Lumbar epidural steroid injections
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Introduction
Spinal pain and related problems continue to be among the most challenging musculoskeletal problems, if not general health problems, facing health-care providers today. In this respect, the use of epidural steroid injections in the management of axial spine pain or sciatica continues to be commonplace since the original Contemporary Concepts in Spine Care paper was published in 1994 [1]. This revised article will again discuss the pertinent literature and, through theoretical, practical and clinical parameters, will conclude with recommendations regarding the current use of lumbar epidural steroid injections.

Historical review
Injection of corticosteroids into the epidural space for treatment of lumbosacral radicular pain was first recorded in 1952 [2]. Subsequently, numerous reports have been published regarding this procedure, but only a very few have been randomized controlled trials. In fact, only 18 such studies have been published in nearly a half century. Four randomized clinical trials on the use of epidural steroid injections [3–6] have been published in the literature since 1994, when the original Contemporary Concepts in Spine Care paper was published.

Despite its widespread use and acceptance, this procedure remains controversial, especially outside of North America [7] and has prompted several independent critical reviews [8–13]. One basis for these controversies is the relative inconsistency in techniques by which epidural steroid injections are performed. Some variable factors include the requirement of fluoroscopic guidance, route of administration, components and quantity of injectate and necessity of physiologic monitoring. Further, the relatively limited support in the scientific literature, combined with the current medicoeconomic climate that emphasizes randomized clinical trials, has prompted some to question the value of these procedures in clinical practice.

Rationale
Within the past decade, a definite trend toward nonsurgical management of lumbosacral disc herniations with radicular symptoms has occurred with research supporting the following concepts: 1) resorption of lumbar herniated disc fragments [14–16]; 2) occurrence of asymptomatic lumbar disc pathology, including herniations [17–19] and 3) an inflammatory component to low back and/or radicular pain in the presence of disc herniation [20–23]. An implication of these observations is that an inflammatory reaction may be necessary to precipitate symptoms, even in the presence of mechanical compression. An obvious corollary is that control of inflammation may diminish clinical symptoms, which in turn may reduce patient dysfunction and disability.

An autoimmune or “chemical” basis for lumbar radicular pain was postulated in 1977 [24,25]. Within the past decade, data have been published indicating enzymatic deregulation as a potential catalyst of low back and radicular pain. Phospholipase A$_2$ (PLA$_2$) is released from an intact disc after injury, and in vitro PLA$_2$ has neurotoxic properties [26,27]. PLA$_2$ propagates an inflammatory cascade by means of liberation of arachidonic acid, resulting in both chemotactic and noncellular mediated responses through leukotrienes and prostaglandins. Inflammatory substances in the epidural space may also directly or indirectly induce increased vascular permeability of endoneural blood vessels [28].

There is evidence that supports a neurogenic inflammatory basis for pain generation [29]. Neuropeptides, such as substance P and calcitonin gene-related peptide, are activated and released from the dorsal root ganglion after noxious mechanical stimulation [30–33]. Receptor sites for substance P are present in the outer annulus of the intervertebral disc and
in the posterior longitudinal ligament [34]. These neuropeptides may sensitize nociceptors in the lumbar disc to the effects of prostaglandins and leukotrienes, and they may stimulate leukotriene activity directly.

Although inflammation occurs in response to a noxious stimulus, uncontrolled inflammation can retard or prevent adequate healing; thus, the rationale for using any anti-inflammatory agent. Corticosteroids have known anti-inflammatory properties related to inhibition of prostaglandin synthesis [35], but they can also impede cell-mediated and immunologic responses [36–38]. In distinction, nonsteroidal anti-inflammatory agents inhibit prostaglandin synthesis only. Postulated modes of action of corticosteroids include membrane stabilization, inhibition of neuropeptide synthesis or action and blocking PLA₂ activity [39]. In addition, corticosteroids may exert an “anesthetic”-like action, blocking nociceptive C-fiber conduction [40] independent of their anti-inflammatory properties. Corticosteroids may also block the vascular response of the vasomotorum induced by nucleus inflammagens [28].

Corticosteroids are frequently administered orally, but the effectiveness of the corticosteroid is presumed proportional to the dose of medication delivered to the site of pathology. Theoretically, local instillation of steroid preparation yields higher local concentrations than oral dosing. Further, epidural injection of corticosteroids is not dependent on local blood flow, which is frequently impaired with compressive lesions [41]. Target site concentration also depends on injection variables, including the route of epidural administration. Caudal or translaminar epidural injections are affected by the presence or absence of epidural plica or scarring, which may prevent migration of the posteriorly administered injectate to the anterior epidural space, where the primary pathology is usually located. Other variables, such as volume of injectate and the rate and/or pressure of injection, may affect outcome. Studies of epidural injections performed “blindly” (without fluoroscopic control) have revealed misplacement of up to 40% of the caudal and 30% of the translaminar techniques, even in experienced hands [42–47]. One study suggested that the accuracy of “blind” caudal epidural steroid injections can be as high as 91% if anatomic landmarks were easily identifiable and if careful attention was given to the absence of palpable subcutaneous air over the sacrum [48]. However, unsuspected venous plexus uptake can occur, and this study still recommended that fluoroscopy should be used if available and not contraindicated. Another study concluded that the overall success rate of nonfluoroscopically guided epidural steroid injections was 93% for lumbar (translaminar) and 64% for caudal injections with unsuccessful placement proportional to the size of the patient [49]. Finally, the classic loss of resistance technique used to identify the epidural space, as performed with a small-gauge Touhy needle, has been demonstrated to be 99% sensitive but only 27% specific when compared with fluoroscopic confirmation [50].

Transforaminal injection, which delivers medication to the anterior epiradicular space of a single segmental level, may ultimately prove to be the most effective means of administering epidural corticosteroids [51]. Certainly for purely diagnostic value, placement of a small volume of anesthetic at a single segmental epiradicular site will have the most value in determining whether a single nerve root is responsible for radicular symptoms [52]. This route of administration may also be more effective for radiculopathy resulting from foraminal or extraforaminal disc herniations rather than a translaminar approach [53]. Variations of these injections have also been described [54,55].

Epidural steroid injections have been demonstrated to be generally very safe [56,57]. Risks are from the injection itself and potential pharmacologic side effects from the corticosteroid, local anesthetics or contrast agent used. The most common adverse reaction occurring during the procedure is a vasovagal reaction. The most common adverse reactions after epidural injection include nausea, facial flushing, insomnia, fever (usually less than 100 F), nonpositional headache and transient increased pain, all corticosteroid phenomena that may be dose dependent. Dural puncture with subsequent spinal (positional) headache is much more common after translaminar (up to 5%) than caudal (0.6%) injections, because the thecal sac usually ends at the S2 level. Very rare adverse consequences include spinal infection [58–60] (eg, epidural abscess or meningitis), allergic reactions, myopathy [61], nerve root injury and transient paralysis [62]. Cushingoid syndrome has been reported after administration of epidural methylprednisolone [63], and suppression of the pituitary–adrenal axis [64,65] may have implications for patients undergoing surgery soon after epidural steroid administration.

Absolute contraindications to performing epidural injections include local infection at injection site; use of exogenous blood thinners, including anticoagulants and antiplatelet pharmaceuticals; bleeding diathesis; congestive heart failure and uncontrolled diabetes mellitus. In diabetics, particularly insulin-dependent diabetics, substantial elevation of serum glucose levels can occur after epidural steroid injection. Duration of pain greater than 1 year and psychosocial barriers to recovery would be relative contraindications to performing an epidural steroid injection.

Results

Three reports have systematically reviewed the randomized studies of lumbar epidural steroid injections present in the literature through the 1990s [9,10,12]. A total of 15 controlled clinical trials evaluating epidural steroid injections for the treatment of low back pain and/or sciatica (either presumed or demonstrated by imaging to be the result of disc herniation) have been published in the past three decades: 5 in the 1970s [48,66–69], 5 in the 1980s [70–74], 2 in the 1990s [3,75] and 3 since 2000 [4–6]. One study evaluated patients with spinal stenosis only [76], and 1 reported a mixed study population with herniated discs or spinal stenoses [70]. Two trials studied patients with low back pain only, including postsurgical patients [77] and those with extremely chronic
pain of several years’ duration [78]. Salient features of some of these studies are presented below.

Almost all of the published randomized studies have methodological flaws that limit the impact of their respective results. These include noncomparable control groups, small number of study patients, lack of comparison to other relevant treatments, inconsistent primary and co-interventions and limited long-term follow-up. Possibly the most significant procedural flaw in all of the studies published before 2000 (receiving only brief mention in one of the reviews [9,10] and only implied in the other [12]) is that none used fluoroscopic-guided needle placement and contrast confirmation when performing the epidural steroid injections. Aside from the obvious potential result of placement of the injectate in a location other than the epidural space, a more subtle but very relevant consequence is inadequately directed medication to the precise location of pathology despite adequate placement of the injectate within the epidural space. The lack of true confirmation of the location of the injected material makes the results of all studies suspect, with the possibility that a more positive response than reported would have been realized.

Both Koes et al. [9,10] and Rozenburg et al. [12] used the same 100-point scale to assess methodological quality (the greater the number, the better the quality), reporting a range of 17 to 72 and 12 to 85, respectively. This quality index was not clearly correlated with the results, although three of the top five rated studies did not demonstrate significant benefit of the steroid over the nonsteroid group, the decade in which the study was published or the type of injection (ie, caudal vs. translaminal). Each review identified essentially equal numbers of studies with significant benefit versus no significant benefit from the use of epidural steroid injections. Although neither review could ultimately conclude from the available literature whether epidural steroid injections are beneficial for the treatment of lumbar pain syndromes, Koes et al. [9,10] commented that there did appear to be short-duration benefit from epidural steroid injections in some studies.

Overall, seven studies reported significantly better response in the epidural steroid group than the comparison group [67–69,71,73–75]. The study by Helliwell et al. [71] was not included in the reviews by Rozenburg et al. [12]. The study by Breivik et al. [67] received quite discrepant scores in the two reviews. For purposes of this discussion, the results of Mathews et al. [73] demonstrated a positive benefit from the use of epidural steroid injections, although Rozenburg et al. [12] concluded this to be a negative study. The remaining five studies were scored and ranked similarly. Among the seven positive studies, there was a large range of technical variation, including the location, volume and components of the injectate in both the study and control groups and outcome measures. The following is a summary of these studies.

Mathews et al. [73] evaluated patients with low back pain and sciatica of less than 3 months’ duration from presumed discogenic basis, although not proven by advanced imaging. The steroid group was administered from one to three caudal injections of bupivacaine 20 mL and methylprednisolone 80 mg (2 mL) 2 weeks apart, whereas the control group received lidocaine 2 mL subcutaneously over the sacral hiatus or tender spot. Other treatments were not restricted. Outcome measures included pain relief, improvement in straight leg raise (SLR) and improvement in neurologic deficit. Significant improvement in pain level was identified at 3 months but not at 1, 6 or 12 months.

The study by Bush and Hillier [75] examined patients with low back pain and sciatica of 1 to 13 months’ duration with two caudal injections of either procaine 25 mL, saline and triamcinolone 80 mg (steroid group) or saline 25 mL only (control group), separated by 2 weeks. Additional physical therapy and limited non–anti-inflammatory analgesics were allowed in both groups. Visual analog scale and SLR assessments of pain were the outcome measures assessed at 4 weeks and 1 year. Improvement was significantly greater in the steroid group at 4 weeks, but no significant difference in pain was recorded at 1 year after treatment, although SLR was described as significantly better in the steroid group at 1 year.

In a study by Helliwell et al. [71], patients with low back pain and radicular pain of greater than 2 months’ duration (average of 9 months in the steroid group and 13 months in the control group) were administered either a single translaminal injection of saline 10 mL plus methylprednisolone 80 mg or saline 5 mL in an interspinous ligament. No additional treatment was allowed except for analgesics, and follow-up assessment was made at 1 and 3 months after injection. Significant improvement was noted in lumbar range of motion, SLR, and patient’s subjective report of “definite improvement” at 3 months in the steroid group.

Dilke et al. [68] performed a study on patients with low back pain and sciatica ranging from 1 week to more than 2 years (although the majority less than 1 year). All patients were admitted to the hospital and received either up to two (separated by 1 week) translaminal injections of saline 10 mL and methylprednisolone 80 mg or an interspinous injection of saline 1 mL. A variety of other therapeutic modalities were given in each group. Outcome measures for up to 3 months included pain reduction, reduction in analgesic use, length of hospitalization, duration of disability and requirement for subsequent surgery. The steroid group showed significant differences in pain reduction at 1 month and reduction of analgesic use and return to work at 3 months.

In the study by Breivik et al. [67], patients with chronic low back pain and sciatica (ranging from several months to years, including postsurgical patients) were randomized to receive from one to three caudal injections with bupivacaine 20 mL and methylprednisolone 80 mg (2 mL) 1 week apart, with the control group receiving a caudal injection of bupivacaine 20 mL plus saline 100 mL. The outcome assessment with follow-up from 3 to 20 months included pain level, SLR, neurologic deficit and return to work. Although improvement was identified in both groups, a significantly greater positive response was found in the steroid group.

Ridley et al. [74] studied patients with low back pain and sciatica of mean duration approximately 8 months. The ste-
roid group received up to two translaminar injections of saline 10 mL and methylprednisolone 80 mg (2 mL) versus saline 2 mL in the interspinous ligament at 1-week intervals. The outcome measures were pain control and improvement in SLR at 1, 2 and 4 weeks, and 3 and 6 months. Significant improvement was found in the steroid group at 2 weeks and 3 months but not at 6 months.

The study by Yates [69] included patients with low back pain and presumably sciatica, as SLR reduction was an outcome measure. There were four groups: two steroid groups that received triamcinolone 60 mg (3 mL) in either saline or lidocaine 47 mL, and the corresponding two control groups received the carrier (but not the corticosteroid) all by means of the caudal approach. Up to four injections were given separated by 1 week each. Outcome measurements at 1 week after each injection included improvement in spinal range of motion and SLR. No data on pain reduction were presented, but significant benefit was noted for SLR in the steroid group at each interval.

As indicated above, three of the five top-rated studies [3,70,79] did not reveal a significant benefit from the use of lumbar epidural steroids, as compared with the control group. Carette et al. [3] studied patients with low back pain and sciatica of 1 month to approximately 1 year duration. The steroid group received up to three translaminar injections of saline 8 mL plus methylprednisolone 80 mg (2 mL), compared with the control group, which received saline 1 mL epidurally, separated by 3 weeks. Concurrent treatments were discouraged but were not totally eliminated. The outcome measures assessed at 3, 6 and 12 weeks included pain level, improved function, reduction in analgesic use, neurologic examination findings, reduction in work disability and need for subsequent surgery. Significant improvement in the steroid group was noted for lumbar range of motion and reduced sensory disturbance at 3 weeks, reduction in sciatica at 6 weeks and improved functional scale scores at 3 and 12 weeks. Otherwise, no other outcomes demonstrated significant differences between the two groups. The authors concluded that epidural steroid injections may provide short-term improvement in sciatica but do not result in functional benefit or reduce the need for subsequent surgery.

In the study by Snoek et al. [79], patients with low back pain and sciatica were randomized to either a treatment group with a single translaminar injection with saline 8 mL and methylprednisolone 80 mg (2 mL) or a control group receiving a single translaminar injection of saline 2 mL. Outcome was assessed acutely, within 3 days of the injection, and long term at an average of 14 months after injection. No significant differences were noted between the two groups with regard to relief of low back pain, radicular pain, SLR, subjective improvement in pain level or need for subsequent surgery.

Cuckler et al. [70] studied patients with chronic low back pain and sciatica of greater than 6 months’ duration with a single translaminar epidural steroid injection of procaine 5 mL and methylprednisolone 80 mg (2 mL) or equal volumes of procaine and saline. Assessment was performed at 24 hours postinjection, and no significant difference in symptom reduction was identified. If patients did not experience at least 50% improvement within 24 hours, a second nonblinded epidural steroid injection was administered regardless of which group they were in, for ethical reasons. At an average of 20 months after injections, no significant difference was noted between the groups relating to the need for surgery.

These three studies are frequently referenced to demonstrate the lack of efficacy of epidural steroid injections but are also subject to methodologic scrutiny. The study of Cuckler et al. [70] highlights the methodologic flaw regarding intervention that is inconsistent with practical management. Epidural steroid injections are not typically administered within 24 hours regardless of the degree of subjective improvement at that early interval, and the expectation of a 50% to 75% improvement within 24 hours is not reasonable. In all three of these no-benefit studies, the control group may have derived therapeutic benefit from the administration of a fluid volume of any agent (saline or anesthetic) into the epidural space by means of washout effect on inflammatory mediators, release of epiradicular adhesions or other as yet undetermined neurophysiologic effects. In all three studies, the control group received an epidural injection, albeit a sham injection. In none of the positive studies in which a translaminar injection was performed did the control group receive an epidural sham injection. All received an interspinous ligament injection. Thus, the question arises as to whether the lack of a significant difference between the groups in the studies by Carette et al. [3], Snoek et al. [79] and Cuckler et al. [70] indicates that a positive response may be mediated in part through a volume or mass effect in the epidural space independent of the specific injectate. Indeed, substantial improvement was identified in all of the control groups in these studies.

Finally, the study of Carette et al. [3] also has significant weaknesses, some of which are common to all of the studies mentioned, such as absence of fluoroscopic guidance, relatively diluted concentration of corticosteroid, and all of the injections were performed at the L3–L4 level regardless of the presumed level of pathology. Other weaknesses of the Carette et al. [3] study include a greater dropout rate for the placebo versus steroid group (which although not technically considered a flaw of the study design, may influence interpretation of the results) and intentional limitation of multimodal treatment, which may delay functional progression during the window of symptomatic improvement. In fact, the results of this study do indicate symptomatic reduction in sciatica at 6 weeks after injection.

A paper on this subject addresses the use of fluoroscopically guided transforaminal epidural steroid injections for therapeutic purposes in patients with refractory radicular pain secondary to a known lumbar disc herniation [80]. Although not a randomized clinical trial, this is the first published prospective case series study using the transforaminal route of injection with contrast-enhanced fluoroscopic guidance in this patient population. Both pain relief and functional outcome
were assessed with results indicating substantial benefit up to 20 months after injection without the need for surgical intervention. All patients had not responded previously to standard nonoperative measures but were enrolled in a functional reactivation program emphasizing trunk-strengthening exercises concurrent with the injection treatments. The proposed benefits of this route of injection included more precise localization to the site of pathology (both at the segmental level of pathology and at the anterior epidural space), smaller total volume of injectate with less steroid dilutional effect and lower risk of dural puncture.

As indicated, three randomized clinical trials have been published since 2000 [4–6]. The study by Buchner et al. [4] has a number of methodological flaws. It was performed in a prospective manner but not performed in a double-blinded fashion. Thirty-six patients with low back and leg pain were randomized to either an epidural steroid group or no injection. Both groups received similar treatment, including rest, medication, manual therapy and back-strengthening exercises. The injection group received three injections (all within 2 weeks during an inpatient hospitalization) of methylprednisolone 100 mg and bupivicaine 10 cc not performed with fluoroscopic guidance; the segmental levels at which the injections were given were not reported. The follow-up period ranged from 2 weeks to 6 months, with the outcome results indicating a significant improvement in SLR at 2 weeks, a nonsignificant improvement in visual analog scale pain levels and lumbar mobility at 2 weeks and an overall improvement but no significant difference between the two groups in any of the parameters by 6 months.

The study by Riew et al. [6] is a very intriguing one. This is the first prospective, randomized clinical trial performed in a double-blind fashion using selective nerve root injections (although the description of the procedure was basically a transforaminal epidural steroid injection). The premise of the study was to determine if patients who were considered good surgical candidates for lumbar radicular pain resulting from nerve root compression (either from herniated nucleus pulposus or spinal stenosis) could avoid surgery by the administration of epidural steroids. Fifty-five patients were randomized to either bupivicaine and betamethasone (28 patients) or bupivicaine alone (27 patients). Up to four selective nerve root injections per patient were performed from a transforaminal approach using radiographic and contrast confirmation. Injectate volumes were 1 or 2 mL, depending on the group to which the patient was randomized. The mean follow-up time was 23 months. Twenty-six of the 55 patients eventually underwent surgery. Eighteen of those were in the bupivicaine-only group. Therefore, only 8 of the 28 in the steroid group required surgery. The difference in the surgery rates for the two groups was highly significant. The authors concluded that selective nerve root injections should be considered for patients with lumbar radicular pain at one or two levels before the consideration of surgery.

The most recent randomized controlled double-blind trial in the literature was published by Karppinen et al. [5] also using the transforaminal injection approach. A total of 160 patients with unilateral sciatica resulting from lumbar disc protrusion or herniation were randomized to either a control group receiving saline or a treatment group receiving 40 mg per mL methylprednisolone and bupivicaine 5%, both groups receiving relatively small volumes of 2 to 3 mL. Only one injection was offered to any given patient. No concurrent treatment was offered at the outset. At 2 weeks, a back school program was given to both groups. Additional analgesic medication and physical therapy was prescribed for patients with unrelenting pain, and surgical intervention was offered for those with severe sciatic pain. The outcome measurements included leg pain (visual analog scale), straight leg raising (goniometric), lumbar flexibility (modified Schober test) and patient satisfaction (Oswestry questionnaire) and were assessed at 2 and 4 weeks, 3 and 6 months and 1 year. The outcome results indicated that at 2 weeks, significant improvements in leg pain, SLR, flexibility and satisfaction was present in the steroid group as compared with the saline group. However, at 3 months the saline group demonstrated significantly less back pain and, at 6 months, less leg pain. By 1 year, 18 patients in the steroid group and 15 in the saline group underwent surgery. The overall operative rate was 21% without a significant difference between the two groups. The authors concluded that the benefits of epidural corticosteroid and anesthetic are only slightly better than saline and only in the early stages of symptoms. Although this study is compelling for limiting the use of epidural steroid injections, it demonstrates some of the same methodological flaws that have been delineated above, including a control injection that was a sham epidural injection as opposed to a ligamentous injection, and the limited number of injections performed in any given patient.

Discussion

In the current medical environment in which randomized clinical trials represent the gold standard of evidence-based medicine, much of the extensive literature regarding epidural steroid injections does not come close to meeting this standard. To date, there are no more than 18 published randomized trials of epidural steroid injections with varying degrees of methodological purity. Approximately half of these randomized studies reported positive benefit from the use of lumbar epidural steroid injections for the treatment of sciatica with or without concomitant low back pain. Often the improvement was relatively short-term. Currently, no randomized studies of the use of lumbar epidural steroid injections in nonchronic low back pain without radicular pain exist.

One of the clear challenges of completing this gold standard model study, as observed in the study of Cuckler et al. [70] and summarized in editorials by Hopwood and Manning [81] and Warfield [82], is physician bias driven by “ethical concerns” regarding withholding epidural steroid injections from certain patients enrolled in a randomized clinical trial. However, these physicians’ aggregate, long-standing prac-
Conclusions and current recommendations

1. There is biochemical, neurophysiologic and basic science support for the role of epidural steroid injections.

2. A limited number of randomized controlled trials are available in the literature regarding the use of lumbar epidural steroid injections demonstrating mixed results—approximately equal studies demonstrate significant benefit and no significant benefit as compared with a control group. Most of these studies have substantial methodological flaws.

3. Lumbar epidural steroid injections can be endorsed as a treatment component for lumbosacral radicular pain syndromes resulting from disc herniation. There are substantial theoretical constructs, research data and practical experience demonstrating that epidural steroid injections are beneficial for the treatment of radicular pain, especially in the early stages.

4. The location of pathology should determine the appropriate injection technique. Preliminary results from transformaminal epidural steroid injections suggest that this route may be the most effective for radicular pain. For this epidural procedure, fluoroscopic guidance is necessary and, in general, fluoroscopic guidance and contrast enhancement are recommended for all epidural steroid injections to place the medication at the proper target site, yield more diagnostic feedback and maximize therapeutic results. In certain cases, an advanced spinal imaging study to support the clinical diagnosis and to determine the best location for medication placement before planning an injection should be considered, particularly if the diagnosis is unclear, the patient is older or the patient has had previous lumbar surgery.

5. The literature contains no data on the use of epidural steroid injection for low back pain only (without a radicular component), but this may be beneficial for some discogenic axial pain syndromes, such as those caused by an annular tear.

6. The risk–benefit ratio of injection of epidural steroid injections in the lumbar spine reveals extremely limited risk for serious complications.

7. There is probably no role for a “series” (of three) lumbar epidural steroid injections without consideration of the response to the initial injection. Additional injections, if any, should be administered at least 1 week apart to allow time to assess the therapeutic effect of the corticosteroid.

8. Epidural steroid injections should be performed by experienced physicians who have demonstrated competence in the technical aspects of these procedures. Informed consent must be obtained from the patient after review of the risks and benefits of the procedure. The need for physiologic monitoring is up to the individual physician.

9. Generally, therapeutic success from the nonoperative treatment of lumbar radicular pain syndromes may be optimized by the concurrent use of other measures emphasizing restoration of function, by taking advantage of the early window of improvement that epidural steroid injections seem to provide.
References


