TREATMENT OF COMMON SPINE DISORDERS

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Introduction

Neurologists should play a pivotal role in the evaluation and treatment of patients with spine and limb pain with or without neurologic signs and symptoms. We can do this by recognizing when surgical intervention is indicated, when medical treatment will suffice, referring patients to the right surgeon if one is needed, and recommending or prescribing appropriate non-operative therapy. This syllabus describes indications for surgery on the spine, medical therapy for patients who do not need an operation, diagnostic and therapeutic needle procedures performed on the spine, surgical treatment options, and some recent developments in spine care. The syllabus includes material that will not be presented.

Cervical Radiculopathy, Myelopathy, and Axial Neck Pain

Cervical Nerve Root Compression

Cervical root compression is usually due to a herniated intervertebral disk or narrowing of the intervertebral foramen from degenerative hypertrophic changes or a combination of the two. C7 root compression by a C6-7 disk is most common followed by C6 radiculopathy secondary to C5-6 disk followed by C8 radiculopathy and C5 radiculopathy.

While less common than lumbar root compression, cervical radiculopathy follows a similar course in that most patients improve spontaneously with or without conservative treatment.

Surgical referral should be considered for the patient with persistent brachialgia (pain in the arm and/or forearm and/or scapula) which has been present for weeks with strong physiologic evidence of dysfunction of a specific nerve root and confirmation of nerve root compression at the appropriate level and side on an imaging study, usually magnetic resonance imaging (MRI), or less commonly, myelography with CT scanning. Similar to the situation for patients with low back and lower limb pain, imaging and surgical referral are typically delayed in the patient with neck and upper limb pain until there is significant or progressive weakness, evidence of myelopathy, uncontrolled pain, or symptoms that last > 4 to 6 weeks.

The following factors may influence the referral decision and prompt one to seek help sooner:

- Severe or progressive radicular deficit or the development of spinal cord impingement.
- A larger-sized herniated disk on the imaging study.
- Profound pain.
- The neurologic deficits are critically important to the patient's vocation or avocation.
- C5 or C8 radiculopathy.
- Be aware that cervical root weakness, especially triceps paresis (C7), is not always recognized by the patient.
- As root compression progresses, pain can actually lessen at the same time that the patient's deficit worsens.

C5 and C8 nerve root compressions seem to have a worse prognosis possibly because these two spinal nerves do not have as much dual innervation from adjacent spinal levels as the other cervical roots. In the case of the C8 nerve root persistent weakness of the hand can be disabling. The C5 root is important for elbow flexion which facilitates feeding.

Surgery for spondylotic radiculopathy can be performed from a posterior approach in which part of the lamina on one side is removed, and the offending bony spurs and/or protruding intervertebral disk are cut out. Alternatively, surgery can be performed from an anterior approach in which the appropriate intervertebral disk is removed and either autologous or cadaver bone or synthetic materials are implanted. Additionally, a metal plate and screws are usually permanently placed into the two adjacent vertebral bodies in order to promote solid bony fusion. The

plates and screws are not removed if the fusion is successful. Placement of an artificial cervical disk (the Prestige Cervical Disc made by Medtronic, the ProDisc-C made by Synthes Spine, the Bryan Cervical Disc also made by Medtronic, the Secure-C made by Globus Medical, the Mobi-C made by LDR Spine, etc.) from an anterior approach is another option for spondylotic radiculopathy.

The anterior and posterior surgical approaches have comparable results with ≥ 75% of patients experiencing substantial relief from their radicular symptoms.

Complications occur in a small percentage of patients and include nerve root more likely than spinal cord injury and hoarseness and swallowing difficulty after anterior cervical spine surgery.

Both anterior and posterior cervical (and lumbar) procedures are performed by both neurosurgeons and orthopedic surgeons.

Medical Treatment of Cervical Radiculopathy

Because cervical radiculopathy is much less common than lumbar radiculopathy, evidence-based recommendations for the treatment of cervical radiculopathy and neck pain are relatively lacking. Analgesics, especially nonsteroidal anti-inflammatory drugs and acetaminophen, can be used for most patients, and opioid analgesics can be given for severe pain. Muscle relaxants may be helpful for acute pain associated with muscle spasm. Interlaminar and, increasingly in recent years, transforaminal epidural steroid injections have been used to treat patients with persisting neck and referred upper limb pain. Such injections require pre-injection advanced imaging of the cervical spine, usually with MRI or alternatively with CT myelography. Firm evidence of benefit from injection therapy is lacking and very rare complications including root injury, epidural hematoma, spinal cord and cerebellar infarction, infection, and even death are reported. Physical measures can also be utilized. These include heat and/or ice, intermittent cervical traction, short-term use of a cervical collar, use of a cervical pillow at night, and various physical therapies including range of motion and strengthening exercises. Typically, exercises are only recommended after an acute cervical radiculopathy has subsided and are thought to help prevent future recurrence. Anecdotally, a brief, 7 to 10 day course of oral corticosteroids is sometimes given to patients with acute radicular pain with unproven benefit.

Cervical Spondylotic Myelopathy

This common condition can be due to congenital narrowing, degenerative changes, or a combination of the two. Worldwide, cervical spondylosis is the most common cause of myelopathy. The differential diagnosis of myelopathy is lengthy. In general, the cause of the patient's apparent myelopathy will be established by MRI and other imaging studies of the cervical spine (e.g., spondylosis, tumor, syringomyelia, multiple sclerosis), MRI of the brain (e.g., multiple sclerosis, parasagittal lesion, stroke), EMG (e.g., ALS and peripheral nerve disease), and blood tests (e.g., B_{12} deficiency, copper deficiency myelopathy, neuromyelitis optica, HTLV-I and HIV infection, and syphilis). Of course, cervical spondylosis is common, and mild to moderate degrees of cervical spinal canal narrowing can be seen in patients with an additional, non-spondylotic cause for their apparent or actual myelopathy. While the most common presentation of spondylotic myelopathy is a slowly evolving myeloradiculopathy related to slowly progressive degenerative and hypertrophic changes in the cervical spine, patients can present acutely or subacutely if they experience trauma or in the setting of an acute, large central disk herniation. Increased T_2 signal within the cervical spinal cord on MRI indicates edema or myelomalacia and increases the likelihood of symptomatic myelopathy. Increased T_1 signal in the cord indicates some degree of permanent damage. While patients who are found to have spondylotic myelopathy should probably be referred to a spine surgeon for consultation, it is not always clear that they need to undergo surgery. Many patients with spondylotic myelopathy remain stable for several years or longer with conservative treatment. A small percentage actually improves over time.

Surgical Considerations in Cervical Spondylotic Myelopathy

Decompression is clearly indicated for patients with moderate or severe deficits and for patients with progressive neurologic worsening. Surgery from an anterior approach (discectomy with fusion of one or more levels) or from a posterior approach (multi-level laminectomy often done without fusion) is associated with low morbidity, initial improvement in up to 70% of patients, and then a tendency to deteriorate over many years. Another posterior surgical procedure is laminoplasty in which several cervical laminae are cut through on one side of the spinal canal and grooved on the other side allowing the freed laminae to swing open like a hinged door. Placement of an artificial cervical disk from an anterior approach is another, less commonly used option for spondylotic myelopathy confined to 1 or possibly 2 levels. If stenosis is multi-level, then posterior decompressive

laminectomy may be favored. Anterior decompression and fusion are favored in patients with an unstable spine or stenosis confined to one or two levels. In general, the outcome from surgery is worse in patients with a longer preoperative duration of symptoms and in older patients.

What about prophylactic surgery to prevent spinal cord injury in the patient with cervical spinal stenosis without myelopathy? While commonly advised, in this author's opinion, such patients should not undergo cervical spinal decompression for asymptomatic cervical stenosis. Acute cervical spinal cord injury is uncommon and usually occurs in the setting of young men behaving recklessly. A significant minority of acute spinal cord injuries, about 20%, are related to falls. The percentage of spinal cord injuries due to falls has increased over time, and such falls are more likely to affect the elderly. Although unproven, it is assumed that many older individuals who fall and injure their spinal cord have underlying cervical spinal stenosis, but most victims are not known to have a narrowed cervical spinal canal before their fall. The incidence of spinal cord injury for the entire population is only between 15 and 50 per million population per year (about 10,000 per year in the United States). Even if patients with cervical spinal stenosis have a risk of traumatic spinal cord injury that is many times higher than the population at large, this risk is surely less than the risk of prophylactic cervical spine surgery which carries with it a risk of injuring the spinal cord that is about 1% (10,000 per million people undergoing the operation) and a mortality rate of about 0.5% (0.39 to 0.6% or 3900 to 6000 per million people undergoing surgery). Cervical spinal cord injury can occur whether or not there is underlying antecedent stenosis. Moreover, decompression with or without fusion does not provide absolute protection against traumatic spinal cord injury. Additionally, the risk of surgery is taken when it is performed whereas the risk of an injury is some indefinite time in the future, and quite possibly never. Patients with cervical spinal stenosis (and everyone) should avoid falls and head and neck injuries. The patient with asymptomatic or minimally symptomatic cervical spinal stenosis should be advised to shun activities that could result in head and neck injuries, drive large automobiles that have lots of airbags, drive defensively, use supportive footwear, safety-proof their homes (use night-lights, eliminate loose rugs and clutter in the home, modify uneven and slippery surfaces, obtain and use handrails, sit when showering, use a gait aid, etc.), and eliminate or take precautions when using medications and alcohol that could result in hypotension or imbalance. This author believes that the risks of prophylactic decompression for asymptomatic and mildly symptomatic cervical spinal stenosis outweigh any possible benefit. A recent report of 199 patients with "asymptomatic spondylotic cervical spinal cord encroachment" found a low incidence of spinal cord injury after minor trauma (Bednarik, 2011).

Medical Therapy of Cervical Spondylotic Myelopathy

In addition to avoidance of head and neck injury, use of a cervical pillow at night and use of a soft or rigid cervical collar are often recommended without evidence of benefit. Patients should avoid postures and positions which place a strain on the neck. Physical therapy directed at neurologic deficits is appropriate. Cervical spine range of motion and paraspinal muscle strengthening exercises and intermittent traction are probably best avoided.

The Evidence Regarding Surgical Treatment of Cervical Spondylotic Radiculopathy and Myelopathy

A recent Cochrane Systematic Review of Surgery for Cervical Radiculomyelopathy concludes that "the available small randomised trials do not provide reliable evidence on the effects of surgery for cervical spondylotic radiculopathy or myelopathy. It is not clear whether the short-term risks of surgery are offset by any long-term benefits." Having said this, the authors could only find two randomized controlled trials – one trial of 81 patients comparing surgery versus conservative treatment for cervical radiculopathy and one trial of 49 patients comparing surgery with conservative treatment for cervical myelopathy. The single prospective, randomized trial comparing surgical treatment with physical therapy or a cervical collar for spondylotic cervical radiculopathy found that the operated patients were better three to four months after intervention, but the outcomes were the same after 15 to 16 months. In the cervical spondylotic myelopathy trial, there was no significant difference between the operated and conservatively treated patients with mild and moderate myelopathy after six months and two years. This same cohort of patients with 19 additional recruits was followed, and there was also no difference between operated and conservatively treated patients after three years. The same authors also reported on a prospective study of 64 patients followed for 10 years and there was no difference in the conservatively treated versus surgically treated patients (Kadanka, et al. 2011). Cervical radiculopathy and spondylotic myelopathy are extraordinarily common, and it is remarkable that there have been so few randomized trials of surgery versus conservative treatment for these conditions. The paucity of studies is no doubt related to the strongly held belief that surgical intervention is helpful for cervical nerve root and cervical spinal cord compression. The author hereby discloses that he believes decompression for significantly symptomatic spondylotic root and spinal cord compression is beneficial for many patients.

Axial Neck Pain Alone

Neck pain is less common than low back pain (LBP). Acute neck pain is more common than chronic (> 3 months). A supplement to Spine published in 2008 was dedicated to "A Best Evidence Synthesis on Neck Pain: Findings From the Bone and Joint Decade 2000-2010 Task Force on Neck Pain and its Associated Disorders." The supplement article summaries include the following statements.

Nonsurgical therapies (education, mobilization, manual therapy, exercises, heat and perhaps acupuncture) appear to have some benefit but none is superior. Interventions focusing on regaining function and returning to work are more effective than those that do not.

Medial branch blocks with local anesthetics followed by radiofrequency lesioning of the medial branches which relieved neck pain are used to treat facetogenic neck pain with moderate evidence of benefit, especially in patients with neck pain following whiplash-type injury. The medial branch nerves are the medial branches of the dorsal rami of the spinal nerves and innervate the facet joints and multifidus muscles. Each facet joint is innervated by two adjacent medial branches. Evidence does not support intra-articular facet joint injections.

Although interlaminar or selective transforaminal epidural steroid injections can temporarily help cervical radiculopathy, they are not given for neck pain alone.

What about surgery for neck pain alone? There is no evidence to support surgery (e.g., anterior cervical discectomy with fusion or placement of an artificial cervical disk) for neck pain without radiculopathy, myelopathy, or spinal instability. Nonetheless, operations are performed on the neck for neck pain alone. If necessary, refer your patients with neck pain to a very conservative surgeon.

There is a very nice recent review of the epidemiology, diagnosis and treatment of neck pain published in the Mayo Clinic Proceedings (Cohen 2015).

Lumbar Radiculopathy, Cauda Equina Syndrome, and Lumbar Spinal Stenosis

Lumbar Radiculopathy

Low back problems are substantially more common than neck problems. The common causes of non-neoplastic lumbar nerve root compression include herniated disks, osteophytic spurring, spondylolisthesis, spinal stenosis, and synovial cysts. The most common low back syndrome is acute LBP with or without sciatica. It is estimated that about 85% of patients who present with acute LBP alone cannot be given a specific diagnosis.

Herniated Lumbar Disk

The natural history of acute LBP and most lumbar disk herniations is benign. Patients with acute LBP with or without lower limb pain do not need imaging let alone surgical consultation for 4 to 6 weeks so long as they do not have symptoms or signs of cauda equina syndrome, a severe or progressive neurologic deficit, or 'red flags' suggesting tumor, infection, or fracture. Early lumbar spine imaging should be considered only if one or more of the following red flags are present:

> The red flags for possible fracture are major trauma (e.g., fall from a height, motor vehicle accident) or minor trauma (lifting an object) in a patient at risk for fracture (e.g., known osteoporosis, is on corticosteroids).
>
> The red flags for possible tumor or infection include: age < 20 or > 50; history of cancer, fever, chills, weight loss, recent infection, IV drug use, immunosuppression; and pain that worsens when supine or wakes the patient from sleep.
>
> Neurologic red flags are possible cauda equina syndrome and severe or progressive lower limb weakness.

Patients who do not have and do not develop any of the red flags listed above should be considered for imaging and possible surgery if their radicular pain and associated neurologic signs and symptoms persist without improvement for > 4 to 6 weeks. Most of these patients will have strong physiologic evidence for dysfunction of a specific nerve root (e.g., loss of a reflex, some motor weakness).

Surgery can be considered with the above symptoms and signs only if there is confirmation of nerve root compression (usually by a herniated lumbar disk) at the appropriate level on an imaging study, usually MRI.

Similar to the situation in the cervical spine, progressive nerve root compression can result in the paradox of increasing neurologic deficit associated with lessening axial or limb pain. This is often seen with disk extrusion. A patient's weakness can be asymptomatic or minimally symptomatic, especially with S1 radiculopathy and weakness of plantar flexion of the foot.

What is sciatica? Sciatica is "pain in the distribution of the sciatic nerve." Since the sciatic nerve innervates sensation above and below the knee, this author believes that sciatica can involve the thigh alone or the leg alone or both; the pain of sciatica does not need to go below the knee. Upper lumbar radiculopathies commonly cause pain in the thigh but not the leg. Pain radiating below the knee is more likely to represent a true radiculopathy than pain radiating only to the posterior thigh. By the way, anatomically speaking, the thigh is the portion of the lower extremity from the hip to the knee, and the leg is the part between the knee and the ankle. In the upper limb, the arm extends from the shoulder to the elbow and the forearm extends from the elbow to the wrist.

A history of sciatica is the strongest piece of evidence that the patient could have radiculopathy. Its absence makes compressive lumbosacral radiculopathy very unlikely. Deyo (1992) estimates the likelihood of a surgically important disk in a patient without sciatica as 1 in 1,000.

More than 90% of disk herniations occur at L4-5 (and typically produce L5 radiculopathy) and L5-S1 (and cause S1 radiculopathy); herniation at L3-4 (with L4 radiculopathy) and L2-3 (with L3 radiculopathy) are less common. Lumbar disk herniation typically impinges on the nerve root that is migrating laterally to exit beneath the pedicle of the next numbered vertebra. Far lateral disk protrusions can affect the root at the same level as the disk herniation. Large disk herniations can compress more than one nerve root on one side and, if large enough, can cause bilateral root compression and the cauda equina syndrome.

About one-third of asymptomatic adults will have one or more herniated lumbar disks on MRI. Thus, clinical correlation between the imaging findings and the patient's signs and symptoms is critical. Treat the patient, not their image.

There is pretty clear evidence that laminectomy and lumbar discectomy can relieve symptoms more quickly than non-surgical medical and physical therapies in patients with significant lumbar disk protrusions and radicular symptoms who have not improved after one to two months of conservative treatment. A Cochrane Systematic Review of Surgery for Lumbar Disc Prolapse concluded that "surgical discectomy for carefully selected patients with sciatica due to lumbar disc prolapse provides faster relief from the acute attack than conservative management, although any positive or negative effects on the lifetime natural history of the underlying disc disease are unclear." A recent evidence-based clinical practice guideline from the American Pain Society concurs that there is good evidence that discectomy for radiculopathy due to prolapsed disk provides moderate benefit for short-term outcomes (through 3 months). Note that patients in the trials reported had mild to moderate lumbosacral radiculopathy. Most authors agree that patients with severe and progressive deficits should undergo surgery sooner rather than later. Unless they have cauda equina syndrome, the surgery does not need to be done emergently or even urgently.

<u>Acute Cauda Equina Syndrome (CES) Usually Due to Large Lumbar Disk Protrusion</u>
The most common cause of a subacute to acute CES is a large lumbar disk protrusion or extrusion. Many, but not all patients, have a past history of LBP and/or radicular pain. Presenting clinical symptoms can include LBP, bilateral or unilateral sciatica, perineal/perianal/saddle sensory loss, lower limb motor weakness, lumbosacral root sensory deficits, difficulty with bladder more often than bowel sensation and control, and sexual dysfunction. Neurologic findings include weakness and sensory loss in the distribution of multiple lumbosacral nerve roots on one or both sides, positive straight-leg raising signs, reduced perianal and perineal sensation, reduced anal sphincter tone and strength, and absent superficial anal and bulbocavernosus reflexes. Most authors use the term CES only when the patient also has impairment of bladder, bowel, or sexual function and perianal, perineal, or "saddle" numbness. The onset of CES can be leisurely or sudden. A subacute onset is more typical. Left untreated or under-treated, adverse neurologic outcomes (e.g., sphincter and sexual function, chronic pain, weakness, and sensory loss) can greatly affect an individual's ability to work and function socially. Many articles try to determine optimal timing for surgical intervention (i.e., less than 24 hours after the onset of CES versus less than 48 hours after the onset versus greater than 48 hours). Authors differentiate between patients who have CES with urinary retention and overflow incontinence which is thought to be due to complete damage to the nerves innervating the bladder and urinary sphincter as opposed to patients with incomplete CES in which there is

impaired urinary sensation, reduced desire to void, or poor stream, but without urinary retention or overflow incontinence. Complete denervation of the bladder and urinary sphincter implies complete bilateral injury to the spinal nerves involved. Patients with incomplete CES are thought to have a better prognosis and a better chance of improvement with surgical intervention. The urgency of surgical intervention on CES due to disk compression remains controversial. Early surgical intervention is thought to be appropriate for incomplete CES but is less established in complete CES. For incomplete CES, operating less than 24 hours from onset is probably better than operating between 24 and 48 hours from onset which is probably better than operating more than 48 hours from onset. The benefit from earlier surgery in patients with complete CES is less clear.

Neurologists and primary care physicians should be on the lookout for warning symptoms of CES (bilateral sciatica, perineal/perianal/saddle numbness, urinary more likely than rectal sphincter disturbance, sexual dysfunction, and often LBP). Unless the time course of onset of symptoms is very leisurely or the symptoms are longstanding and fixed, these patients should be evaluated emergently (or at the least very urgently). In general, this will mean sending the patient to an emergency department that has the capability of performing MRI (the diagnostic imaging procedure of choice) and performing urgent or emergent spine surgery. If you refer a patient with suspected CES directly to a spine surgeon, I suggest that you call them to arrange expedited imaging and in-person evaluation.

Lumbar Spinal Stenosis (LSS)

This common condition can be due to congenital narrowing, degenerative changes, or a combination of both. In descending order, the most commonly affected levels are L4-5, L3-4, L2-3, L5-S1, and L1-2. A majority of patients have stenosis at more than one level.

Narrowing of the lumbar spinal canal can be asymmetric or only affect the lateral recess or neural foramen on one side and produce asymmetric or unilateral symptoms.

Electromyography can show usually chronic neurogenic changes consistent with lumbosacral nerve root injury in up to 90% of patients. Muscles supplied by the L5 and S1 nerve roots are most often affected.

If there is a need to confirm the clinical diagnosis or if surgery is being considered, MRI is the imaging study of choice for LSS. Surgery can be performed on the basis of MRI alone. Plain CT can be used as a screening test in patients who are unable to undergo MRI.

Because lumbar spinal stenosis is symptomatic and more easily demonstrated when the patient is standing, vertical MRI machines and imaging with simulated weight-bearing (axial loading while the patient is supine) have been promoted as helpful diagnostic studies. The vertical MRI machines often use a lower field strength magnet which compromises image quality. Vertical myelography, usually with CT, is another imaging option. The vast majority of patients with symptomatic LSS will have stenosis on MRI when they are supine.

Asymptomatic central canal LSS is seen in 3-5% of middle-aged and older adults on MRI and CT. The clinician must be sure that the patient's symptoms are consistent with the imaging findings, especially before suggesting surgical treatment.

Therapy can be divided into physical, pharmacologic, and surgical categories. Exercises to strengthen abdominal muscles and reduce lumbar lordosis may be helpful. Use of a short cane or walker may allow the patient to stand longer and walk farther. The four-wheeled rolling walker with hand brakes, seat, and a basket is a wonderful invention. Corsets and braces may help to reduce lumbar lordosis when standing and thereby delay the onset of symptoms. Much of the excess weight in obese patients is carried in their abdomen forcing them to extend their lumbar spine in order to maintain their sagittal balance. By reducing the degree of lumbar lordosis needed to stand erect and by reducing the axial load on the lumbar spine, substantial weight loss (20 to 30 kg or more) can be very helpful to patients with LSS. However, such weight loss is exceedingly difficult to achieve.

Analgesics are usually not very helpful because the patient's pain is intermittent and can be relieved by change in posture. Muscle relaxants are of no help. While epidural steroid injections are frequently used to provide temporary symptomatic improvement, a recent double-blind trial found no difference in benefit in LSS patients receiving an injection of glucocorticoid with lidocaine versus lidocaine alone (Friedly, 2014). Despite this evidence, epidural injections are still commonly given to patients with LSS and pseudoclaudication.

The mainstay of effective treatment of LSS is surgical decompression from a posterior approach with laminectomies at 1 or more levels. Decompression may need to be coupled with fusion if there is preoperative or the potential for postoperative spondylolisthesis with instability. Because LSS is frequently a multi-level process, the majority of patients require decompression of more than one spinal level. Foraminotomies for lateral recess or intervertebral foraminal stenosis may be needed, and part or all of one or more facet joints may need to be removed. Simultaneous fusion, especially if performed with instrumentation, increases the cost and complication rate without necessarily improving clinical outcomes. Complications from surgery are reported in up to 10 to 15% of patients. About three-fourths of patients have good to excellent relief of their lower limb symptoms for one to several years or longer. LBP, even if postural, is not reliably helped by surgery for LSS. Because of the risk of complications, the less than complete benefit, and the intermittency of symptoms, surgery for LSS is completely elective.

Surgery should be considered as follows:

Symptoms have been present for greater than three months.

Pseudoclaudication symptoms significantly interfere with the patient's work, lifestyle, or both. Alternatively or additionally, the patient has severe or progressive neurologic deficits.

Imaging studies show significant lumbar spinal canal stenosis at one or more levels.

The patient is otherwise in good health and elects surgery with a full understanding of the risks and benefits.

Factors associated with disappointing results include absence of appropriate indication(s) for surgery, inadequate decompression, older age, re-stenosis at the same or another level, an instrumented fusion, a previous low back operation, an increase in mechanical back pain following surgery, and "co-morbidities." By co-morbidities, we mean other medical problems which increase the risk of operating and limit the patient's functional ability and survival. Osteoarthritis of major lower limb joints may require surgical attention either before or after spinal stenosis surgery.

In summary, surgical decompression is a reasonable treatment for lumbar spinal stenosis if the patient has significant pseudoclaudication, has appropriate imaging findings, is in reasonably good health otherwise, and elects operative intervention with a full understanding of the risks and hoped-for benefits.

X Stop® and Other Interspinous Implants for Lumbar Spinal Stenosis

In November, 2005, the X Stop® interspinous process decompression (or distraction) system made by Medtronic was approved by the FDA to relieve symptoms of LSS. The X Stop® is a cylindrical titanium rod that is implanted horizontally between adjacent posterior projecting spinous processes at a level of lumbar spinal stenosis. The device markedly limits extension of the lumbar spine at the implanted level. The X Stop® implant is indicated for treatment of patients aged 50 or older with neurogenic claudication secondary to a confirmed diagnosis of LSS. The X Stop® reportedly can be implanted at one or two lumbar levels. The procedure can be performed as an outpatient usually under local anesthesia.

The X Stop® implant makes some sense. By separating the posterior spinous processes and forcing flexion at one or two lumbar spinal levels, we would expect that the degree of lumbar spinal stenosis would be reduced, and the patient's pseudoclaudication would be delayed in onset or lessened in severity or possibly prevented altogether. It is not clear what long-term effect this forced flexion will have on the disk at this level or the spine above and below the level(s) of distraction. X Stop® is clearly less invasive than surgical decompression and can be considered in selected patients, including patients with increased surgical risk related to decompressive laminectomy and/or limited life expectancy.

A recent study by van den Akker-van Marle, et al found that implantation of an interspinous decompressive device is highly unlikely to be cost effective when compared to bony decompression for patients with symptomatic LSS.

Other devices include Superion, Coflex, Affix, Axle, BacFuse, BridgePoint, Inspan, Lanx, PrimaL, SPFix, and Spire. High complication and reoperation rates and poor outcomes limit the use of these devices.

Pharmacologic Treatment of LBP With and Without Sciatica and Recently Published Guidelines

Pharmacologic therapies for LBP are best viewed as comfort control measures. There is evidence of efficacy for the following medications which can be used for LBP and/or lower extremity pain.

There is strong evidence that nonsteroidal anti-inflammatory drugs (NSAIDs) are an effective short-term treatment for patients with acute and chronic LBP. There does not seem to be one specific NSAID that is clearly superior. The benefit is modest, and NSAIDs are probably no more effective for LBP than other analgesics or non-drug therapies. COX2 inhibitors are similarly effective. COX2 inhibitors have fewer side effects than traditional NSAIDs, but this class of drugs may be associated with a slightly higher risk of cardiovascular side effects.

Acetaminophen has not been studied as much as NSAIDs, but is reasonably safe and acceptable for treating patients with acute and chronic LBP, although a recent double-blind study in *Lancet* found no benefit of acetaminophen in acute LBP (Williams, 2014).

There is strong evidence to suggest that muscle relaxants are more effective than placebo for short-term pain relief in patients with acute LBP. Different types of muscle relaxants are equally effective. There is also evidence for the effectiveness of muscle relaxants for short-term pain relief in patients with chronic LBP. Adverse side effects of muscle relaxants require that they be used with caution. Muscle relaxants are probably no more effective than NSAIDs, and there is probably no additional benefit obtained by combining muscle relaxants with NSAIDs.

Opioid analgesics are an option for time-limited use in patients with acute, severe LBP. Not everyone agrees that opioids should be used in this situation for fear of diversion, overuse, addiction, and death from accidental overdose.

Have you ever given a course of oral corticosteroids to a patient with an acute radiculopathy? A recent randomized clinical trial of patients with ≤ 3 months of significant radicular pain and a herniated disk on MRI found that a 15-day tapering dose of prednisone starting with 60 mg per day resulted in modestly improved function and no improvement in pain at 3 weeks and 52 weeks (see Goldberg, et al. 2015). There were more adverse events in the prednisone group and no difference in the surgical treatment rate at 1 year follow-up. The results give very modest support to this practice.

Tricyclic and tetracyclic antidepressants are moderately effective for chronic LBP. Patients with radicular pain are more likely to benefit. Gabapentin can also be used for radicular pain.

Clinical practice guidelines recommend that clinicians should conduct a focused history and physical and neurologic examinations to help place patients into one of three broad categories:

Nonspecific LBP

Back pain potentially associated with radiculopathy or spinal stenosis

Back pain potentially associated with another specific spinal cause

The history should include assessment of psychosocial risk factors which predict risk for developing chronic disabling back pain.

Clinicians should not routinely obtain imaging or other diagnostic tests in patients with nonspecific LBP. Harms that can follow routine imaging that is not indicated include being labeled as having a disease, exposure to radiation and contrast agents, findings that lead to additional unnecessary procedures now and in the future, the risk of useless treatments directed at findings unrelated to the patient's symptoms, and the cost of the extraneous diagnostic and therapeutic interventions. Clinicians should perform diagnostic imaging and testing for patients with LBP when it is severe, if progressive neurologic deficits are present, or when serious underlying conditions are suspected on the basis of the history and physical and neurologic examinations.

Magnetic resonance imaging (preferred) or computed tomography should be obtained only if patients are potential candidates for surgery or epidural steroid injection (for suspected radiculopathy).

Clinicians should provide patients with evidence-based information on LBP with regard to their expected course, advise patients to remain active, and provide information about effective self-care options.

For LBP, clinicians should consider the use of medications with proven benefits in conjunction with back care information and instruction in self-care. Clinicians should assess severity of baseline pain and functional deficits, potential benefits, risks, relative lack of long-term efficacy, and safety before initiating therapy. For most patients, first-line medication options are NSAIDs or acetaminophen.

For patients who do not improve with these measures, clinicians should consider the addition of nonpharmacologic therapy with proven benefits. Nonpharmacologic therapies with good evidence of moderate efficacy for chronic or subacute LBP are cognitive-behavioral therapy, exercise, spinal manipulation (I disagree for chronic LBP), and interdisciplinary rehabilitation. For acute LBP, the only nonpharmacologic therapy with good evidence of efficacy is superficial heat. There is fair evidence that spinal manipulation is helpful for acute LBP. There is fair evidence that acupuncture, massage, yoga (viniyoga), and functional restoration are helpful for chronic LBP.

There are some good results for individual herbal medicines in individual short-term trials. The herbal medications with reported short-term benefit were oral Harpagophytum procumbens (devil's claw), oral Salix alba (white willow bark), and topical Capsicum (cayenne pepper or capsaicin).

A very nice review of the evaluation and treatment of low back pain was published in the Mayo Clinic Proceedings in late 2015 (Hooten and Cohen).

Diagnostic and Therapeutic Spinal Interventions

Facet Joint Interventions

The facet or zygapophyseal joints are a significant source of axial spine pain. The medial branches of the dorsal rami of the spinal nerves innervate the facet joints. Medial branch blocks are indicated in patients with chronic (> 6 months) axial spine pain that is inadequately explained and poorly controlled. If diagnostic blocks of the nerves which supply specific facet joints relieve the patient's pain, it is assumed that those facet joints are pain generators, and radiofrequency lesioning of the same medial branch nerves can be offered to provide prolonged benefit. Separately, or coupled with a medial branch block, patients may receive intra-articular facet joint injections of a corticosteroid in hopes of providing prolonged pain relief by an anti-inflammatory effect. There is, however, no high quality literature support for therapeutic benefit from such facet joint injections at any level of the spine. The diagnostic blocks and radiofrequency treatments are better validated in the cervical than in the lumbar than in the thoracic spine and may be especially helpful in patients with neck pain following a whiplash-type of injury.

Epidural Injections

Radicular pain, with or without radiculopathy, is a common clinical condition. Studies of the pathophysiology of radicular pain suggest that both mechanical compression and an inflammatory response are needed for pain production. The inflammatory component provides rationale for the targeted injection of corticosteroids as a therapy for radicular pain. Local anesthetics alone may be used diagnostically to identify the spinal nerve mediating the patient's pain. A corticosteroid and/or a local anesthetic can be delivered to the dorsal epidural space using an interlaminar approach, or via the sacral hiatus with a so-called caudal approach. Corticosteroid and/or a local anesthetic can be delivered directly to a symptomatic spinal nerve in the intervertebral foramen using a transforaminal approach. Advanced spinal imaging with MRI, plain CT, or CT myelography is considered a prerequisite for epidural injections by most interventionalists. There is a historical practice of providing a series of epidural injections (e.g., weekly for 3 weeks), but this is not supported by the literature. A decision to repeat an injection should be based on the response to one or more previous injections.

Very rare catastrophic vertebrobasilar and spinal cord infarction can occur with transforaminal epidural injections due to damage to a vertebral artery or the artery of Adamkiewicz or another large thoracolumbar radiculomedullary artery. Risks of all epidural injections are greater in the cervical spine. There are also risks inherent in any spine-related interventional procedure that include allergy to medications, radiation exposure, dural puncture, needle injury to neural or vascular structures, and infection (e.g., epidural abscess and recently reported iatrogenic fungal meningitis and spinal and paraspinal infections). Multiple corticosteroid injections can

cause systemic side effects. Benefits of epidural steroid injections include pain relief, functional recovery, avoidance of surgical intervention, and identification of the patient's pain generator. There is more evidence of temporary symptomatic benefit than avoidance of surgery. While there is support for their use in the cervical spine, there is much stronger evidence of benefit in the lumbar spine. Patients who have failed conservative therapy or who are not candidates for surgical intervention can be considered for epidural steroid injections to relieve their radicular pain temporarily.

Discography

Discography (provocation discography, disk stimulation) is an invasive diagnostic test performed to confirm or exclude internal disk disruption (IDD) as the cause of axial spine pain. One of the most common causes of chronic low back pain is IDD or discogenic pain which may account for 40% of chronic low back pain. The purpose of discography is to make the diagnosis of IDD or discogenic pain. In general, discography is only performed in patients with significant, persistent axial spine pain of at least six months' duration that is undiagnosed despite noninvasive testing and unresponsive to conservative therapy. Advanced spinal imaging (MRI, CT myelography, or plain CT) is considered a prerequisite to performing discography. Discography is performed under fluoroscopic guidance with minimal sedation, as the patient's provoked pain responses are critical to the interpretation. Small caliber (25 gauge) needles are placed into the target disk and one or two adjacent control disk(s). Under manometric control, contrast material with an antibiotic is slowly instilled into the nucleus pulposus of each disk sequentially while the patient's pain response is monitored. In lumbar discography, CT is performed after the procedure to better define the presence and grading of annular fissures. Provocation of the patient's characteristic spine pain is the key outcome measure. A discogram is positive if injection of the targeted disk causes pain that is significant (≥ 6/10), concordant with the patient's baseline pain, caused by no more than moderate pressure (≤ 50 pounds/square inch), and not brought on by stimulation of any adjacent disk.

There is a substantial body of evidence supporting the assertion that discography can identify a painful disk. The false positive rate for lumbar diskography is low, in the range of 10%. However, there is no consensus on appropriate interventional therapy for a painful disk. Surgical fusion as a treatment for discogenic pain remains highly controversial. Compared to the lumbar spine, the evidence is more tenuous, and the role of discography is less certain in the cervical spine. There is even less experience with use of discography in the thoracic spine. Discography is often utilized to define the extent of a proposed fusion or qualify patients for disk arthroplasty or a fusion, but there is no high quality evidence to support these uses. The lack of a proven therapy does not discount the value of reaching a diagnosis. By establishing a diagnosis, a positive discogram protects the patient from futile invasive and expensive diagnostic and therapeutic endeavors directed at other causes of axial spine pain. The primary risk of disk stimulation is bacterial discitis which is very low.

Lumbar Fusion

Lumbar fusion is sometimes recommended for patients with chronic LBP who have not responded to conservative measures. In addition, fusion may need to be coupled with laminectomy performed for disk herniation or spinal stenosis if there is preoperative or the potential for postoperative spinal instability. For patients with low back pain, lumbar fusion should be considered in patients with demonstrated spondylolisthesis (subluxation forward or backward of one lumbar vertebra on its neighbor below) who have severe back pain and/or sciatica associated with substantial functional impairment that has persisted for a year or longer. Fusion for degenerative disk and facet joint disease with LBP alone is controversial and often unnecessary.

For LBP without radiculopathy associated with spondylotic degenerative changes, fusion is no more effective than intensive rehabilitation, but is associated with small to moderate benefits compared to standard non-surgical therapy. Fusion may be helpful for some patients with chronic LBP and disability associated with degenerative disk disease limited to one or two levels, and referral to a spine surgeon can be considered in this situation.

Intermediate and long-term (mean of 12.8 years) prospective randomized trials of lumbar fusion for chronic LBP have shown largely negative results. Studies have been conducted in Sweden (Fritzell, et al and Hedlund, et al), in Norway (Brox, et al and Mannion, et al), and in the UK (Fairbank, et al and Mannion, et al).

Fusion (and referral for possible fusion) should be considered for patients with significant chronic LBP and/or sciatica associated with spondylolisthesis especially in the presence of spinal instability.

For patients with spondylolisthesis and significant or progressive neurologic deficits, surgery including fusion is indicated.

Fusion can be needed following major spine trauma and with some tumor operations.

Additional Considerations for Surgery on the Lumbar and Cervical Spine – Artificial Disks

Artificial Lumbar Disks

In October, 2004, the U.S. Food and Drug Administration approved the first artificial disk, the Charité model, for use in the lumbar spine. In August, 2006, the FDA approved a second artificial lumbar disk, the ProDisc-L. The Charité is manufactured by DePuy Spine, a Johnson & Johnson Company, and the ProDisc-L is manufactured by Synthes Spine. Five artificial cervical disks have been approved (see below), and additional artificial disks are being tested for use in the lumbar and cervical spine. World-wide, there have been tens of thousands artificial disks implanted over the last 20 years in the lumbar and cervical spine combined. These devices have been approved by the FDA because they have been shown to produce results equivalent to spinal fusion procedures.

Arthroplasty is more attractive conceptually and functionally than arthrodesis (fusion). We do not (usually) fuse hips or knees. Fusions run the risk of causing increased degenerative changes in the adjacent nonfused spinal levels. Artificial disks can restore normal disk height, reduce "discogenic pain," and potentially preserve motion in the operated vertebral segment.

Indications for total disk arthroplasty include a mature skeleton with degenerative disk disease usually at just one level from L3 or L4 to S1, which is most often at L4-5 or L5-S1. The patient must have significant discogenic LBP confirmed by history, examination, and radiographic studies often including discography. They should have failed at least six months of conservative therapy. Many insurance carriers judge the devices to be experimental and do not cover artificial disk placement surgery.

There is concern that continued (or increased) movement at the level of the replaced disk could result in ongoing or increased degenerative change in the same level facet joints. The artificial disks can migrate out of the disk space. There is also concern about "subsidence" which means sinking of the metallic component into the vertebral body above or below the implanted artificial disk. The lumbar device is placed from an anterior approach and additional surgical complications include hematomas, infection, erectile dysfunction, and injury to structures such as viscera, blood vessels, and peripheral and spinal nerves.

Please note that the indication for artificial lumbar disk replacement surgery is discogenic LBP and not nerve root compression (radiculopathy, lumbar spinal stenosis, or cauda equina syndrome). In fact, nerve root compression is a contraindication to this procedure.

Artificial Cervical Disks

To date, at least six artificial cervical disks have been approved by the U.S. Food and Drug Administration: The Prestige Cervical Disc made by Medtronic, the ProDisc-C made by Synthes Spine, the Bryan Cervical Disc also made by Medtronic, the Secure-C made by Globus Medical, the PCM made by NuVasive, and the Mobi-C, made by LDR Spine. Additional artificial cervical disks with different designs are being tested. As with anterior cervical discectomy and interbody fusion, the operation requires removal of the native intervertebral disk utilizing an anterior approach. Insurance coverage is variable.

Cervical artificial disk replacement, like anterior cervical discectomy with fusion, can help to decompress the spinal cord and cervical nerve roots. With fusion, same-level osteophytes will recede, but with artificial disk replacement they may persist because motion is retained. By preserving motion, artificial disk placement may help to avoid accelerated spondylotic changes of the adjacent segments above and below the implanted artificial disk, which continues to move, unlike a fused segment.

The six approved cervical devices are indicated for one-level or possibly two-level disk replacement from C3 through C7 in skeletally mature patients with "intractable radiculopathy and/or myelopathy." Note that chronic neck pain alone is not an indication.

Complications of artificial cervical disk placement include hematoma (epidural and retropharyngeal), vertebral fracture during implantation, dislodgement of the implanted artificial disk from the interspace, paravertebral ossification, failure to improve clinically, hoarseness, swallowing difficulty, infection, and infrequent neurologic complications. Late failure may prove difficult to treat surgically.

Additional Considerations for Surgery on the Spine – Spinal Cord Stimulation

Spinal cord stimulation (SCS) has been used for more than 40 years for the treatment of chronic, refractory pain. SCS is most commonly used for failed back syndrome (FBS) of the lumbar spine when the patient has both low back and lower limb pain. FBS is also known as post-laminectomy syndrome. FBS is defined as chronic low back and/or lower limb pain that occur after back surgery, usually lumbar laminectomy. SCS is also used for lower more often than upper limb complex regional pain syndrome type I, refractory angina pectoris, and inoperable, painful peripheral vascular disease. There is evidence of benefit for these indications.

Vertebral Compression Fractures

Fractures, especially compression fractures, are very common and are often discovered by neurologists. If there is neural compression or significant deformity, the patient will likely require referral to a surgeon. Most patients with benign compression fractures improve with conservative therapy and do not require intervention. Malignant spinal fractures do require treatment. New techniques for treating compression fractures with persistent pain (vertebroplasty and kyphoplasty) are available with help from interventional radiologists and some spine surgeons. However, two recent double-blind, placebo-controlled trials failed to show benefit from vertebroplasty. A meta-analysis of the two blinded studies did not show any subgroups who benefited from vertebroplasty, but an open-label, prospective, randomized trial did find a subgroup of patients whose pain relief was prompt and sustained for at least one year after vertebroplasty. The debate continues and additional trials are under way. Vertebroplasty and kyphoplasty are still used to treat patients, especially if they have severe pain and functional impairment for 6 weeks or longer. Kyphoplasty is more expensive and less studied than vertebroplasty. Kyphoplasty may be as effective as vertebroplasty. While kyphoplasty is touted to be safer and capable of increasing vertebral body height to a limited degree, there is little evidence to support these claims. Kyphoplasty may be preferred for treating malignant vertebral compression fractures.

Percutaneous Image-Guided Lumbar Decompression (PILD) for Lumbar Spinal Stenosis (LSS)

PILD is a new technique for LSS patients and is often performed with a commercially available technology owned by Vertos Medical which they term minimally invasive lumbar decompression or mild®. This procedure is indicated for patients with central lumbar canal stenosis secondary to hypertrophy of the ligamentum flavum associated with classic symptoms of neurogenic claudication. Using an epidural catheter, an epidurogram reveals the stenotic area and provides an anterior boundary for the decompression. A trocar is advanced into the interlaminar space and fixed in position. A bone sculptor is advanced through this portal and is used to remove small pieces of the lamina on one or both sides at one or more levels. The bone sculptor is replaced with a tissue sculptor, which is used to incrementally shave off bits of ligamentum flava. The epidurogram helps determine adequacy of the decompression. Typically, the procedure is performed as an outpatient without general anesthesia. A number of published studies report that the risk of complications is very low, back care health-related costs are reduced, and pain and disability are substantially improved. The procedure helps central canal stenosis, but does not help pain related to neuroforaminal stenosis, facet hypertrophy, or bulging disks.

CMS has determined that Medicare would not cover PILD (i.e., mild®) for LSS, except in patients participating in a clinical trial. They cited lack of consensus on diagnostic criteria, lack of consensus on appropriate treatment of LSS, and uncertainty of the efficacy of PILD as a treatment.

Disk Infection as a Cause of Chronic Low Back Pain

Four years ago, Albert et al, published consecutive articles in the *European Spine Journal* supporting infection as the cause of chronic LBP. In the first article, they answered the question “Does nuclear tissue infected with bacteria following disc herniations lead to Modic changes in the adjacent vertebrae?” in the affirmative. Modic type 1 changes are thought to be due to edema and inflammation and are highly associated with chronic LBP (> 6 months’ duration). Modic type 1 changes are hyper-intense on T_2-weighted and hypo-intense on T_1-weighted

MRI. Nuclear disk material from 61 patients undergoing discectomy for a herniated lumbar disk was cultured and 46% had a positive culture (>90% of these had positive anaerobic cultures). 80% of the patients with positive anaerobic cultures developed new Modic type 1 changes in the vertebra adjacent to the previous disk herniation while only 44% of patients with negative cultures developed the imaging changes. Proprionibacterium acnes was the infectious agent most commonly identified.

In the companion article, Albert et al reported on "Antibiotic treatment in patients with chronic low back pain and vertebral edema (Modic type 1 changes): a double-blind randomized clinical controlled trial of efficacy." Patients were included if they had chronic low back pain of at least six months' duration which occurred after a previous disk herniation and who also had bone edema as demonstrated by Modic type 1 changes in the vertebrae adjacent to the previous herniation. Operated and unoperated patients were included. They reported that 100 days of antibiotic treatment with amoxicillin-clavulanate resulted in highly statistically significant improvement on all outcome measures (disability, LBP, leg pain). Improvement continued from 100 days' follow-up (the end of antibiotic therapy) until one-year follow-up.

Needless to say, the publications were met with skepticism and a flurry of letters to the editor. The response to these two publications is reminiscent of the reaction to a 1982 report by Marshall and Warren that peptic ulcer disease was due to Helicobacter pylori infection. The editorial accompanying the Albert et al articles suggests that patients with LBP of > 6 months' duration, a symptomatic lumbar disk herniation in the last 1 year, and Modic type 1 changes on MRI be considered for a course of antibiotics! Antibiotics for low-grade infection of the intervertebral disk has not caught on as a form of therapy for chronic LBP.

American College of Physicians Clinical Practice Guidelines for the Treatment of Low Back Pain

Updated American College of Physicians Clinical Practice Guidelines for the Treatment of Low Back Pain were published in February, 2017. See the end of the bibliography section for the references.

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Pharmacologic Treatment of Low Back Pain With and Without Sciatica and Recently Published Guidelines

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