Review Article

Low Back Pain

Rohitha Jayamaha, Specialist Physician and Fellow Interventional Pain Practice
Provincial General Hospital, Badulla

Low back pain(LBP) is extremely common. Even if we don't have our own data, in the U.S., nonspecific mechanical low back pain is the fifth most common reason for all physician visits, and the second most common symptomatic reason, accounting for approximately 2.3% of all physician visits.[1]The most frequently quoted epidemiological studies cite lifetime adult prevalence rates varying from 50% to 80%, and point prevalence rates from 15% to 30%.[2]

Back pain can be classified into acute (less than 4 weeks), subacute (between 4 weeks and 3 months) or chronic (greater than 3 months) according to time durations.

Box 1
Causes of LBP [3]

1. **Mechanical (80-90%) – pain mainly confined to back**
   - 1.1. Unknown cause—usually attributed to muscle strain (myofascial) or ligamentous injury (65%-70%)
   - 1.2. Degenerative disc or joint disease
   - 1.3. Vertebral fracture
   - 1.4. Congenital deformity (eg. scoliosis, kyphosis, transitional vertebrae)
   - 1.5. Spondylolysis
   - 1.6. Instability
2. **Neurogenic (5-15%) – mainly radicular pain ± back pain**
   - 2.1. Herniated disc
   - 2.2. Annular fissure with chemical irritation of nerve root
   - 2.3. Spinal stenosis
   - 2.4. Osteophytic nerve root compression
   - 2.5. Failed back surgery syndrome (eg. arachnoiditis, epidural adhesions, recurrent herniation); may also cause mechanical back pain
   - 2.6. Infection (eg. herpes zoster and post herpetic neuralgia)
3. **Non-mechanical spinal conditions (1-2%)**
   - 3.1. Neoplastic (eg. primary or metastatic) disease
   - 3.2. Infection (eg. osteomyelitis, discitis, abscess)
   - 3.3. Inflammatory arthritis (eg. rheumatoid arthritis and spondyloarthropathies, including ankylosing spondylitis, reactive arthritis, enteropathic arthritis)
   - 3.4. Paget's disease
   - 3.5. Other (eg. Scheuermann's disease, Baastrup's disease)
4. **Referred visceral pain (1-2%)**
   - 4.1. Gastrointestinal disease (eg. inflammatory bowel disease, pancreatitis, diverticulitis)
   - 4.2. Renal disease (eg nephrolithiasis, pyelonephritis)
   - 4.3. Abdominal aortic aneurysm
5. **Other (2-4%)**
   - 5.1. Fibromyalgia
   - 5.2. Somatoform disorder (such as somatization disorder, pain disorder)
   - 5.3. Malingering

As seen in Box 1 the origin of pain can be broadly classified as mechanical, neuropathic, or secondary to another cause. Mechanical back pain implies that the source of pain is in the spine or its supporting structures. Neuropathic back pain denotes the presence of symptoms that stem from irritation of a nerve root(s).

History

There are several ways to distinguish mechanical (musculoskeletal) from neuropathic (nerve root) low back pain in history taking. Patients with neuropathic pains are more likely to describe a radicular type "shooting" / "stabbing" pain. Where as mechanical/musculoskeletal pains are described as "throbbing" or "aching" pains.

Sometimes a mechanical/musculoskeletal pain may radiate into the upper thigh and buttocks but extension below the knee is less common than with radicular pain.

Several instruments (eg. painDITECT questionnaire) can facilitate distinguishing neuropathic from nociceptive pain.[3,4]

The rationale for distinguishing between neuropathic and non-neuropathic back pain is that mechanistically basedpain treatments may be more effective than aetiologically based treatments.

Mechanical causes of back pain, including muscle strains, are typically worsened with movement and improved by rest. In patients with disc disorders, prolonged sitting or forward flexion may aggravate symptoms. Where as pains arising from Zygapophyseal Joints (Z joints) are worsened on spinal extension. The pain associated with spinal stenosis is classically relieved by forward flexion, and worsened with extension. These patients can often walk up hills or ride a bicycle with minimal difficulty. Sensory

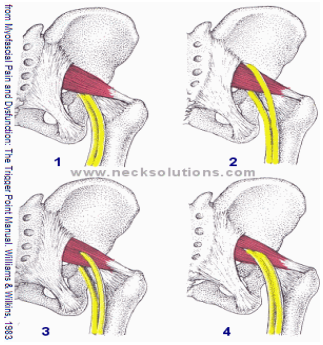
changes such as tingling and numbness may indicate lumbosacral radiculopathy.

Although episodes of serious low back pain are as likely to begin during activities of daily living as after minor trauma, a precipitating event can occasionally help pinpoint a pain source. Among the various aetiologies of mechanical low back pain, sacroiliac joint pain is most often associated with a traumatic event such as a fall or motor vehicle collision.[5] In patients presenting with a neuropathic pain, a herniated disc is more likely than spinal stenosis to be associated with an abrupt onset and specific inciting event.

Examination

A physical examination is generally used to direct further investigation, but is rarely diagnostic for a specific aetiology. The physical examination is performed with special attention to spinal and pelvic girdle symmetry, posture, and spinal and lower extremity flexibility. Palpation can assess for both bony and soft tissue tenderness, hypertonicity, and spasm. Abdominal palpation and auscultation are performed for suspicion of possible abdominal process, such as aortic aneurysm. The hip joint should also be checked, particularly for complaints of buttock, hip region, or groin pain. Neurologic examination of the lower extremities is done; however, findings are rare in non-radiating pain.

In a systematic review, the straight leg raising test was found to be the most sensitive sign for radiculopathy, but it was limited by low specificity (pooled sensitivity 0.85, specificity 0.52).[6] Similar analyses conducted for range of motion have generally found them to be limited by low to moderate inter-examiner reliability and a poor relation with functional impairment.[7]Spinal palpation is often used to evaluate low back pain. Compared with motion assessment, palpation has been found in systematic reviews to have better reliability,[8] but neither test has proved benefit in directing clinical care or establishing a diagnosis. For suspected sacroiliac joint pain and facet arthropathy, no history or physical examination sign is reliably predictive of response to diagnostic injections.



Piriformis syndrome is characterized by pain and instability. The location of the pain is often imprecise, but it is often present in the hip, coccyx, buttock, groin, or distal part of the leg. The history and physical findings are key elements in differentiating the more common forms of LBP and piriformis syndrome. The literature and general knowledge on piriformis syndrome is limited, compared with that of sciatica or disc herniation. The sciatic nerve exits the pelvis via 4 routes: (1) The nerve passes anteriorly to the piriformis between the rims of the greater sciatic foramen. (2) The peroneal portion of the sciatic nerve passes through the piriformis; the tibial portion passes anterior to the piriformis muscle. (3) The peroneal branch of the sciatic nerve loops above and posterior to the piriformis muscle, whereas the tibial branch passes anterior to the piriformis muscle. (4) The undivided sciatic nerve penetrates the piriformis muscle.

Piriformis syndrome is a diagnosis of exclusion. At physical examination, the most important factor that differentiates sciatic pain from piriformis syndrome is the absence of neurologic deficit in piriformis syndrome. Herniation or disc compression results in intraneural derangement of the nerve root structure, whereas piriformis syndrome causes a qualitative epineural irritation. In piriformis syndrome, the only true-positive sign is tenderness over the gluteal region.The pain can be reproduced with maximum elongation of the piriformis muscle in flexion, adduction, and internal rotation (FlAIR test) of the hip. Sometimes weakness can be observed with resisted external rotation and abduction of the hip.

After the history taking and examination following important points should be picked up for further revaluation.

1. Features suggestive of nerve root involvement

- Leg pain greater than back pain
- Radiation into foot or lower leg
- Numbness and paraesthesias in dermatomal distribution
- Diminished leg reflexes
- Positive straight leg raising test (L4-S1 nerve roots)
- Positive femoral stretch test (L2-L4 nerve roots)
- Leg pain exacerbated by LSCS - laughing, sneezing, coughing, straining (Valsalva manoeuvre)

2. Features suggestive of serious underlying disorders **"red flag"** signs and symptoms

Age >50 years	Metastases, vertebral fractures, herpes zoster, and life threatening conditions such as aortic rupture or perforated bowel
Age <20	May suggest congenital anomalies (such as spina bifida), early onset disorders (such as Scheuermann's disease), or conditions associated with substance misuse (osteomyelitis)
Trauma	Vertebral fractures, sacroiliac joint pain
Systemic illness	Vertebral fractures, spinal infections, and metastases
Constitutional symptoms	Metastases and spinal infections
Immunosuppression or steroid use	May predispose patients to infectious process, malignancy, or vertebral fractures
Widespread neurological symptoms	Cauda equina syndrome, myelopathy, multiple sclerosis
Unrelenting pain	Psychogenic pain or somatoform disorder, malingering, malignancy, life threatening abdominal pathology

3. Features suggestive of chronicity; the **"yellow flags"** (Box 2)

Box 2
Clinical factors
• Previous episodes of back pain
• Multiple previous musculoskeletal complaints
• Hysteria or hypochodriasis
• Alcohol, drugs, cigarettes
• Waddell signs$
Pain experience
• Rates pain as severe
• Blames others for pain
• Legal issues or compensation
Premorbid factors
• Rates job as physically demanding
• Patient believes he or she will not be working in 6 months
• Not getting along with supervisors
• Near retirement
• Spouse too supportive
• Unmarried or have been married multiple times
• Low socioeconomic status
• Troubled childhood (abuse, parental death, alcohol, difficult divorce)

Among patients evaluated for low back pain in primary care setting, 80-90% will no longer seek care after three months. However, recent studies suggest that 30-40% may continue to experience persistent symptoms.[13]Numerous studies have tried to identify predictors of episodes of acute low back pain, and the transition from acute to chronic pain and disability.
In general, some psychological factors (such as coexisting depression and anxiety; coping mechanisms and attitudes; work related stress and job satisfaction; and perceived health and activity levels) in history and some physical findings (such as waddle signs) in examination are associated with the development and persistence of back pain.

$Waddell signs
1. Superficial and Widespread tenderness or Nonanatomic tenderness. (Skin discomfort on light palpation or tenderness crossing over non-anatomical boundaries)
2. Stimulation tests: Axial loading and Pain on simulated rotation. (eliciting pain when pressing down on the top of the patient's head or rotating the shoulders and pelvis together should not be painful)
3. Distracted straight leg raise. (if a patient complains of pain on straight leg raise, but not if the examiner extends the knee with the patient seated at another time during the initial evaluation)
4. Non-anatomic sensory changes: Regional sensory changes and Regional weakness.(sensory loss in an entire extremity or side of the body or weakness that is non consistent and jerky, ie "cogwheeling")
5. Overreaction. (Exaggerated painful response to a stimulus, that is not reproduced when the same stimulus is given later)
If there are more than 3 of 5 present then there is high probability that patient has non-organic pain.

Imaging

The utility of diagnostic imaging for back pain in the absence of major structural abnormalities (such as tumour or infection) is limited by the high prevalence of degenerative disorders in asymptomatic adults. About 30% of adults without low back pain have evidence of a protruded disc on magnetic resonance imaging, over half have bulging or degenerative discs, and a fifth have annular fissures.[11] In acute low back pain plain radiographs have failed to show a benefit for early imaging, although patients who have radiography may have higher satisfaction rates.[8] Prospective studies evaluating early magnetic resonance imaging MRI) and other imaging methods in patients with low back pain (regardless of whether they have radicular symptoms) have also failed to show benefit.[9] Therefore it is recommended, imaging for low back pain **only when severe or progressive neurological deficits are present**, **when a serious underlying condition is suspected**, or **when evaluating patients for surgery or interventional pain procedures (IPM)**. When evaluating disc disorders or

neurological symptoms or ruling out vertebral fractures or metastases, magnetic resonance imaging without contrast is the most sensitive method.[12]

Management

Most people with low back pain do not seek medical care. Many self-treat with over the counter medications and lifestyle changes.[11]Most cases of acute, non- specific low back pain resolve within **two weeks**.

Reassurance and counselling patients to stay active are cornerstones of treating such pain, though some may benefit from short-term pharmacotherapy. A Cochrane review found that advice to stay active had a small but consistently beneficial effect for pain reduction and functional improvement compared with bed rest in patients with acute, non-specific back pain.[15]

For sciatica, the same authors found high quality evidence that bed rest has little or no effect on functional status or pain.[15] In patients with persistent pain with or without radiculopathy, a multimodal treatment regimen that includes a regular exercise programme, weight loss, and if indicated, psychotherapy, medications, and injections can be beneficial.[12] When the pain is the result of a serious systemic cause (such as cancer), symptom palliation should be started concurrently with primary treatment.

Pharmacotherapy
Several systematic reviews have concluded that strong evidence supports the use of non-steroidal anti-inflammatory drugs for non-neuropathic low back pain, though the treatment effect is small and the evidence is greater for acute than chronic pain.

Paracetamol (acetaminophen) is slightly less effective than non-steroidal anti-inflammatory drugs but has fewer or less severe side effects. Minimal evidence exists that non-steroidal anti-inflammatory drugs are effective for radiculopathy, or that one drug is better than others.[12]

In patients with acute non-specific back pain, strong evidence exists to support a small effect size for non-benzodiazepine muscle relaxants (such as Tizanidine), and weaker evidence exists to support benzodiazepines (such as diazepam and clonazepam).[16] Given the side effect profile of benzodiazepines and their potential for addiction, many experts believe benzodiazepines should be prescribed only when other muscle relaxants have proved ineffective, and with clearly defined goals and time frames.[12] For chronic low back pain the evidence supporting muscle relaxants is less convincing.[14]

Most but not all systematic reviews have found that tricyclic antidepressants, but not selective serotonin reuptake inhibitors, are more effective thanplacebo for chronic, non-specific low back pain. For neuropathic pain, the number needed to treat for one patient to obtain significant relief with selective serotonin reuptake inhibitors is more than three times higher than the number for tricyclic antidepressants; the efficacy for serotonin and noradrenaline (norepinephrine) reuptake inhibitors falls between that for these two drug classes.[16]

Scant evidence exists to support any drug class for radiculopathy, but two studies have shown a small benefit for gabapentin.[18,19] Opioids are generally regarded as a reasonable option for some episodes of acute back pain, but the evidence for use in chronic low back pain is unclear. In meta-analysis it was concluded that, although opioids can provide short term relief in some patients with chronic low back pain, their long term benefits remain unproved.[17] If opioids are used for chronic low back pain or other non- malignant conditions, many guidelines advocate their use only when more conservative treatments have failed, in conjunction with risk assessment tools and an opioid contract, and with clearly defined goals and exit strategies.[20]

Alternative therapies

Physicians are increasingly referring patients for complementary and alternative medical treatments, with some studies showing that more than half of primary care doctors routinely recommend or prescribe them for backache.[18] In practice guidelines published jointly by the American College of Physicians and the American Pain Society, fair to good evidence is cited supporting numerous alternative treatments for chronic and subacute (1 – 3 months) low back pain, including acupuncture, yoga, massage, spinal manipulation, and functional restoration.[18] For acute, non-specific back pain (myofascial and ligament sprains), evidence of efficacy was found only for spinal manipulation and superficial heat. Evidence was insufficient to fully evaluate any therapy for radiculopathy or to support one effective treatment over another.

Spinal manipulation	Manual therapy designed to maximise painless movement, reduce muscle tightness, improve joint mobility, and correct alignment problems	Superior to sham therapy for patients with acute and chronic (axial and radicular) low back pain. It is no more effective than conventional or other alternative treatments
Acupuncture	Inserting needles into the skin at various anatomical locations to reduce pain or induce anesthesia. Needles may be manipulated manually or through electrical stimulation	The benefits of acupuncture for acute low back pain are unclear. For chronic pain, acupuncture is more effective in the short term for pain relief and functional improvement compared with no treatment or sham treatment. It is no more effective than conventional or other alternative treatments
Massage therapy	The manipulation of muscle and connective tissue to enhance function and promote relaxation and wellbeing	May provide short term relief for subacute and chronic non- specific low back pain
Exercise therapy	Active or passive physical exercises designed to strengthen or stabilise the spine, which may reduce pain, prevent injuries, and improve posture and body mechanics	Stronger evidence for chronic than acute non-specific low back pain. May facilitate return to work, but no evidence for prevention of work injuries. No clear evidence supporting one technique over another
Other therapies	Includes interferential therapy, low level laser therapy, shortwave diathermy, electrical muscle stimulation, transcutaneous electrical nerve stimulation (TENS), yoga, ultrasonography, heat/cold, and traction.	Weak evidence to support TENS for short-term pain relief. No evidence to support the use of traction. For other modalities, there is insufficient evidence to support their use for acute or chronic low back pain

Also importantly, many evidence based guidelines found that **lumbar corset and support belts** had not been proven beneficial for treating patients with **acute and chronic** low back problems.[1]There was insufficient evidence to judge the relative effectiveness mattress types in patients with acute low back pain. For chronic low back pain, one higher-quality trial found a firm mattress slightly inferior to a medium-firm mattress for pain-related disability and pain while in bed. Anyway there were no differences in other pain outcomes. [1]

Interventional Pain Procedures (IPP)

In patients whose symptoms last longer than **six weeks**, IPP may offer **diagnostic and therapeutic** benefits. Low back pain is best treated based on diagnosis and the available evidence, with various interventional pain therapies including epidural injections, percutaneous adhesiolysis, intradiscal therapy or annular thermal therapy, and mechanical disc decompression for disc-related pain(either discogenic or secondary to disc herniation, radiculitis, spinal stenosis, or failed back surgery syndrome (FBSS)). Facet joint interventions and sacroiliac joint interventions are utilized in managing facet joint and sacroiliac joint pain. Following is a summary of currently available evidence for such procedure.[22]

1. The evidence for accuracy of **diagnostic** selective nerve root blocks is **limited**; whereas for lumbar provocation discography, it is **fair.**
2. The evidence for **diagnostic** lumbar **facet joint nerve blocks** (medial branch blocks) and **diagnostic sacroiliac interarticularis injections** is **good** with 75% to 100% pain relief as criterion standard with controlled local anesthetic or placebo blocks.
3. The evidence is **good** in managing **disc herniation or radiculitis** for **caudal and transforaminal epidural injections**; **fair** for axial or discogenic pain **without disc herniation, radiculitis** or **facet joint pain** with **caudal, and interlaminar epidural injections**, and limited for transforaminal epidural injections; fair for spinal stenosis with caudal, interlaminar, and transforaminal epidural injections; and fair for failed back surgery syndrome (FBSS) with caudal epidural injections and limited with transforaminal epidural injections. The evidence is mainly for **subacute than chronic pain**.[23] In a small, randomised, placebo controlled study, **transforaminal epidural steroids reduced the rate of later surgical intervention**.[24]

4. The evidence for **therapeutic facet joint interventions is good** for **conventional radiofrequency**, limited for pulsed radiofrequency, fair to good for lumbar facet joint nerve blocks, and **limited for intra-articular injections**.
5. For **sacroiliac joint** interventions, the evidence for **cooledradiofrequency neurotomy is fair**; **limited for intra-articular injections and peri-articular injections**; and limited for both pulsed radiofrequency and conventional radiofrequency neurotomy.
6. For **lumbar percutaneous adhesiolysis**, the evidence is fair in managing chronic low back and lower extremity pain secondary to **failed back surgery syndrome (FBSS) and spinal stenosis**.
7. For intradiscal procedures for discogenic pain, the evidence for intradiscal electrothermal therapy(IDET) and biaculoplasty is limited to fair and is limited for discTRODE.
8. For **percutaneous disc decompression**, the **evidence is limited** for automated percutaneous lumbar discectomy (APLD), percutaneous lumbar laser disc decompression, and Dekompressor; and limited to fair for nucleoplasty.

For all these interventions, systematic reviews found evidence only if they were performed under image guidance(**fluoroscopically guided)**.[24]**Interventional treatments for axial low back pain are less effective than for radiculopathy.**

Surgery

Surgical interventions for low back pain secondary to major pathologies such as infections, tumours, and fractures are often effective in protecting neurological structures, preventing deformity, and relieving pain.

In patients with persistent radiculopathy resulting from common degenerative conditions, surgery can reduce pain and improve function. For disc herniations without severe neurological deficits, the main benefit of surgery may be a more rapid return of function compared with the natural course.

Compared with non-operative therapy, surgical intervention for spinal stenosis and spondylolisthesis results in superior outcomes, which persist for at least two years after surgery.[25]In patients with chronic low back pain who present with common degenerative changes seen on imaging, surgical interventions (fusion or disc arthroplasty) are less effective. Whether surgery in this group gives much better results than a comprehensive rehabilitation programme with cognitive behavioural therapy is not clear. Only 15-40% of patients can expect a highly functional outcome after surgery in this context and rest might end up with "failed back surgery syndrome" (FBSS) a nightmare situation.

Interdisciplinary rehabilitation (IDR)

IDR is defined by the reviews as an intervention consisting of a **physician's consultation** plus a **psychological, social, or vocational intervention**, or a combination of these. For **chronic low back pain**, intensive interdisciplinary rehabilitation with functional restoration is **moderately more effective** than usual care or non-interdisciplinary rehabilitation for reducing pain and improving function, though effects on work-related outcomes are inconsistent.[1]In higher-risk patients (with yellow flags) with **acute or subacute low back pain**, one higher-quality trial found interdisciplinary rehabilitation moderately more effective than usual care for pain relief, use of analgesic medications, and return to work . [1]

Issues to be addressed

Specific findings from this evidence reviewshave identified several key research gaps:[1]

• Nearly all trials are efficacy trials conducted in ideal setting and selected populations, usually with short-term follow-up. More effectiveness studies assessing long-term outcomes in more generalizable populations are needed to determine the effectiveness of interventions in realworld settings.Additional long-term trials with adequate follow-up and appropriate comparison interventions are needed specifically for patients with chronic non-specific low back pain.
• More research is needed on effective interventions for identification and treatment of 'yellow flags' in order to prevent the development of chronic disabling low back pain.
• The optimal use of combinations of medications has not been well studied. In addition, emerging data on potential cardiovascular risks with non-selective NSAIDs may alter risk- benefit assessments. There is also little evidence on opioids specifically for low back pain. In particular, evidence on long-term use of opioids and risks of abuse and addiction remains sparse.
• Decision tools or classification schemes for matching patients to interventions (such as manipulation, specific exercise regimens or other interventions) that they are more likely to benefit from are promising, but require

additional validation. In addition, currently available tools include assessment of physical exam findings that many primary care clinicians are unfamiliar with or that have uncertain reliability and reproducibility. More research on decision tools or classification schemes that can be reliably used by most clinicians need to be developed and tested in clinical settings.

• For most interventions, data on harms are sparse, with disproportionate attention paid to benefits. Many interventions for low back pain appear to have similar effects on patient outcomes. Higher quality studies of cost-effectiveness could help clarify optimal choices between such interventions.

• There is no evidence on optimal sequencing of interventions, and limited evidence on optimal combinations of interventions. In many cases, combinations of interventions were not much more effective than monotherapy, but more research is needed to clarify when and how treatments should be combined.High quality research on management of failed back surgery syndrome and back pain during pregnancy is lacking and provides little guidance for appropriate management in these populations.

Conclusion

Low back pain is a common clinical problem. According to available evidence, there are many gaps in management of low back pain clinically and logistically. While waiting for more scientific evidence, an interdisciplinary approach with available scientific evidence is deemed cost effective.

References

1. Roger C, Laurie HH. Diagnosis and Treatment of Low Back Pain: A Joint Clinical Practice Guideline from the American College of Physicians and the American Pain Society. Annals of Internal Medicine, 2007; 147:478-491
2. Andersson GB. Epidemiology of low back pain. ActaOrthopScandSuppl1998;281:28-31.
3. Deyo RA, Weinstein JN. Low back pain. N Engl J Med 2001;344:363-70.
4. 3Freynhagen R, Baron R, Gockel U, Tolle TR. painDETECT: a new screening questionnaire to identify neuropathic components in patients with back pain. Curr Med Res Opin2006;22:1911-20.
5. Cohen SP. Sacroiliac joint pain: a comprehensive review of anatomy, diagnosis and treatment. AnesthAnalg2005;101:1440-53.
6. Rubinstein SM, vanTulder M. A best-evidence review of diagnostic procedures for neck and low-back pain. Best Pract Res ClinRheumatol2008;22:471-82.
7. Seffinger MA, Najm WI, Mishra SI, Adams A, Dickerson VM, Murphy LS, et al. Reliability of spinal palpation for diagnosis of back and neck pain. A systematic review of the literature. Spine 2004;29:E413-25.
8. Stochkendahl MJ, Christensen MW, Hartvigsen J, Vach W, Haas M, Hestbaek L, et al. Manual examination of the spine: a systematic critical literature review of reproducibility. J Manipulative PhysiolTher2006;29:475-85.
9. Hancock MJ, Maher CG, Latimer J, Spindler MF, McAuley JH, Laslett M, et al. Systematic review of tests to identify the disc, SIJ or facet joint as the source of low back pain. Eur Spine J 2007;16:1539-50.
10. Cohen SP, Raja SN. Pathogenesis, diagnosis and treatment of lumbar zygapophysial (facet) joint pain. Anesthesiology 2007;106:591-614.
11. Jarvik JG, Deyo RA. Diagnostic evaluation of low back pain with emphasis on imaging. Ann Intern Med 2002;137:586-97.
12. Chou R, Qaseem A, Snow V, Casey D, Cross JT Jr, Shekelle P, et al. Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. Ann Intern Med 2007;147:478-91.
13. Henschke N, Maher CG, Refshauge KM, Herbert RD, Cumming RD, Bleasel J, et al. Prognosis in patients with recent onset low back pain in Australian primary care: inception cohort study. BMJ 2008;337:a171, doi:10.1136/bmj.a171
14. Pincus T, Burton AK, Vogel S, Field AP. A systematic review of psychological factors as predictors of chronicity/disability in
15. back pain and sciatica. Cochrane Database Syst Rev 2004;(4):CD001254.
16. Van Tulder MW, Touray T, Furlan AD, Solway S, Bouter LM; Cochrane back review Group. Muscle relaxants for nonspecific low back pain: a systematic review within the framework of the Cochrane collaboration. Spine 2003;1:1978-92.
17. Martell BA, O'Connor PG, Kerns RD, Becker WC, Morales KH,
18. Kosten TR, et al. Systematic review: opioid treatment for chronic back pain: prevalence, efficacy, and association with addiction. Ann Intern Med 2007;146:116-27.
19. 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.
20. Assendkelft WJ, Morton SC, Yu EI, Suttorp MJ, Shekelle PG. Spinal manipulative therapy for low back pain. Cochrane Database Syst Rev 2004;(1):CD000447.

21. Furlan AD, van Tulder MW, Cherkin DC, Tsukayama H, Lao L, Koes BW, et al. Acupuncture and dry-needling for low back pain. Cochrane Database Syst Rev 2005;(1):CD001351.
22. Laxmaiah M et al. An Update of Comprehensive Evidence-Based Guidelines for Interventional Techniques in Chronic Spinal Pain. Pain Physician 2013; 16:S49-S283
23. Hayden JA, vanTulder MW, Malmivaara AV, Koes BW. Meta-analysis: exercise therapy for non-specific low back pain. Ann Intern Med 2005;142:765-75.
24. Airaksinen O, Brox JI, Cedraschi C, Hildebrandt J, Klaber-Moffett J, Kovacs F, et al. Chapter 4: European guidelines for the management of chronic nonspecific low back pain. Eur Spine J 2006;15:S192-300. Riew KD, Park JB, Cho YS, Gilula L, Patel A, Lenke LG, et al. Nerve root blocks in the treatment of lumbar radicular pain. A minimum five-year follow-up. J Bone Joint Surg (Am) 2006;88:1722-5.
25. Weinstein JN, Tosteson TD, Lurie JD, Tosteson AN, Blood E, Hanscom B, et al. Surgical versus nonsurgical therapy for lumbar spinal stenosis. N Engl J Med 2008;358:794-810.