ABSTRACT
The use of Epidural Steroid Injections (ESIs) to treat radicular back pain (sciatica) due to nerve root irritation can be very effective and safe when proper patient selection and contemporary techniques are used. Just as there are multiple types of back pain, several techniques and approaches for delivering ESIs have evolved with experience. Historically, ESI was performed at the bedside without radiographic guidance by an anesthesiologist or other physician trained in spinal injections. There was little or no insight into the anatomic origin of the pain, and the goal was simply to get the drug somewhere in the lumbar epidural space. Modern techniques employ fluoroscopic guidance as the standard of care to ensure safe and accurate placement of injectate in the epidural space via one of three routes of entry – transforaminal, caudal, and interlaminar. All have excellent efficacy and safety profiles, but different pluses and minuses. Transforaminal ESI delivers the most site-specific concentrated steroid but at an increased risk of neurological complication; caudal ESI delivers the least drug but with the lowest risk; interlaminar ESI delivers more site-specific drug than the caudal approach with limited long term benefit . . .

ESI should be part of a multimodal approach to reduce painful symptoms of nerve root irritation with the goal of avoiding surgery and achieving self-healing. Although there is inevitably a subset of patients for whom surgery is the best option, it is usually elective, and should be reserved for those who do not respond to non-surgical treatment or have serious neurological compromise.

INTRODUCTION
Low Back Pain (LBP) and sciatica continue to be a leading cause of disability in the United States with documented socio-economic impact. The conventional wisdom that in most cases the pain will resolve on its own within a few weeks is true, but recent evidence indicates that the relief from such “self-healing” is followed by a significant incidence of recurrence, usually in less than a year. It is an unfortunate fact that symptoms result from degenerative changes in the spine – an ongoing process that has no cure. Nonetheless, symptoms can usually be successfully managed with a multimodal approach.

Any plan for treating these problems must take into account the natural history of symptomatic Herniated Nucleus Pulposus (HNP) and Spinal Stenosis (SS). In most cases HNPs resolve, as do the symptoms they cause, so the goal should be to relieve symptoms during the phase of self healing. Concurrently, patients need to be educated that most HNPs do not need to be removed, and that most bulging discs, bone spurs, and acquired spinal stenosis, occur “naturally” with aging. Furthermore, ESIs do not remove, shrink, or change the appearance of HNPs. Rather; they are indicated to reduce sciatica and LBP caused by suspected nerve root irritation and/or inflammation.

Spinal Stenosis (Fig. 1) is primarily an acquired condition in which bulging discs, hypertrophic facet joints, and bone spurs “grow” into and narrow the epidural space and neural foramina. This process leads to both direct compression and ischemia of the nerve roots. Early symptoms often wax and wane and often respond to ESI, whereas severe stenosis requires surgical care or permanent modification of activity.

The rapid relief of symptoms often provided by ESI hopefully provides patients with a more positive outlook about their chronic ailment, so they are more willing and able to weather the weeks to months required to determine whether they will self-heal. That ESIs are only a “temporary fix” is no reason to discount their utility in a well planned, multi-modal approach to therapy. After all, with the current state of the art, no treatment option – even surgery – provides guaranteed long-term relief of LBP.

ESI’s have been used for decades to treat both LBP and sciatica. Unfortunately, they have been, and likely will continue to be, over utilized. Because they have a very safe track record and are viewed as an alternative to surgery, some practitioners have been willing to perform...
them without careful clinical deliberation, and “work order” epidurals for most any type of back pain have been too commonplace. Since all anesthesiologists are trained to enter the epidural space without fluoroscopic guidance, such “blind” ESI is readily available. But although there are indeed many clinical reports of excellent outcomes and low risk, controlled studies of outcomes have been – at best – limited, and the best designed studies have utilized the outdated and unreliable blind interlaminar approach (see below) to the epidural space. Indeed, several of these studies have refuted the benefits of ESIs in this patient population.4,5

MECHANISMS OF LOW BACK PAIN AND SCIatica

Spinal pain usually arises from damage to or degenerative changes in the spinal nerves, intervertebral discs, facet joints, muscle/fascia, and dural tissue surrounding the spinal nerve roots.6

Facet joints may be responsible for 14–45% of cases of LBP6 most often as a result of degenerative changes or trauma that causes inflammation of the joint capsule from overloading. Degenerated and herniated discs are other common causes of LBP and sciatica. Though the mechanism is still not certain, animal studies indicate that when there is nucleus pulposus tissue in the epidural space, it induces an inflammatory response, neurotoxicity, and thrombosis, all of which can lead to nerve root ischemia and irritation.7 Fissured, degenerative discs are thought to cause pain by allowing growth of sensory fibers from the sinuvertebral nerve into the inner layer of the annulus fibrosis and nucleus pulposus which are normally not innervated.8

It remains unclear to what degree nerve root compression or irritation is responsible for radicular pain and LBP. In general, sciatica type pain is most-likely due to nerve root compromise (radiculopathy), while axial back pain is more indicative of a “mechanical origin” such as facet syndrome, discogenic pain, or muscular pain. Radicular-dominant pain is many fold more likely to respond to epidural steroid injections than back-dominant pain.
BASIC MECHANISMS AND ANATOMY OF ESI

Although the actual mechanism of action is not fully known, there is evidence that corticosteroids achieve pain relief by inhibition of pro-inflammatory mediators (e.g. neural peptides, phospholipase A, acid hydrolases, histamine, and kinin) and by causing a reversible local anesthetic effect (decreased sensitivity of nerve roots to irritants).9,10

The epidural space is a potential space that surrounds the thecal sac circumferentially from the foramen magnum to the sacral hiatus (Fig. 2). It is bordered anteriorly by the posterior longitudinal ligament, posteriorly by the ligamentum flavum, and laterally by the intervertebral foramina and pedicles. Its contents include neural tissue (spinal cord and nerve roots), as well as fat and vascular tissue. The posterior epidural space is highly compartmentalized with connective tissue planes and a medial divider (plica mediana dorsalis), all of which influence the direction of flow of injectate within the epidural space. In one study, 84% of interlaminar injections resulted in unilateral flow,11 which can be a critical issue when treating unilateral or bilateral symptoms. Blind injections cannot confidently be placed on the right or left, or at a specific level, let alone in the epidural space.12,13 Contrast enhanced, image-guided, fluoroscopic injections are the only reliable method to place injected agents accurately in the epidural space.

INDICATIONS FOR ESI

ESIs are indicated for treating radicular and LBP caused by annular tears, HNPs (with or without image-confirmed nerve root compression), chemical neuritis, spondylosis, and spinal stenosis. ESIs are most effective during the acute phase of pain and inflammation.14 Response rates that range up to 90% in patients with symptoms for less than 3 months fall under 50% in patients with symptoms for more than a year. As discussed earlier, ESI is best used in conjunction with other conservative measures to relieve painful symptoms with the understanding that most of these conditions will self–heal with time. The longer symptoms persist without responding to conservative measures, the less likely they are to improve, and the more likely they will require surgical treatment. Other than duration of symptoms, a poorer response rate to ESI is predicted by prior lumbar surgery, and severe compression by spinal stenosis or a large HNP.

ESIs have been repetitively shown to be unreliable and ineffective in treating pure LBP, for which they have little or no indication. Musculoskeletal pain (muscle, joint, disc) can mimic true neurogenic radicular pain, and there is no reliable diagnostic test other than provocative neuro-blockade to identify pain mediators.

METHODS AND ROUTES OF ADMINISTRATION

There is little agreement regarding the type, dosage, frequency, or total number of ESIs that will yield the greatest efficacy and safety. The recommendations often quoted are for a series of 3 injections spaced two weeks apart: 3 mg/kg body weight; 210 mg annually, or 240 mg in a lifetime. These figures are based on old studies done with poorly supported rationale.10 Considering the plethora of agents available, the three routes of entry, and the countless personal preferences of providers, it is not surprising that analysis of outcome studies often leads to confusion and uncertainty.

The three routes of entry to the epidural space are Caudal, Interlaminar, and Transforaminal. All are actively practiced today and have their own unique risks and benefits. The superiority of one route over the others is controversial because of documented as well as perceived differences in efficacy and safety.9 Evidence-based guidelines have been drafted by the American Society of Interventional Pain Physicians in an ongoing project with continuous updates. For the current management of chronic low back or radicular pain, Interlaminar ESIs offer strong short-term relief and limited long-term relief;
Caudal and Transforaminal ESIs provide strong short-term relief and moderate long-term relief. All techniques have limited benefit in managing post laminectomy syndrome and spinal stenosis.

**Caudal ESI** (Fig. 3) was the first to be used for sciatica symptoms. The caudal injection is made below the level of involvement at the most caudal aspect of the epidural space. It is the easiest route into the epidural space with the lowest risk of inadvertent puncture of the dura, so it was popular among those who had limited training. It is the least target-specific approach; it requires the largest volume of injectate (which dilutes the steroid that reaches the site of pathology); it reaches only the lower lumbar nerve roots; and it is not useful to treat problems above the lower lumbar region. More recent catheter-assisted techniques have made it more useful in patients with prior lumbar surgery.

**Interlaminar ESI** (Fig. 4) gained popularity because it permits placement of steroid from the low lumbar region to the cervical spine at the level and site of pathology. Classically, this technique was performed blindly, but those experienced in interventional pain management now performed it with fluoroscopic guidance, which greatly increases the accuracy of drug placement. Still, the drug is mostly confined to the posterior epidural space, with anterior spread in less than 40% of injections. Though there is an increased risk of dural puncture with this technique, the risk is still quite low (approximately 1%).

**Transforaminal ESI** (Fig. 4) has been developed only in the past 10-15 years, and is designed to administer the most target-specific agent to the affected nerve root. Injections are made directly into the neural foramen, which allows spread of steroid to the anterior epidural space, which is believed to be the site of disc-nerve interface. Recent outcome studies reveal greater duration of pain relief and avoidance of surgery compared with interlaminar injections.

**COMPLICATIONS**

The list of most common complications of ESIs can be divided into those caused by needle placement and those caused by the administered drug. Complications from needle placement are fortunately rare, and include dural puncture, spinal cord trauma, epidural hematoma, nerve damage, headache, vascular injury, and death. Relatively few complications have been reported due to the pharmacology of the injected steroid. Review of the literature on intrathecal steroid injections reveals no proof of clinically significant arachnoiditis. In addition, no direct evidence of neurotoxicity of steroids administered in the lumbar region has been confirmed in the clinical setting. Transient suppression of the pituitary-adrenal axis has been universally observed with ESIs. Other potential side effects include weight gain, osteoporosis, and avascular necrosis of bone.

The most worrisome complications of ESIs are permanent neurological injury and death. Though rare, there are several case reports of nerve root and spinal cord injury that led to paralysis and death. In particular, several case reports of transforaminal injections have described catastrophic complications such as spinal cord infarction, massive cerebral edema, anterior spinal artery syndrome, and intra-cord injections. This experience has tamed the enthusiasm to embrace transforaminal-ESIs and abandon other approaches, though with proper precautions and standardized techniques, major complications should be rare.

Epidural hematoma is the most frequent concern, since more and more patients are receiving anti-platelet and anticoagulant therapy. Guidelines for spinal injections in patients taking anticoagulants have been drafted by the American Society of Regional Anesthesia.
should be discontinued 4-5 days in advance, to yield an INR <1.5. Plavix and Ticlid should be withheld for 7 and 14 days respectively. Epidural hematomas, though rare, can lead to rapid compression of the spinal cord with paralysis if not recognized promptly and treated aggressively.

CONCLUSIONS
Epidermal steroid injections are effective and safe in providing short and long term pain relief for sciatica and LBP when proper patient selection and contemporary techniques are used. They are not intended to replace surgical treatment for spinal disorders, but to aid in avoiding surgery if possible. Surgery should be reserved for those who do not respond to non-surgical treatment or have serious neurological compromise. Unfortunately, patients are usually not given the option of ESI until their condition has become chronic, and after multiple and often more costly conservative therapies have been exhausted. ESIs are most effective during the acute phase of pain and inflammation, and response rates fall as the duration of symptoms increases. For the patient with LBP with a radicular component, they should be utilized early, in combination with other non-surgical treatments, to provide relief of pain and suffering during the period of self-healing.

REFERENCES


