# Epidural Injection in Chronic Pain Management

39

Ramana K. Naidu

## Key Points

- Epidural analgesia has been used for chronic pain management for over a century.
- The anatomy of the spinal epidural space has several misconceptions, and proceduralists should be knowledgeable about the details of this unique space.
- There are several approaches, trajectories, and techniques to enter the epidural space and several needle designs have been employed and modified over time.
- Therapy using local anesthetics, steroids, and normal saline has been widely used for epidural analgesia.
- The risks of epidural analgesia include post-meningeal puncture headache, hematoma, infection, and neurologic complications.
- Outcomes are mixed. Certain conditions that result in radicular pain appear to have short-term benefit.

## Introduction

The reported first attempt and successful injection of drug for chronic pain management occurred in 1901 in France by Jean-Athanase Sicard and Fernand Cathelin. The unique features of injection into this relatively small space were the production of *segmental neural blockade* and *influence on neuraxial pathology*. Since then, the epidural space has been used to manage acute, chronic, and cancer pain. This review will focus on its use in chronic pain management.

R.K. Naidu, MD (✉)
University of California at San Francisco, Department of Anesthesia & Perioperative Care, Division of Pain Medicine, 2255 Post St, San Francisco, CA 94115, USA
e-mail: ramana.naidu@ucsf.edu

## Anatomy

A discussion regarding epidural analgesia is founded on an understanding of the anatomy, which has gone through several controversies over the past century. It is imperative that practitioners are aware of the details and potential aberrancies of this unique anatomical structure (Fig. 39.1). The spinal epidural space is distinct from the cranial epidural space; the spinal epidural space is often coined a potential space, but the fact that it is filled with fat, arterioles, Batson's venous plexus, and lymphatics with millimeters of thickness which can be viewed with any MRI or CT contradicts the definition of a potential space—it is in fact an actual space (Figs. 39.2, 39.3, and, 39.4). The cranial epidural space is a potential space. The posterior spinal epidural space is between dura and ligamentum flavum and runs from foramen magnum to the sacrococcygeal ligament (Fig. 39.5). On the lateral aspects of the epidural space, rootlets exit the neuroforamina. Injectate flows along rootlets, to the nerve roots and dorsal root ganglia (Fig. 39.6) [1]. The thickest portion of the epidural space is typically at the interlaminar interspace in the posterior midline. The anterior aspect of the epidural space which is between the posterior longitudinal ligament, and the dura is of interest because this is where injectate may have an impact on disc pathology. There is significant heterogeneity of the epidural space with changes in thickness anterior to the spinous process and around pedicles; although often depicted as a contiguous and symmetric sheet based on artists' renderings in textbooks, it is more often not (Fig. 39.7). In rare instances, *plica mediana dorsalis*, a midline septum, can prevent bilateral spread of injectate [2].

## Anatomy of Pathology

There are several etiologies for neck and back pain with or without radicular pain. The discs have nerves, including the sinuvertebral and gray rami innervating the annu-



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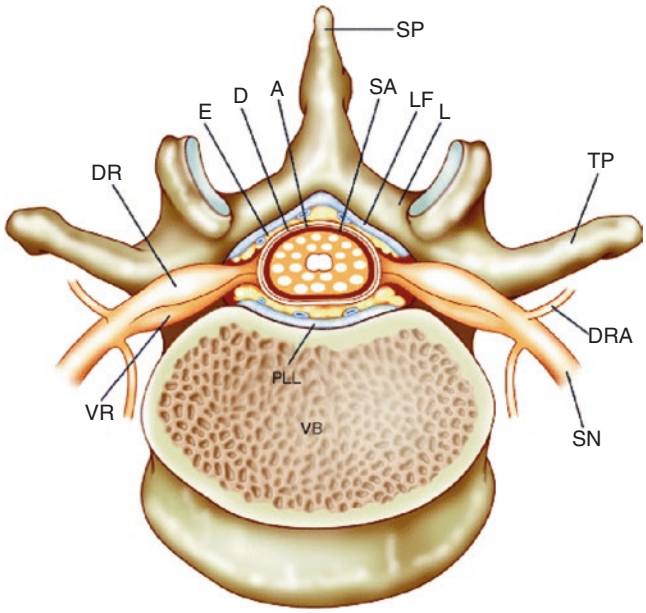


**Fig. 39.1** Local anesthetics may abolish sensation in various parts of the body by topical application, injection in the vicinity of peripheral nerve endings and along major nerve trunks, or instillation within the epidural or subarachnoid space. The ensuing sensory block occurs locally and spreads to areas distal along the nerve pathway (With permission from Deer et al. [27]. © American Academy of Pain Medicine 2013)

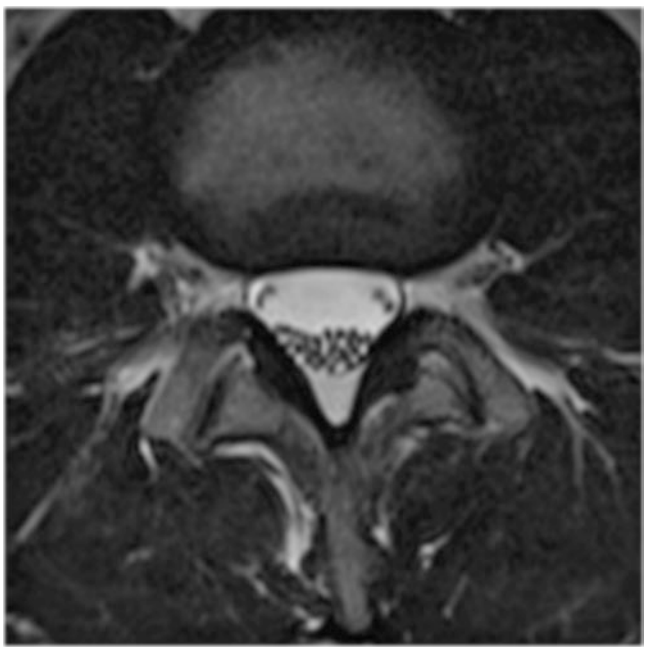

**Fig. 39.2** Magnetic resonance imaging of lumbar spine. Axial T2-weighted image demonstrates the dorsal and lateral epidural space (Acknowledgement of Dr. Alex Schabel)

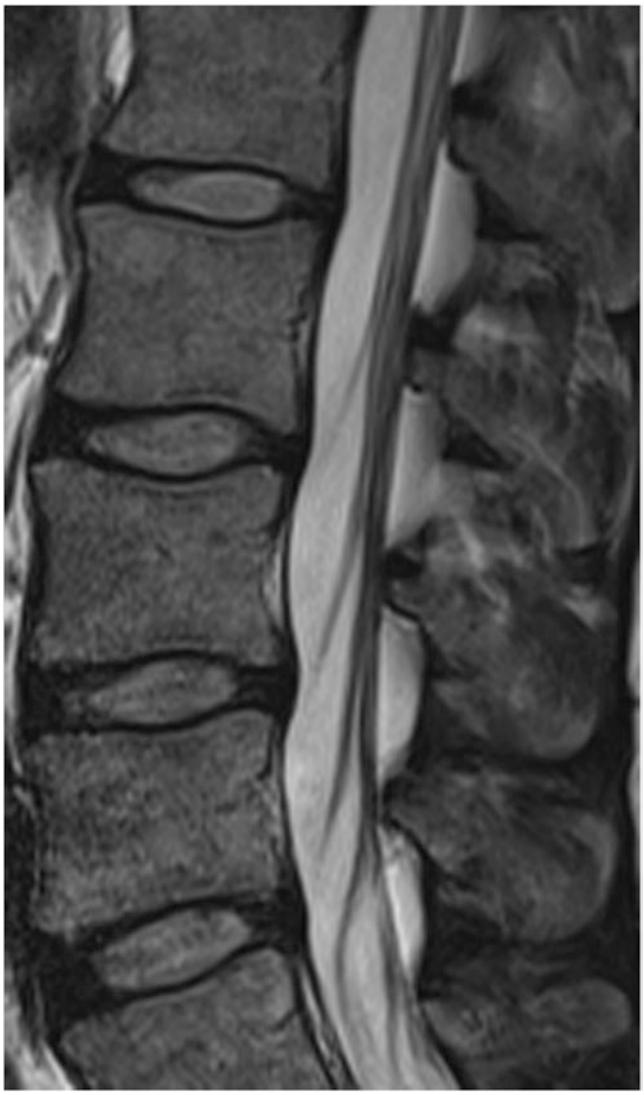

**Fig. 39.3** Magnetic resonance imaging of lumbar spine. Sagittal T2-weighted images revealing the typical "sawtooth" pattern of the posterior midline epidural fat (Acknowledgement of Dr. Alex Schabel)

lus fibrosus which can be nociceptive due to inflammation or trauma. When there is a disc herniation, the nucleus pulposus, a vestige of the notochord, may extrude its contents of proteoglycans onto nerves causing significant nociceptive input. The natural history of disc herniation is not as dire as once believed—most patients recover without intervention as discs do protrude, extrude, and absorb over time [3] although predicting where resorption versus continued pathology will occur and in whom is difficult to predict at this time.

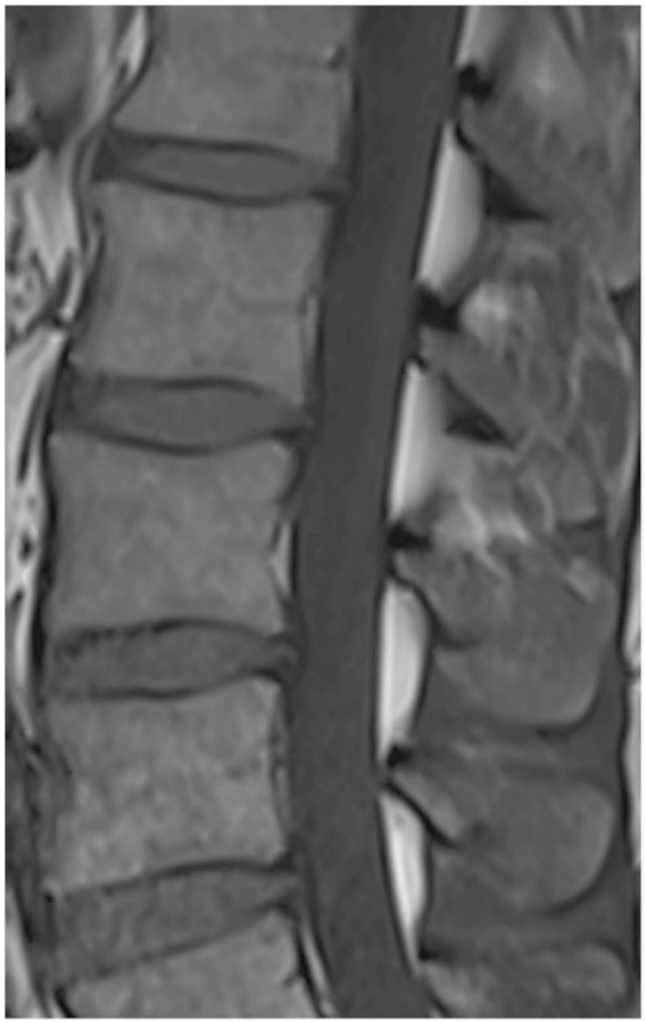

**Fig. 39.4** Magnetic resonance imaging of lumbar spine. Sagittal T1-weighted images revealing the typical "sawtooth" pattern of the posterior midline epidural fat (Acknowledgement of Dr. Alex Schabel)

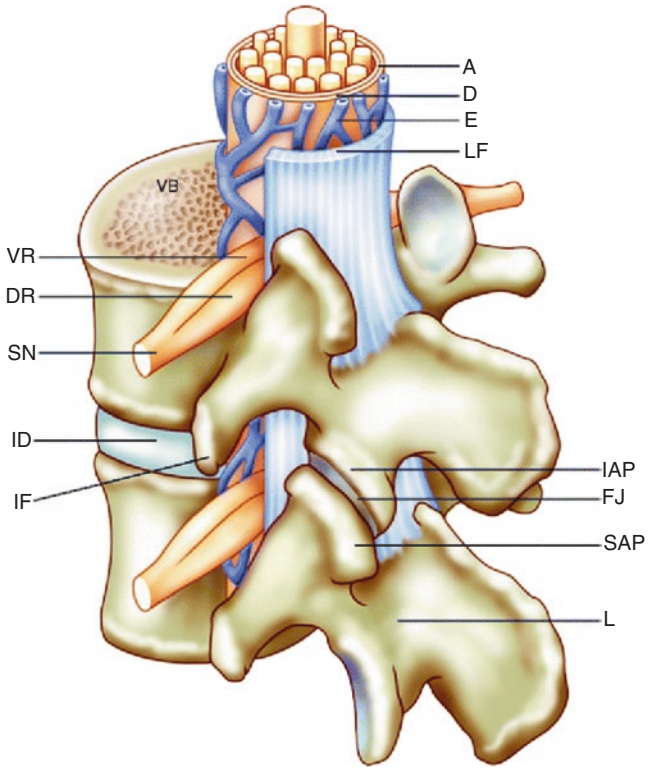


**Fig. 39.5** Layers of the neuraxial canal (With permission from Deer et al. [27]. © American Academy of Pain Medicine 2013)

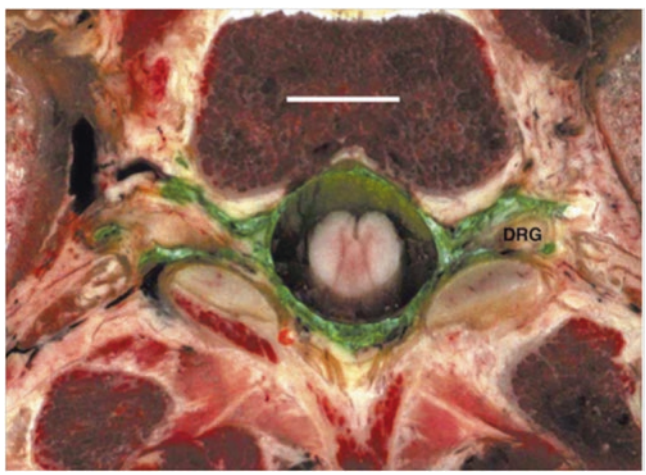


**Fig. 39.6** Axial cryomicrotome of the second thoracic vertebrae and spinal nerve. *DRG* dorsal root ganglion (With permission from Hogan [1]. © Wolters Kluwer Health Inc.)

The neuroforamina may be occluded causing lateral stenosis due to disc herniation, facet hypertrophy, synovial cyst, or scarring. Central spinal canal stenosis can be congenital, as in short pedicle syndrome, or due to degeneration which has an estimated prevalence of 19.4% of the US population aged 60–69 years with absolute spinal stenosis based on CT imaging; not all of these patients have symptoms of neurogenic claudication [4]. Stenosis can result from disc herniation, ligamentum flavum hypertrophy, vertebral osteophytosis, posterior longitudinal ligament osteosis, facet hypertrophy, compression fracture, or spondylolisthesis.

In addition to understanding these structural components that may stimulate nociceptive inputs, it is crucial that practitioners understand that pain is an unpleasant sensory and emotional experience [5]. An individuals' genotype, phenotype, and psychological makeup also play a role in what they describe as pain, or suffering.

## Techniques

The epidural space can be approached via several routes in the sacral, lumbar, thoracic, and cervical spine (Fig. 39.8). The *interlaminar* approach involves a posterior introduction of the needle between the superior and inferior lamina

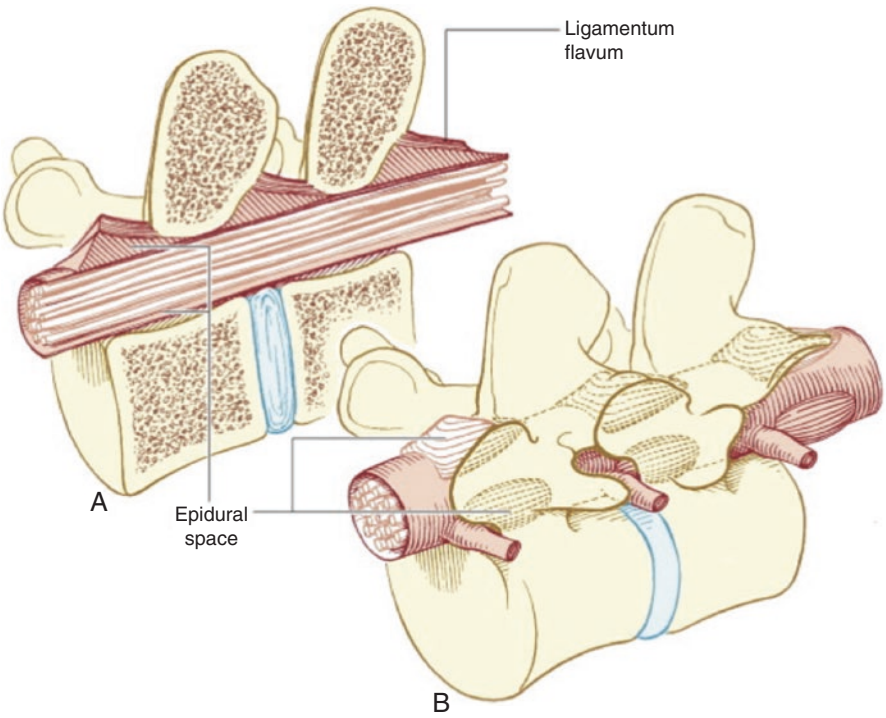


**Fig. 39.7** Conceptual model of the epidural space with pockets of incongruity (Image obtained from SlideShare.net)

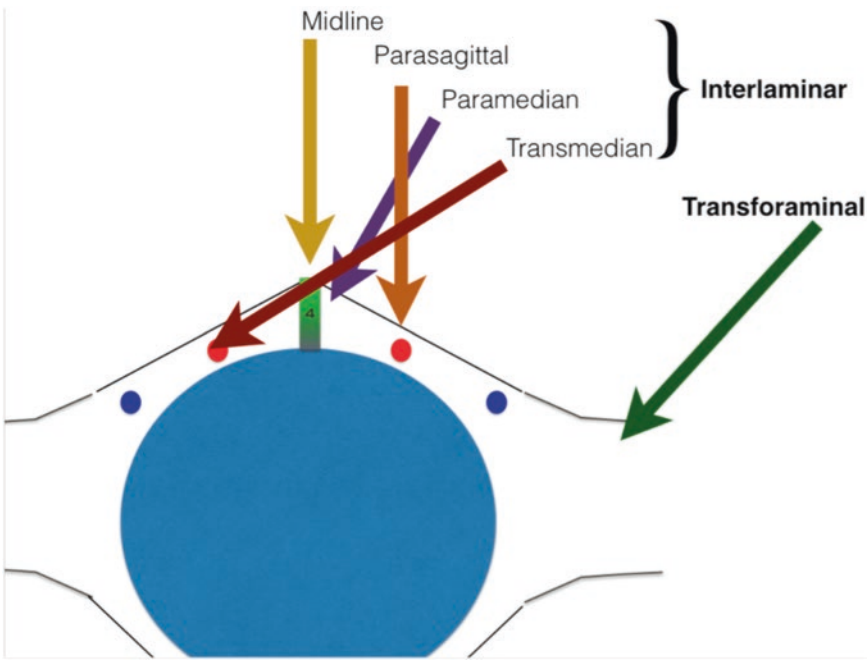


**Fig. 39.8** Conceptual approaches to enter the epidural space. The interlaminar approach has four trajectories: midline, paramedian, parasagittal, and transmedian. The transforaminal approach enters laterally in the neuroforamina

of the desired levels of the cervical, thoracic, and lumbar spine. The commonly performed trajectories include the midline and paramedian. Some operators choose to perform a parasagittal or transmedian trajectory to deposit injectate to one side of the epidural space, ideally where the pathology exists. The *caudal* approach enters the epidural space from its most inferior aspect at the sacral hiatus with piercing of the sacrococcygeal ligament. Determination of the approach or interspace to enter and/or target depends on where a patient describes their nociceptive input, which is often corroborated by provocative testing and radiologic imaging. The type of needle used for this approach is generally a more blunt needle so operators can feel the differences in tissue, with particular attention on the pressure

gradient on the syringe plunger between the ligamentum flavum and into the epidural space [6]. The Tuohy, Coudé, Weiss, Crawford, or Hustead needles are blunt needles and come in a variety of sizes for entry from 14 gauge for spinal cord stimulator leads to 22 gauge for single-shot injection. Catheters come in various materials and can be steered through needles to the area of pathology. The Tuohy needle was developed by Ralph Huber, DMD, and utilized by Edward Tuohy, MD, for continuous spinal anesthesia in 1945 [7]. This needle has since been used for continuous epidural analgesia, starting with Curbelo on 1949, and has the advantageous property of a scalloped surface on the back of the bevel allowing for laminar sliding.

Entry into the epidural space can be determined via several approaches, most commonly, the loss-of-resistance technique, which was developed by the Italian physician, Achille Mario Dogliotti, in 1933. The loss-of-resistance technique can be accomplished with a low-friction piston/syringe (glass or plastic) using saline or air. Saline is recommended to reduce the potential risk of pneumocephalus if there is intrathecal infiltration, although there may be situations where using air has advantages [8]. Other ways to determine entry into the epidural space include the hanging drop method, catheter insertion, fiber optics, and epiduroscopy.

The *transforaminal* approach starts at a point lateral on a patient's back and enters via the neuroforamen. This technique does not use loss-of-resistance and utilizes a spinal needle (Quincke, Chiba) to end either adjacent to the nerve root or at a point contiguous with the epidural space. All of these techniques are confirmed using contrast media under fluoroscopy to watch spread along nerve roots or with epidural fat delineation and spinous process sparing, to differentiate from intrathecal spread, or a myelogram. The landmark or blind approach has fallen out of favor due to poor reliability in entering the epidural space, although was the pioneering method for entry. Ultrasound can be a useful imaging tool, particularly in pregnant patients where radiation is contraindicated [9].

The epidural space can be used to place catheters for continuous infusion such as in acute or cancer pain, to place leads for dorsal column stimulation, or if adhesions in the epidural space are a source of nociception, adhesiolysis can be performed. The remainder of this chapter will focus on epidural steroid injections.

## Therapeutic Injectate

Local anesthetics were the first drugs injected into the epidural space, starting with cocaine in 1901. Even with the longest-acting local anesthetics to date, relief is still on the order of several hours via voltage-gated sodium channel blockade. For this reason, other agents have been added to prolong relief. Steroids were introduced into the epidural space in 1952 by Robecchi and Capra, and despite a voluminous history of epidural steroid injections, steroids are not FDA approved for use in the epidural space today. The North American Spine Society, Agency for Healthcare Research and Quality, and Department of Health and Human Services recognize that epidural steroid injections are part of the management for radicular pain. It is hypothesized that steroids work by decreasing inflammation by inhibiting PLA2 [10], reducing vascular permeability, or by epigenetic mechanisms [11], which may account for the delay (days) in relief. The relief from steroids is variable from patient to patient. Glucocorticoids are preferred for epidural steroid injections because of their greater anti-inflammatory activity (Fig. 39.9). There has been controversy regarding the ability of particulate steroids to aggregate and their potential role in neurologic injury. For this reason, non-particulate steroids are recommended. Greater scrutiny of steroids occurred in 2012 when the New England Compounding Center (NECC) shipped methylprednisolone that was contaminated by fungi leading to 800 individuals developing meningitis—this tragic event led to 64 deaths [12]. Steroid frequency and dose must be monitored; chronic neuraxial steroid use can lead to hypercorticism, adrenal suppression, osteopenia, impaired glucose tolerance, and increased intraocular pressure, among other side effects.

Other adjuvants in chronic pain management have not been as widely explored as compared to acute pain management where adjuvants such as epinephrine, clonidine, neostigmine, cyclooxygenase inhibitors, etc. have been used. One of the unspoken therapeutics in epidural injections is normal saline, a substance that is not benign. Future development of therapeutics in the epidural space to modulate pathology, dorsal root ganglia, or nerves could be impactful in chronic pain management (Table 39.1).

## Contraindications and Risks

The contraindications to this procedure include patient refusal, an allergy to any of the substances in contact with the patient, systemic infection or local infection at the needle entry site, bleeding dyscrasia, or on an antithrombotic agent without proper cessation. In a retrospective study of 4265 ESIs, the most common complications were increased pain (1.1%), pain at site of injection (0.33%), persistent numbness (0.14%), and "others" (0.80%) [13].

Meningeal puncture is a risk and can result in postural headache. Bernards expressed that the term post-dural puncture headache (PDPH) is inaccurate because dura mater is actually porous; he advocated the term post-meningeal puncture headache (PMPH) [14]. PMPH appears to be more rare in chronic pain management, with one retrospective analysis

| STEROIDS | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Drug | Approximate Equivalent Dose | Relative Anti-Inflammatory Potency | Relative Mineralcorticoid Potency | 0-10 micrometers (Benzon) | 11-20 micrometers (Benzon) | 21-50 micrometers (Benzon) | >50 micrometers (Benzon) | Particle Image (Benzon) |
| Short | Cortisone | 25 mg | 0.8 | 2 | | | | | |
| | Hydrocortisone | 20 mg | 1 | 2 | | | | | |
| Intermediate | Prednisone | 5 mg | 4 | 1 | | | | | |
| | Prednisolone | 5 mg | 4 | 1 | | | | | |
| | Triamcinolone | 4 mg | 5 | 0 | 71 | 8 | 9 | 12 |  |
| | Methylprednisolone (Depo-Medrol®) | 4 mg | 5 | 0 | 53 | 11 | 8 | 27 |  |
| Long | Dexamethasone (Decadron®) | 0.75 mg | 25-30 | 0 | 0 | 0 | 0 | 0 |  |
| | Betamethasone Sodium Phosphate (Celestone®) | 0.6 - 0.75 mg | 25 | 0 | 61 | 7 | 10 | 22 |  |
| | Betamethasone Sodium Phosphate | 0.6 - 0.75 mg | 25 | 0 | 0 | 0 | 0 | 0 |  |
| | Betamethasone Sodium Phosphate/ Betamethasone Acetate | 0.6 - 0.75 mg | 25 | 0 | | | | |  |

**Fig. 39.9** Relative properties of steroids (Adapted from Benzon et al. [26])

**Table 39.1** Indications in chronic pain management

| |
|---|
| Disc herniation |
| Central spinal canal stenosis |
| Neuroforaminal stenosis |
| Facet or nerve root cyst with radicular pain |
| Compression fracture of the spine with radicular pain |
| Postherpetic neuralgia |

stating an incidence of 0.004% [13], than it is in labor analgesia, approximately 1% [15], due to several factors including imaging and age differences. Management of PMPH is generally time, fluid, caffeine, cyclooxygenase inhibitors, potentially triptans, and epidural blood patch [16].

Risks of epidural injections include bleeding in the epidural space resulting in an epidural hematoma, which could lead to paralysis if not identified and evacuated [17, 18]. In 2015, the American Society of Regional Anesthesia and Pain Medicine (ASRA) developed their guidelines on the use of antithrombotics for interventional pain procedures. [19] It should be noted that even strict adherence to these guidelines does not prevent this complication absolutely. Patients are at rare risk for segmental medullary artery or artery of Adamkiewicz vasospasm or occlusion from transforaminal injections; this can result in anterior spinal artery ischemia and paralysis [20]. Infections can occur locally at the site of injection, cellulitis; into the epidural space, becoming an epidural abscess; or along the meninges, to become meningitis. Needles and catheters have found their way into and around nerve roots, as well as into the spinal cord. Development of cauda equina syndrome has been reported [21]. A review of methods to reduce neurologic complications related to epidural steroid injections was published in *Anesthesiology* and is worth review [22].

## Outcomes

The study on the outcomes of epidural steroid injections has historically been poor, and in order to understand the impact of this intervention on individuals and populations, better studies must be conducted and published without bias. Various operators, routes, injectate properties, approaches, pathologies, and individual differences make

studying outcomes an extraordinary challenge. After the NECC fungal meningitis outbreak, *The New England Journal of Medicine* published a perspective article that stated "clinicians persist in clinical practices despite weak evidence of efficacy." [23] A review and meta-analysis in *Pain Physician* in 2015 looked at 52 articles that met manuscript criteria (with 72 excluded) and concluded that there is level II evidence for ESI for disc herniation, discogenic pain, postsurgery back syndrome, and spinal canal stenosis [24]. In 2013, Cohen et al. summarized the evidence regarding the use of epidural steroid injections, their impact on patient beneficence, cost-effectiveness, prevention of surgery, return to work, and healthcare utilization [25].

## Conclusion

At 115 years since the first epidural for chronic pain management, we are still in our developing stages of understanding the power of the epidural space. Its physiologic purpose is enigmatic, and some interventionists feel its purpose is to provide a conduit to one of the most epidemiologically pressing health problems in humans—low back and neck pain. We have a long way to go to understand which candidates are best for such intervention to improve our currently shaky outcomes. Refining understanding, techniques, and development of drugs will progress the utility of epidural analgesia in chronic pain management.

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