Minimally-Invasive Interventional Spine Treatment – Part 1

Flouroscopically-directed spinal injection techniques may improve the efficacy of physical therapy and functional restoration protocols.

Minimally invasive interventional spine treatment has grown in importance during the past several years. In this first of a two-part series, we examine the common intradiscal procedures and spinal injections not involving the discs and we present the most common spinal procedures utilized to diagnose and treat spinal pain. Part 1 examines injections for diagnosing, localizing, and ultimately blocking spinal pain generators.

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The patient diagnostic work-up should commence with a well-documented, detailed, and directed historical account, together with a focused physical examination of the involved body parts. Historical emphasis should be on the duration of symptoms, previous attempts at procedures, and the functional approach to their disability. The signs of symptom magnification and malingering should be noted and documented. Notwithstanding potential discomfort, a thorough functional, social, and psychological history should be reviewed with the patient. Selective imaging studies (e.g., plain radiographs, MRI, CT scan, myelography, bone scans) can be useful added screening aids in further evaluating the patient, when compared to the history and physical. During the evaluation, additional screening and diagnostic studies (such as laboratory work-up and electromyography) can be useful in determining the correct diagnosis, and possibly ruling out other diagnoses. Electrodiagnostic studies (i.e., EMG/NCS) are useful for detecting neurogenic changes, denervation activity, differentiating multiple root vs. plexus lesion involvement, as well as the extent or severity of these changes, and the level of involvement. Unlike imaging studies, electrodiagnostic studies provide electrophysiological information, which is helpful to the clinician in determining an accurate diagnosis. Lastly, diagnostic interventional procedures can be useful in providing valuable insight into the patient's primary pain generator(s), anatomic defect(s), pain threshold, and psychological response to treatments given.[1-14]

Role of Spinal Injections in Spinal-related Pain

The use of selective spinal injections in the treatment of spinal-related pain disorders have obvious diagnostic and therapeutic values for the affected patient. They involve discrete, well-controlled injection techniques directed at specific target sites in and around the spine, which usually involve the use of fluoroscopy to aid in the proper needle placement and in so doing, may help increase the accuracy and efficacy of the specific injection technique (see Figure 1). Spinal injections are an extremely useful adjunct to other clinical evaluation tools, in precisely diagnosing and localizing the clinically significant spinal pain generators. This technique is especially valuable for multilevel disc disease, suspected chemically-mediated symptoms (when demonstrated by imaging or electromyographic testing in situations with no obvious abnormalities), or in post-operative cases where anatomic boundaries are disrupted and imaging studies are difficult to interpret accurately. When combined with corticosteroids (which interfere with inflammatory mediators, membrane stabilization, and suppression of ectopic neuronal discharges) and other anesthetic solutions (which cause reversible nerve conduction block), they provide a dramatic therapeutic benefit. They provide a specific beneficial role for individuals by relieving pain and increasing the range of motion prior to, or during, the rehabilitative process and so allows the patient to participate more fully in the therapy program. These selective spinal injections are indicated specifically in the medically-stable patient and are considered outpatient, minimally-invasive, minor surgical procedures. For patients with failed back surgical syndrome (FBSS) and prior to proceeding with operative interventions (e.g., spinal fusion, microdiscectomy, or laminectomy and decompression), the coupling of injection procedures with an accurate history and physical exam, and the confirmation with the appropriate imaging and/or electrophysiologic study, can greatly assist the spinal surgeon and treating physician to make an informed decision regarding a more directed and efficient treatment program.

The goal of diagnostic selective bocks is to differentiate the qualitative and quantitative contributions of discogenic, radicular, and posterior element pain sources. Because of the required precision in needle localization—and technical difficulty in performing these procedures—the use of fluoroscopy and contrast dye is essential. Epidural injections are frequently performed without radiographic guidance, but incorrect needle placement can occur in up to 25% of cases, including subcutaneous, intraligamentous, and intravenous locations. Therefore, fluoroscopic visualization with an epidurogram, perisheathogram, or arthrogram is highly recommended, especially in postoperative cases.

Pain reproduction during these procedures may also help to more accurately identify the painful structure. Typically, nonaffected nerve roots will not trigger as severe a pain response when mechanically irritated by a spinal needle or contrast dye. Often, comparison of pain levels prior to and after the injections — by patient verbalization, pain diaries, or visual analog scale — is very helpful in gauging the response to the anesthetic procedure. Afterwards, provocative maneuvers such as evaluating spinal range of motion, straight leg raise, and ambulatory capabilities pre- and post-injection may also assist in identifying a particular site as the actual pain source. Exaggerated or extreme pain behaviors during the procedure provide information regarding nonphysiologic causes for pain.[15-31]

Diagnostic and Therapeutic Spinal Injections

Selective spinal injections are being performed with increasing frequency in the management of acute and chronic pain syndromes. A few of the most common indications for these diagnostic and therapeutic spinal procedures are noted as follows:

- Spinal nerve radiculopathy;
- Spinal stenosis;
- Discogenic pain (i.e., symptomatic, internal disc disruption);
- Contained, disc bulge, or protrusion vs. extruded or sequestered herniated disc;
- Multilevel degenerative disc disease;
- Facet joint arthropathy or associated facet joint nerve pain;
- Sacroiliac joint pain dysfunction;
- Failed back surgery syndrome (FBSS);
- Epidural and/or perineural fibrosis/granulation with associated symptomatic pain;
- Complex regional pain syndrome (CRPS) (formerly known as reflex sympathetic dystrophy, RSD).

Although numerous interventional procedures are used to treat spinal-related pain conditions, a few of the most common types of diagnostic and therapeutic spinal injections are noted as follows:

- Epidural steroid injections (translaminar, transforaminal, caudal);
- Facet joint nerve blocks and facet joint intra-articular injections;
- Neurolytic and radiofrequency (RF) nerve ablation procedures;
- Sacroiliac joint and other intra-articular joint injections;
- Sympathetic ganglion nerve blocks;
- Diagnostic discographic injections;

The following sections will briefly describe these interventional spine procedures in common use for spinal-related pain.[32-50]

Epidural Steroid Injections (ESIs)

Lumbar epidural injections, as a treatment for lower back pain (LBP) and sciatica, were first introduced in 1901 when several investigators injected cocaine epidurally. In 1952, Robechhi and Capra reported the first experience with epidural injection of cortisone for the treatment of LBP and sciatica via the sacral route. Clinical use of epidural injections for the treatment of LBP and sciatica preceded well-controlled clinical trials to evaluate efficacy and so has led to much controversy and a poorly-formed body of literature. Inconsistencies in indications and protocols have been striking. Epidural cortisone injections (specifically lumbar) are primarily indicated for the treatment of acute, relapsing, and chronic radiculopathy, epiduritis, central canal stenosis, and foraminal stenosis. They are also indicated for other conditions, such as spondylolisthesis, scoliosis, compression fractures, herniated or painful discogenic conditions, arachnoiditis, and occasional sympathetic pain. The mechanism of action primarily involves reducing the inflammatory response and reducing the painful state through the use of cortisone and anesthetic solutions. Due to studies which have documented improper needle positioning even in experienced hands using a "blind technique" (i.e., not using fluoroscopic visualization with errors reported to be 40% using the caudal route and 30% using the translaminar route); most injectionists believe that fluoroscopic visualization of needle positioning and contrast flow is critical to optimize a proper and safe outcome

Contraindications for ESIs

Contraindications to the injections are separated into absolute and relative categories, as follows.

Absolute:

- bacterial infection,
- pregnancy (fluoroscopy),
- bleeding diathesis;

Relative:

- post-surgical altered anatomy,
- injectant allergies,
- NSAIDs,
- other antiplatelet agents,
- hyperglycemia,
- andrenal suppression,
- congestive heart failure, or
- steroid psychosis.

Potential Complications

- local infection,
- sepsis,
- pilonidal cyst,
- congenital abnormalities of the dural sac and its contents, and
- potential hematogenous spread via the Batson's plexus.

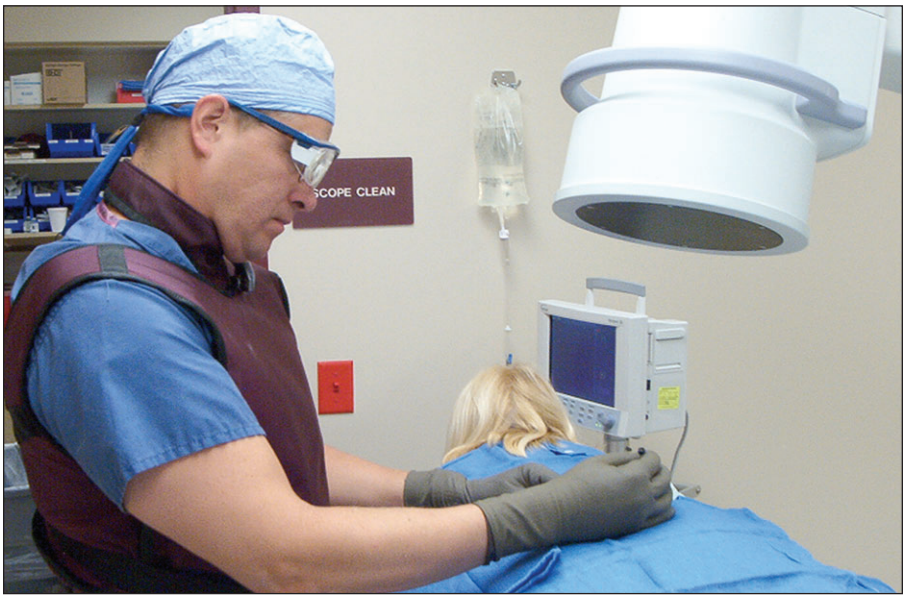

FIGURE 1. *Fluoroscopically-guided soinal injection procedure in a patient with lower back pain.*

Relative complications of epidural steroid injections (and other injections to be named subsequently) include: dural puncture, post-injection headaches, subarachnoid injection, intravascular injection, spinal cord or nerve root injury, epidural hematoma and abscess, aseptic meningitis, anaphylactic reactions, local anesthetic toxicity, and corticosteroid side effects, among others. Since these procedures require a needle to be placed in or around the spine, there are always relative and absolute risk of complications, which should always be realized by the injectionist, referring physician and, most importantly, by the patient.

The three most popular types of ESIs are briefly discussed: translaminar, transforaminal, caudal. It is noted that the term lumbar is used to describe epidural steroid injections, but when used generically, will refer to other levels of ESIs (i.e., cervical, thoracic, caudal).

Translaminar

The lumbar translaminar epidural injection is primarily indicated to relieve lower extremity radicular symptoms recalcitrant to conservative interventions including NSAIDs or oral corticosteroids, and appropriate physical therapy or exercises. The objective is to precisely deliver anesthetic and corticosteroid to the epidural disc and nerve root interface. No true role exists for a series of lumbar epidural injections given without regard to response to the initial injection. It is noted that since stenosis (central, foraminal, or lateral) and herniated nucleus pulposus can induce nerve root inflammation and functional nerve root changes; the nerve root inflammation causes radicular symptoms. Corticosteroid reduces morphologic and functional nerve root changes, while lidocaine and/or bupivacaine decreases nerve root inflammation and increases intraradicular blood flow. In so doing, this reduces the need for surgery because the natural history of lumbar radiculopathy is likely one of gradual resolution over a period of months to years. The procedure can be performed by either a midline or paramedian approach, although the latter is preferred by the author and many other injectionists who use fluoroscopic guidance.

Transforaminal

Transforaminal (including selective nerve root blocks) epidural injections instill medications along the affected nerve root and into the anterior epidural space adjacent to the disc herniation at the inflammatory tissue. Foraminal stenosis and herniated nucleus pulposus can induce nerve root inflammation and functional nerve root changes. Nerve root inflammation causes radicular symptoms. Corticosteroid reduces morphologic and functional nerve root changes, and lidocaine decreases nerve root inflammation, while increasing intraradicular blood flow. Therefore, a lumbar transforaminal selective epidural injection of corticosteroid relieves radicular symptoms. This may serve as a means of avoiding surgery since the natural history of lumbar radiculopathy is likely one of gradual resolution over a period of months to years. Successful long-term outcome is reported at approximately 75%. Transforaminal epidural injections are generally performed for two reasons: 1) medication did not flow in the desired direction because of local anatomic variation or abnormality or previous surgery at the level of pathology, or 2) the injectionist wishes to place the medication within the epidural space more precisely than may be done with the above techniques. In addition, if the volume of injectant is limited to 0.5-1.0 cc of local anesthetic, it may be used for diagnostic purposes. If the patient's extremity pain dramatically reduces within 30-60 seconds, it may be reasonably assumed that the anesthetized nerve root mediates the pain. The assumption is buttressed when a previous nerve root block performed in the same manner at a distant level failed to reduce pain. The contraindications and complications are similar to that for the translaminar approach.

Caudal

Although the discovery of a practical way to administer drugs via the caudal approach to the epidural space preceded that for the lumbar approach by almost 20 years, the popularity of the caudal epidural block has waxed and waned. Caudal epidural injections are similar to the translaminar approach noted above in many respects. The sacral hiatus has been used as a portal of entry to the spine for many reasons. Initially it was used to inject large volumes of local anesthetic or normal saline and cortisone in order to treat LBP. More recently it has been used to pass catheters selectively into the anterior epidural space and to inject specific regions. In addition, selective catheter placement has been used to disrupt and inject structures such as perineural adhesions and perineural cysts. In addition to applications for surgical and obstetric anesthesia, caudal epidural nerve block with local anesthetics can be utilized as a diagnostic tool when differential neural blockade is performed on an anatomic basis to evaluate pelvic, bladder, perineal, genital, rectal, anal, and lower extremity pain. It is useful in the treatment of a variety of chronic benign pain syndromes, including:

- lumbar radiculopathy,
- lower back pain syndrome,
- spinal stenosis,
- postloaminectomy syndrome,
- vertebral compression fractures,
- diabetic polyneuropathy,
- postherpetic neuralgia,
- reflex sympathetic dystrophy,
- phantom limb pain,
- orchalgia,
- proctalgia, and
- pelvic syndromes.

The caudal approach is especially useful in patients who have previously undergone low back surgery, which may make the lumbar approach to the epidural space less efficacious. Because of the simplicity, safety, and reduction in pain associated with the caudal approach to the epidural space, this technique is used in many pain centers instead of the lumbar epidural approach.[51-82]

Facet Joint and Medial Branch Blocks

The lumbar z-joints (zygapophyseal) were first identified as a source of pain in 1911. In 1933, Ghormley coined the term "facet syndrome" referring to the symptom complex associated with pain emanating from these joints. Subsequently, various types of localized, pseudoradicular, and sclerotogenous referred pain have been described initially from these joints in the lumbar and subsequently in the cervical and thoracic region. Injections to diagnose and control pain originating from the zygapophyseal (z) joint should always be used as an adjunct to aggressive, conservative spine care. These injections have become an

important yet sometimes controversial part of non-surgical spine care. The value of these injections has been disputed but, when appropriately used, they can provide both diagnostic and therapeutic benefit and value. Fluoroscopically-guided contrast-enhanced z-joint injection procedures help to specifically evaluate the z-joint as an isolated source of spine-related pain. These injection procedures also may provide short- and long-term pain relief through the therapeutic effects of the anesthetic and corticosteroid used. Pain relief allows patients to advance through their rehabilitation program more efficiently and rapidly and may result in overall improved patient function.

Because no reliable, non-invasive clinical findings or techniques exist for the accurate diagnosis of z-joint-mediated pain, and because the clinical features of z-joint pain, discogenic pain, ligamentous/muscular, and sacroiliac joint pain overlap greatly; fluoroscopically-guided z-joint injections of local anesthetics are commonly considered the gold standard for isolating or excluding the z-joints as the primary source of spine or extremity pain. Either intraarticular or medial branch blocks can be used in the diagnostic work-up. Physiologic analgesia is the underlying principle; pain relief after blockade of the nociceptive fibers implicates the blocked structure as the source of pain. Therefore, analgesia after local anesthetic blocks of a z-joint or its nerve supply indicates that the blocked site or joints are indeed the primary pain source. The primary indications for z-joint and medial branch block (MBB) injections are:

- Failure of greater than 4 weeks of appropriate, directed, conservative management in bringing pain relief;
- Used in combination with orthopedic manual techniques (OMT), as performed by select physical therapists and physicians;
- Chronic or subacute, whiplash-associated injuries;
- Certain types of cervicogenic headaches;
- Pain of significant intensity with associated loss of function.

Potentially important but not diagnostic clinical findings include:

- Site of maximal segmental or direct articular tenderness;
- Concordant pain on provocative segmental testing;
- Articular restriction and local soft tissue changes such as increased muscle spasticity;
- Pain in recognized z-joint referral zones.

Studies show that certain levels appear to be more commonly involved, including C2-C3, C5-C6, L4-L5, and L5-S1. Contraindications to these injections are the same as for the ESIs, but specifically to avoid the procedures if patients show signs of abnormal clotting status, infection (local or systemic), or have allergies to injectants. Complications for z-joint block procedures are rare: increased z-joint pain, local needle site pain, and chemical meningism. Spinal nerve root or subdural injections are rarer still. The studies that evaluate treatment of spine pain of z-joint origin—documented by analgesia after single diagnostic blocks—assess the efficacy of isolated corticosteroid z-joint injections, posterior lumbar fusion, and radiofrequency denervations (RFNAs).

Radiofrequency Nerve Ablation

Facet or zygapophyseal (z) joint nerve ablation (FJNA or RFNA, also known as facet denervation, facet rhizotomy, or radiofrequency facet nerve ablation) is used to treat chronic posterior element pain unresponsive to more conservative measures. It involves interruption of the facet joint nerve (medial articular branch of the posterior primary ramus). It was originallly described by Rees in 1971, who percutaneously inserted a long knife into the paravertebral muscles and claimed a success rate of 99.8% in 1000 patients. In 1972, Shealy attempted to repeat Rees' results and achieved only a 50% success rate in 29 patients. Subsequently, Shealy described the modern percutaneous radiofrequency technique of FJNA. FJNA is indicated in patients with chronic, recalcitrant pain of cervical, thoracic, or lumbar facet joint origin. Clinically, facet joint nerve pain is difficult to evaluate but may be suspected when axial pain is greater than extremity pain, extremity pain is in a vague distribution, no neurological changes are noted, and pain is greatest with extension. Because the pain complex may be mimicked by other conditions, facet joint nerve pain should be confirmed by diagnostic anesthetic facet joint nerve blocks (FJNBs). If the FJNBs do not substantially (>50%) relieve the pain complex for at least the life of the local anesthetic, other sources of pain should be explored. Although this procedure remains controversial, numerous studies have reported its efficacy in the treatment of chronic posterior element pain.[83-116]

Sacroiliac Intra-articular Joint Injections

The sacroiliac joint (SIJ) is a controversial source of primary lower back pain, but recent studies have shown it can cause significant pain. Its importance is often overlooked because its anatomical location makes it difficult to examine in isolation, and many SIJ clinical tests place mechanical stresses on contiguous structures. Furthermore, many other structures may refer pain to the SIJ. Before 1934, the SIJ was felt to be the primary cause of lower back pain. However, Mixter and Barr's study in 1934 focused attention on the disc as the primary cause of lower back pain. Only more recently has attention been refocused on the SIJ as a primary or secondary cause of lower back pain and disability. Dating as far back as the 1930's, research has focused on basic anatomy, biomechanics, and treatment strategies. Fluoroscopically-guided, contrast-enhanced, intra-articular injections are one subset of the treatment techniques available for SIJ pain.

Sacroiliac joint dysfunction is first suspected when a patient presents with a suggestive mechanism of injury (e.g., direct fall on the buttocks, rear-end motor vehicle accident with ipsilateral foot on the brake at the moment of impact; fall into a hole with one leg in the hole and the other leg extended outside). Pain diagrams, which document a predominant pain zone extending from the posterosuperior iliac spine to the caudal portion of the joint, can accurately predict which patients with suspected discogenic or posterior element pain have symptomatic sacroiliac joints upon provocative injection. Physical examination findings include a positive seated flexion test, standing flexion test, or Gillet test for aberrant sacroiliac motion, positive Patrick's maneuver for ipsilateral sacroiliac joint pain, tenderness over the ipsilateral sacroiliac joint, sacrotuberous ligament, piriformis muscle, and pubic symphysis. Diagnostic confirmation is attained when symptoms are reproduced upon distention of the joint capsule by provocative injection and subsequently abated with an analgesic block. The ligamentous integrity of the joint is established arthrographically.

Because diagnostic (with anesthetic only) and therapeutic (with corticosteroid and anesthetic agents) sacroiliac joint injec-

tions are invasive, the procedure should be reserved for patients who have the profile of a potentially painful sacroiliac joint and have failed to respond to aggressive functional restoration or who have reached a plateau in the therapy process. In these cases, sacroiliac joint injection can be applied for diagnostic affirmation as well as for the therapeutic benefit of the intra-articular injecton of anesthetic and long-lasting corticosteroid. Fluoroscopic evaluation is essential to ensure accurate intra-articular injections due to the irregular and convoluted joint surface and anatomy. The procedure has a very low morbidity and complication rate, however the need for preprocedural patient education, precautions, and preparation is still essential. The author recommends post-operative application of ice to the affected area, short course of muscle relaxants and NSAIDs, and the initiation of a short, but intense, course of physical therapy with emphasis on sacroiliac joint mobilization and stabilization exercises. The SIJ can be a primary or secondary source of lower back pain or dysfunction, and should therefore be thoroughly investigated and considered. When aggressive, conservative care fails to relieve SIJ pain, fluoroscopically-guided, contrast-enhanced, intra-articular injections can potentially provide both diagnostic and therapeutic benefits.[117-126]

Sympathetic Ganglion Nerve Blocks

The sympathetic nervous system is thought to play a role in many painful disorders, including the face and extremities. The most common of these disorders are called causalgia, reflex sympathetic dystrophy (RSD), and sympathetically-maintained pain (SMP). In 1994, the International Association for the Study of Pain (IASP) recommended replacing these terms for these disorders with the terms complex regional pain syndrome, CRPS type I (which is not associated with a classic nerve injury), and CRPS type II (which is associated with a classic nerve injury, as in causalgia). Their criteria for the diagnosis of CRPS included: 1) an initiating noxious event; 2) continued pain, allodynia, or hyperalgesia; 3) evidence of edema, vasomotor changes, and temperature changes; and 4) exclusion of other causes which would explain the symptoms. It is noted that pain and swelling may be due to various other medical conditions. Therefore, the failure to exclude a condition that is potentially treatable with sympathetic blockade or any other condition that may account for the symptoms was the impetus behind the changes to the 1994 criterion. Based upon these criteria, the history and physical examination are the most sensitive and accurate assessment tools (ie- not just subjective complaints of a chronically cold limb or positive response to sympathetic blockade or other diagnostic testing). Other diagnostic tools to assist with the correct diagnosis include plain radiographs, triple-phase bone scans, thermography, and response to sympathetic blockade.

Successful diagnosis and treatment of these conditions is best accomplished by an aggressive, multimodal approach involving medical, surgical, and percutaneous treatments. Local treatments for sympathetic blockade are the cornerstone of these therapeutic programs. From a physiological standpoint, sympathetic blockade may have a transient effect on pain or temperature changes. The literature reflects variable responses to sympathetic blockade with a success rate in the range of 18-50%. Since sympathetic pain is difficult to treat and responds best to early intervention, aggressive early treatment protocols are indicated to improve the success and duration of pain relief and prevent chronic dystrophic changes. Local sympathetic blocks are the cornerstone of treatment and are thought to interrupt and disorganize the inapproprate efferent sympathetic activity and result in the restoration of normal central processing of nociceptive and non-nociceptive afferent sensory input. However, sympathetic blocks should be supported by oral medications, epidural injections, physical therapy, and psychological intervention, when indicated. For refractory patients who do not receive adequate pain relief with cervicothoracic or lumbar sympathetic chain blocks, regional sympathetic blockade (percutaneous) or surgical sympathectomy may be a treatment option. Dorsal column stimulation has shown some success with recalcitrant cases to other treatments. Local anesthetic blockade of the cervicothoracic and lumbar sympathetic chains are valuable tools in the diagnosis and treatment of sympathetic pain disorders. All physicians performing these techniques must remain informed concerning the regional anatomy, potential complications, and safety protocols to safely perform these interventional procedures.[127-144]

Conclusion

Compartmentalization of pain problems into physiological, physical, and psychosocial categories may be useful diagnostically, but must be synergistically joined to achieve therapeutic success. The interventional pain specialist is a valuable and often crucial member of the pain management team. Injury and tissue-specific therapeutic exercise programs must form the basis of physical rehabilitation and functional restoration protocols. Importantly, the protocols must expand to encompass psychotherapeutic intervention in chronic pain conditions. Neuromuscular reconditioning must be included to ensure a function-specific, task-oriented program in order to enhance and foster functional recovery among the affected patient.

Injection techniques play a major role in the management of disorders of the musculoskeletal system. Various procedures and techniques have been used over the years, and are being developed for the interventional management of pain. During the 1990's, more novel injection techniques have been developed, and traditional injection techniques have been refined concurrent with the technologic advances in imaging modalities and a clearer understanding of the pathomechanics and the physiochemistry of pain. Many of the painful states seen by an interventional physiatrist and pain specialist can be helped greatly by using a rehabilitation program that may include injection techniques. Some of these interventional procedures are relatively simple and common to perform, whereas others can be technically challenging and should be done only by a specialist with adequate experience and knowledge to perform these procedures accurately and in a timely fashion. It is important to emphasize that the use of fluoroscopy to aid in proper needle placement is now the standard and norm. Fluoroscopic direction of needle placement increases the accuracy and efficacy of several types of selective spinal procedures.

In Part II of this series to appear in our next issue, we address diagnostic and therapeutic intradiscal interventions. ■

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References

1. Saal, JA and Saal JS. Physical Medicine and Rehabilitation as They Relate to Pain Management (Ch. 32) In Weiner RS. *Pain Management: A practical Guide for Clinicians* (5th Ed). CRC Press LLC. Boca Raton, FL. 1998. pp 481-493.

2. Lennard TA. *Physiatric Procedures in Clinical Practice* (1st Ed). Hanley & Belfus. Phila, PA. 1995.

3. Deyo RA, et al. What can the history and physical examination tell us about low back pain? *JAMA*. 1992. 268(6):760-765.

4. Waddell G, McCulloch JA, et al. Nonorganic physical signs in low-back pain. *Spine*. 1980. 5:117-125.

5. Weinstein SM and Herring SA. Rehabilitation of the Patient with Low Back Pain (Ch. 47). In Delisa JA. *Rehabilitation Medicine: Principles and Practice* (2nd Ed). J.B. Lippincott Co. Philadelphia, PA. 1993. pp 996-1017.

6. Cailliet R. *Low Back Pain Syndrome* (4th Ed). FA Davis. Philadelphia, PA. 1988.

7. Boden SD, Davis DO, et al. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. *J Bone Joint Surg Am*. 1990. 72:403.

8.White AA and Panjabi MM. *Clinical Biomechanics of the Spine* (2nd Ed). Lippincott-Raven. Phila, PA. 1990.

9. Saal JA. Electrophysiologic evaluation of lumbar pain: Establishing the rationale for therapeutic management. In J.A. Saal (Ed). *Spine State-of the-Arts Reviews*. Hanley & Belfus. Philadelphia, PA. 1987. pp 21-46.

10. Saal JA. Diagnostic studies for the industrial low back. *Topics in Acute Care and Trauma Rehabilitation*. 1988. 2(3):31-49.

11. Saal JA. Nonoperative treatment of herniated lumbar intervertebral disc with radiculopathy: An outcome study. *Spine.* 1989. 14(4):431-437.

12. LaRocca H. Scientific approach to the assessment and management of activity-related spinal disorders. *Spine.* 1997. 12(S7):S8-S59.

13. Sinaki M and Mokri B. Low back pain and disorders of the lumbar spine (Ch. 39). In Braddom RL. *Physical Medicine & Rehabilitation.* W.B. Saunders. Philadelphia, PA. 1996. pp 813-850.

14. Bogduk N. *Clinical Anatomy of the Lumbar Spine and Sacrum* (3rd Ed). Churchhill-Livingsone. New York, NY. 1997.

15. Kirkaldy-Willis W and Farfan HF. Instability of the lumbar spine. *Clinical Orthopedics*. 1983. 615:110-123.

16. Yong-Hing K and Kirkaldy-Willis WH. The pathophysiology of degenerative disease of the lumbar spine. *Orthopedic Clinics of North America*. 1983. 14:491-504.

17. Mooney V, Vleeming A, et al (eds). The Integrated Function of the Lumbar Spine and Sacroiliac Joint (Part 1 & 2). From the 2nd Interdisciplinary World Congress on Low Back Pain, San Diego, CA. November 9-11, 1995. Published as an educational grant from Phillips Medical Systems.

18. Coppes MH, Marani E, et al. Innervation of "painful" lumbar discs. *Spine.* 1997. 20:2342-2349.

19. Freemont AJ, et al. Nerve ingrowth into diseased intervertebral disc in chronic back pain. *Lancet.* 1997. 9072:178-181.

20. Bigos SJ, et al. Acute low back pain problems in adults. *AHCPR Clinical Practice Guidelines, No. 14.* U.S. Department of Health and Human Services, Public Health Service. Publication 95-0642. December, 1994.

21. Deyo RA, et al. How many days of bed rest for acute low back pain? A randomized clinical trial. *N Engl J Med*. 1986. 315(17):1064-1070.

22. Malmivaara A, et al. The treatment of acute low back pain: Bed rest, exercises, or ordinary activity? *NEJM*. 1995. 332:351-355.

23. Lahad A, et al. The effectiveness of four interventions for the prevention of low back pain. *JAMA*. 1994. 272(16):1286-1291.

24. Van Tulder MW, et al. Conservative treatment of acute and chronic nonspecific low back pain: a systematic review of randomized controlled trials of the most common interventions. *Spine.* 1997. 22:2128-2156.

25. Waddell G. A new clinical model for the treatment of low back pain. *Spine.* 1987. 12:632-644.

26. Ward NG. Tricyclic antidepressants for chronic low back pain: Mechanisms of action and predictors of response. *Spine.* 1986. 11(7):661-665.

27. Ghosh P. Influence of drugs, hormones, and other agents on the metabolism of the disc and the sequelae of its degeneration. In Ghosh P (Ed). *The biology of the disc*. CRC Press. Boca Raton, FL. 1988. 121-160.

28. Goodman AG, et al. *The pharmacological basis of therapeutics*. Macmillan Press. New York, NY. 1980.

29. Carey TS, et al. The outcomes and costs of care for acute low back pain among patients seen by primary care practitioners, chiropractors, and orthopedic surgeons. The North Carolina Back Pain Project. *NEJM*. 1995. 333:913-917.

30. Shekelle PG, et al. Spinal manipulation for low back pain. *Ann Intern Med*. 1992. 117:590-598.

31. Von Korff M, et al. Effects of practice style in managing back pain. *Ann Intern Med*. 1994. 121(3):187-195.

32. Adams MA and Hutton WC. The mechanical function of the lumbar apophyseal joints. *Spine.* 1983. 8:327-330.

33. Farfan HF. Muscular mechanism of the lumbar spine and the position of power and efficiency. *Orthopedic Clinics of North America*. 1975. 6:135-144.

34. McKenzie R. Mechanical disorders and treatment of lumbar spine disorders. *Spinal Publications*. Waikanae, New Zealand. 1981.

35. Schnebel BF, et al. *A digitizing technique for the study of movement of intradiscal dye in response to flexion and extension of the lumbar spine*. Presented at the International Society for the Study of the Lumbar Spine Meeting. Rome, Italy. 1987.

36. McQuain MT, et al. Effect of electrical stimulation on lumbar paraspinal muscles. *Spine.* 1993. 18:1787.

37. Murphy DA and Cornish RD. Prediction of chronicity in acute low back pain. *Arch Phys Med Rehabil*. 1984. 65:334.

38. Mayer TG, et al. Objective assessment of spine function following industrial injury: A prospective study with comparison group and one-year follow—up. *Spine.* 1985. 10(6):482-493.

39. Moffett JA, et al. A controlled prospective study to evaluate the effectiveness of a back school in the relief of chronic low back pain. *Spine.* 1986. 11:120-123.

40. Cherkin DC, et al. Physician variation in diagnostic testing for low back pain. Who you see is what you get. *Arthritis Rheum*. 1994. 37(1):15-22.

41. Windsor RE and Lox DM (Eds). *Soft Tissue Injuries: Diagnosis and Treatment*. Hanley & Belfus. Philadelphia, PA. 1998.

42. Weinstein SM (Ed). Injection Techniques: Principles and Practice. In *Physical Medicine and Rehabilitation Clinics of North America*. W.B. Saunders. Philadelphia, PA. November, 1995. pp 671-926.

43. Waldman SD and Winnie AP (Eds.). *Interventional Pain Management.* W.B. Saunders. Philadelphia, PA. 1996. 151-406.

44. Derby R. Diagnostic block procedure: Use in pain localization. Hanley & Belfus. Philadelphia, PA. 1986.

45. Lippitt AB. The facet joint and its role in spine pain: Management with facet joint injections. *Spine.* 1984. 9:746-750.

46. Takeshi T, et al. Selective lumbosacral radiculography and block. *Spine.* 1980. 5: 68-78.

47. White LR and Mayer DJ. Injection techniques for the diagnosis and treatment of low back pain. *Orthopedic Clinics of North America.* 1983. 14:553-567.

48. Destouet JM. Lumbar facet syndrome: diagnosis and treatment. *Surg Rounds Orthop*. 1988. 2:22-27.

49. Dooley JF, et at. Nerve root infiltration in the diagnosis of radicular pain. *Spine.* 1988. 13:79-83.

50. Jeffries B. Facet steroid injections. *Spine: State of the Art Reviews.* 1988. 2:409-417.

51. Jeffries B. Epidural steroid injections. *Spine: State of the Art Reviews.* 1988. 2:419-26.

52. Krempen JF. Selective nerve root infiltration for the evaluation of sciatica. *Orthop Clin North Am*. 1975. 6:311-314.

53. White AH, Derby R, and Wynne G. Epidural injection for the diagnosis and treatment of low back pain. *Spine.* 1980. 5:78-82.

54. El-Khoury GY, et al. Epidural steroid injection: a procedure ideally performed with fluoroscopic control. *Radiology*. 1988. 168:554-557.

55. Gamburd R. Use of selective injections for the diagnosis and management of lumbar spine problems. *Phys Med & Rehab Clin North Am*. 1991. 2:79-96.

56. Falco FJ. Lumbar spine injection procedures in the management of low back pain. *Occup Med*. 1998. 13(1):121-149.

57. Moskovich R. Epidural injections for the treatment of low back pain. *Bull Hosp Jt Dis*. 1996. 55(4):178-184.

58. Kinard RE. Diagnostic spinal injection procedures. *Neurosurg Clin N Am*. 1996. 7(1):151-165.

59. Cuckler J, et al. The use of epidural steroids in the treatment of lumbar radicular pain. A prospective, randomized, double-blind study. *J Bone Joint Surg*. 1985. 67:63-66.

60. Dallas T, et al. Epidural morphine and methylprednisolone for low back pain. *Anesthesiology*. 1987. 408-411.

61. Gordon J. Caudal extradural injections in the treatment of low back pain. *Anesthesia*. 1980. 35:515-516.

62. Rowlingson JC, et al. Epidural analgesic techniques in the management of cervical pain. *Anesth Analg*. 1986. 65:938-942.

63. Snoek W, et al. Double-blind evaluation of methylprednisolone for herniated lumbar discs. *Acta Orthop Scand*. 1977. 48:635-641.

64. Stanley D, et al. A prospective study of nerve root infiltration in the diagnosis of sciatica: A comparison with radiculography, computed tomography, and operative findings. *Spine.* 1990. 15:540-543.

65. White AH, et al. Epidural injections for the diagnosis and treatment of low back pain. *Spine.* 1980. 5:78-86.

66. Bush K, et al. Controlled studies of caudal epidural injections of triamcinolone plus procaine for the management of intractable sciatica. *Spine.* 1991. 16:572-575.

67. Ridley M, et al. Outpatient lumbar epidural steroid injections in the management of sciatica. *Br J Rheum. 1988. 27:295-299.*

68. White AH, et al. Injection techniques for the diagnosis and treatment of low back pain. *Orthop Clin North Am*. 1983. 553-567.

69. Mehta M and Salmon N. Extradural block. Confirmation of the injection site by x-ray monitoring. *Anaesthesia*. 1985. 40:1009-1012.

70. Renfrew D, et al. Correct placement of epidural steroid injections: Fluoroscopic guidance and contrast administration. *AJNR*. 1991. 12:1003-1007.

71. Windsor RE, Pinzon EG, and Gore HC. Complications of Common Selective Spinal Injections, Prevention and Management: a Focused Review. *The American Journal of Orthopedics*. 2000.

72. McCarron RF, et al. The inflammatory effects of

nucleus pulposus. A possible element in the pathogenesis of low back pain. *Spine.* 1987. 12:760-764.

73. Olmarker K, et al. Autologous nucleus pulposus induces neurophysiologic and histologic changes in porcine cauda equina nerve roots. *Spine.* 1993. 18:1425-1432.

74. Murphy RW. Nerve roots and spinal nerves in degenerative disk disease. *Clin Orthop Rel Research*. 1977. 46-60.

75. Smyth MJ and Murphy V. Sciatica and the intervertebral disc. *J Bone Joint Surg*. 1958. 40:1401.

76. Olmarker K, et al. Effects of methylprednisolone on nucleus pulposus-induced nerve root injury. *Spine.* 1994. 19:1803-1808.

77. Yabuki S, et al. Effects of lidocaine on nucleus pulposus-induced nerve root injury. *Spine.* 1998. 23:2383-2389.

78. Yabuki S, et al. Nerve root infiltration and sympathetic block. *Spine.* 1995; 20: 901-6.

79. Weber H. Lumbar disc herniation: A controlled prospective study with ten years of observation. *Spine.* 1983. 8:131-40.

80. Lutz GE, et al. Fluoroscopic transforaminal lumbar epidural steroids: An outcome study. *Arch Phys Med Rehabil*. 1998. 79:1362-1369.

81. Derby R, et al. Precision percutaneous blocking procedures for localizing spinal pain. Part 2. The lumbar neuroaxial compartment. *Pain Digest*. 1993. 3:175-88.

82. Goldthwaith JE. The lumbosacral articulation: An explanation of many cases of lumbago, sciatica, and paraplegia. *Boston Med Surg J.* 1911. 164:365-372.

83. Ghormley RK. Low back pain with special reference to the articular facets, with presentation of an operative procedure. *JAMA*. 1933. 101:1773-1777.

84. Aprill C and Bogduk N. The prevalence of cervical zygapophyseal joint pain- A first approximation. *Spine.* 1992. 17:744-747.

85. Bogduk N and Marsland A. The cervical zygapophyseal joints as a source of neck pain. *Spine.* 1988. 13:610-617.

86. Dreyfuss P, et al. Thoracic zygapophyseal joint pain patterns: A study in normal volunteers. *Spine.* 1994. 19:807-811.

87. Dwyer A, et al. Cervical zygapophyseal joint pain patterns. A study in normal volunteers. *Spine.* 1990. 15:453-457.

88. McCall IW, et al. Induced pain referral from posterior lumbar elements in normal subjects. *Spine.* 1979. 4:441-446.

89. Mooney V, et al. Facet joint syndrome. *Clin Orthop*. 1976. 115:149-156.

90. Carette S, et al. A controlled trial of corticosteroid injections into the facet joints for chronic low back pain. *N Engl J Med*. 1991. 325:1002-1007.

91. Esses SI and Moro JK. The value of facet blocks in patient selection for lumbar fusion. *Spine.* 1993. 18:185-190.

92. Jackson RP. The facet syndrome: Myth or reality? *Clin Orthop*. 1992. 279:110-121.

93. Jackson RP, et al. Facet joint injection in low back pain: A prospective statistical study. *Spine.* 1988. 13:966-971.

94. Lilius G, et al. Lumbar facet joint syndrome: a randomized clinical trial. *J Bone Joint Surg*. 1989. 71B:681-684.

95. Derby R, et al. Precision percutaneous blocking procedures for localizing spinal pain. Part 1. The posterior lumbar compartment. *Pain Digest*. 1993. 3:89-100.

96. Revel ME, et al. Facet joint block for low back pain: identifying predictors of a good response. *Arch Phys Med Rehabil*. 1992. 73:824-828.

97. Dreyer P, et al. Zygapophyseal (facet) joint injections. *Phys Med Rehab Clin North Am*. 1995. 6:715-741.

98. Bogduk N. Back pain: Zygapophysial blocks and epidural steroids. In Cousins MJ and Bridenbaugh PO (Eds). *Neural Blockade in Clinical Anesthesia and Management of Pain* (2nd Ed). J.B. Lippincott. Philadelpia, PA. 1989. 935-954.

99. Barnsley L, et al. Comparative local anesthetic blocks in the diagnosis of cervical zygapophyseal joint pain. *Pain*. 1993. 55:99-106.

100. Bonica JJ. Local anesthesia and regional blocks. In Wall PD, Melzack R (Eds). *Textbook of Pain* (2nd Ed). Churchill-Livingstone. Edinburgh. 1989. 724-743.

101. Carrera GF, et al. Current concepts in evaluation of the lumbar facet joints. *Crit Rev Diagn Imaging*. 1984. 21:85-104.

102. Murtagh FR. Computed tomography and fluoroscopy-guided anesthesia and steroid injection in facet syndrome. *Spine.* 1988. 13:686-689.

103. Schwarzer AC, et al. Clinical features of patients with pain stemming form the lumbar zygopaphysial joints: Is the lumbar facet syndrome a clinical entity? *Spine.* 1994. 19:1132-1137.

104. Lora J and Long D. So-called facet denervation in the management of intractable back pain. *Spine.* 1976. 1:121-126.

105. Ogsbury JS, et al. Facet "denervation" in the treatment of low back syndrome. *Pain*. 1977. 3:257-263.

106. Silvers R. Lumbar percutaneous facet rhizotomy. *Spine.* 1990. 15:36-40.

107. Rees W. Multiple bilateral subcutaneous rhizolysis of segmental nerves in the treatment of the intervertebral disc syndrome. *Ann Gen Pract*. 1971. 26:126-127.

108. Shealy C. Percutaneous radiofrequency denervation of spinal facets. *J Neurosurg*. 1975. 43:448-451.

109. Shealy C. Facet denervation in the management of back and sciatic pain. *Clin Orthop*. 1976. 115:157-164.

110. Jackson R, et al. Facet joint injections in low back pain. *Spine.* 1988. 13: 967-971.

111. Mooney V, et al. The facet syndrome. *Clin Orthop*. 1976. pp 149-156.

112. Anderson D, et al. Percutaneous radiofrequency facet joint denervation in low back and extremity pain. *ActaNeurochir*. 1987. 87:48-51.

113. Burton C. Percutaneous radiofrequency facet denervation. *Appl Neurophysiol*. 1977. 39:80-86.

114. Ignelzi R and Cummings T. A statistical analysis of percutaneous radiofrequency lesions in the treatment of chronic low back pain and sciatica. *Pain*. 1980. 8:181-187.

115. Oudenhoven R. Paraspinal electromyography following facet rhizotomy. *Spine.* 1977. 2:299-304.

116. Silvers R. Lumbar percutaneous facet rhizotomy. *Spine.* 1990. 15:36-40.

117. Dreyfuss P, et al. Sacroiliac joint injection techniques. In Weinstein SM (Ed). Injection Techniques. *Physical Medicine & Rehabilitation Clinics of North America*. 6(4):785-813.

118. Aprill C. *The role of anatomically specific injections into the sacroiliac joint*. Presented at the 1st Interdisciplinary Word Congress on Low Back Pain and its Relation to the Sacroiliac Joint. San Diego, CA, Nov. 5-6, 1992.

119. Bernard TN Jr and Cassidy JD. The sacroiliac joint syndrome- pathophysiology, diagnosis, and management. In Frymoyer JW (ed.). *The Adult Spine: Principles and Practice*. Raven Press. New York, NY. 1991. pp 2107-2130.

120. Fortin JD, et al. Sacroiliac joint: Pain referral maps upon applying a new injection/arthrography technique. Part 1: Asymptomatic volunteers, Part 2: Clinical evaluation. *Spine.* 19:1475-1489. 1994.

121. Schwarzer AC, Aprill CN, and Bogduk N. The sacroiliac joint in chronic low back pain. *Spine.* 1995. 20:31-37.

122. Mixter WJ and Barr JS. Rupture of the intervertebral disc with involvement of the spinal canal. *NEJM*. 1934. 211:210.

123. DonTigny RL. Function and pathomechanics of the sacroiliac joint: A review. *Phys Ther*. 1985. 65:35-44.

124. Vleeming A, et al. Mobility in the sacroiliac joints in the elderly: A kinematic and radiographic study. *Clin Biomechanics*. 1992. 166:1243-1247.

125. White AA, et al. *Biomechanics of lumbar spine and sacroiliac articulation: Relevance to idiopathic low back pain*. In White AA, Gordon SL (Eds). American Academy of Orthopedic Surgeons Symposium on Idiopathic Low Back Pain. CV Mosby. St. Louis, MO. 1982. pp 269-322.

126. Fortin JD and Tolchin R. Sacroiliac arthrograms and postarthrography CT. *Arch Phys Med Rehabil*. 1993. 74:1259.

127. Dotson R. Causalgia- reflex sympathetic dystrophy- sympathetically-maintained pain: Myth and reality. *Muscle & Nerve*. 1993. 16:1049-1055.

128. Doury P. Algodystrophy: Reflex sympathetic dystrophy syndrome. *Clin Rheum*. 1988. 7:173-180.

129. McMahon S. Mechanisms of sympathetic pain. *Br Med Bull*. 1991. 47:584-600.

130. Schott G. Visceral afferents: Their contribution to 'sympathetic dependent' pain. *Brain*. 1994. 117:397-413.

131. Racz GB, et al. Definitions, classifications, and taxonomy: An overview. *Phys Med Rehabil State*. Art Rev. 1996. 10:2.

132. Kozin F, et al. Bone scintigraphy in the reflex sympathetic dystrophy syndrome. *Radiology*. 1981. 138:437-443.

133. Smith F and Powe J. Effect of sympathetic blockade on bone imaging. *Clin Nucl Med*. 1992. 1:665-669.

134. Weiss L, et al. Prognostic value of triple phase bone scanning for reflex sympathetic dystrophy in hemiplegia. *Arch Phys Med Rehabil*. 1993. 74:716-719.

135. Charlton J. Management of sympathetic pain. *Br Med Bull*. 1991. 47:601-618.

136. Lofstrom J and Cousins M. Sympathetic neural blockade of upper and lower extremity. In Cousins M (ed.). *Neural Blockade in Clinical Management of Pain* (2nd Ed). Lippincott. Philadelphia, PA. 1988. p 461.

137. Stanton-Hicks M. Upper and lower extremity pain. In Racz G (Ed). *Pain Management* (2nd Ed). Mosby, St.Louis. 312 pgs.

138. Veldman P, et al. Signs and symptoms of reflex sympathetic dystrophy: Prospective study of eight-hundred and twenty-nine patients. *Lancet.* 1993. 342:1012-1016.

139. Bonica JJ. *The Management of Pain*, Vol. 1. (2nd Ed). Lea & Febiger. Philadelphia. 1990. pp 226-227.

140. Mandel S, Rothrock R. Sympathetic dystrophies: Recognizing and managing a puzzling group of syndromes. *Postgrad Med*. 1990. 87:213-218.

141. Duncan, et al. Treatment of upper extremity reflex sympathetic dystrophy with joint stiffness using sympatholytic bier blocks and manipulation. *Orthopedics*. 1988. 11:883-886.

142. Ford S, et al. The treatment of reflex sympathetic dystrophy with regional bretylium. *Anesthesiol*. 1988. 68:137-140.

143. Brosetta J, et al. Chronic epidural dorsal column stimulation in the treatment of causalgic pain. *Appl Neurophysiol*. 1982. 45:190-194.

144. Spiegelmann R and Friedman W. Spinal cord stimulation: A contemporary series. *Neurosurgery*. 1991. 28:65-70.