliminary results of a comparative effectiveness evaluation of adhesiolysis and caudal epidural injections in managing chronic low back pain secondary to spinal stenosis: a randomized, equivalence controlled trial. *Pain physician*. 2009;12:E341-354.

1811.   Arinzon ZH, Fredman B, Zohar E, et al. Surgical management of spinal stenosis: a comparison of immediate and long term outcome in two geriatric patient populations. *Archives of gerontology and geriatrics*. 2003;36:273-279.

1812.   Atlas SJ, Chang Y, Kammann E, Keller RB, Deyo RA, Singer DE. Long-term disability and return to work among patients who have a herniated lumbar disc: the effect of disability compensation. *The Journal of bone and joint surgery*. 2000;82:4-15.

1813.   Deer TR, Kapural L. New image-guided ultra-minimally invasive lumbar decompression method: the mild procedure. *Pain physician*. 2010;13:35-41.

1814.   Kovacs FM, Urrutia G, Alarcon JD. Surgery versus conservative treatment for symptomatic lumbar spinal stenosis: a systematic review of randomized controlled trials. *Spine (Phila Pa 1976)*. 2011;36:E1335-1351.

1815.   Mekhail N, Vallejo R, Coleman MH, Benyamin RM. Long-term results of percutaneous lumbar decompression mild((R)) for spinal stenosis. *Pain practice : the official journal of World Institute of Pain*. 2012;12:184-193.

1816.   Overdevest GM, Luijsterburg PA, Brand R, et al. Design of the Verbiest trial: cost-effectiveness of surgery versus prolonged conservative treatment in patients with lumbar stenosis. *BMC musculoskeletal disorders*. 2011;12:57.

1817.   Phillips FM, Cunningham B. Managing chronic pain of spinal origin after lumbar surgery: the role of decompressive surgery. *Spine*. 2002;27:2547-2553; discussion 2554.

1818.   Podichetty VK, Spears J, Isaacs RE, Booher J, Biscup RS. Complications associated with minimally invasive decompression for lumbar spinal stenosis. *Journal of spinal disorders & techniques*. 2006;19:161-166.

1819.   Ragab AA, Fye MA, Bohlman HH. Surgery of the lumbar spine for spinal stenosis in 118 patients 70 years of age or older. *Spine*. 2003;28:348-353.

1820.   Schomer DF, Solsberg D, Wong W, Chopko BW. mild((R)) Lumbar Decompression for the Treatment of Lumbar Spinal Stenosis. *The neuroradiology journal*. 2011;24:620-626.

1821.   Weinstein JN, Tosteson TD, Lurie JD, et al. Surgical versus nonoperative treatment for lumbar spinal stenosis four-year results of the Spine Patient Outcomes Research Trial. *Spine*. 2010;35:1329-1338.

1822.   Katz JN, Lipson SJ, Larson MG, McInnes JM, Fossel AH, Liang MH. The outcome of decompressive laminectomy for degenerative lumbar stenosis. *The Journal of bone and joint surgery American volume*. 1991;73:809-816.

1823.   Silvers HR, Lewis PJ, Asch HL. Decompressive lumbar laminectomy for spinal stenosis. *Journal of neurosurgery*. 1993;78:695-701.

1824.   Cheng JS, Lee MJ, Massicotte E, et al. Clinical guidelines and payer policies on fusion for the treatment of chronic low back pain. *Spine (Phila Pa 1976)*. 2011;36:S144-163.

1825.   Li G, Patil CG, Lad SP, Ho C, Tian W, Boakye M. Effects of age and comorbidities on complication rates and adverse outcomes after lumbar laminectomy in elderly patients. *Spine*. 2008;33:1250-1255.

1826.   Brown LL. A double-blind, randomized, prospective study of epidural steroid injection vs. the mild(R) procedure in patients with symptomatic lumbar spinal stenosis. *Pain Pract*. 2012;12:333-341.

1827.   Malmivaara A, Slatis P, Heliovaara M, et al. Surgical or nonoperative treatment for lumbar spinal stenosis? A randomized controlled trial. *Spine*. 2007;32:1-8.

1828.   Thome C, Zevgaridis D, Leheta O, et al. Outcome after less-invasive decompression of lumbar spinal stenosis: a randomized comparison of unilateral laminotomy, bilateral laminotomy, and laminectomy. *Journal of neurosurgery Spine*. 2005;3:129-141.

1829.   Grob D, Humke T, Dvorak J. Degenerative lumbar spinal stenosis. Decompression with and without arthrodesis. *The Journal of bone and joint surgery*. 1995;77:1036-1041.

1830.   Choudhri TF, Mummaneni PV, Dhall SS, et al. Guideline update for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 4: radiographic assessment of fusion status. *Journal of neurosurgery Spine*. 2014;21:23-30.

1831.   Dailey AT, Ghogawala Z, Choudhri TF, et al. Guideline update for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 14: brace therapy as an adjunct to or substitute for lumbar fusion. *Journal of neurosurgery Spine*. 2014;21:91-101.

1832.   Dhall SS, Choudhri TF, Eck JC, et al. Guideline update for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 5: correlation between radiographic outcome and function. *Journal of neurosurgery Spine*. 2014;21:31-36.

1833.   Eck JC, Sharan A, Ghogawala Z, et al. Guideline update for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 7: lumbar fusion for intractable low-back pain without stenosis or spondylolisthesis. *Journal of neurosurgery Spine*. 2014;21:42-47.

1834.   Eck JC, Sharan A, Resnick DK, et al. Guideline update for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 6: discography for patient selection. *Journal of neurosurgery Spine*. 2014;21:37-41.

1835.   Ghogawala Z, Resnick DK, Watters WC, 3rd, et al. Guideline update for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 2: assessment of functional outcome following lumbar fusion. *Journal of neurosurgery Spine*. 2014;21:7-13.

1836.   Ghogawala Z, Whitmore RG, Watters WC, 3rd, et al. Guideline update for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 3: assessment of economic outcome. *Journal of neurosurgery Spine*. 2014;21:14-22.

1837.   Groff MW, Dailey AT, Ghogawala Z, et al. Guideline update for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 12: pedicle screw fixation as an adjunct to posterolateral fusion. *Journal of neurosurgery Spine*. 2014;21:75-78.

1838.   Kaiser MG, Eck JC, Groff MW, et al. Guideline update for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 17: bone growth stimulators as an adjunct for lumbar fusion. *Journal of neurosurgery Spine*. 2014;21:133-139.

1839.   Kaiser MG, Eck JC, Groff MW, et al. Guideline update for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 1: introduction and methodology. *Journal of neurosurgery Spine*. 2014;21:2-6.

1840.   Kaiser MG, Groff MW, Watters WC, 3rd, et al. Guideline update for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 16: bone graft extenders and substitutes as an adjunct for lumbar fusion. *Journal of neurosurgery Spine*. 2014;21:106-132.

1841.   Mummaneni PV, Dhall SS, Eck JC, et al. Guideline update for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 11: interbody techniques for lumbar fusion. *Journal of neurosurgery Spine*. 2014;21:67-74.

1842.   Resnick DK, Watters WC, 3rd, Mummaneni PV, et al. Guideline update for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 10: lumbar fusion for stenosis without spondylolisthesis. *Journal of neurosurgery Spine*. 2014;21:62-66.

1843.   Resnick DK, Watters WC, 3rd, Sharan A, et al. Guideline update for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 9: lumbar fusion for stenosis with spondylolisthesis. *Journal of neurosurgery Spine*. 2014;21:54-61.

1844.   Sharan A, Groff MW, Dailey AT, et al. Guideline update for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 15: electrophysiological monitoring and lumbar fusion. *Journal of neurosurgery Spine*. 2014;21:102-105.

1845.   Wang JC, Dailey AT, Mummaneni PV, et al. Guideline update for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 8: lumbar fusion for disc herniation and radiculopathy. *Journal of neurosurgery Spine*. 2014;21:48-53.

1846.   Watters WC, 3rd, Resnick DK, Eck JC, et al. Guideline update for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 13: injection therapies, low-back pain, and lumbar fusion. *Journal of neurosurgery Spine*. 2014;21:79-90.

1847.   Andersson GB, Mekhail NA, Block JE. Treatment of intractable discogenic low back pain. A systematic review of spinal fusion and intradiscal electrothermal therapy (IDET). *Pain Physician*. 2006;9:237-248.

1848.   Carreon LY, Glassman SD, Howard J. Fusion and nonsurgical treatment for symptomatic lumbar degenerative disease: a systematic review of Oswestry Disability Index and MOS Short Form-36 outcomes. *The spine journal : official journal of the North American Spine Society*. 2008;8:747-755.

1849.   Choma TJ, Schuster JM, Norvell DC, Dettori JR, Chutkan NB. Fusion versus nonoperative management for chronic low back pain: do comorbid diseases or general health factors affect outcome? *Spine (Phila Pa 1976)*. 2011;36:S87-95.

1850.   Daubs MD, Norvell DC, McGuire R, et al. Fusion versus nonoperative care for chronic low back pain: do psychological factors affect outcomes? *Spine (Phila Pa 1976)*. 2011;36:S96-109.

1851.   Freeman BJ, Davenport J. Total disc replacement in the lumbar spine: a systematic review of the literature. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2006;15 Suppl 3:S439-447.

1852.   Gertzbein SD, Betz R, Clements D, et al. Semirigid instrumentation in the management of lumbar spinal conditions combined with circumferential fusion. A multicenter study. *Spine*. 1996;21:1918-1925; discussion 1925-1916.

1853.   Mirza SK, Deyo RA. Systematic review of randomized trials comparing lumbar fusion surgery to nonoperative care for treatment of chronic back pain. *Spine*. 2007;32:816-823.

1854.   Nguyen TH, Randolph DC, Talmage J, Succop P, Travis R. Long-term outcomes of lumbar fusion among workers' compensation subjects: a historical cohort study. *Spine (Phila Pa 1976)*. 2011;36:320-331.

1855.   Parker SL, Adogwa O, Paul AR, et al. Utility of minimum clinically important difference in assessing pain, disability, and health state after transforaminal lumbar interbody fusion for degenerative lumbar spondylolisthesis. *Journal of neurosurgery Spine*. 2011;14:598-604.

1856.   Patel PN, Upasani VV, Bastrom TP, et al. Spontaneous lumbar curve correction in selective thoracic fusions of idiopathic scoliosis: a comparison of anterior and posterior approaches. *Spine*. 2008;33:1068-1073.

1857.   Phillips FM, Lee JY, Geisler FH, et al. A prospective, randomized, controlled clinical investigation comparing PCM cervical disc arthroplasty with anterior cervical discectomy and fusion. 2-year results from the US FDA IDE clinical trial. *Spine*. 2013;38:E907-918.

1858.   Turner JA, Ersek M, Herron L, et al. Patient outcomes after lumbar spinal fusions. *Jama*. 1992;268:907-911.

1859.   van den Eerenbeemt KD, Ostelo RW, van Royen BJ, Peul WC, van Tulder MW. Total disc replacement surgery for symptomatic degenerative lumbar disc disease: a systematic review of the literature. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2010;19:1262-1280.

1860.   Wolfer LR, Derby R, Lee JE, Lee SH. Systematic review of lumbar provocation discography in asymptomatic subjects with a meta-analysis of false-positive rates. *Pain physician*. 2008;11:513-538.

1861.   Zhou J, Li X, Dong J, et al. Three-level anterior cervical discectomy and fusion with self-locking stand-alone polyetheretherketone cages. *J Clin Neurosci*. 2011;18:1505-1509.

1862.   Djurasovic M, Glassman SD, Howard JM, Copay AG, Carreon LY. Health-related quality of life improvements in patients undergoing lumbar spinal fusion as a revision surgery. *Spine (Phila Pa 1976)*. 2011;36:269-276.

1863.   Saltychev M, Eskola M, Laimi K. Lumbar fusion compared with conservative treatment in patients with chronic low back pain: a meta-analysis. *International journal of rehabilitation research Internationale Zeitschrift fur Rehabilitationsforschung*. 2014;37:2-8.

1864.   Deyo RA, Mirza SK. Trends and variations in the use of spine surgery. *Clinical orthopaedics and related research*. 2006;443:139-146.

1865.   Brox JI, Reikeras O, Nygaard O, et al. Lumbar instrumented fusion compared with cognitive intervention and exercises in patients with chronic back pain after previous surgery for disc herniation: a prospective randomized controlled study. *Pain*. 2006;122:145-155.

1866.   Brox JI, Sorensen R, Friis A, et al. Randomized clinical trial of lumbar instrumented fusion and cognitive intervention and exercises in patients with chronic low back pain and disc degeneration. *Spine*. 2003;28:1913-1921.

1867.   Fairbank J, Frost H, Wilson-MacDonald J, Yu LM, Barker K, Collins R. Randomised controlled trial to compare surgical stabilisation of the lumbar spine with an intensive rehabilitation programme for patients with chronic low back pain: the MRC spine stabilisation trial. *BMJ (Clinical research ed*. 2005;330:1233.

1868.   Froholdt A, Holm I, Keller A, Gunderson RB, Reikeraas O, Brox JI. No difference in long-term trunk muscle strength, cross-sectional area, and density in patients with chronic low back pain 7 to 11 years after lumbar fusion versus cognitive intervention and exercises. *The spine journal : official journal of the North American Spine Society*. 2011;11:718-725.

1869.   Keller A, Brox JI, Gunderson R, Holm I, Friis A, Reikeras O. Trunk muscle strength, cross-sectional area, and density in patients with chronic low back pain randomized to lumbar fusion or cognitive intervention and exercises. *Spine*. 2004;29:3-8.

1870.   Keller A, Gunderson R, Reikeras O, Brox JI. Reliability of computed tomography measurements of paraspinal muscle cross-sectional area and density in patients with chronic low back pain. *Spine*. 2003;28:1455-1460.

1871.   Moller H, Hedlund R. Surgery versus conservative management in adult isthmic spondylolisthesis--a prospective randomized study: part 1. *Spine*. 2000;25:1711-1715.

1872.   Caputy AJ, Luessenhop AJ. Long-term evaluation of decompressive surgery for degenerative lumbar stenosis. *Journal of neurosurgery*. 1992;77:669-676.

1873.   Bakhsheshian J, Dahdaleh NS, Fakurnejad S, Scheer JK, Smith ZA. Evidence-based management of traumatic thoracolumbar burst fractures: a systematic review of nonoperative management. *Neurosurgical focus*. 2014;37:E1.

1874.   Dai LY, Jiang LS. Single-level instrumented posterolateral fusion of lumbar spine with beta-tricalcium phosphate versus autograft: a prospective, randomized study with 3-year follow-up. *Spine (Phila Pa 1976)*. 2008;33:1299-1304.

1875.   Wood K, Buttermann G, Mehbod A, et al. Operative compared with nonoperative treatment of a thoracolumbar burst fracture without neurological deficit. A prospective, randomized study. *The Journal of bone and joint surgery*. 2003;85-A:773-781.

1876.   Yi L, Jingping B, Gele J, Baoleri X, Taixiang W. Operative versus non-operative treatment for thoracolumbar burst fractures without neurological deficit. *Cochrane Database Syst Rev*. 2006:CD005079.

1877.   Alvin MD, Derakhshan A, Lubelski D, et al. Cost-utility Analysis of One and Two-level Dorsal Lumbar Fusions With and Without Recombinant Human Bone Morphogenic Protein-2 at 1-year Follow-up. *Journal of spinal disorders & techniques*. 2014.

1878.   Boden SD. Biology of lumbar spine fusion and use of bone graft substitutes: present, future, and next generation. *Tissue engineering*. 2000;6:383-399.

1879.   Boden SD, Kang J, Sandhu H, Heller JG. Use of recombinant human bone morphogenetic protein-2 to achieve posterolateral lumbar spine fusion in humans: a prospective, randomized clinical pilot trial: 2002 Volvo Award in clinical studies. *Spine (Phila Pa 1976)*. 2002;27:2662-2673.

1880.   Burkus JK, Sandhu HS, Gornet MF, Longley MC. Use of rhBMP-2 in combination with structural cortical allografts: clinical and radiographic outcomes in anterior lumbar spinal surgery. *The Journal of bone and joint surgery*. 2005;87:1205-1212.

1881.   Burkus JK, Transfeldt EE, Kitchel SH, Watkins RG, Balderston RA. Clinical and radiographic outcomes of anterior lumbar interbody fusion using recombinant human bone morphogenetic protein-2. *Spine*. 2002;27:2396-2408.

1882.   Carragee EJ. Re: BMP-2 augmented fusion in the low-risk, healthy subjects: a confirmation of effectiveness and harms highlights the need for study in high risk patients. *Spine*. 2014;39:341-342.

1883.   Chen BL, Zhong Y, Huang YL, et al. Systematic back muscle exercise after percutaneous vertebroplasty for spinal osteoporotic compression fracture patients: a randomized controlled trial. *Clin Rehabil*. 2012;26:483-492.

1884.   Dawson E, Bae HW, Burkus JK, Stambough JL, Glassman SD. Recombinant human bone morphogenetic protein-2 on an absorbable collagen sponge with an osteoconductive bulking agent in posterolateral arthrodesis with instrumentation. A prospective randomized trial. *The Journal of bone and joint surgery*. 2009;91:1604-1613.

1885.   Dimar JR, 2nd, Glassman SD, Burkus JK, Pryor PW, Hardacker JW, Carreon LY. Clinical and radiographic analysis of an optimized rhBMP-2 formulation as an autograft replacement in posterolateral lumbar spine arthrodesis. *The Journal of bone and joint surgery*. 2009;91:1377-1386.

1886.   Dimar JR, Glassman SD, Burkus KJ, Carreon LY. Clinical outcomes and fusion success at 2 years of single-level instrumented posterolateral fusions with recombinant human bone morphogenetic protein-2/compression resistant matrix versus iliac crest bone graft. *Spine (Phila Pa 1976)*. 2006;31:2534-2539; discussion 2540.

1887.   Fu R, Selph S, McDonagh M, et al. Effectiveness and harms of recombinant human bone morphogenetic protein-2 in spine fusion: a systematic review and meta-analysis. *Ann Intern Med*. 2013;158:890-902.

1888.   Glassman SD, Carreon LY, Djurasovic M, et al. RhBMP-2 versus iliac crest bone graft for lumbar spine fusion: a randomized, controlled trial in patients over sixty years of age. *Spine (Phila Pa 1976)*. 2008;33:2843-2849.

1889.   Glassman SD, Dimar JR, Carreon LY, Campbell MJ, Puno RM, Johnson JR. Initial fusion rates with recombinant human bone morphogenetic protein-2/compression resistant matrix and a hydroxyapatite and tricalcium phosphate/collagen carrier in posterolateral spinal fusion. *Spine (Phila Pa 1976)*. 2005;30:1694-1698.

1890.   Glassman SD, Howard J, Dimar J, Sweet A, Wilson G, Carreon L. Complications with recombinant human bone morphogenic protein-2 in posterolateral spine fusion: a consecutive series of 1037 cases. *Spine*. 2011;36:1849-1854.

1891.   Guppy KH, Paxton EW, Harris J, Alvarez J, Bernbeck J. Does bone morphogenetic protein change the operative nonunion rates in spine fusions? *Spine*. 2014;39:1831-1839.

1892.   Haid RW, Jr., Branch CL, Jr., Alexander JT, Burkus JK. Posterior lumbar interbody fusion using recombinant human bone morphogenetic protein type 2 with cylindrical interbody cages. *The spine journal : official journal of the North American Spine Society*. 2004;4:527-538; discussion 538-529.

1893.   Mannion RJ, Nowitzke AM, Wood MJ. Promoting fusion in minimally invasive lumbar interbody stabilization with low-dose bone morphogenic protein-2--but what is the cost? *The spine journal : official journal of the North American Spine Society*. 2011;11:527-533.

1894.   Alexander DI, Manson NA, Mitchell MJ. Efficacy of calcium sulfate plus decompression bone in lumbar and lumbosacral spinal fusion: preliminary results in 40 patients. *Canadian journal of surgery Journal canadien de chirurgie*. 2001;44:262-266.

1895.   Alsaleh KA, Tougas CA, Roffey DM, Wai EK. Osteoconductive bone graft extenders in posterolateral thoracolumbar spinal fusion: a systematic review. *Spine (Phila Pa 1976)*. 2012;37:E993-1000.

1896.   Carragee EJ, Comer GC, Smith MW. Local bone graft harvesting and volumes in posterolateral lumbar fusion: a technical report. *The spine journal : official journal of the North American Spine Society*. 2011;11:540-544.

1897.   Delecrin J, Takahashi S, Gouin F, Passuti N. A synthetic porous ceramic as a bone graft substitute in the surgical management of scoliosis: a prospective, randomized study. *Spine*. 2000;25:563-569.

1898.   Korovessis P, Koureas G, Zacharatos S, Papazisis Z, Lambiris E. Correlative radiological, self-assessment and clinical analysis of evolution in instrumented dorsal and lateral fusion for degenerative lumbar spine disease. Autograft versus coralline hydroxyapatite. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2005;14:630-638.

1899.   Lerner T, Griefingholt H, Liljenqvist U. Bone substitutes in scoliosis surgery. *Der Orthopade*. 2009;38:181-188.

1900.   Ransford AO, Morley T, Edgar MA, et al. Synthetic porous ceramic compared with autograft in scoliosis surgery. A prospective, randomized study of 341 patients. *J Bone Joint Surg Br*. 1998;80:13-18.

1901.   Wood KB, Fritzell P, Dettori JR, Hashimoto R, Lund T, Shaffrey C. Effectiveness of spinal fusion versus structured rehabilitation in chronic low back pain patients with and without isthmic spondylolisthesis: a systematic review. *Spine (Phila Pa 1976)*. 2011;36:S110-119.

1902.   Bono CM, Lee CK. Critical analysis of trends in fusion for degenerative disc disease over the past 20 years: influence of technique on fusion rate and clinical outcome. *Spine*. 2004;29:455-463; discussion Z455.

1903.   Errico TJ, Gatchel RJ, Schofferman J, et al. A fair and balanced view of spine fusion surgery. *The spine journal : official journal of the North American Spine Society*. 2004;4:S129-138.

1904.   Gibson JN, Grant IC, Waddell G. The Cochrane review of surgery for lumbar disc prolapse and degenerative lumbar spondylosis. *Spine*. 1999;24:1820-1832.

1905.   Kwon B, Katz JN, Kim DH, Jenis LG. A review of the 2001 Volvo Award winner in clinical studies: lumbar fusion versus nonsurgical treatment for chronic low back pain: a multicenter randomized controlled trial from the Swedish lumbar spine study group. *Spine*. 2006;31:245-249.

1906.   Turner JA, Herron L, Deyo RA. Meta-analysis of the results of lumbar spine fusion. *Acta orthopaedica Scandinavica*. 1993;251:120-122.

1907.   Fritzell P, Hagg O, Nordwall A. Complications in lumbar fusion surgery for chronic low back pain: comparison of three surgical techniques used in a prospective randomized study. A report from the Swedish Lumbar Spine Study Group. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2003;12:178-189.

1908.   Zigler J, Delamarter R, Spivak JM, et al. Results of the prospective, randomized, multicenter Food and Drug Administration investigational device exemption study of the ProDisc-L total disc replacement versus circumferential fusion for the treatment of 1-level degenerative disc disease. *Spine*. 2007;32:1155-1162; discussion 1163.

1909.   Martin BI, Mirza SK, Comstock BA, Gray DT, Kreuter W, Deyo RA. Reoperation rates following lumbar spine surgery and the influence of spinal fusion procedures. *Spine*. 2007;32:382-387.

1910.   Christensen FB, Hansen, E.S., Laursen M., Thomsen K., Bunger C.E. Long-term functional outcome of pedicle screw instrumentation as a support for posterolateral lumber spinal fusion: randomized clinical study with 5-year follow-up. *Spine*. 2002;27:1269-1277.

1911.   Fischgrund JS, Mackay M, Herkowitz HN, Brower R, Montgomery DM, Kurz LT. 1997 Volvo Award winner in clinical studies. Degenerative lumbar spondylolisthesis with spinal stenosis: a prospective, randomized study comparing decompressive laminectomy and arthrodesis with and without spinal instrumentation. *Spine*. 1997;22:2807-2812.

1912.   Fritzell P, Hagg O, Wessberg P, Nordwall A. 2001 Volvo Award Winner in Clinical Studies: Lumbar fusion versus nonsurgical treatment for chronic low back pain: a multicenter randomized controlled trial from the Swedish Lumbar Spine Study Group. *Spine*. 2001;26:2521-2532; discussion 2532-2524.
1913.   Fritzell P, Hagg O, Wessberg P, Nordwall A. Chronic low back pain and fusion: a comparison of three surgical techniques: a prospective multicenter randomized study from the Swedish lumbar spine study group. *Spine*. 2002;27:1131-1141.
1914.   Kim KT, Lee SH, Lee YH, Bae SC, Suk KS. Clinical outcomes of 3 fusion methods through the posterior approach in the lumbar spine. *Spine*. 2006;31:1351-1357; discussion 1358.
1915.   Thomsen K, Christensen FB, Eiskjaer SP, Hansen ES, Fruensgaard S, Bunger CE. 1997 Volvo Award winner in clinical studies. The effect of pedicle screw instrumentation on functional outcome and fusion rates in posterolateral lumbar spinal fusion: a prospective, randomized clinical study. *Spine*. 1997;22:2813-2822.
1916.   Zhao J, Wang X, Hou T, He S. One versus two BAK fusion cages in posterior lumbar interbody fusion to L4-L5 degenerative spondylolisthesis: a randomized, controlled prospective study in 25 patients with minimum two-year follow-up. *Spine*. 2002;27:2753-2757.
1917.   Weinstein JN, Lurie JD, Tosteson TD, et al. Surgical versus nonsurgical treatment for lumbar degenerative spondylolisthesis. *N Engl J Med*. 2007;356:2257-2270.
1918.   Luers P. Spinal alteration of motion segment integrity *The Guides Newsletter, American Medical Association*; 2007.
1919.   Fritzell P, Hagg O, Jonsson D, Nordwall A. Cost-effectiveness of lumbar fusion and nonsurgical treatment for chronic low back pain in the Swedish Lumbar Spine Study: a multicenter, randomized, controlled trial from the Swedish Lumbar Spine Study Group. *Spine*. 2004;29:421-434; discussion Z423.
1920.   Hagg O, Fritzell P, Ekselius L, Nordwall A. Predictors of outcome in fusion surgery for chronic low back pain. A report from the Swedish Lumbar Spine Study. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2003;12:22-33.
1921.   Mooney V. Re: Surgery versus conservative medical and adult isthmic spondylolisthesis (Spine 2000; 25: 1711-15). *Spine (Phila Pa 1976)*. 2001;26:594-595.
1922.   Deyo RA, Nachemson A, Mirza SK. Spinal-fusion surgery - the case for restraint. *N Engl J Med*. 2004;350:722-726.
1923.   Brox JI, Nygaard OP, Holm I, Keller A, Ingebrigtsen T, Reikeras O. Four-year follow-up of surgical versus non-surgical therapy for chronic low back pain. *Annals of the rheumatic diseases*. 2010;69:1643-1648.
1924.   DeBerard MS, Masters KS, Colledge AL, Schleusener RL, Schlegel JD. Outcomes of posterolateral lumbar fusion in Utah patients receiving workers' compensation: a retrospective cohort study. *Spine*. 2001;26:738-746; discussion 747.
1925.   Franklin GM, Haug J, Heyer NJ, McKeefrey SP, Picciano JF. Outcome of lumbar fusion in Washington State workers' compensation. *Spine*. 1994;19:1897-1903; discussion 1904.
1926.   Fernández-Fairen M, Sala P, Ramírez H, Gil J. A prospective randomized study of unilateral versus bilateral instrumented posterolateral lumbar fusion in degenerative spondylolisthesis. *Spine*. 2007;32:395-401.
1927.   Cinotti G, Roysam GS, Eisenstein SM, Postacchini F. Ipsilateral recurrent lumbar disc herniation. A prospective, controlled study. *J Bone Joint Surg Br*. 1998;80:825-832.
1928.   Fu TS, Lai PL, Tsai TT, Niu CC, Chen LH, Chen WJ. Long-term results of disc excision for recurrent lumbar disc herniation with or without posterolateral fusion. *Spine*. 2005;30:2830-2834.
1929.   Jonsson B, Stromqvist B. Repeat decompression of lumbar nerve roots. A prospective two-year evaluation. *J Bone Joint Surg Br*. 1993;75:894-897.
1930.   Suk KS, Jeon CH, Park MS, Moon SH, Kim NH, Lee HM. Comparison between posterolateral fusion with pedicle screw fixation and anterior interbody fusion with pedicle screw fixation in adult spondylolytic spondylolisthesis. *Yonsei medical journal*. 2001;42:316-323.
1931.   Hallett A, Huntley JS, Gibson JN. Foraminal stenosis and single-level degenerative disc disease: a randomized controlled trial comparing decompression with decompression and instrumented fusion. *Spine (Phila Pa 1976)*. 2007;32:1375-1380.
1932.   Goodwin CB, Brighton CT, Guyer RD, Johnson JR, Light KI, Yuan HA. A double-blind study of capacitively coupled electrical stimulation as an adjunct to lumbar spinal fusions. *Spine*. 1999;24:1349-1356; discussion 1357.
1933.   Linovitz RJ, Pathria M, Bernhardt M, et al. Combined magnetic fields accelerate and increase spine fusion: a double-blind, randomized, placebo controlled study. *Spine*. 2002;27:1383-1389; discussion 1389.
1934.   Mooney V. A randomized double-blind prospective study of the efficacy of pulsed electromagnetic fields for interbody lumbar fusions. *Spine*. 1990;15:708-712.
1935.   de Kleuver M, Oner FC, Jacobs WC. Total disc replacement for chronic low back pain: background and a systematic review of the literature. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2003;12:108-116.

1936.	Jacobs W, Van der Gaag NA, Tuschel A, et al. Total disc replacement for chronic back pain in the presence of disc degeneration. *Cochrane Database Syst Rev*. 2012;9:CD008326.
1937.	Kanayama M, Hashimoto T, Shigenobu K, Togawa D, Oha F. A minimum 10-year follow-up of posterior dynamic stabilization using Graf artificial ligament. *Spine*. 2007;32:1992-1996; discussion 1997.
1938.	Yue J, Zhang K, Bai HX, et al. A comparison of patients who have undergone 1-Level versus 2-Level ProDisc arthroplasty: a prospective study with minimum of 5-year follow-up. *Spine*. 2013;38:1194-1198.
1939.	Rampersaud YR, Wai EK, Fisher CG, et al. Postoperative improvement in health-related quality of life: a national comparison of surgical treatment for focal (one- to two-level) lumbar spinal stenosis compared with total joint arthroplasty for osteoarthritis. *The spine journal : official journal of the North American Spine Society*. 2011;11:1033-1041.
1940.	U.S. Food and Drug Administration. Center for Devices and Radiological Health.Available at: https://www.fda.gov/about-fda/fda-organization/center-devices-and-radiological-health.
1941.	Hellum C, Berg L, Gjertsen O, et al. Adjacent level degeneration and facet arthropathy after disc prosthesis surgery or rehabilitation in patients with chronic low back pain and degenerative disc: second report of a randomized study. *Spine (Phila Pa 1976)*. 2012;37:2063-2073.
1942.	Hellum C, Johnsen LG, Gjertsen O, et al. Predictors of outcome after surgery with disc prosthesis and rehabilitation in patients with chronic low back pain and degenerative disc: 2-year follow-up. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2012;21:681-690.
1943.	Hellum C, Johnsen LG, Storheim K, et al. Surgery with disc prosthesis versus rehabilitation in patients with low back pain and degenerative disc: two year follow-up of randomised study. *BMJ*. 2011;342:d2786.
1944.	Johnsen LG, Brinckmann P, Hellum C, Rossvoll I, Leivseth G. Segmental mobility, disc height and patient-reported outcomes after surgery for degenerative disc disease: a prospective randomised trial comparing disc replacement and multidisciplinary rehabilitation. *Bone Joint J*. 2013;95-B:81-89.
1945.	Berg S, Tullberg T, Branth B, Olerud C, Tropp H. Total disc replacement compared to lumbar fusion: a randomised controlled trial with 2-year follow-up. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2009;18:1512-1519.
1946.	Blumenthal S, McAfee PC, Guyer RD, et al. A prospective, randomized, multicenter Food and Drug Administration investigational device exemptions study of lumbar total disc replacement with the CHARITE artificial disc versus lumbar fusion: part I: evaluation of clinical outcomes. *Spine*. 2005;30:1565-1575; discussion E1387-1591.
1947.	Gornet MF, Burkus JK, Dryer RF, Peloza JH. Lumbar disc arthroplasty with Maverick disc versus stand-alone interbody fusion: a prospective, randomized, controlled, multicenter investigational device exemption trial. *Spine (Phila Pa 1976)*. 2011;36:E1600-1611.
1948.	Guyer RD, McAfee PC, Banco RJ, et al. Prospective, randomized, multicenter Food and Drug Administration investigational device exemption study of lumbar total disc replacement with the CHARITE artificial disc versus lumbar fusion: five-year follow-up. *The spine journal : official journal of the North American Spine Society*. 2009;9:374-386.
1949.	Skold C, Tropp H, Berg S. Five-year follow-up of total disc replacement compared to fusion: a randomized controlled trial. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2013;22:2288-2295.
1950.	Zigler JE, Burd TA, Vialle EN, Sachs BL, Rashbaum RF, Ohnmeiss DD. Lumbar spine arthroplasty: early results using the ProDisc II: a prospective randomized trial of arthroplasty versus fusion. *Journal of spinal disorders & techniques*. 2003;16:352-361.
1951.	Sasso RC, Foulk DM, Hahn M. Prospective, randomized trial of metal-on-metal artificial lumbar disc replacement: initial results for treatment of discogenic pain. *Spine*. 2008;33:123-131.
1952.	Regan JJ, McAfee PC, Blumenthal SL, et al. Evaluation of surgical volume and the early experience with lumbar total disc replacement as part of the investigational device exemption study of the Charite Artificial Disc. *Spine*. 2006;31:2270-2276.
1953.	Galibert P, Deramond H, Rosat P, Le Gars D. Preliminary note on the treatment of vertebral angioma by percutaneous acrylic vertebroplast. *Neurochirurgie*. 1987;33:166-168.
1954.	Anderson PA, Froyshteter AB, Tontz WL, Jr. Meta-analysis of vertebral augmentation compared with conservative treatment for osteoporotic spinal fractures. *Journal of bone and mineral research : the official journal of the American Society for Bone and Mineral Research*. 2013;28:372-382.
1955.	Han S, Wan S, Ning L, Tong Y, Zhang J, Fan S. Percutaneous vertebroplasty versus balloon kyphoplasty for treatment of osteoporotic vertebral compression fracture: a meta-analysis of randomised and non-randomised controlled trials. *Int Orthop*. 2011;35:1349-1358.

1956.   Ma XL, Xing D, Ma JX, Xu WG, Wang J, Chen Y. Balloon kyphoplasty versus percutaneous vertebroplasty in treating osteoporotic vertebral compression fracture: grading the evidence through a systematic review and meta-analysis. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2012;21:1844-1859.

1957.   Papanastassiou ID, Phillips FM, Van Meirhaeghe J, et al. Comparing effects of kyphoplasty, vertebroplasty, and non-surgical management in a systematic review of randomized and non-randomized controlled studies. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2012;21:1826-1843.

1958.   Robinson Y, Olerud C. Vertebroplasty and kyphoplasty--a systematic review of cement augmentation techniques for osteoporotic vertebral compression fractures compared to standard medical therapy. *Maturitas*. 2012;72:42-49.

1959.   Shi MM, Cai XZ, Lin T, Wang W, Yan SG. Is there really no benefit of vertebroplasty for osteoporotic vertebral fractures? A meta-analysis. *Clinical orthopaedics and related research*. 2012;470:2785-2799.

1960.   Zhang Z, Fan J, Ding Q, Wu M, Yin G. Risk factors for new osteoporotic vertebral compression fractures after vertebroplasty: a systematic review and meta-analysis. *Journal of spinal disorders & techniques*. 2013;26:E150-157.

1961.   Cotten A, Boutry N, Cortet B, et al. Percutaneous vertebroplasty: state of the art. *Radiographics*. 1998;18:311-320; discussion 320-313.

1962.   Deen HG, Jr., Rizzo TD, Fenton DS. Sudden progression of lumbar disk protrusion during vertebral axial decompression traction therapy. *Mayo Clinic proceedings Mayo Clinic*. 2003;78:1554-1556.

1963.   DePalma MJ, Ketchum JM, Frankel BM, Frey ME. Percutaneous vertebroplasty for osteoporotic vertebral compression fractures in the nonagenarians: a prospective study evaluating pain reduction and new symptomatic fracture rate. *Spine (Phila Pa 1976)*. 2011;36:277-282.

1964.   Garfin SR, Reilley MA. Minimally invasive treatment of osteoporotic vertebral body compression fractures. *The spine journal : official journal of the North American Spine Society*. 2002;2:76-80.

1965.   Garfin SR, Yuan HA, Reiley MA. New technologies in spine: kyphoplasty and vertebroplasty for the treatment of painful osteoporotic compression fractures. *Spine*. 2001;26:1511-1515.

1966.   Hulme PA, Krebs J, Ferguson SJ, Berlemann U. Vertebroplasty and kyphoplasty: a systematic review of 69 clinical studies. *Spine*. 2006;31:1983-2001.

1967.   Ploeg WT, Veldhuizen AG, The B, Sietsma MS. Percutaneous vertebroplasty as a treatment for osteoporotic vertebral compression fractures: a systematic review. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2006;15:1749-1758.

1968.   Taylor RS, Taylor RJ, Fritzell P. Balloon kyphoplasty and vertebroplasty for vertebral compression fractures: a comparative systematic review of efficacy and safety. *Spine*. 2006;31:2747-2755.

1969.   Staples MP, Kallmes DF, Comstock BA, et al. Effectiveness of vertebroplasty using individual patient data from two randomised placebo controlled trials: meta-analysis. *BMJ*. 2011;343:d3952.

1970.   Lad SP, Patil CG, Lad EM, Hayden MG, Boakye M. National trends in vertebral augmentation procedures for the treatment of vertebral compression fractures. *Surg Neurol*. 2009;71:580-584; discussion 584-585.

1971.   Buchbinder R, Osborne RH, Ebeling PR, et al. A randomized trial of vertebroplasty for painful osteoporotic vertebral fractures. *N Engl J Med*. 2009;361:557-568.

1972.   Kallmes DF, Comstock BA, Heagerty PJ, et al. A randomized trial of vertebroplasty for osteoporotic spinal fractures. *N Engl J Med*. 2009;361:569-579.

1973.   Farrokhi MR, Alibai E, Maghami Z. Randomized controlled trial of percutaneous vertebroplasty versus optimal medical management for the relief of pain and disability in acute osteoporotic vertebral compression fractures. *Journal of neurosurgery Spine*. 2011;14:561-569.

1974.   Klazen CA, Lohle PN, de Vries J, et al. Vertebroplasty versus conservative treatment in acute osteoporotic vertebral compression fractures (Vertos II): an open-label randomised trial. *Lancet*. 2010;376:1085-1092.

1975.   Aslam E, Muhammad T, Sharif S. Percutaneous vertebroplasty in osteoporotic vertebral compression fractures: our initial experience. *J Pak Med Assoc*. 2008;58:498-501.

1976.   Evans AJ, Jensen ME, Kip KE, et al. Vertebral compression fractures: pain reduction and improvement in functional mobility after percutaneous polymethylmethacrylate vertebroplasty retrospective report of 245 cases. *Radiology*. 2003;226:366-372.

1977.   Hochmuth K, Proschek D, Schwarz W, Mack M, Kurth AA, Vogl TJ. Percutaneous vertebroplasty in the therapy of osteoporotic vertebral compression fractures: a critical review. *European radiology*. 2006;16:998-1004.

1978.   Jensen ME, Evans AJ, Mathis JM, Kallmes DF, Cloft HJ, Dion JE. Percutaneous polymethylmethacrylate vertebroplasty in the treatment of osteoporotic vertebral body compression fractures: technical aspects. *Am J Neuroradiol*. 1997;18:1897-1904.

1979.   Kallmes DF. Randomized vertebroplasty trials: Current status and challenges. *Academic radiology*. 2006;13:546-549.

1980.   Levine SA, Perin LA, Hayes D, Hayes WS. An evidence-based evaluation of percutaneous vertebroplasty. *Manag Care*. 2000;9:56-60, 63.

1981.   Miller FG, Kallmes DF. The case of vertebroplasty trials: promoting a culture of evidence-based procedural medicine. *Spine*. 2010;35:2023-2026.

1982.   Trout AT, Gray LA, Kallmes DF. Vertebroplasty in the inpatient population. *Am J Neuroradiol*. 2005;26:1629-1633.

1983.   Trout AT, Kallmes DF, Gray LA, et al. Evaluation of vertebroplasty with a validated outcome measure: the Roland-Morris Disability Questionnaire. *Am J Neuroradiol*. 2005;26:2652-2657.

1984.   Voormolen MH, Mali WP, Lohle PN, et al. Percutaneous vertebroplasty compared with optimal pain medication treatment: short-term clinical outcome of patients with subacute or chronic painful osteoporotic vertebral compression fractures. The VERTOS study. *Am J Neuroradiol*. 2007;28:555-560.

1985.   Lieberman I, Reinhardt MK. Vertebroplasty and kyphoplasty for osteolytic vertebral collapse. *Clinical orthopaedics and related research*. 2003:S176-186.

1986.   Vats HS, McKiernan FE. Infected vertebroplasty: case report and review of literature. *Spine*. 2006;31:E859-862.

1987.   McGirt MJ, Parker SL, Wolinsky JP, Witham TF, Bydon A, Gokaslan ZL. Vertebroplasty and kyphoplasty for the treatment of vertebral compression fractures: an evidenced-based review of the literature. *The spine journal : official journal of the North American Spine Society*. 2009;9:501-508.

1988.   Trout AT, Kallmes DF. Does vertebroplasty cause incident vertebral fractures? A review of available data. *Am J Neuroradiol*. 2006;27:1397-1403.

1989.   Tseng YY, Yang TC, Tu PH, Lo YL, Yang ST. Repeated and multiple new vertebral compression fractures after percutaneous transpedicular vertebroplasty. *Spine*. 2009;34:1917-1922.

1990.   Bouza C, Lopez-Cuadrado T, Cediel P, Saz-Parkinson Z, Amate JM. Balloon kyphoplasty in malignant spinal fractures: a systematic review and meta-analysis. *BMC palliative care*. 2009;8:12.

1991.   Burton AW, Hamid B. Kyphoplasty and vertebroplasty. *Current pain and headache reports*. 2008;12:22-27.

1992.   Coumans JV, Reinhardt MK, Lieberman IH. Kyphoplasty for vertebral compression fractures: 1-year clinical outcomes from a prospective study. *J Neurosurg*. 2003;99:44-50.

1993.   Deramond H, Saliou G, Aveillan M, Lehmann P, Vallee JN. Respective contributions of vertebroplasty and kyphoplasty to the management of osteoporotic vertebral fractures. *Joint, bone, spine : revue du rhumatisme*. 2006;73:610-613.

1994.   Grafe IA, Da Fonseca K, Hillmeier J, et al. Reduction of pain and fracture incidence after kyphoplasty: 1-year outcomes of a prospective controlled trial of patients with primary osteoporosis. *Osteoporosis international : a journal established as result of cooperation between the European Foundation for Osteoporosis and the National Osteoporosis Foundation of the USA*. 2005;16:2005-2012.

1995.   Heini PF, Orler R. Kyphoplasty for treatment of osteoporotic vertebral fractures. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2004;13:184-192.

1996.   Huang Z, Wan S, Ning L, Han S. Is unilateral kyphoplasty as effective and safe as bilateral kyphoplasties for osteoporotic vertebral compression fractures? A meta-analysis. *Clinical orthopaedics and related research*. 2014;472:2833-2842.

1997.   Karlsson MK, Ohlin A, Hasserius R. Could vertebroplasty and kyphoplasty be regarded as evidence-based treatment of osteoporotic vertebral fractures? *Acta Radiol*. 2010;51:828-831.

1998.   Kasperk C, Grafe IA, Schmitt S, et al. Three-year outcomes after kyphoplasty in patients with osteoporosis with painful vertebral fractures. *Journal of vascular and interventional radiology : JVIR*. 2010;21:701-709.

1999.   Taylor RS, Fritzell P, Taylor RJ. Balloon kyphoplasty in the management of vertebral compression fractures: an updated systematic review and meta-analysis. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2007;16:1085-1100.

2000.   Zampini JM, White AP, McGuire KJ. Comparison of 5766 vertebral compression fractures treated with or without kyphoplasty. *Clinical orthopaedics and related research*. 2010;468:1773-1780.

2001.   Blattert TR, Jestaedt L, Weckbach A. Suitability of a calcium phosphate cement in osteoporotic vertebral body fracture augmentation: a controlled, randomized, clinical trial of balloon kyphoplasty comparing calcium phosphate versus polymethylmethacrylate. *Spine*. 2009;34:108-114.

2002.   Becker S, Garoscio M, Meissner J, Tuschel A, Ogon M. Is there an indication for prophylactic balloon kyphoplasty? A pilot study. *Clinical orthopaedics and related research*. 2007;458:83-89.

2003.   Wardlaw D, Cummings SR, Van Meirhaeghe J, et al. Efficacy and safety of balloon kyphoplasty compared with non-surgical care for vertebral compression fracture (FREE): a randomised controlled trial. *The Lancet*. 2009;373:1016-1024.

2004.   Chung HJ, Chung KJ, Yoon HS, Kwon IH. Comparative study of balloon kyphoplasty with unilateral versus bilateral approach in osteoporotic vertebral compression fractures. *International orthopaedics*. 2008;32:817-820.

2005.   Schmelzer-Schmied N, Cartens C, Meeder PJ, Dafonseca K. Comparison of kyphoplasty with use of a calcium phosphate cement and non-operative therapy in patients with traumatic non-osteoporotic vertebral fractures. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2009;18:624-629.

2006.   Bouza C, Lopez T, Magro A, Navalpotro L, Amate JM. Efficacy and safety of balloon kyphoplasty in the treatment of vertebral compression fractures: a systematic review. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*. 2006;15:1050-1067.

2007.   Eck JC, Nachtigall D, Humphreys SC, Hodges SD. Comparison of vertebroplasty and balloon kyphoplasty for treatment of vertebral compression fractures: a meta-analysis of the literature. *The spine journal : official journal of the North American Spine Society*. 2008;8:488-497.

2008.   Gill JB, Kuper M, Chin PC, Zhang Y, Schutt R, Jr. Comparing pain reduction following kyphoplasty and vertebroplasty for osteoporotic vertebral compression fractures. *Pain Physician*. 2007;10:583-590.

2009.   Liu JT, Liao WJ, Tan WC, et al. Balloon kyphoplasty versus vertebroplasty for treatment of osteoporotic vertebral compression fracture: a prospective, comparative, and randomized clinical study. *Osteoporosis international : a journal established as result of cooperation between the European Foundation for Osteoporosis and the National Osteoporosis Foundation of the USA*. 2010;21:359-364.

2010.   Movrin I, Vengust R, Komadina R. Adjacent vertebral fractures after percutaneous vertebral augmentation of osteoporotic vertebral compression fracture: a comparison of balloon kyphoplasty and vertebroplasty. *Archives of orthopaedic and traumatic surgery*. 2010;130:1157-1166.

2011.   Fribourg D, Tang C, Sra P, Delamarter R, Bae H. Incidence of subsequent vertebral fracture after kyphoplasty. *Spine*. 2004;29:2270-2276; discussion 2277.

2012.   Harrop JS, Prpa B, Reinhardt MK, Lieberman I. Primary and secondary osteoporosis' incidence of subsequent vertebral compression fractures after kyphoplasty. *Spine*. 2004;29:2120-2125.

2013.   Frankel BM, Monroe T, Wang C. Percutaneous vertebral augmentation: an elevation in adjacent-level fracture risk in kyphoplasty as compared with vertebroplasty. *The spine journal : official journal of the North American Spine Society*. 2007;7:575-582.

2014.   Schutz U, Grob D. Poor outcome following bilateral sacroiliac joint fusion for degenerative sacroiliac joint syndrome. *Acta orthopaedica Belgica*. 2006;72:296-308.

2015.   Gaenslen F. Sacroiliac arthodesis: indications, author's technic and end results. *JAMA : the journal of the American Medical Association*. 1927;89:2031-2035.

2016.   Moore MR. Results after sacroilliac joint fusion. *Proceed of Third World Indisciplinary Congress on Low Back Pain and its Relation to the Sacroilliac Joint* Vienna; 1998.

2017.   Waisbrod H, Krainick JU, Gerbershagen HU. Sacroiliac joint arthrodesis for chronic lower back pain. *Archives of orthopaedic and traumatic surgery*. 1987;106:238-240.

2018.   Simpson EL, Duenas A, Holmes MW, Papaioannou D, Chilcott J. Spinal cord stimulation for chronic pain of neuropathic or ischaemic origin: systematic review and economic evaluation. *Health Technol Assess*. 2009;13:iii, ix-x, 1-154.

2019.   Stojanovic MP, Abdi S. Spinal cord stimulation. *Pain Physician*. 2002;5:156-166.

2020.   Al-Kaisy A, Van Buyten JP, Smet I, Palmisani S, Pang D, Smith T. Sustained effectiveness of 10 kHz high-frequency spinal cord stimulation for patients with chronic, low back pain: 24-month results of a prospective multicenter study. *Pain Med*. 2014;15:347-354.

2021.   Levy R, Henderson J, Slavin K, et al. Incidence and avoidance of neurologic complications with paddle type spinal cord stimulation leads. *Neuromodulation : journal of the International Neuromodulation Society*. 2011;14:412-422; discussion 422.

2022.   North RB, Wetzel FT. Spinal cord stimulation for chronic pain of spinal origin: a valuable long-term solution. *Spine*. 2002;27:2584-2591; discussion 2592.

2023.   Mailis-Gagnon A, Furlan AD, Sandoval JA, Taylor R. Spinal cord stimulation for chronic pain. *Cochrane Database Syst Rev*. 2004:CD003783.

2024.   Turner JA, Loeser JD, Deyo RA, Sanders SB. Spinal cord stimulation for patients with failed back surgery syndrome or complex regional pain syndrome: A systematic review of effectiveness and complications. *Pain*. 2004;108:137-147.

2025.   Kumar K, Taylor RS, Jacques L, et al. Spinal cord stimulation versus conventional medical management for neuropathic pain: a multicentre randomised controlled trial in patients with failed back surgery syndrome. *Pain*. 2007;132:179-188.

2026.   North RB, Kidd DH, Farrokhi F, Piantadosi SA. Spinal cord stimulation versus repeated lumbosacral spine surgery for chronic pain: a randomized, controlled trial. *Neurosurgery*. 2005;56:98-106; discussion 106-107.

2027.   Turner JA, Hollingworth W, Comstock BA, Deyo RA. Spinal cord stimulation for failed back surgery syndrome: outcomes in a workers' compensation setting. *Pain*. 2010;148:14-25.

2028.   Kemler MA, de Vet HC, Barendse GA, van den Wildenberg FA, van Kleef M. Spinal cord stimulation for chronic reflex sympathetic dystrophy--five-year follow-up. *N Engl J Med*. 2006;354:2394-2396.

2029.   Ohnmeiss DD, Rashbaum RF, Bogdanffy GM. Prospective outcome evaluation of spinal cord stimulation in patients with intractable leg pain. *Spine*. 1996;21:1344-1350; discussion 1351.

2030.   North RB, Kidd DH, Zahurak M, James CS, Long DM. Spinal cord stimulation for chronic, intractable pain: experience over two decades. *Neurosurgery*. 1993;32:384-394; discussion 394-385.

2031.   Turner JA, Loeser JD, Bell KG. Spinal cord stimulation for chronic low back pain: a systematic literature synthesis. *Neurosurgery*. 1995;37:1088-1095; discussion 1095-1086.

2032.   Bernacki EJ, Yuspeh L, Tao X. Determinants of escalating costs in low risk workers' compensation claims. *Journal of occupational and environmental medicine / American College of Occupational and Environmental Medicine*. 2007;49:780-790.

2392.   Gomes, T., D. N. Juurlink, T. Antoniou, M. M. Mamdani, J. M. Paterson and W. van den Brink (2017). "Gabapentin, opioids, and the risk of opioid-related death: A population-based nested case–control study." PLoS medicine 14(10): e1002396.

2393.   Mannion, A. F., J. I. Brox and J. C. Fairbank (2013). "Comparison of spinal fusion and nonoperative treatment in patients with chronic low back pain: long-term follow-up of three randomized controlled trials." The Spine Journal 13(11): 1438-1448.

2394.   Mannion, A. F., G. Leivseth, J.-I. Brox, P. Fritzell, O. Hägg and J. C. Fairbank (2014). "ISSLS Prize winner: long-term follow-up suggests spinal fusion is associated with increased adjacent segment disc degeneration but without influence on clinical outcome: results of a combined follow-up from 4 randomized controlled trials." Spine 39(17): 1373-1383.

2395.   Mannion, A. F., J.-I. Brox and J. C. Fairbank (2016). "Consensus at last! Long-term results of all randomized controlled trials show that fusion is no better than non-operative care in improving pain and disability in chronic low back pain." The Spine Journal 16(5): 588-590.

2396.   Froholdt, A., I. Holm, A. Keller, R. B. Gunderson, O. Reikeraas and J. I. Brox (2011). "No difference in long-term trunk muscle strength, cross-sectional area, and density in patients with chronic low back pain 7 to 11 years after lumbar fusion versus cognitive intervention and exercises." The Spine Journal 11(8): 718-725.

2397.   Froholdt, A., O. Reikeraas, I. Holm, A. Keller and J. I. Brox (2012). "No difference in 9-year outcome in CLBP patients randomized to lumbar fusion versus cognitive intervention and exercises." European Spine Journal 21(12): 2531-2538.

2398.   Hedlund, R., C. Johansson, O. Hägg, P. Fritzell, T. Tullberg and S. L. S. S. Group (2016). "The long-term outcome of lumbar fusion in the Swedish lumbar spine study." The Spine Journal 16(5): 579-587.

2399.   Xie, L., Z.-G. Zhao, S.-J. Zhang and Y.-B. Hu (2017). "Percutaneous vertebroplasty versus conservative treatment for osteoporotic vertebral compression fractures: An updated meta-analysis of prospective randomized controlled trials." International Journal of Surgery 47: 25-32.

2400.   Zhao, S., C.-y. Xu, A.-r. Zhu, L. Ye, L.-l. Lv, L. Chen, Q. Huang and F. Niu (2017). "Comparison of the efficacy and safety of 3 treatments for patients with osteoporotic vertebral compression fractures: A network meta-analysis." Medicine 96(26).

2401.   Jacobson, R. E., O. Palea and M. Granville (2017). "Progression of Vertebral Compression Fractures After Previous Vertebral Augmentation: Technical Reasons for Recurrent Fractures in a Previously Treated Vertebra." Cureus 9(10).

2402.   Li, Y.-x., D.-q. Guo, S.-c. Zhang, D. Liang, K. Yuan, G.-y. Mo, D.-x. Li, H.-z. Guo, Y. Tang and P.-j. Luo (2018). "Risk factor analysis for re-collapse of cemented vertebrae after percutaneous vertebroplasty (PVP) or percutaneous kyphoplasty (PKP)." International Orthopaedics: 1-9.

2403.   Shanthanna, H., I. Gilron, M. Rajarathinam, R. AlAmri, S. Kamath, L. Thabane, P. J. Devereaux and M. Bhandari (2017). "Benefits and safety of gabapentinoids in chronic low back pain: A systematic review and meta-analysis of randomized controlled trials." PLoS Medicine 14(8): e1002369.

2404.   McCleane, G. J. (2001). "Does gabapentin have an analgesic effect on background, movement and referred pain? A randomised, double-blind, placebo controlled study." The Pain Clinic 13(2): 103-107.

2405.   Atkinson, J. H., M. A. Slater, E. V. Capparelli, S. M. Patel, T. Wolfson, A. Gamst, I. S. Abramson, M. S. Wallace, S. D. Funk and T. R. Rutledge (2016). "A randomized controlled trial of gabapentin for chronic low back pain with and without a radiating component." Pain 157(7): 1499.

2406.   Mathieson, S., C. G. Maher, A. J. McLachlan, J. Latimer, B. W. Koes, M. J. Hancock, I. Harris, R. O. Day, L. Billot and J. Pik (2017). "Trial of pregabalin for acute and chronic sciatica." New England Journal of Medicine 376(12): 1111-1120.

2407.   Chou, R., R. Hashimoto, J. Friedly, R. Fu, T. Dana, S. Sullivan, C. Bougatsos and J. Jarvik (2015). "Pain management injection therapies for low back pain." Rockville, MD: Agency for Healthcare Research and Quality.

2408. Ribeiro, L. H., R. N. V. Furtado, M. S. Konai, A. B. Andreo, A. Rosenfeld and J. Natour (2013). "Effect of facet joint injection versus systemic steroids in low back pain: a randomized controlled trial." Spine 38(23): 1995-2002.

2409. Freeman, B. J., G. L. Ludbrook, S. Hall, M. Cousins, B. Mitchell, M. Jaros, M. Wyand and J. R. Gorman (2013). "Randomized, double-blind, placebo-controlled, trial of transforaminal epidural etanercept for the treatment of symptomatic lumbar disc herniation." Spine 38(23): 1986-1994.

2410. Friedly, J. L., B. A. Comstock, J. A. Turner, P. J. Heagerty, R. A. Deyo, Z. Bauer, A. L. Avins, S. S. Nedeljkovic, D. R. Nerenz and X. R. Shi (2017). "Long-Term Effects of Repeated Injections of Local Anesthetic With or Without Corticosteroid for Lumbar Spinal Stenosis: A Randomized Trial." Archives of Physical Medicine and Rehabilitation 98(8): 1499-1507.

2411. Carette, S., S. Marcoux, R. Truchon, C. Grondin, J. Gagnon, Y. Allard and M. Latulippe (1991). "A controlled trial of corticosteroid injections into facet joints for chronic low back pain." New England Journal of Medicine 325(14): 1002-1007.

2412. Marks, R. C., T. Houston and T. Thulbourne (1992). "Facet joint injection and facet nerve block: a randomised comparison in 86 patients with chronic low back pain." Pain 49(3): 325-328.

2413. Lilius, G., E. Laasonen, P. Myllynen, A. Harilainen and G. Gronlund (1989). "Lumbar facet joint syndrome. A randomised clinical trial." The Journal of Bone and Joint Surgery. British volume 71(4): 681-684.

2414. Sae-Jung, S. and K. Jirarattanaphochai (2016). "Outcomes of lumbar facet syndrome treated with oral diclofenac or methylprednisolone facet injection: a randomized trial." International Orthopaedics 40(6): 1091-1098.

2415. Murata, Y., Y. Kato, K. Miyamoto and K. Takahashi (2009). "Clinical study of low back pain and radicular pain pathways by using l2 spinal nerve root infiltration: a randomized, controlled, clinical trial." Spine 34(19): 2008-2013.

2416. Lakemeier, S., M. Lind, W. Schultz, S. Fuchs-Winkelmann, N. Timmesfeld, C. Foelsch and C. D. Peterlein (2013). "A comparison of intraarticular lumbar facet joint steroid injections and lumbar facet joint radiofrequency denervation in the treatment of low back pain: a randomized, controlled, double-blind trial." Anesthesia & Analgesia 117(1): 228-235.

2417. Do, K. H., S. H. Ahn, Y. W. Cho and M. C. Chang (2017). "Comparison of intra-articular lumbar facet joint pulsed radiofrequency and intra-articular lumbar facet joint corticosteroid injection for management of lumbar facet joint pain: A randomized controlled trial." Medicine 96(13).

2418. Rauck, R., R. J. Coffey, D. M. Schultz, M. S. Wallace, L. R. Webster, S. E. McCarville, E. J. Grigsby and L. M. Page (2013). "Intrathecal gabapentin to treat chronic intractable noncancer pain." Anesthesiology: The Journal of the American Society of Anesthesiologists 119(3): 675-686.

2419. Kumar, K., G. Hunter and D. D. Demeria (2002). "Treatment of chronic pain by using intrathecal drug therapy compared with conventional pain therapies: a cost-effectiveness analysis." Journal of neurosurgery 97(4): 803-810.

2420. Coffey, R. J., M. L. Owens, S. K. Broste, M. Y. Dubois, F. M. Ferrante, D. M. Schultz, L. J. Stearns and M. S. Turner (2009). "Mortality associated with implantation and management of intrathecal opioid drug infusion systems to treat noncancer pain." The Journal of the American Society of Anesthesiologists 111(4): 881-891.

2421. Koh, W., S.-S. Choi, M. H. Karm, J. H. Suh, J. G. Leem, J. D. Lee, Y. K. Kim and J. Shin (2015). "Treatment of chronic lumbosacral radicular pain using adjuvant pulsed radiofrequency: a randomized controlled study." Pain Medicine 16(3): 432-441.

2422. Juch, J. N., E. T. Maas, R. W. Ostelo, J. G. Groeneweg, J.-W. Kallewaard, B. W. Koes, A. P. Verhagen, J. M. van Dongen, F. J. Huygen and M. W. van Tulder (2017). "Effect of radiofrequency denervation on pain intensity among patients with chronic low back pain: the Mint randomized clinical trials." Jama 318(1): 68-81.

2423. Cohen, S. P., H. Hameed, C. Kurihara, P. F. Pasquina, A. M. Patel, M. Babade, S. R. Griffith, M. E. Erdek, D. E. Jamison and R. W. Hurley (2014). "The effect of sedation on the accuracy and treatment outcomes for diagnostic injections: A randomized, controlled, crossover study." Pain Medicine 15(4): 588-602.

2424. Phan, K., J. Xu, K. Schultz, M. A. Alvi, V. M. Lu, P. Kerezoudis, P. R. Maloney, M. E. Murphy, R. J. Mobbs and M. Bydon (2017). "Full-endoscopic versus micro-endoscopic and open discectomy: A systematic review and meta-analysis of outcomes and complications." Clinical Neurology and Neurosurgery 154: 1-12.

2425. Dorow, M., M. Löbner, J. Stein, A. Konnopka, H. J. Meisel, L. Günther, J. Meixensberger, K. Stengler, H.-H. König and S. G. Riedel-Heller (2017). "Risk Factors for Postoperative Pain Intensity in Patients Undergoing Lumbar Disc Surgery: A Systematic Review." PloS One 12(1): e0170303.

2426. Deyo RA, Nachemson A, Mirza SK. Spinal-fusion surgery - the case for restraint. N Engl J Med. 2004;350(7):722-6.

2427. Johnsen, L., P. Brinckmann, C. Hellum, I. Rossvoll and G. Leivseth (2013). "Segmental mobility, disc height and patient-reported outcomes after surgery for degenerative disc disease." Bone Joint J 95(1): 81-89.

2428. Zigler, J. E., J. Glenn and R. B. Delamarter (2012). "Five-year adjacent-level degenerative changes in patients with single-level disease treated using lumbar total disc replacement with ProDisc-L versus circumferential fusion." Journal of Neurosurgery: Spine 17(6): 504-511.

2429. Sköld, C., H. Tropp and S. Berg (2013). "Five-year follow-up of total disc replacement compared to fusion: a randomized controlled trial." European Spine Journal 22(10): 2288-2295.

2430.   Kroon, F., M. Staples, P. R. Ebeling, J. D. Wark, R. H. Osborne, P. J. Mitchell, C. H. Wriedt and R. Buchbinder (2014). "Two-Year Results of a Randomized Placebo-Controlled Trial of Vertebroplasty for Acute Osteoporotic Vertebral Fractures." Journal of Bone and Mineral Research 29(6): 1346-1355.

2431.   Polly, D., D. Cher, P. G. Whang, C. Frank, J. Sembrano and I. S. Group (2016). "Does level of response to SI joint block predict response to SI joint fusion?" International Journal of Spine Surgery 10.

2432.   Polly, D. W., J. Swofford, P. G. Whang, C. J. Frank, J. A. Glaser, R. P. Limoni, D. J. Cher, K. D. Wine, J. N. Sembrano and I. S. Group (2016). "Two-year outcomes from a randomized controlled trial of minimally invasive sacroiliac joint fusion vs. non-surgical management for sacroiliac joint dysfunction." International Journal of Spine Surgery 10.

2433.   Duhon, B. S., D. J. Cher, K. D. Wine, D. A. Kovalsky, H. Lockstadt and S. S. Group (2016). "Triangular titanium implants for minimally invasive sacroiliac joint fusion: a prospective study." Global Spine Journal 6(03): 257-269.

2434.   Sachs, D., R. Capobianco, D. Cher, T. Holt, M. Gundanna, T. Graven, A.N. Shamie, and J. Cummings (2014). "One-year outcomes after minimally invasive sacroiliac joint fusion with a series of triangular implants: a multicenter, patient-level analysis." Med Devices (Auckl) 7: 299–304.

2435.   Vanaclocha, V., J.M. Herrera, N. Saiz-Sapena, M. Rivera-Paz, and F. Verdu-Lopez. (2018). "Minimally invasive sacroiliac joint fusion, radiofrequency denervation, and conservative management for sacroiliac joint Pain: 6-Year comparative case series." Neurosurgery 82(1): 48-55.

2436.   Lorio, M.P. (2016). "ISASS Policy 2016 Update – Minimally invasive sacroiliac joint fusion." Int J Spine Surg 10: 26.

2437.   North American Spine Society. (2015). "Coverage Recommendations." Available at https://www.spine.org/policypractice/coveragerecommendations/aboutcoveragerecommendations.

2438.   Whang, P., D. Cher, C. F. David Polly, H. Lockstadt, J. Glaser, R. Limoni and J. Sembrano (2015). "Sacroiliac joint fusion using triangular titanium implants vs. non-surgical management: six-month outcomes from a prospective randomized controlled trial." International Journal of Spine Surgery 9.

2439.   Dengler, J., B. Duhon, P. Whang, C. Frank, J. Glaser, B. Sturesson, et al. (2017). "Predictors of outcome in conservative and minimally invasive surgical management of pain originating from the sacroiliac joint: a pooled analysis." Spine (Phila Pa 1976) 42(21): 1664–1673.

2440.   Sturesson, B., D. Kools, R. Pflugmacher, A. Gasbarrini, D. Prestamburgo, and J. Dengler. (2017). "Six-month outcomes from a randomized controlled trial of minimally invasive SI joint fusion with triangular titanium implants vs conservative management." Eur Spine J. 26(3): 708-719.

2441.   Sitzman, B.T., and D.A. Provenzano. (2017). "Best practices in spinal cord stimulation." Spine (Phila Pa 1976) 42 Suppl 14: S67-S71.

2442.   Manca, A., K. Kumar, R.S. Taylor, L. Jacques, S. Eldabe, M. Meglio, et al. (2008). "Quality of life, resource consumption and costs of spinal cord stimulation versus conventional medical management in neuropathic pain patients with failed back surgery syndrome (PROCESS trial)." Eur J Pain 12(8): 1047-1058.

2443.   Grider, J., L. Manchikanti, A. Carayannopoulos, M. Sharma, C. Balog, M. Harned, V. Grami, R. Justiz, K. Nouri and S. Hayek (2016). "Effectiveness of Spinal Cord Stimulation in Chronic Spinal Pain: A Systematic Review." Pain physician 19(1): E33-54.

2444.   Frey, M., L. Manchikanti, R. Benyamin, D. Schultz, H. Smith and S. Cohen (2009). "Spinal cord stimulation for patients with failed back surgery syndrome: a systematic review." Pain physician 12(2): 379-397.

2445.   Muhammad, S., S. Roeske, S. R. Chaudhry and T. Mehari Kinfe (2017). "Burst or High-Frequency (10 kHz) Spinal Cord Stimulation in Failed Back Surgery Syndrome Patients With Predominant Back Pain: One Year Comparative Data." Neuromodulation: Technology at the Neural Interface.

2446.   Bicket, M. C., R. Y. Dunn and S. U. Ahmed (2016). "High-frequency spinal cord stimulation for chronic pain: pre-clinical overview and systematic review of controlled trials." Pain Medicine 17(12): 2326-2336.

2447.   Hollingworth, W., J. A. Turner, N. J. Welton, B. A. Comstock and R. A. Deyo (2011). "Costs and cost-effectiveness of spinal cord stimulation (SCS) for failed back surgery syndrome: an observational study in a workers' compensation population." Spine 36(24): 2076-2083.

2448.   Kapural, L., B. Vrooman, S. Sarwar, L. Krizanac-Bengez, R. Rauck, C. Gilmore, J. North and N. Mekhail (2015). "Radiofrequency intradiscal biacuplasty for treatment of discogenic lower back pain: a 12-month follow-up." Pain Medicine 16(3): 425-431.

2449.   Taylor, R. S., M. J. Desai, P. Rigoard and R. J. Taylor (2014). "Predictors of Pain Relief Following Spinal Cord Stimulation in Chronic Back and Leg Pain and Failed Back Surgery Syndrome: A Systematic Review and Meta-Regression Analysis." Pain Practice 14(6): 489-505.

2450.   Lazaro, R. (2015). "Electromyography in musculoskeletal pain: A reappraisal and practical considerations." Surgical neurology international 6.

2451.   Chiodo, A., A. J. Haig, K. S. Yamakawa, D. Quint, H. Tong and V. R. Choksi (2007). "Needle EMG has a lower false positive rate than MRI in asymptomatic older adults being evaluated for lumbar spinal stenosis." Clinical Neurophysiology 118(4): 751-756.

2452.   Haig, A. J., H. C. Tong, K. S. Yamakawa, D. J. Quint, J. T. Hoff, A. Chiodo, J. A. Miner, V. R. Choksi and M. E. Geisser (2005). "The sensitivity and specificity of electrodiagnostic testing for the clinical syndrome of lumbar spinal stenosis." Spine 30(23): 2667-2676.

2453.   Hasankhani, E. and F. Omidi-Kashani (2013). "Magnetic resonance imaging versus electrophysiologic tests in clinical diagnosis of lower extremity radicular pain." ISRN Neuroscience 2013.

2454.   Tong, H. C. (2012). "Incremental ability of needle electromyography to detect radiculopathy in patients with radiating low back pain using different diagnostic criteria." Archives of Physical Medicine and Rehabilitation 93(6): 990-992.

2455.   Yagci, I., O. H. Gunduz, G. Ekinci, D. Diracoglu, O. Us and G. Akyuz (2009). "The utility of lumbar paraspinal mapping in the diagnosis of lumbar spinal stenosis." American journal of physical medicine & rehabilitation 88(10): 843-851.

2456.   Coster, S., S. F. De Bruijn and D. L. Tavy (2010). "Diagnostic value of history, physical examination and needle electromyography in diagnosing lumbosacral radiculopathy." Journal of Neurology 257(3): 332-337.